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Commentary

# Effect of Pre-Treatment with Midazolam Alone and Midazolam in Combination with Fentanyl on Etomidate-Induced Myoclonus

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## INTRODUCTION

Etomidate came into clinical practice in 1972 and the first report of etomidate was published in 1965. Etomidate is Ethyl 1-[(1R)-1-phenylethyl]-1H-imidazole-5-carboxylate containing compound that is not having any connection chemically to other drug used for the IV induction of anaesthesia. It is considered as an alternate for propofol, particularly when there is an unstable cardiovascular system, due to its relatively high safety margin for hemodynamic stability and the fact that it has no negative effects on the sympathetic nervous system or the baroreceptors' ability to function. The exclusive properties of etomidate include cerebral protection, haemodynamic stability, and pharmacokinetics quick recovery after a single dose or continuous infusion. In the 1970s, due to these advantages of etomidate, common use for induction and maintenance of anaesthesia, and sedation in critical care unit but the popularity among clinicians for etomidate decreased in the 1980s by reports that the drug could cause temporary inhibition of steroid synthesis after single doses and infusions. This effect, combined with other side effects e.g. Increase in intraocular pressure, myoclonus, and hiccups, superficial thrombophlebitis, myoclonus and a frequent incidence of nausea and vomiting, led to questioning the role of etomidate in modern anaesthetic practice. In some patients, there can be serious side effects e.g. risk of regurgitation and aspiration, in open globe injury risk of prolapse of vitreous material, increased intraocular pressure, increased oxygen demand, and hyperkalemia and interfere with monitoring.

Myoclonus refers to sudden, brief, involuntary jerking of a muscle or group of muscles either irregular or rhythmic. Myoclonus is a common side-effect with an incidence as high as 50-80% in the absence of premedication. The exact mechanism of action is not clear; it may be because of etomidate-inducedmyoclonus duo to subcortical disinhibition that normally inhibited extrapyramidal motor activity. Another potential mechanism may be that etomidate acts on the Gamma-Amino Butyric Acid (GABAA). A receptor leading to inhibition of the central nervous reticular activating system. Due to inhibition of neurotransmission through GABAA receptor activation, there may be the skeletal muscle control of relevant signal transmission which allows the occurrence of autonomic nervous conduction. The excitatory neuronal pathways are inhibitory neuronal pathways after injection of etomidate. Excitatory movements are parallel with the early slow phase of the EEG, which corresponds to the starting of deep anaesthesia. Myoclonus occurs on awakening if the extrapyramidal system arises quickly than to cortex that inhibits it. Many drugs have been used for prevention of etomidate-induced myoclonus. These include fentanyl, midazolam, remifentanil, sufentanil, low dose etomidate, dexmedetomidine, dezocine, magnesium, butorphanol, and lignocaine and low dose ketamine.

Etomidate-induced myoclonus can be prevented by pre-treatment with benzodiazepines or opioids, which inhibit subcortical neuronal activity. By inhibiting the central nervous system and acting on various receptors, benzodiazepines (midazolam) with or without opioids (fentanyl) reduce the incidence of myoclonus caused by etomidate. In addition to stimulating receptors on GABA-nergic neurons in the basal ganglia, fentanyl also minimizes the incidence of myoclonus caused by the etomidate. Midazolam and fentanyl (one or both) are drugs that commonly use with etomidate during induction of anesthesia, and we can use these drugs (fentanyl and/or midazolam followed by induction doses of etomidate after 2 min of wait period to allow the onset of action of midazolam and/or fentanyl) to decrease the incidence of etomidate-induced myoclonus and it can avoid the use of additional use of other drugs for prevention of etomidate-induced myoclonus.

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