# HAEMODYNAMIC RESPONSE TO LARYNGOSCOPY AND ENDOTRACHEAL INTUBATION AND ITS TREATMENT

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## INTRODUCTION

***Both laryngoscopy and endotracheal intubation are associated with marked changes in the cardiovascular parameters and this is commonly labelled as the pressor, haemodynamic or sympathoadrenal response. Anaesthesiologists first became aware of the changes related to airway manipulation in the 1940's. It was initially suggested that an increase in vagal tone was the causative factor (1). However, in 1951 Burnstein and then King et al (2,3) implied an increase in the cardiac sympathetic activity as an alternative mechanism.*** *PJCTS 1999;1: 22-26*

## MECHANISM

The pressor response is a reflex characterized by a rise in both systolic and diastolic blood pressure, tachycardia, and an increase in the circulating catecholamines leading to increased myocardial workload. The mean blood pressure generally rises 20 to 50 mm of Hg compared to awake control (4). The blood pressure begins to rise 15 seconds after laryngoscopy, is marked at 30 to 45 seconds and takes 4 to 5 minutes to come back to control values (5). A similar but lesser response accompanies extubation of the trachea (6). It has been suggested that the major cause of the sympathoadrenal response is laryngoscopy alone which causes stimulation of the supraglottic region, leading to a reflex increase in the sympathetic nervous activity. Endotracheal intubation on the other hand causes much smaller increase in the mean arterial pressure but is mainly responsible for the tachycardia component (7). Several studies have reported an increase in the plasma concentration of noradrenaline following laryngoscopy, which is related to the changes in mean arterial pressure but no firm association has been established between heart rate changes and increase in plasma adrenaline. This response is of little value in ASA I patients but may be potentially dangerous in patients with ischaemic heart disease, vascular disease, cerebral aneurysm, raised intracranial pressure, aortic aneurysm, hypertensives and intensive care patients, where it can lead to life threatening complications. The response has also been elicited brain dead organ donors where plasma catecholamines increase, correlated with the onset of surgical stimuli (8). Moorthy recently measured the changes in blood flow in common carotid, middle cerebral and femoral arteries associated with endotracheal intubation. He demonstrated increased cerebral blood flow and decreased peripheral flow in the femoral artery secondary to laryngoscopy and tracheal intubation and attributed it to delay in cerebral autoregulatory response due to increased systemic vascular resistance (9). The changes have also been related to EEG and the haemodynamic changes due to laryngoscopy, and tracheal intubation caused an increase in the activity (10).

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## RECENT TRENDS

### 1. EFFECT OF DISEASE PROCESS

13 patients with nephropathy and 12 with uremia of other origins were compared to 12 ASA I controls. The systolic arterial pressure response was greater in diabetic uraemic patients than in other groups. The uraemic patients irrespective of baseline pathology had higher catecholamine concentrations than the control patients. This could be attributed to impaired clearance of catecholamine and higher sympathetic activity needed to maintain cardiac function in these patients. (11).

### 2. EFFECT OF SMOKING

Haemodynamic response in 30 patients who smoked more than 10 cigarettes a day was

compared to the same number who did not smoke. The heart rate response was significantly greater in smokers. The blood pressure response, however, showed no change. It has been suggested that smoking induces chronic changes in upper airway epithelium resulting in greater exposure of subepithelial airway receptors to stimuli (12).

### 3. RESPONSE RELATED TO AGE

Children also exhibit the response. 60 children had plasma catecholamines measured after endotracheal intubation, diazepam and clonidine pre-medication. Children receiving diazepam or clonidine 2 ugkg$^{-1}$ showed a significantly higher increase in both systolic and diastolic arterial pressures, heart rate and catecholamine concentration compared to those who received 4 ugkg-1 (13). The effect of aging on the haemodynamic response was evaluated and compared in 3 groups of patients aged 18-25 years, 40-50 years and 65-80 years. The elderly had significantly lower chronotropic and blood pressure response after intubation compared to the young age group (14).

### 4. EFFECT OF LARYNGOSCOPE DESIGN AND OTHER AIRWAY DEVICES

McCoy studied the cardiovascular changes and catecholamine concentration in 20 patients before and after laryngoscopy with either Mackintosh or McCoy laryngoscope blades (15). Use of the McCoy blade did not result in any significant change in either heart rate or arterial blood pressure compared to the Mackintosh blade where significant increase did occur. The plasma adrenaline concentrations did not change compared to prelaryngoscopy value in either group. This was probably due to the fact that the epiglottis is elevated by the hinged tip of the McCoy blade rather than forward displacement of the whole larynx. 75 Patients scheduled for routine urological or gynaecological surgery were studied after insertion of combitube (CT), laryngeal mask airway (LMA), or endotracheal tube (ETT). Insertion of CT caused a more pronounced haemodynamic and catecholamine stress response than LMA or ETT. With CT this may be due to the pressure of the pharyngeal cuff on the anterior pharyngeal wall and may represent a serious hazard to patients with cardiovascular disease (16). A cuffed oropharyngeal airway (COPA) which is a modified Guedel airway device with a specially designed cuff at its distal end was compared with LMA in 35 female patients. Changes in arterial pressure and heart rate after insertion were found to be similar in both groups and not significantly different from baseline values (17).

### 5. COMPARISON OF DIFFERENT INTUBATION TECHNIQUES

Pernerstorfer et al compared the stress response induced by direct laryngoscopy with the blind oral intubation using the Augustine guide (18). The direct laryngoscopy group showed a significant increase in SAP and MAP accompanied by a significant rise in plasma adrenaline and prolactin levels even though it took less time. The authors concluded that the major determinant was increased sympathetic reactivity and not the duration of laryngoscopy.

### 6. PHARMACOLOGICAL METHODS FOR ATTENUATION OF THE RESPONSE

Heropoulos in 1995 reported the use of angiotension-converting enzyme inhibitor enalaprilat (1.25 mgs intravenous, 20 minutes prior to induction) on haemodynamic and hormonal responses to endotracheal intubation (19). Arterial blood pressure and heart rate increased significantly in the placebo compared to enalaprilat group. Although no significant change was seen in the catecholamine levels, plasma rennin activity was significantly increased at post-incision in the enalaprilat group. This suggests that activation of the rennin - angiotensin system may play a key role in this response. Puri studied 36 patients with coronary artery disease (20). Patients received either 0.1 ml$^{-1}$kg magnesium sulphate or isotonic sodium chloride. Magnesium sulphate administration was associated with a significant decrease in mean arterial pressure (MAP) and had a significantly lesser increase in MAP compared with a control group who received lignocaine before endotracheal intubation. The underlying mechanism with magnesium sulphate is said to be inhibition of the release of catecholamines. Haemodynamic changes associated with endotracheal intubation after induction with propofol alone or propofol/fentanyl were studied by Billard (21). There was no effect on the

response when the propofol dose was increased from 2 to 3.5 $mgkg^{-1}$ but post-intubation hypertension was decreased with increase in the fentanyl dose to 4 $ugkg^{-1}$. The effect of 3 bolus doses of remifentanil on the pressor response during rapid sequence induction was assessed in a placebo controlled study. 0.5 $ugkg^{-1}$ was ineffective whereas 1 and 1.25 $ugkg^{-1}$ dose was associated with a decrease in systolic arterial pressure to less than 90 mm of Hg in 7 out of 20 patients. (22). The same authors had found that remifentanil 0.25, 0.5 or 1.0 $ugkg^{-1}$ was inadequate in obtunding the response when reocuronium was used as a muscle relaxant. The response was short lived and of smaller magnitude after 1 $ugkg^{-1}$ compared to placebo (23). Remifentanil 1 $ugkg^{-1}$ bolus over 30 seconds, followed by an infusion of 5 $ugkg^{-1}min^{-1}$ was found to effectively attenuate the pressor response by Thompson et al. Concurrent vagolytic agents were used otherwise bradycardia or hypotension was seen (24). Sufentanil has also been used to attenuate the response. 3 $ugkg^{-1}$ sufentanil in patients premedicated with 60 $ugkg^{-1}$ lorazepam produced a near maximal haemodynamic effect and is adequate for induction and intubation in patients undergoing CABG. Increasing the dose to 15 $ugkg^{-1}$ had no additional benefit and caused transient cardiovascular stimulation (25). In a study of 60 ASA class I and II patients alfentanil 0.03 $ugkg^{-1}$ and a combination of alfentanil 0.05 $ugkg^{-1}$ and esmolol 1 $ugkg^{-1}$ was used prior to induction. The half dose combination was found to be as effective as alfentanil and superior to 2 $ugkg^{-1}$ esmolol (26). Lignocaine was used by two methods in a group of patients undergoing ophthalmic surgery. One group received direct laryngeal/tracheal lignocaine spray immediately before intubation, whereas another received orolaryngeal lignocaine spray before induction of anaesthesia. The later method resulted in a reduction of the response compared to the former (27). A combination of lignocaine 1.5 $ugkg^{-1}$ with esmolol 1 $ugkg^{-1}$ was superior in attenuating the response compared to both drugs used alone (28). Nebulized bupivacaine 15 minutes before induction of anaesthesia attenuated the heart rate but not the hypertensive response to laryngoscopy and intubation (29). Nifedepine 10 mgs sublingually 20 minutes before induction of anaesthesia has been recommended in patients with pregnancy induced hypertension undergoing caesarian section under general anaesthesia. The dose was effective in attenuating the hypertensive response to laryngoscopy and intubation but not tachycardia (30). The optimal dose of nicardipine which maintained cardiovascular stability at induction and during intubation was found to be 1 mg intravenously 2 minutes before tracheal intubation (31).

## 7. EFFECT OF TRACHEAL EXTUBATION

Extubation response can cause myocardial ischemia in patients with coronary artery disease. Haemodynamic response to cessation of mechanical ventilation and extubation was studied in 84 patients after coronary artery bypass grafting. Patients were randomly allocated to extubation while awake or still sedated with propofol 1-3 $mgkg^{-1}hr^{-1}$. 20 patients (53%) in the awake group and 3 (7.5%) in the sedated group required treatment for systolic hypertension. Significant ST segment changes were seen in 5 patients in awake group. The authors recommended that removal of tracheal tube while patients were still sedated after CABG was safer (32). Lignocaine alone and prostaglandin E1 (PGE1) have been used for blunting the extubation response (33). PGE1 alone was effective for hypertensive response but not tachycardia but a combination of the two above agents was effective for both hypertension and an increase in heart rate (34). Removal of LMA was associated with an extubation response but this was lesser than a tracheal tube. In case of hypertensive patients use of LMA resulted in a cardiovascular change of lesser magnitude when compared to ETT (35).

## CONCLUSION

Inspite of enormous data pertaining to the haemodynamic response, some aspects are still misunderstood or controversial. There are difficulties in comparing different studies due to variation in patients population, depth of anaesthesia, methods of stress evaluation, etc. Each method recommended so far has variable effectiveness and undesirable side effects. Methods based on combination of drugs whereby the total dosage of each drug decreases seem promising for further use.

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