INTRODUCTION
INTRODUCTION

Tracheal intubation is usually indicated in anaesthetized and unconscious patients for adequate maintenance of airway, to protect the respiratory tract from aspiration of foreign materials and for positive pressure ventilation. Usually this is carried out under direct vision made possible by direct laryngoscopy. In most cases of otherwise healthy patients these procedures, direct laryngoscopy and tracheal intubation, do not leave behind any serious complications. However, a potential hazard of these procedures is the reflex cardiovascular responses, mainly in the form of hypertension, tachycardia and dysrhythmias, even when the laryngoscopy and tracheal intubation are uncomplicated by hypoxia, hypercarbia or cough. Other contributory causes can be anxiety, hypercarbia and hypoxaemia, atropine premedication, and a reflex baroreceptor effect as a result of the decrease in arterial pressure after thiopentone.

These reflex responses are due to the stimulation of the nerve endings of the vagus, glossopharyngeal and trigeminal nerves and mediated by increased sympathetic nervous activity. Although direct recording of the
sympathetic activity is difficult in man measurements of the plasma concentration of catecholamines have consistently demonstrated increase in noradrenaline levels following laryngoscopy. Thus sympathetic mediation is confirmed in this response.

The increases in blood pressure and heart rate are usually transitory, variable and unpredictable. The maximum pressure occurs within 1 minute after intubation and gradually returns towards normal within 5-10 minutes in normotensive patients. This transitory hypertension and tachycardia are probably of no consequence in normotensive individuals but either or both may be hazardous to those with hypertension, myocardial insufficiency or cerebro-vascular disease.

Hypertensive patients are more prone to develop significant increase in blood pressure, whether they have been treated or untreated beforehand. In normotensive patients mean arterial pressure increases by about 20 to 30 mm Hg. and heart rate by about 15-20/mt, following direct laryngoscopy and this results in an increase of the rate pressure product from about 7500 to 12500, whereas in untreated hypertensive patients the same stimulus can increase the systolic arterial pressure from 140 (after induction) to 250 mm Hg. and heart rate from 70 to 110 beats per minute. The rate pressure product in
this case will increase from about around 10,000 to a peak value of 27,500 well above the angina threshold in patients with known ischaemic heart disease.

The sudden rise in blood pressure may cause left ventricular failure, myocardial ischaemia, and cerebral haemorrhage especially in patients with intra-cranial aneurysms and these complications are more likely in the presence of coronary or cerebral atheroma or hypertension. In these conditions it can even become life threatening. This may be particularly dangerous in patients with leaking aortic aneurysm.

Sudden death presumably from ventricular fibrillation has been reported to result reflexly from tracheal intubation and tracheal suction. Surgical patients with ischemic heart disease or hypertension or both have been shown to carry an increased risk of perioperative complications. In pre-eclamptic patients convulsions may be precipitated due to the sudden rise in blood pressure following laryngoscopy and tracheal intubation.

In view of these findings it is generally agreed that the pressor response to laryngoscopy and intubation may be harmful and attempts must be made to prevent its occurrence especially in patients at a higher risk of developing these complications. Various
attempts have been made to attenuate these responses, but none has achieved widespread acceptance. Deep inhalational anaesthesia (King et al, 1951), systemic and topical lignocaine (Kenneth et al, 1974; Hamill et al, 1981; Jerrold and Anthony, 1985), anti-hypertensive drugs (Stoelting, 1979; Davis et al, 1981), beta blockers (Prys-Roberts et al, 1973; Magnusson et al, 1986), peripheral vasodilators (Curren et al, 1980; Passaulaki and Kaniarić, 1983; Kotôk et al, 1986) and intravenous narcotics (Black et al, 1984; Crawford et al, 1987; Sweeney et al, 1989) have all been used and recommended.

Considering the theoretical as well as clinical significance of these reflex changes, the available techniques were found to be unreliable and unpredictable. Hence it was thought worthwhile to study attenuation of these reflex responses to laryngoscopy and intubation with various drug combinations.

******