

# Attenuation Of The Hemodynamic Response To Endotracheal Intubation: Fentanyl Versus Lignocaine

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## Abstract

**Aim:** A double blind, randomized, controlled, study was designed to compare the efficacy of single bolus doses of Fentanyl (2µg/kg) or Lignocaine (1.5 mg/kg) for attenuation of pressor response to laryngoscopy and endotracheal intubation (ETI).

**Methods:** Ninety patients received either fentanyl or lignocaine or saline 5 minutes before intubation. Rest of the procedure was standardized.

**Results:** The fentanyl group showed significantly lesser rise (5.46%) in HR compared to lignocaine (16.23%) (p=0.018) and Control group (43.68%) (p=0.000). The rise persisted for 2, 5 and 10 minutes in fentanyl, lignocaine & control groups respectively. The lignocaine group showed lesser rise in SBP (12.1%) compared to Control group (25.8%) (p=0.000) at intubation. The rise persisted for 3 minutes and 10 minutes in lignocaine and control groups respectively. The fentanyl group showed significant decrease in SBP after administration, which came back to normal at 1 to 3 minutes following intubation and again decreased 4 minutes after intubation. In the control group 50% of the patients had hypertension and 80% had tachycardia by definition, while no adverse effects were noted in lignocaine and fentanyl groups.

**Conclusion:** Lignocaine and fentanyl both attenuated the rise in pulse rate, though fentanyl was better. Lignocaine attenuated the rise in blood pressure with intubation whereas fentanyl prevented it totally.

## ABBREVIATIONS

SBP - Systolic Blood pressure  
DBP - Diastolic Blood pressure  
MAP - Mean Arterial Pressure  
RPP - Rate Pressure product  
PrSD - Prior to study drug injection  
PoSD - Post study drug injection  
At Intb - At the time of intubation  
Min (1-10) - Number of minutes following intubation

## INTRODUCTION

In 1940, Reid and Brace<sup>1</sup> first described hemodynamic response to laryngoscopy and intubation. The rise in the pulse rate and blood pressure is usually transient, variable and unpredictable. Usually these changes are well tolerated by healthy individuals. However, these changes may be fatal in patients with hypertension, coronary artery disease or

intracranial hypertension. To 'blunt' this pressor response, various methods have been tried including adrenergic blockers, vasodilators, calcium channel blockers, alpha 2 agonists etc. These methods require administration of an additional costly drug, which not only have no role for induction and maintenance of anesthesia but also can cause dangerous complications.

Narcotics or inhalational anesthetics can also attenuate pressor response by maintaining proper depth of anesthesia. As such administration of one or the other analgesic is needed during surgery. If a small dose of fentanyl, administered 5 minutes before intubation can prevent this hemodynamic response, it would be worth. Few studies have shown that fentanyl is effective in blunting pressor response to laryngoscopy and endotracheal intubation.<sup>2,3,4,5,6,7</sup> We undertook a study comparing the effect of Fentanyl (2µg/kg)

or Lignocaine (1.5 mg/kg), a most widely used drug with control group for attenuation of hemodynamic response.

### MATERIALS AND METHODS

Following approval from the Institutional review board and a written informed consent this prospective randomized double blind study was carried out on 90 ASA I patients, aged 18-65 years, scheduled for elective surgery requiring general anesthesia with endotracheal intubation (ETI). Thorough history was obtained. Patients on drugs affecting autonomic nervous system, significant medical co-morbidities, ASA II and above, with known allergies to study drugs, airway abnormalities, expected difficult intubation and patients undergoing procedures requiring head/neck manipulations, nasogastric tube insertion, throat packing during study period were excluded.

Patients were randomly divided into three groups according to computer generated randomization table. Group F: Fentanyl 2 µg/kg diluted to 10cc normal saline; Group L: lignocaine 1.5 mg/kg diluted to 10cc normal saline; Group C: Control 10cc normal saline. Person A injected study drug as per study protocol. Person B monitored heart rate (HR), systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP) with respect to time, while Person C intubated the patient (Person C was kept constant throughout the study). Person B and C were kept unaware of the drug injected.

All patients were given 0.2 mg Glycopyrrolate bromide intramuscularly 30 min prior to surgery. Inside operating room, after applying routine noninvasive monitors (cardio scope / pulse oximeter / non-invasive blood pressure monitor) intravenous access was secured and infusion of ringer lactate 5ml/kg/hr was started. At minute 0, midazolam 0.04mg/kg was administered intravenously over 30 seconds as premedication. At minute 5, study drug (Fentanyl 2 µg/kg or lignocaine 1.5 mg/kg or saline) as per group was injected over 20 seconds in double blinded fashion. Then the patient was oxygenated with 100% oxygen. At minute 7, intravenous thiopentone Na 5mg/kg was administered in incremental doses until loss of eyelash reflex occurred. This was followed by inj. Vecuronium bromide 0.15mg/kg over 20 seconds. Patients were then ventilated with 60% N<sub>2</sub>O in Oxygen up to minute 10. Then at minute 10, patients were intubated. Tube was fixed and secured. After minute 15 only, surgery was allowed to commence.

Thereafter anesthesia was maintained with 60%N<sub>2</sub>O + 0.5% Halothane in oxygen. Hemodynamic parameters monitored

were HR, SBP, DBP and MAP. These were measured by putting non-invasive blood pressure monitor (NIBP) on manual mode at that particular time and recorded at min 0(baseline), min 5(prior to injection of study drug), min 7(post study drug), min 10(at intubation), and every minute thereafter for 5 minutes and then at every 5 minutes.

We had defined following parameters for study: 1) Hypotension was defined as SBP < 25% of baseline value or 90 mm Hg, whichever was lower; 2) Hypertension was defined as SBP > 25% of baseline value or 150mm Hg whichever was higher; 3) Tachycardia was defined as HR > 25% of baseline value; 4) Bradycardia was defined as HR < 60 beats/min; 5) An arrhythmia was defined as any ventricular or supra-ventricular premature beat or any rhythm other than sinus. Incidences of all these parameters were recorded in all three groups.

If there was hypotension as per definition in between 10 to 15 min, then fluid challenge was given. If there was hypertension as per definition in the above period halothane was started. If there was tachycardia associated with hypotension, fluid challenge was given or if associated with hypertension, then halothane was started. If there was bradycardia as per definition in above period, that was treated with injection Atropine. After 15 minutes, if there was hypotension, halothane was shut off; if it remained persistent intravenous fluid challenge was given. If there was hypertension, halothane was started or increased in incremental doses, still if persisted, bolus dose of injection Esmolol 0.5-2 mg/kg was given.

### STATISTICAL ANALYSIS

Demographic data were analyzed by ANOVA followed by unpaired 't' test. Data using binary scale were analyzed using chi square test. Within the group, changes in hemodynamic parameters with respect to baseline were compared using paired 't' test. Inter group comparisons of percentage change of hemodynamic parameters compared to baseline were done by repeated measure ANOVA followed by unpaired 't' test with Bonferoni's correction.

The sample size, n=24, in each group was required based on following four assumptions: a) significant difference as 10% difference in the percentage rise in HR or SBP; b) 20% variability in sample; c) a type I (α) error of 5%; d) a type II (β) error of 20%. To be on safer side we selected 30 patients in each group.

**RESULTS**

Demographic data were comparable in all three groups (Table 1).

**Figure 1**

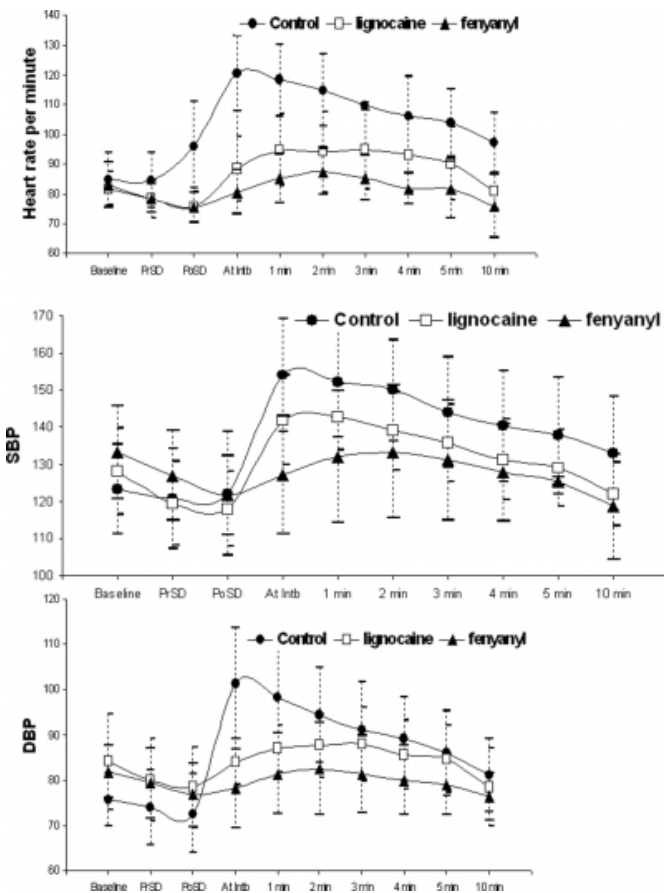
Table 1: Demographic Data

Group	Lignocaine	Fentanyl	Control	P value
Age (yrs) Mean ± SD	30.17±11.39	30.47±13.7	31.97±16.13	0.960
Weight (kg) Mean ± SD	50.73±8.3	47.47±10.02	49.37±9.53	0.987
Sex (male/ female) Number	22 / 08	16 / 14	17 / 13	0.120

After giving midazolam, HR decreased in all the three groups signifying anxiolysis and sedation. HR further decreased after giving the study drug in lignocaine and fentanyl groups. With intubation, HR increased in all the three groups (Figure 1).

**Figure 2**

Figure 1: Serial Hemodynamic Changes

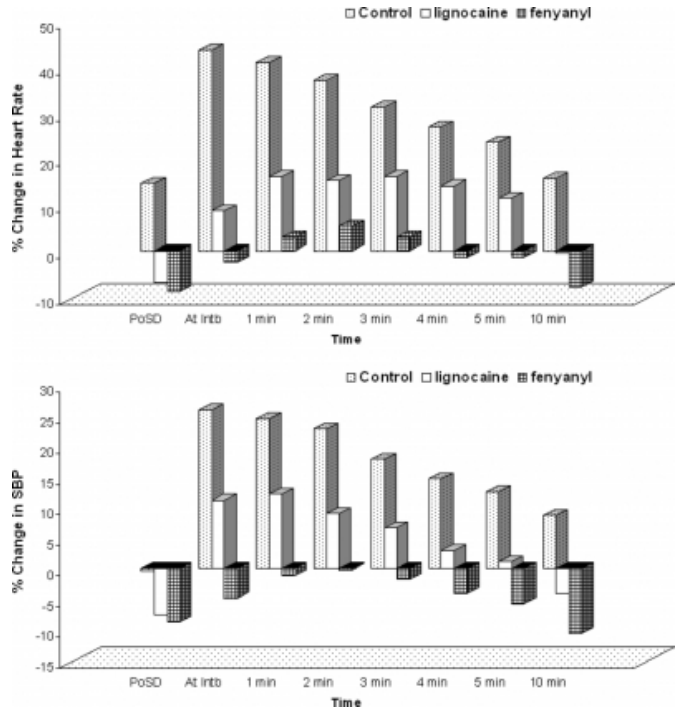


The fentanyl group showed significantly lesser rise (5.46%) in HR compared to lignocaine (16.23%) (p=0.018) and Control group (43.68%) (p=0.000). The rise persisted for 2, 5 and 10 minutes in fentanyl, lignocaine & control groups

respectively (Figure 2). At 10 minutes fentanyl group showed decrease (7.92%) in HR (Figure 2).

**Figure 3**

Figure 2: Percentage change from baseline in Heart Rate and Systolic Blood Pressure



After giving midazolam, SBP decreased from baseline in all the groups (Figure 1). SBP further decreased, after giving lignocaine and fentanyl by 7.6% and 8.8% respectively from baseline. This fall from baseline in fentanyl group was not significantly different when compared with lignocaine (p=0.611) (Figure 2). The lignocaine group showed lesser rise in SBP (12.1%) compared to control group (25.8%) (p=0.000) at intubation (Figure 2). The rise persisted for 3 minutes and 10 minutes in lignocaine and control group respectively. The fentanyl group showed significant decrease in SBP after administration, which came back to normal at 1 to 3 minutes following intubation, again decreased and remained significantly lower (10.63%) compared to baseline even 10 minutes after intubation (P=0.000) (Figure 2).

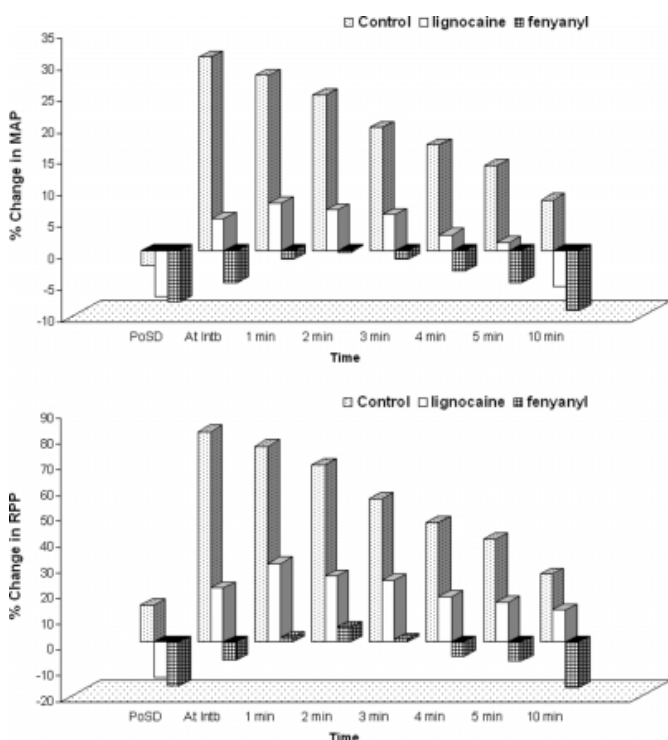
In both lignocaine and fentanyl groups there was initial decrease, followed by rise with intubation (coming back to baseline) & then again decrease in DBP. There was no significant difference between lignocaine and fentanyl group in DBP changes. Control group had significantly higher DBP compared to lignocaine and fentanyl group (P=0.000) from the time of intubation onwards. (Figure 1)

The control group showed maximum rise in MAP of 30.8%

at intubation ( $P=0.000$ ) (Figure 3). These changes persisted even at 10 minutes ( $P=0.000$ ). The lignocaine group showed decrease (7.38%) in MAP after administration; but from the time of intubation onwards up to 3 minutes, there was significant rise (7.54%); MAP again decreased from baseline at 10 minutes. The fentanyl group showed significant decrease (8.08%) in MAP following administration only to come back to baseline from 1 to 3 minutes after intubation and again a decrease, which persisted even at 10 minutes.

**Figure 4**

Figure 3: Percentage change from baseline in Mean Arterial Pressure and Rate Pressure product



The control group had maximum rise of rate pressure product (RPP) at intubation (82%) and changes persisted even at 10 minutes (26.5%) ( $P=0.000$ ). The lignocaine group had significant decrease in RPP following administration. However, RPP increased with intubation, maximum increase being at 1 min (30.7%) and changes persisted up to 5 minutes (Figure 3). The fentanyl group had significant decrease (16.85%) in RPP following its administration; The RPP came to baseline with intubation; maximum rise was insignificant (5.54%); it decreased again at 4 minutes after intubation.

In the control group, 80% patients had tachycardia and 50% had hypertension after intubation. Out of these, 18 (60%) patients required halothane to mitigate this sudden rise in pulse rate and blood pressure. No adverse events occurred in

either lignocaine or fentanyl group.

## DISCUSSION

Intubation is associated with a cardiovascular response of elevated blood pressure and pulse, occasional dysrhythmias, cough reflexes, increased intracranial pressure, and increased intraocular pressure. If no specific measures are taken to prevent hemodynamic response, the HR can increase from 26%-66% depending on the method of induction, <sup>8,9,10,11</sup> and SBP can increase from 36%-45%. <sup>3,10,11</sup> In our study also there was 44% rise in HR and 26% rise in SBP in control group. In patients with atherosclerotic heart disease, intracranial lesions, and potential penetrating eye injuries, these responses to intubation are of greater risk. About half the patient with coronary artery disease experience episodes of myocardial ischaemia during intubation when no specific prevention is undertaken.

Various studies have reviewed the effect of lignocaine to blunt these responses. It is tried in various forms like viscous lignocaine <sup>12</sup>, aerosol <sup>13</sup>, orolaryngeal spray before the induction of anesthesia <sup>14,15</sup>, and inhalation of lignocaine prior to induction of anesthesia <sup>16</sup>.

Some studies note a response of intravenous lignocaine in blunting rises in pulse, blood pressure, intracranial and intraocular pressure. Yukioka et al <sup>17</sup> showed that Cough reflex was suppressed completely by IV lignocaine. Aouad et al <sup>18</sup> showed that supplementing sevoflurane induction of anesthesia in children with IV lignocaine 2 mg/kg can suppress cough after tracheal intubation and thus improve intubating conditions. In addition, lignocaine minimizes blood pressure fluctuations after tracheal intubation.

Abou-Madi et al <sup>19</sup> have discussed the possible mechanisms to account for these observations with IV lignocaine. These include a direct myocardial depressant effect, a peripheral vasodilating effect and finally an effect on synaptic transmission. Lev & Rosen <sup>20</sup> wrote a review on "Prophylactic lidocaine use preintubation". They said that a dose of prophylactic lidocaine of 1.5 mg/kg given intravenously 3 minutes before intubation is optimal. No studies document any harmful effects of prophylactic lidocaine given preintubation.

Recent studies, however, have questioned lignocaine's efficacy. In Singh et al's <sup>9</sup>, van den Berg et al's <sup>21</sup> and Kindler et al's <sup>22</sup> study IV Lignocaine 1.5 mg/kg was ineffective in controlling the acute hemodynamic response following laryngoscopy and intubation. In two different

studies, it was shown that lignocaine 1.5 and 2 mg/kg is ineffective in blunting the responses during rapid sequence induction. <sup>11,23</sup> Bachofen <sup>24</sup> studied blood pressure responses to ETI with 1.5 mg/kg lignocaine in patients with intracranial vessel malformations or brain tumors. In both groups no significant effect of lignocaine on the pressure response could be observed.

Perhaps timing of administration of lignocaine is equally important. Tam et al <sup>25</sup> in their article "Intravenous lidocaine: optimal time of injection before tracheal intubation", showed that, when given intravenously 3 minutes before intubation, esmolol and lidocaine appear to have similar efficacies to attenuate moderate hemodynamic changes secondary to emergency intubation in patients with isolated blunt head injury. Wang et al <sup>26</sup> showed that the values of systolic and diastolic pressures 1 min after intubation were significantly less in groups where lignocaine was given either 3 or 5 minutes before intubation. However In Miller and Warren's study <sup>27</sup>, IV lignocaine failed to attenuate the cardiovascular response to laryngoscopy and tracheal intubation irrespective of the timing of administration i.e. 1, 2, or 3 min before laryngoscopy. Wilson et al <sup>8</sup> showed that irrespective of the timing of administration of injection of lignocaine 2, 3 or 4 minutes before tracheal intubation, there was a significant increase in heart rate of 21-26% in all groups. There was no significant increase in MAP in response to intubation in any group of patients given lignocaine before intubation, but in the placebo group, MAP increased by 19% compared to baseline values.

Bruder et al <sup>28</sup> in a review article wrote that in clinical practice, lignocaine is particularly effective in preventing the pressor response to tracheal intubation, whatever its route of administration (intravenous or intratracheal), but not the increase in heart rate. In our study, in lignocaine group there was significant rise in SBP for 3 minutes after ETI; SBP increased up to 12%; significant rise in HR was present for 5 minutes after ETI; HR increased up to 16%. We injected lignocaine 5 minutes before intubation.

Beta-blockers with bradycardic, antihypertensive, antiarrhythmic and antiischaemic properties have been advocated. As opposed to lignocaine, these agents are more effective in preventing the changes in heart rate than the pressor response. <sup>9</sup> Because of their depressor effect on the myocardium, their place still remains to be defined, especially in the cardiac risk patient. Nitroglycerin is specifically indicated in coronary artery disease; however it

causes tachycardia. In clinical practice, prevention will first rely on a sufficient dose of narcotics.

We reviewed number of such studies on the cardiovascular response to laryngoscopy and ETI. It was shown that adequate depth of anesthesia and quick, smooth laryngoscopy is the mainstay for blunting this response. Narcotics have advantage of having perioperative role in anesthesia. They can be used as sole or supplementary agent for induction of anesthesia.. Narcotics are very commonly used for intraoperative analgesia; therefore there is no additional cost involved. Various narcotic drugs like morphine, fentanyl, alfentanil, sufentanil and remifentanil have been tried for attenuation of pressor response associated with laryngoscopy and ETI.

Fentanyl was found to be more effective than remifentanil at preventing increases in cerebral blood flow velocity during intubation in children undergoing sevoflurane anesthesia. Fentanyl also seemed to provide a more stable hemodynamic profile prior to laryngoscopy and tracheal intubation when compared to remifentanil. <sup>29</sup> Salihoglu et al <sup>30</sup> compared the effects of 1 µg/kg fentanyl (F), 10 µg/kg alfentanil (A), 1 µg/kg followed by an infusion of 0.5 µg/kg/min remifentanil (R) or saline (P) in morbidly obese patients. After induction of anesthesia, arterial pressures decreased significantly in all groups, but the decrease was more pronounced in Groups A and R. After induction, heart rate decreased significantly in all groups except in Group P. After intubation, hemodynamic responses were similar in the remifentanil, fentanyl and alfentanil groups and were within normal limits.

Fentanyl is available in our country since 1998 and has various advantages like no histamine release or bronchospasm, cardiostability, rapid onset and short duration of action. So our study was designed to find out its efficacy for attenuation of pressor response.

Fentanyl has been tried in various bolus doses and infusion forms. Kautto <sup>2</sup> found that supplementation of fentanyl 2 µg/kg significantly attenuated and 6 µg/kg completely abolished the arterial pressure and heart rate increases during laryngoscopy and intubation. In addition, decreased the amount of fentanyl needed during the operation. Respiratory depression was not observed during recovery. Chung et al <sup>3</sup> showed that when given two minutes before laryngoscopy, fentanyl 2 µg/kg & 5 µg/kg resulted in 24% and 6% rise in maximum SBP during rapid-sequence induction in healthy patients. Black et al <sup>4</sup> and Kay et al <sup>5</sup> found complete

attenuation of hemodynamic response with 5 µg/kg fentanyl. Dahlgren and Messeter<sup>6</sup> found that Fentanyl 5 µg/kg treatment caused a significant attenuation of the blood pressure and pulse response to laryngoscopy and intubation in patients undergoing elective intracranial surgery. Cork et al<sup>7</sup> found that fentanyl 5 µg/kg reduced norepinephrine rise during rapid-sequence induction of anesthesia. Fentanyl 6 µg/kg was found to have a useful place in attenuating the cardiovascular effects of fiberoptic intubation under general anaesthesia<sup>31</sup>. Martin et al<sup>32</sup> used thiopental, 3 mg/kg, along with fentanyl, 8 µg/kg, for induction of anaesthesia. MAP rise was attenuated compared to plain thiopentone 6 mg/kg group.

Ebert et al<sup>33</sup> did a comparative study of attenuation by esmolol (500 µg/kg/min X 6 minutes, followed by 300 µg/kg/min X 9 minutes), or fentanyl (0.8 µg/kg/min X 10 minutes). Fentanyl decreased the SBP, MAP and DBP significantly below the baseline, while these pressures were either retained at or elevated slightly above control in the esmolol group. In these doses, the HR response to laryngoscopy was more effectively blocked by fentanyl, while esmolol better retained perfusion pressure. There were no complications or ischaemic electrocardiographic changes in any patient.

In order to find out the optimal dose Iyer and Russell<sup>34</sup> studied, 80 patients undergoing coronary artery surgery. Patients received, either 0, 2, 5, 10 or 15 µg/kg of fentanyl. Mean MAP fell at all dose levels after induction, the mean fall being about 30 mmHg at 5 µg/kg and greater. Mean MAP exceeded pre-induction values after intubation with 0 and 2 µg/kg, and progressive attenuation of the MAP rise was found as the dose of fentanyl increased. They concluded that, if a minimal fall in mean MAP after induction with no rise above preinduction MAP is the sole criterion, a fentanyl dose of about 3 µg/kg is recommended.

There are very few negative studies for fentanyl. Helfman et al<sup>10</sup> did not find any attenuation with either 200 mg lignocaine or 200 µg fentanyl, however they intubated 2 minutes after study drug injection. Maximum percent increases in HR during and after intubation were similar in the placebo (44% +/- 6%), lignocaine (51% +/- 10%), and fentanyl (37% +/- 5%) groups, Maximum SBP percent increases were lower in the lignocaine (20% +/- 6%), fentanyl (12% +/- 3%), and esmolol (19% +/- 4%) groups than in the placebo (36% +/- 5%) group (P < 0.05), but not different from each other (P > 0.05).

Feng et al<sup>35</sup> found 75% and 45% incidence of tachycardia and 70% and 40% incidence of hypertension with lignocaine (2mg/kg) and fentanyl (3 µg/kg) respectively. However, their definition of tachycardia was HR > 100 per minute. Secondly they intubated their patients 3 minutes after the study drug administration.

Fusciardi et al<sup>36</sup> randomly assigned 46 patients to receive either fentanyl, 3 µg/kg (group 1, n = 6), fentanyl, 8 µg/kg (group 2, n = 20), or fentanyl 3 µg/kg plus a continuous intravenous nitroglycerin infusion, 0.9 µg/kg/min (group 3, n = 20), in addition to thiopental-pancuronium anaesthetic induction, prior to laryngoscopy and intubation. In the group 1, myocardial ischemia occurred during laryngoscopy and intubation in four patients, and MAP, HR, and mean PCWP increased significantly. Despite greater stability in MAP and HR in the group 2, myocardial ischemia still occurred in four patients. Probable reasons for these results are inadequate time given for fentanyl action, inadequate sedation, and use of pancuronium. We sedated our patients with midazolam, avoided pancuronium. We standardized the dose as per Kg dose

Ko et al<sup>37</sup> designed a study to examine the optimal time of injection of fentanyl. Patients received fentanyl (2 µg/kg) 1, 3, 5, or 10 min before tracheal intubation. They concluded that the most effective time to administer fentanyl to protect circulatory responses to laryngoscopy and tracheal intubation is 5 min before tracheal intubation. Fentanyl is often used to reduce the hemodynamic response to tracheal intubation. However, large doses may cause unwanted side effects. Administration of fentanyl at the optimal time reduces the dose required. Therefore, we selected fentanyl dose of 2 µg/kg given 5 minutes prior to intubation.

Splinter and Cervenko<sup>38</sup> compared the hemodynamic responses to ETI after induction of anesthesia with thiopentone alone or in combination with 1.5 mg/kg lidocaine and/or 1.5 or 3.0 µg/kg fentanyl in geriatric patients. Fentanyl reduced the rises in SBP, DBP, MAP, HR, and RPP and lignocaine decreased the rises in arterial blood pressure and RPP.

Besides minimizing the cardiovascular response, anesthesia induction for patients at risk must also satisfy the following requirements: it must be applicable regardless of patient group, prevent impairment of cerebral blood flow, and avoid arousal of the patient; it should neither be time-consuming nor affect the duration or modality of the ensuing anesthesia. Among the recommended procedures, intravenous

lignocaine or fentanyl appears to best fulfill the criteria. We used fentanyl dose of 2 µg/kg, as this dose is found to be effective in attenuating the pressor response, in quite a few studies; moreover, higher doses would lead to undue bradycardia, and hypotension. With 2µg/kg fentanyl, we noted 9.5% decrease in MAP.

Our study was designed as controlled, double blind, randomized, prospective, comparative study. We restricted our study period to 15 min because after commencement of surgery multiple factors like various surgical stimuli play role in hemodynamic response, as compared to laryngoscopy & ETI are the only two factors playing role in pressor response. We chose midazolam (0.04mg/kg) as a premedication agent as it is having sedative, anxiolytic properties and has rapid and short duration of action. After giving midazolam, due to anxiolysis and sedation, pulse rate in all study groups decreased from baseline. While after giving the study drug, there was decrease from baseline of 7% and 9% in lignocaine and fentanyl group respectively. Fentanyl is known to cause bradycardia but the fall in pulse rate associated with lignocaine can't be explained.

In response to laryngoscopy and ETI, HR increased in all the three groups. The peak rise of 43.68% was seen at intubation in the control group, the rise persisted even at 10 minutes, indicating need for some method to attenuate this response. In the control group 80% patients had tachycardia after intubation. Out of them, 15 patients required halothane in incremental doses. No adverse effects were seen in these patients, mostly because all the patients were of ASA I class. But, in patients with ischemic heart disease, with fixed cardiac output states, sudden tachycardia associated with laryngoscopy and intubation can cause adverse effects. Both fentanyl and lignocaine were effective in attenuating rise in HR; the peak rise in the HR being 5.46% and 16.23% in fentanyl and lignocaine groups, respectively.

The lignocaine group showed lesser rise in SBP (12.1%) for shorter time (3 minutes) compared to Control group (25.8%) (10 minutes). At intubations, 50% patients in control group suffered from hypertension, which was uneventfully corrected by using incremental doses of halothane. In patients with hypertension or intracranial pathology, this hypertensive response can cause adverse consequences. The fentanyl group showed significant decrease in SBP after administration, which came back to normal at 1 to 3 minutes following intubation, again decreased and remained significantly lower compared to baseline even 10 minutes after intubation. Though none of the patients in our study

had hypotension, by definition, 10.6% mean reduction in SBP in fentanyl group suggests its potential for hypotension, particularly in hemodynamically unstable patients. Fentanyl blunted the rise in the systolic blood pressure at laryngoscopy and ETI better than lignocaine.

MAP is a derived value and is important in relation to the auto-regulatory responses of the heart, brain and kidneys. After ETI, the MAP increased by 30% and 5% in control and lignocaine groups respectively from baseline. In fentanyl group, MAP was below baseline throughout the study period, except during first 3 minutes of intubation. At the end of the study- at min 10, the MAP declined by 9.5% in fentanyl group from baseline as compared to 5% in lignocaine group. This conveys caution for using fentanyl in patients with fixed cardiac output diseases. Control group had heavy rise in RPP (82%) with ETI. Though lignocaine attenuated this rise, still it was 30.7%. Only fentanyl group had insignificant rise of 5.54%. Above data suggests that fentanyl is very useful for attenuation of pressor response in dose of 2µg/kg, 5 min prior to intubation. But as our study included only ASA I patients extending these results to other ASA classes can't be done. This needs further studies in those particular groups.

In conclusion, given 5 minutes prior to intubation, lignocaine (1.5 mg/kg) and fentanyl (2 µg/kg) both attenuated the rise in pulse rate, though fentanyl was better. Lignocaine attenuated the rise in blood pressure and RPP with intubation whereas fentanyl prevented it totally.

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