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A Review of Various Methods for Prevention of Pressor Response to Intubation

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ABSTRACT

Endotracheal intubation has become an integral component of airway management. Intubation is associated with sympathetic surge leading to tachycardia and hypertension. Although these sympathetic responses following intubation are brief, they still carry grave consequences for high risk patients and may cause arrhythmias, myocardial infarction, cardiac failure, intracerebral hemorrhage and raised intracranial pressure (ICP). Since multiple drugs and techniques have been used to prevent the intubation response, the purpose of this review was to study the various drugs available in literature and their success in combating intubation response. The methods used can either (a) block the afferent pathway- superior laryngeal nerve block via topical application or infiltration of local anesthetic; (b) block the central mechanism of integration and sensory input via opioids etc; or (c) block the efferent pathway and effector sites- beta blockers (BB), calcium channel blockers (CCB), lignocaine. Among the various drugs available, dexmedetomidine appears to have better attenuation of intubation response followed by beta blockers and calcium channel blockers. Lignocaine appears to be least effective in prevention of pressor response to intubation and laryngoscopy.

Keywords: pressor response, intubation response, sympathetic surge, esmolol, dexmedetomidine, ligocain, calcium channel blocker, fentanyl.

INTRODUCTION

Protecting the airway is one of the most crucial steps in resuscitation. Since its advent, endotracheal intubation has become an integral component of airway management in both elective and emergency surgeries as well as in intensive care units (ICU). Intubation is associated with tachycardia and hypertension. These circulatory responses following endotracheal intubation were described as caused by reflex sympatho-adrenal stimulation. [1,2] Although these sympathetic responses following intubation are brief, they still carry grave consequences for high risk patients and may cause arrhythmias, myocardial infarction, cardiac failure, intracerebral hemorrhage and raised intracranial pressure (ICP).[3]

Since multiple drugs and techniques have been used to prevent the intubation response, the purpose of this review was to study the various drugs available in literature and their success in combating intubation response.

MECHANISM

Laryngoscopy and tracheal intubation causes sympathetic surge leading to rise in serum catecholamine levels, causing elevation of blood pressure and heart rate. [2,4]

Prevention of these sympathetic responses is essential for smooth induction especially in high risk patients, i.e. patients with hypertension, rhythm disturbances,

myocardial ischemia, raised ICP etc. Various strategies have been developed for combating these responses targeting different levels of the sympathetic reflex pathway.[5] The methods used can either (a) block the afferent pathway- superior laryngeal nerve block via topical application or infiltration of local anesthetic; (b) block the central mechanism of integration and sensory input via opioids etc; or (c) block the efferent pathway and effector sites - beta blockers (BB), calcium channel blockers (CCB), lignocaine etc.[5]

A. BLOCKING THE AFFERENT PATHWAY

1) Superior laryngeal nerve block (SLNB)- It can be done either by using anatomical landmark (AL) technique or under USG (ultrasonographic) guidance. AL technique not only lead to higher patient discomfort but also significantly higher intubation response as compared to USG technique.[6] Apart from SLNB alone , a combination of SLNB with topicalization of the airway has shown greater benefit, better hemodynamics and patient comfort.[7]

2) Topicalization of airway- Local anesthetics used for topicalization of airway as (i) lignocaine nebulisation; (ii) lignocaine sprays have been found to reduce pressor response to intubation.[8,9] Nebulisation can be done either with 5 ml of 0.25% ropivacaine or 5 ml of 2% lignocaine. They both decrease the pressor response to intubation significantly and there is no difference between the two.[8]

B. BLOCKING THE CENTRAL MECHANISM

1) Opioids- Among opioids administered intravenously (IV), all are found to attenuate the pressor response to laryngoscopy and intubation. Intravenous fentanyl 1 µg/kg, sufentanil 0.1 µg/kg, alfentanil 10 µg/kg and remifentanil 1 µg/kg comparably attenuated intubation response. However, hemodynamic stability is better preserved with fentanyl.[11]

2) Alpha 2 agonists- Dexmedetomidine (dexmed) as a bolus dose of 0.5-1 mcg/kg IV over a 10-20 minute period prior to intubation successfully attenuates the hemodynamic response to intubation.[12,13] Clonidine in dose of 2 mcg/kg over 10 minute period also reduces the sympathetic response to intubation, but it slightly lagged behind the attenuation response of dexmedetomidine.[14] Apart from IV route, dexmed administered intranasally (IN) in the dose of 1mcg/kg, 25 to 40 minutes prior to surgery can also provide desirable response at par with the IV route.[15,16] Clonidine 200 mcg orally given 90 minutes prior to surgery also attenuates the hypertensive response to intubation.[21]

3) Gabapentin and pregabalin- Gabapentin 900 mg orally or pregabalin 150 mg orally given 90 minutes prior to surgery attenuate the hemodynamic response to intubation.[21]

C. BLOCKING THE EFFERENT PATHWAY

1) Beta Blockers- Esmolol as a bolus of 1 mg/kg three minutes prior to intubation; or as an infusion of 1.5 mg/kg over 10 minutes prior to intubation suppresses the sympathetic response.[17,18] Labetalol in a dose of 0.25 mg/kg given over 1 minute, five minutes prior to intubation also decreases the pressor response to intubation.[19]

2) Calcium Channel Blockers- Nicardipine in the dose of 30 mcg/kg or verapamil in dose of 100 mcg/kg given three minutes prior to intubation effectively decrease the pressor response.[20]

3) Intravenous lignocaine- A bolus dose of 1.5mg/kg over 10 minutes followed by continuous infusion at the rate of 1.5mg/kg/hr can also be used for preventing intubation response.[10]

COMPARISON BETWEEN VARIOUS DRUGS

1) Esmolol and lignocaine- Although both the drugs attenuate the intubation response, esmolol has a more consistent and effective attenuation than lignocaine.[17,22]

2) Esmolol versus fentayl- both these drugs are effective in attenuating intubation response, but only esmolol provides a consistent and reliable protection against it.[23]

3) Esmolol versus lignocaine- IV lignocaine is poorly effective in controlling intubation response when compared to esmolol. There are widespread fluctuations in blood pressure and heart rate with ligocain when compared to esmolol.[24]

4) Esmolol versus labetalol- both these drugs effectively attenuate the intubation response, but in low doses (esmolol 0.5mg/kg and labetalol 0.25mg/kg) labetalol is better than esmolol.[19,25]

5) Esmolol versus dexmedetomidine- dexmedetomidine acts centrally, via its alpha 2 agonist action, attenuates the stress response, thus creating a more stable hemodynamic effect.[26] It has a better control over hemodynamics and provides better clinical advantage in the perioperative period as compared to esmolol.[27]

6) Esmolol versus nicardipine- both have comparable suppression of intubation response; esmolol (1.5mg/kg) has better heart rate attenuation whereas nicardipine (0.03mg/kg) has better blood pressure control.[28]

7) Labetalol versus dexmedetomidine- dexmed attenuates the intubation response more effectively compared with labetalol.[29]

CONCLUSION

Among the various drugs available, dexmedetomidine appears to have better attenuation of intubation response followed by beta blockers and calcium channel blockers. Lignocaine appears to be least effective in prevention of pressor response to intubation and laryngoscopy.

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