Etomidate

***CoreNotes* by Core Concepts Anesthesia Review, LLC**

What You Must Know

1. Etomidate is a unique intravenous anesthetic agent with a carboxylated imidazole structure.
2. Induction doses are associated with frequent myoclonic movements secondary to loss of cortical inhibition.
3. Etomidate reduces $CMRO_2$, CBF & ICP.
4. Etomidate causes minimal cardiac and respiratory depression, even in the presence of significant cardiopulmonary disease.
5. Etomidate inhibits cortisol synthesis resulting in increased morbidity and mortality in ICU patients when used as a prolonged infusion.
6. The mechanism of action of etomidate appears to be augmentation of GABA-gated chloride currents.

Etomidate is a unique intravenous anesthetic agent possessing a carboxylated-imidazole structure. Only the d-isomer has anesthetic properties. Etomidate is unstable at physiologic pH and is formulated as a 35% propylene glycol solution, which leads to pain on injection, and has been associated with phlebitis and hemolysis.

The distribution and elimination of etomidate follows a tri-exponential decay curve indicating a 3-compartment model. Etomidate has a very high rate of clearance from the plasma as a result of rapid ester hydrolysis by esterases in the liver. Severe hepatic disease results in a delayed clearance of etomidate. The plasma concentration of etomidate increases considerably (by a factor 2 to 3) when combined with fentanyl. The total plasma clearance and volume of distribution of etomidate are decreased by a factor 2 to 3 without a change in half-life when administered with fentanyl.

As with the barbiturates, etomidate causes a reduction in cerebral electrical activity with a concomitant reduction in $CMRO_2$, CBF and ICP. The hemodynamic stability associated with etomidate use maintains cerebral perfusion pressure (CPP). Myoclonic movements are common after induction with etomidate, but are not the result of seizure activity. Rather, these movements seem to be the result of loss of cortical inhibition. Etomidate increases the incidence of PONV to as much as 50% of patients.

Etomidate preserves cardiac and ventilatory function, even in patients with cardiopulmonary disease. In addition, etomidate does not release histamine. For this reason it is a popular induction agent for patients with limited cardiopulmonary reserve.

Etomidate inhibits the action of 11-β-hydroxlyase in the adrenal cortex, causing a reduced production of gluco- and mineralocorticoids and an elevation of ACTH. This effect is apparent even after a single induction dose, but is more pronounced with continuous infusion. This effect has precluded the use of a continuous infusion of etomidate for sedation in critically ill patients.

Additional Reading:

Barash, PG, Cullen, BF, Stoelting, RK, Cahalan, MK, Stock, MC, and Ortega, R. Clinical Anesthesia. Philadelphia: Lippincott Williams & Wilkins, 2013:489