Controversies Surrounding the Use of Etomidate for Rapid Sequence Intubation in Patients with Suspected Sepsis

Stephanie B Edwin and Pamela L Walker

Objective: To evaluate the risk of adrenal insufficiency following a single dose of etomidate in patients with suspected sepsis requiring rapid sequence intubation.

Data Sources: A literature search was conducted using PubMed, MEDLINE, EMBASE, and International Pharmaceutical Abstracts from the dates of database inception until April 2010, utilizing the terms adrenal insufficiency, etomidate, and sepsis.

Study Selection and Data Extraction: Data were synthesized in a qualitative manner, as variable study designs were identified. All studies that evaluated the clinical association between etomidate-induced adrenal insufficiency and sepsis in adults were reviewed and included.

Data Synthesis: A search of the literature revealed 7 studies that specifically evaluated clinical endpoints in septic adults receiving etomidate for induction prior to intubation. Three of the studies evaluated risk factors associated with adrenal insufficiency in critically ill patients. Each of these studies determined that etomidate exposure was independently associated with an inappropriate response to cosyntropin stimulation testing (CST). Two studies found no significant difference in hospital mortality rates when evaluating patients receiving induction with etomidate compared with alternative regimens. Three studies found an increased risk of adrenal insufficiency in patients exposed to etomidate. The majority of studies that evaluated the use of etomidate in sepsis were underpowered, leading to difficulty in establishing a causal relationship between drug-related adrenal insufficiency, morbidity, and mortality.

Conclusions: Until further studies are available, etomidate should be reserved for hemodynamically unstable patients who cannot tolerate an alternative induction agent despite the administration of fluids or vasoactive agents.

Key Words: adrenal insufficiency, critical care, emergency medicine, etomidate, induction, intubation, sepsis.


Published Online, 8 Jun 2010, theannals.com, DOI 10.1345/aph.1M664

Request

Should etomidate be used during rapid sequence intubation in a patient with suspected sepsis?

Response

Background

Rapid sequence intubation is a widely utilized airway management technique for critically ill patients with a rapidly changing respiratory status, as it is not associated with the delayed onset of general anesthesia. Patients at high risk of complications from the procedure may receive a preinduction agent, such as lidocaine or fentanyl, to reduce detrimental effects caused by activation of the sympathetic nervous system upon endotracheal tube placement. Induction agents, most commonly etomidate and benzodiazepines, are routinely used to facilitate a state of unconsciousness. Once a hypnotic state is achieved, a neuromuscular blocking agent is administered to provide profound relaxation of laryngeal skeletal muscles. When utilized appropriately, rapid sequence intubation has been shown to increase the success rate of intubation to as high as 98.9%, with a low rate of serious complications.

Etomidate is widely utilized for induction due to favorable pharmacokinetic and pharmacodynamic effects. A typical induction dose of etomidate (0.2–0.3 mg/kg) provides hypnosis within 5–15 seconds, roughly the amount of time required for the drug to travel from the site of administration to the brain. The duration of hypnosis is proportional to the dose administered, as the drug rapidly redistributes from the central nervous system to peripheral tissues. The majority of patients who receive etomidate return to consciousness within 5–14 minutes. In contrast to alternative agents, such as benzodiazepines or barbiturates,
etomidate produces very little change in hemodynamic parameters. Additionally, etomidate is not subject to regulatory issues associated with scheduled medications. For these reasons, etomidate is an attractive option for a hemodynamically unstable patient. Furthermore, the agent can be used in patients with coronary artery disease, as it produces no significant effect on myocardial oxygen demand or cardiac contractility. Etomidate produces a reliable reduction in intracranial pressure, as seen with propofol and barbiturates; however, it consistently maintains mean arterial pressure and cerebral perfusion pressure.

Critical illness is associated with an acute stress response characterized by activation of the hypothalamic-pituitary-adrenal axis (HPA) and sympathoadrenal system. It has been well recognized that etomidate, when used as an induction agent for intubation, suppresses the HPA through inhibition of 11-β-hydroxylase (Figure 1).

The diagnosis of adrenal insufficiency has historically been guided by cosyntropin stimulation testing (CST); however, the utility of this test in critically ill patients is controversial. In 2002, a prospective randomized trial showed that a 7-day course of hydrocortisone was associated with reductions in 28-day mortality and duration of vasopressor therapy regardless of response to CST. A study published in 2008 refuted the mortality benefit associated with corticosteroids, finding no significant difference in mortality at 28 days in patients receiving hydrocortisone compared with placebo. Although this study was underpowered, it found no distinction between responders and nonresponders to CST. For these reasons, the most recent guidelines for management of severe sepsis and septic shock recommend that the CST should not be used to identify patients who should receive hydrocortisone for adrenal insufficiency.

ETOMIDATE AND ADRENAL INSUFFICIENCY

Initial concerns about etomidate arose in the 1980s, when an increased mortality rate was noted in patients receiving continuous infusion etomidate compared with the rate in similar patients receiving alternative agents. This alarming trend in mortality halted the use of continuous infusion etomidate for sedation of critically ill patients. Shortly after this mortality trend was reported, a prospective controlled study was initiated to determine the effect of a single dose of etomidate on adrenocortical function. The study evaluated patients (N = 29) scheduled for elective surgical procedures. Patients were randomized to induction regimens consisting of either thiopentone 5 mg/kg or etomidate 0.26 mg/kg. Serum cortisol and ACTH levels were measured at baseline, then periodically for 5 hours after administration of the induction agent. Plasma cortisol levels at 120, 150, 180, 210, and 240 minutes postinduction were noted to be significantly lower in patients who had received etomidate (p < 0.05). Furthermore, peak ACTH levels rose much higher in patients receiving etomidate (726 ± 231 pg/mL at 240 minutes postinduction) than in patients receiving thiopentone (104 ± 25 ng/L at 180 minutes postinduction) induction regimens (p < 0.02). This greater rise in ACTH seen in patients who received induction with etomidate indicates a lack of response by the adrenal cortex to endogenous ACTH stimulation.

The effect of etomidate administration on adrenocortical function was not directly assessed in a critically ill population until years later. A prospective, controlled study was initiated in critically ill patients (N = 35) requiring a general anesthetic regimen containing etomidate or thiopentone. No significant differences were noted in plasma cortisol levels at baseline, prior to CST, or following CST. A significant difference was noted in the percent of patients who displayed an inadequate response to CST, defined as less than 200 nmol/L.

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A prospective, randomized study of patients (N = 31) requiring rapid sequence intubation in the emergency department was conducted to assess adrenocortical function in patients receiving a single dose of etomidate. Patients were randomized to receive etomidate 0.3 mg/kg or midazolam 0.05–0.1 mg/kg. All patients received succinylcholine 1–1.5 mg/kg for neuromuscular blockade. Significant differences in adrenocortical function measured by percent of patients with a normal response to a CST were noted only at 4 hours postinduc-

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**Figure 1. Mechanism of adrenal suppression.**
tion (p = 0.004). Significant differences were not found at 12 and 24 hours postinduction. It should be noted that this study was underpowered, leaving potential for a type 1 error.

The first study to associate adrenal insufficiency with clinical outcomes was a prospective, randomized controlled trial of adult trauma patients (N = 30) requiring intubation. Patients received an induction regimen consisting of either etomidate 0.3 mg/kg or fentanyl 100 µg. Significant differences between the cohorts were noted in postintubation cortisol (p = 0.022), change in postintubation cortisol from baseline (p = 0.003), cortisol after CST (p < 0.001), and change in cortisol during CST (p < 0.001). Differences were noted in hospital length of stay (etomidate 13.9 ± 9.5 days vs fentanyl 6.4 ± 4.4 days; p = 0.007), intensive care unit (ICU) length of stay (etomidate 8.1 ± 7.2 days vs fentanyl 3.0 ± 2.4 days; p = 0.011), ventilator days (etomidate 6.3 ± 6.5 days vs fentanyl 1.5 ± 0.8 days; p = 0.007), and units of fresh frozen plasma (etomidate 2.94 ± 3.5 units vs fentanyl 0.33 ± 0.12 units; p = 0.048). Because limited data on clinical characteristics of patients included in this study were provided, it is difficult to elucidate fully whether etomidate influences the risk of bleeding. Furthermore, the choice of fentanyl as an induction agent in the comparator arm of this study is questionable. Differences in length of stay and ventilator days between the cohorts may discourage the use of etomidate in adults requiring intubation.

**Etomidate in Patients with Suspected Sepsis**

The lack of hemodynamic effects associated with etomidate make for an ideal induction agent; however, the potential for adrenocortical suppression is an important consideration. Malerba et al. conducted a prospective evaluation to identify risk factors associated with relative adrenal insufficiency in critically ill patients (Table 1). Acutely ill patients (N = 62) requiring mechanical ventilation for greater than 24 hours were identified for enrollment. Of note, 33.9% of these patients had a diagnosis of severe sepsis and 45.2% received etomidate to facilitate induction prior to intubation. CST performed 24 hours postintubation classified 43.5% of the study patients as nonresponders. A single bolus dose of etomidate was independently associated with a reduced response to CST (OR 12.21; 95% CI 2.99 to 49.74) upon multivariate analysis. Of note, more than 30% of nonresponders were not exposed to etomidate. Unfortunately, this study was not designed or powered to identify additional factors that may contribute to adrenal insufficiency.

Bearing in mind that adrenal insufficiency can be prevalent and multifactorial in trauma patients, Cotton et al. conducted a retrospective analysis to identify risk factors for development of adrenal insufficiency in a critically ill trauma population (Table 1). Of the 137 patients eligible for analysis, 39 (28.5%) were classified as having a diagnosis of sepsis or septic shock. After CST, 83 patients (60.6%) were classified as nonresponders. Patients with hemorrhagic shock (p = 0.005), vasopressor requirements (p = 0.002), etomidate exposure (p = 0.03), and coagulopathy (p = 0.001) had a higher risk of adrenal insufficiency. Etomidate exposure was present in 63.5% of patients, with 56.9% of patients receiving the medication more than 48 hours prior to CST. A significant risk of adrenal insufficiency was noted in all patients who received etomidate.

Mohammad et al. designed a retrospective study to determine the incidence of relative adrenal insufficiency in patients (N = 152) with septic shock after administration of etomidate (Table 1). Twenty-five percent of the study population received etomidate prior to CST; the median time between drug administration and the test was 7 hours. The incidence of adrenal insufficiency was higher in patients who received etomidate compared with patients who were not exposed to the drug (76% vs 51%; p = 0.0077). Significant differences pertaining to in-hospital mortality or cortisol levels (baseline, 30 minutes post-CST, or 60 minutes post-CST) were not found; however, this study was not adequately powered to detect such differences.

A prospective, nonrandomized observational study by Tekwani et al. was designed to examine the effect of etomidate on the mortality and length of stay in septic patients compared with patients receiving alternative induction regimens (Table 1). Patients meeting sepsis criteria who were intubated in the emergency department were followed over a 9-month period. Etomidate was administered to 69.8% of the patients prior to intubation. In-hospital mortality of patients given etomidate (38%; 95% CI 28 to 49) was similar to that of patients receiving alternative induction regimens (44%; 95% CI 28 to 61). Overall hospital length of stay for patients who received etomidate was 8 days (median 3–13 days) compared with 6.5 days (median 3–9.75 days) in patients who did not receive etomidate (p = 0.18). Of patients who survived to hospital discharge, a longer length of stay was noted in those who received etomidate versus alternative induction agents (p = 0.08). The authors disclosed that changes in as few as 2 patients would have resulted in a statistically significant difference in length of stay between the 2 cohorts.

A retrospective study evaluated the incidence of adrenal insufficiency and mortality in patients (N = 65) with severe sepsis or septic shock, receiving either etomidate (mean dose 0.3 mg/kg) or midazolam for induction (Table 1). No significant difference was noted in overall hospital mortality between the 2 groups; however, patients receiving etomidate had a higher incidence of adrenal insufficiency (84% vs 48%; p = 0.003). Of note, the majority of patients in each arm (etomidate 92% vs midazolam 75%; p = 0.109) received glucocorticoid replacement therapy during the hospital admission. As the majority of patients...
### Table 1. Adrenocortical Suppression in Patients with Possible Sepsis Receiving Etomidate

<table>
<thead>
<tr>
<th>Reference</th>
<th>Pts., N</th>
<th>Dosing Regimen</th>
<th>Patient/Study Details</th>
<th>Results</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cotton (2008)(^{14})</td>
<td>137</td>
<td>Not described</td>
<td>Retrospective registry review Trauma pts. admitted to intensive care unit CST performed for suspected septic shock, SIRS criteria with vasopressor use, unexplained hypotension</td>
<td>CST revealed 60.6% (83/137) of pts. were nonresponders Differences noted between responders and nonresponders with incidence of hemorrhagic shock (p &lt; 0.005), need for vasopressors (p &lt; 0.002), etomidate exposure (p &lt; 0.03), coagulopathy (p &lt; 0.001)</td>
<td>Etomidate exposure present in 63.5% (87/137) of pts. Only 9 pts. had been exposed to etomidate within 48 h of CST; after their exclusion from analysis, risk of adrenal insufficiency was still significant</td>
</tr>
<tr>
<td>Malerba (2005)(^{17})</td>
<td>62</td>
<td>Etomidate 0.2–0.4 mg/kg (n = 28) Midazolam + fentanyl (n = 19) Propofol (n = 5) Ketamine (n = 1) Sodium thiopental (n = 1)</td>
<td>Prospective, observational Critical ill pts. requiring mechanical ventilation for &gt;24 h</td>
<td>Single bolus of etomidate an independent risk factor for reduced response to CST (OR 12.21; 95% CI 2.99 to 47.74) &gt;30% of CST nonresponders had no exposure to etomidate</td>
<td>Severe sepsis present in 33.9% (21/62) of pts. Female sex protective against adrenal insufficiency</td>
</tr>
<tr>
<td>Mohammad (2006)(^{19})</td>
<td>152</td>
<td>Not described</td>
<td>Retrospective, cohort Septic shock pts. requiring CST</td>
<td>Pts. who received etomidate had increased risk of relative adrenal insufficiency (etomidate 76% vs no etomidate 51%; p = 0.0077) Hospital mortality not significantly different between groups (63% vs 55%; p = 0.4517) Post-CST cortisol levels not significantly different between groups (38.0 vs 41.0; p = 0.6674)</td>
<td>Small sample size</td>
</tr>
<tr>
<td>Tekwani (2008)(^{20})</td>
<td>106</td>
<td>Etomidate (n = 74) Benzodiazepines (n = 22) Ketamine (n = 3) Propofol (n = 1) Ketamine + benzodiazepine (n = 1) No induction (n = 5)</td>
<td>Prospective, observational Septic pts. requiring intubation</td>
<td>In-hospital mortality of etomidate pts. (38%; 95% CI 28% to 49%) similar to that of pts. receiving alternative regimens (44%; 95% CI 28% to 61%) No difference between groups in survival to hospital discharge</td>
<td>Nonsignificant increase in pt. length of stay with etomidate vs alternative regimens Slightly higher steroid supplementation in non-etomidate group Small sample size</td>
</tr>
<tr>
<td>Kim (2008)(^{21})</td>
<td>65</td>
<td>Etomidate (n = 25; mean dose 0.3 mg/kg) Midazolam (n = 40; mean dose 0.07 mg/kg)</td>
<td>Retrospective, cohort Severe sepsis or septic shock, with CST performed within 24 h of induction</td>
<td>No difference noted between etomidate vs midazolam in rate of in-hospital mortality (p = 0.269) Significantly higher incidence of adrenal insufficiency with etomidate (84% vs 48%; p = 0.003)</td>
<td>Small sample size High rate of glucocorticoid replacement therapy in both groups</td>
</tr>
<tr>
<td>Cuthbertson (2009)(^{22})</td>
<td>499</td>
<td>Not described</td>
<td>A priori substudy of CORTICUS trial Clinical evidence of infection, systemic response to infection, and continued evidence of shock within 72 h Etomidate use discouraged; however, exclusion not warranted</td>
<td>Pts. with etomidate exposure (n = 96) significantly more likely to be classified as nonresponders to CST (61.0% vs 44.6%; p = 0.004) Nonsignificant increase in mortality with etomidate noted in logistic regression model, adjusting for corticosteroid use, response to CST, baseline cortisol level, SAPS II score (OR 1.60; 95% CI 0.98 to 2.62; p = 0.06) Significant increase in mortality in pts. with etomidate exposure when SOFA score added to logistic regression model (OR 1.75; 95% CI 1.06 to 2.90; p = 0.03) Hydrocortisone use among etomidate pts. did not influence mortality</td>
<td>Largest study to evaluate effect of etomidate exposure in septic shock</td>
</tr>
<tr>
<td>Jabre (2009)(^{23})</td>
<td>469</td>
<td>Etomidate 0.3 mg/kg (n = 234) Ketamine 2 mg/kg (n = 235)</td>
<td>Prospective, randomized controlled, single-blind Acutely ill pts. requiring sedation for emergency intubation</td>
<td>No difference in mean maximum SOFA score (10.3 vs 9.6; p = 0.056) Significantly higher incidence of adrenal insufficiency in etomidate pts. (86% vs 48%; p &lt; 0.0001)</td>
<td>Primary endpoint not based on CST Underpowered to evaluate septic pts.</td>
</tr>
</tbody>
</table>

CST = cosyntropin stimulation testing; SAPS II = Simplified Acute Physiology Score II; SIRS = systemic inflammatory response syndrome; SOFA = sequential organ failure assessment.
evaluated in this study had septic shock, corticosteroids may have led to reduced duration of septic shock and overall reduced mortality.

An a priori substudy of the CORTICUS trial evaluated the potential for etomidate administration to induce nonresponders to CST and the effect of etomidate administration on all-cause 28-day mortality (Table 1). Etomidate administration, defined as any dose administered within 72 hours prior to randomization, was present in 96 (19.2%) of the study population. A logistic regression model adjusting for treatment with corticosteroids, response to CST, baseline cortisol level, Simplified Acute Physiology Score II (SAPS II) score, and sequential organ failure assessment (SOFA) score demonstrated a significantly increased risk of mortality in patients who had received etomidate (OR 1.75; 95% CI 1.06 to 2.90; p = 0.03). Use of etomidate was discouraged, but not prohibited, among patients included in the study.

The majority of studies suggesting an association between etomidate exposure, adrenal insufficiency, and increased morbidity in septic patients have been retrospective. The few prospective studies evaluating this topic have not employed a design allowing for the establishment of a causal relationship. Jabre et al. recently conducted a multicenter, prospective, randomized controlled, single-blind trial to evaluate early and 28-day mortality in acutely ill patients receiving a single dose of etomidate (0.3 mg/kg) compared with ketamine (Table 1). The study enrolled patients who were older than 18 years and required sedation for emergency intubation. Of the 469 patients enrolled in the study, only 76 had a diagnosis of sepsis. The primary endpoint was defined as the maximum SOFA score during the first 3 days of ICU admission, selected as a potentially more reliable prognostic indicator, compared with CST response, for critically ill patients. After 3 days of ICU admission, the maximum SOFA score of 10.3 in the etomidate group did not significantly differ from the maximum score of 9.6 in the ketamine group (p = 0.056). A larger percentage of acutely ill patients receiving induction with etomidate had adrenal insufficiency, compared with patients receiving midazolam (86% vs 48%; p < 0.0001). Adrenal axis function was assessed with CST when recommended by the treating physician (n = 232). Patients with etomidate exposure had significantly lower baseline cortisol levels (441 vs 690 nmol/L; p < 0.0001) and a significantly higher percentage of patients were classified as nonresponders (81 vs 42%; p < 0.0001). Assessment of the subgroup of septic patients revealed no significant differences in 28-day mortality (35% etomidate vs 31% ketamine; p = 0.35) or maximum SOFA score (10.3 etomidate vs 9.6 ketamine; p = 0.056).

The small number of sepsis patients enrolled in this study leaves the potential for a type 2 error.

**DISCUSSION**

The majority of studies that evaluate the use of etomidate in sepsis are underpowered and draw conclusions upon the results of CST. Although initial studies suggested that the duration of drug-induced adrenal insufficiency was short-lived, more recent studies have documented impaired cortisol production more than 24 hours after a single dose of etomidate. Studies conducted in patients with suspected sepsis repeatedly recognize etomidate as an independent risk factor for adrenal insufficiency, albeit a number of additional risk factors may also contribute. Furthermore, the incidence of adrenal insufficiency in a population with septic shock has been shown to be significantly higher in patients who received etomidate as an induction agent. Current literature does not provide clinicians with adequately powered studies evaluating septic patients. Furthermore, the majority of literature draws conclusions based on CST, which has fallen out of favor as a means of diagnosing adrenal insufficiency.

In patients with a reasonable blood pressure prior to intubation, induction with midazolam (0.1–0.3 mg/kg) or propofol (0.5–2 mg/kg) should be considered. Another agent that could be considered for induction is sodium thiopental (1.5–3 mg/kg); however, this drug is not readily available in most patient care areas. It has been suggested that midazolam may cause adrenal insufficiency; however, this theory has not been substantiated. Patients with marginal blood pressures may still be considered for treatment with one of these alternative agents, as clinicians can utilize fluids and/or vasopressor support as needed to achieve an adequate blood pressure. For patients who are hypotensive prior to intubation, ketamine (2 mg/kg) may be an alternative induction agent to consider. Due to sympathomimetic characteristics associated with ketamine, it should not be considered in patients with underlying cardiovascular disease. By using alternative induction agents in patients with suspected sepsis until more evidence is available, clinicians can avoid the potential for worsened clinical outcomes as a result of drug-induced adrenal insufficiency.

**Summary**

A single dose of etomidate produces adrenocortical suppression through inhibition of 11-β-hydroxylase. As a means of reducing potential harm, etomidate should be avoided in patients with suspected sepsis until more evidence is available. Alternative induction agents, with fluid and vasopressor support as needed, will reduce the risk of drug-induced adrenal insufficiency in patients with suspected sepsis.

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courtesy of Dr. Stephanie B Edwin
Se ejecutó una búsqueda en los bancos de data de PubMed, MEDLINE, EMBASE, y Abstractos Farmacéuticos Internacionales desde el inicio de estos bancos de datos hasta abril del 2010. Se utilizó términos tales como “insuficiencia adrenal”, “etomidato”, y “sepsis” en la búsqueda.

SELECCIÓN DE ESTUDIOS Y EXTRACCIÓN DE DATOS: Se resumieron los datos de manera cualitativa porque hubo variación en el diseño de los estudios elegidos. Todos los estudios que evaluaron la asociación clínica entre el uso de etomidato y el desarrollo de la insuficiencia adrenal y sepsis fueron repasados e incluidos.

SÍNTESIS DE DATOS: Una búsqueda en la literatura reveló 7 estudios que evaluaron específicamente los puntos clínicos finales en adultos con sepsis que recibieron etomidato para inducción y antes de ser intubados. Tres de los estudios evaluaron los factores de riesgo asociados con la insuficiencia adrenal en pacientes críticamente enfermos. Cada uno de estos estudios determinó que la exposición al etomidato estaba independientemente asociada con una respuesta inadecuada a la prueba de estímulo de cosintropina. Dos de los estudios no reportaron diferencias significativas en las tasas de mortalidad hospitalaria cuando se evaluaron los pacientes que recibieron inducción con etomidato en comparación a regímenes alternativos. Tres de los estudios reportaron un aumento en el riesgo de insuficiencia adrenal en pacientes que fueron expuestos a etomidato. La mayoría de los estudios que evaluaron el uso de etomidato en sepsis tuvieron poco poder estadístico, y resultó difícil establecer una relación causal entre la insuficiencia adrenal inducido por el medicamento y la morbilidad y mortalidad.

CONCLUSIONES: Hasta que haya más estudios disponibles, se debe de reservar el uso de etomidato para pacientes que estén hemodinámicamente inestables y no puedan tolerar un agente de inducción alternativo a pesar de la administración de fluidos o agentes vasoactivos.
une association possible entre l’insuffisance surrénalienne induite par l’étomidate et la présence d’une septicémie ont été revues et évaluées.

SYNTHÈSE DES DONNÉES: Les paramètres cliniques de patients souffrant d’une septicémie et ayant reçu l’étomidate pour l’induction d’une sédation avant une intubation sont rapportés dans 7 études. Trois d’entre elles ont évalué les facteurs de risque associés au développement d’une insuffisance surrénalienne chez les patients hospitalisés à l’unité de soins intensifs. Chacune de ces 3 études rapporte une association entre l’utilisation de l’étomidate et une réponse inappropriée au test de stimulation à l’ACTH. Aucune différence significative entre les taux de mortalité intra-hospitalière chez les patients ayant reçu l’étomidate ou un autre agent utilisé pour l’induction de la sédation n’a été démontrée bien qu’un risque accru d’insuffisance surrénalienne associé à l’utilisation de l’étomidate est rapporté dans 3 études. La majorité de ces publications comportent toutefois des lacunes méthodologiques pouvant limiter l’établissement d’une relation causale entre l’insuffisance surrénalienne, l’utilisation de l’étomidate et la morbi-mortalité observée.

CONCLUSIONS: L’utilisation de l’étomidate devrait être réservée aux patients hémodynamiquement instables qui ne peuvent tolérer un autre agent inducteur de la sédation malgré l’administration de solutés ou d’agents vasoactifs. Cette place dans la thérapie pourra être revue à la lumière de nouvelles données publiées.

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