Original Article

Intravenous esmolol is superior than sublingual nifedipine and intravenous lignocaine for attenuation of haemodynamic responses during laryngoscopy and endotracheal intubation

Utpal Kumar Ray[1], Prasanta Kumar Mandal[2]

Catacholamines released during laryngoscopy and endotrachial intubation (ETI) puts the patients at risk of development of various tachy arrythmias which have a deleterious effect on compromised cardiac functions. During the induction of general anaesthesia the two important events take place. One of them is laryngoscopy and another is endotracheal intubation. During laryngoscopy, the blade of the laryngoscope presses against the base of tongue and lifts up the epiglottis. This incidence gives rise to certain impulses to proceed through the vagus, the result being intense sympathetic stimulation. The cardioaccelerator nerve stimulation gives rise to tachycardia. The increase level of catecholamine gives rise to hypertension. Laryngoscopy produce more intense effects than endotracheal intubation in respect to cardiovascular system. Fifteen patients placed in each group received intravenous (IV) lignocaine (1.5mg), sublingual nifedipine (10mg) and IV esmolol (2mg/kg) 90 seconds,10 minutes 2 minutes before laryngoscopy and ETI respectively. Changes in heart rate, SBP, DBP, MAP RPR were recoded just after ETI and then after 1minute, 2minutes, 5minutes 10 minutes ETI. Results were compared with the control group (n=15, received no study medication). Intravenous lignocaine was not so much effective in countering the cardiovascular responses to laryngoscopy and ETI. Sublingual nifedipine produces a significant attenuation of SBP, DBP and MAP but it was unable to attenuate the pulse rate significantly. Intravenous esmolol is the best attenuator amongst the three drugs studied over here to the cardiovascular responses during laryngoscopy and intubation.

[*J Indian Med Assoc* 2018; **116:** 15-8]

Key words **: Laryngoscopy, endotracheal intubation, cardiovascular responses, lignocaine, esmolol, nifedipine.**

During the induction of general anaesthesia, the two important events take place. One of them is laryngoscopy and another is endotracheal intubation. During laryngoscopy, the blade of the laryngoscope presses against the base of tongue and lifts up the epiglottis. This incidence gives rise to certain impulses to proceed through the vagus, the result being intense sympathetic stimulation. The cardioaccelerator nerve stimulation gives rise to tachycardia. The increase level of catecholamine gives rise to hypertension. Laryngoscopy produce more intense effects than endotracheal intubation in respect to cardiovascular system[1,2].

The changes recorded are a rise in systolic blood pressure by about 30-50 mm of Hg, in diastolic pressure by about 20-30 mm of Hg, thereby increasing the mean arterial pressure. Heart rate increases by about 20-40 beats per minute (BPM) and thus increasing rate pressure product, an index for myocardial oxygen consumption. Various cardiac dysrhythmias, apart from sinus tachycardia or bradycardia, do occur in 5-10% of patients, usually are benign and transient. The sympathoadrenal stimulation may prove detrimental to the health of certain group of patients. Patients with ischaemic heart disease may have acute myocardial infarction. Patients with Ionotropically compromised heart increases the heart rate and thus lapse into heart failure. Patients with aneurysm in the cerebral vessels may have hypertensive haemorrhage in the brain. So to prevent these casualties, the sympathoadrenal system stimulation accompanying laryngoscopy and intubation must be obtunded.

This observations led to use of different techniques and attenuate the cardiovascular responses to laryngoscopy and endotracheal intubation like use of deeper plain of anaesthesia (both local and intravenous), narcotics, beta adrenoceptor blockers, vasodilators etc with various degrees of success. But no single method has gained wide spread acceptance because each method has its own disadvantages. Many newer studies are still being carried out with reevalu-

Department of Anaesthesiology, Murshidabad Medical College & Hospital, Berhampore 742101
[1]DA, MD (Anaesthesiol), Associate Professsor,
[2]MS (Ortho), Associate Professsor, Department of Orthopedics, Bankura Sammilani Medical College, Bankura 722102 and Corresponding author

ation of older ones. With this Idea, an endeavour has been made to evaluate IV lignocaine, sublingual nifedipine and IV esmolol can modify the cardiovascular response to direct laryngoscopy and endotracheal intubation.

Material and Methods

The present study was carried out in the Department of Anaesthesiology, Murshidabad Medical College & Hospital and Department of orthopedics,BS Medical College, Bankura between April 2016 to July 2017. Subjects were selected amongst those were selected amongst those were put for surgery under endotracheal anaesthesia. Sixty patients were randomly chosen for this study. They were of both sexes. Their age ranged from 18 years to 50 years and weight ranged from 40 kg to 70 kg. The patients were carefully selected according to the American Society of Anaesthesiologist Classification and were in the status I & II category. The patients were selected from the general Surgical and orthopedic ward of Murshidabad Medical College&Hospital and BS Medical College & Hospital, Bankura. The procedure were fully explained to them and their written informed consent was obtained.

A detailed history was taken including the history of present and past illness, personal history, family history, past history of any operation and anaesthesia including history of drug intake and drug allergy. Clinical examination included measurement of Height, Weight, Nutrition, Pulse, Blood Pressure, Temperature, Anaemia, Jaundice, Cyanosis, Clubbing and edema. A Careful Clinical Examination of cardiovascular, respiratory, nervous, gastrointestinal and genitourinary system was done. Pre-operative investigations incuded Routine Examination of Blood – Total and differential count of white blood cell, Erythrocyte sedimentation rate, Haemoglobin, Urine – Macroscopic and Microscopic Examination, Stool – Macroscopic and Microscopic Examination, Blood Sugar (fasting and post-prandial), urea, creatinine, Serum Electrolytes – Na^+, K^+, and chloride, Chest X-ray (PA View) and 12 lead ECG.

None of the patients included in this study who have any history of respiratory, cardiovascular, hepatic, renal, endocrinal and metabolic disorders. Their mutritional status was found to be good. The patients has no history of receiving any psychotropic, hypnotic, antihypertensive, antiarrhythmic, diuretic antidiabetic and steroid therapy. All patients waiting for surgery were examined thoroughly in the ward 2 days before the expected day of operation. This opportunity was also utilised for establishing a pleasant report with the patient and allaying his/her anxiety. Altered anatomy of the mouth and neck, particularly dental structure might be a problem to quick and smooth intubation and any such patient was excluded from the study.

All the patients received tablet diazepam 10 mg at bed time on the night before operation. In the ward in the morning of operation, about two hours before induction of anaesthesia, pulse rate, systolic and diastolic pressure were measured and tablet diazepam (10 mg) was given orally.All the patients received Injection Glycopyrrolate (0.3 mg) Intramuscularly half hour before operation.Then the subjects were brought to the operation theatre and they were rested on the operation table for 5 minutes in a calm and quiet atmosphere to get them accustomed with the new environment.The subject were monitored for pulse rate (PR as beats per minute) by palpation of radial artery and for systolic and diastolic pressure (SBP and DBP respectively in mm of Hg) with the help of a mercury sphygmomanometer. After final checking of the subjects, Dependable intravenous channel was instituted. Pulse and Blood pressure were recorded which acted as a preoperative base line value (Before study drug administration).

Altogether sixty subjects were studied. They were placed randomly into four groups, each contains 15 subjects. The groups are –

Group – 1 : Control group – received none of the three drugs under the study

Group – 2 : Received intravenous lignocaine 1.5 mg/kg, 90 seconds before induction of anaesthesia.

Group – 3 : Received sublingual nifedipine (10 mg), 10 minutes before induction of anaesthesia.

Group- 4 : Intravenous esmolol (2mg/kg) 2 minutes before induction of anaesthesia.

In all the four groups, pulse rate and blood pressure were recorded before study drug administration which has been denoted as pre-induction value (Basal value).The patients were preoxygenated with 100% oxygen for 5 minutes from a Boyle's machine via a face mask and mapleson A system. Anaesthesia was induced 90 seconds after lignocaine, 10 minutes after sublingual nifedipine and 2 minutes after esmolol. Anaesthesia was induced with intravenous thiopentone (5 mg/kg of body weight) followed by suxamethonium (1.5 mg/kg of body weight) with proper care and monitoring. After full relaxation, laryngoscopy was done with a MaCintosh laryngoscope to expose the glottis properly and intubation was carried out in one attempt. The calf of endotrachel tube was inflated. Pulse and blood pressure was recorded which denoted as 'O' time (just after laryngoscopy and intubation). Blood pressure and pulse rate was taken 1, 2, 5 and 10 minutes after laryngoscopy and intubation (the 'zero' time). Maintenance of anaesthesia was carried out with nitrous 67%, oxygen (33%) injection Vecuronium 0.08% mg/kg and injection pethidine (1 mg/kg) intravenously. At the End of Surgery, the subjects were reversed from the residual relaxant effect of non-depolarizing muscle relaxants as necessary. During the whole period, the subjects were carefully observed for any untoward effects and specially for those which might be due to lignocaine, nifedipine and esmolol.

Results

In the present study sixty adult patients from both the sexes were divided into four groups. Group 1 served as

control, Group 2, Group 3 and Group 4 were pretreated with intravenous lignocaine (1.5 mg/kg), sublingual nifedipine (10 mg) and Intravenous esmolol (2 mg / kg) respectively.

In all the groups the patients were between 18 to 50 years. The mean age was 36.67±3.12 years in group 1, 31.67±2.53 years in group 2, 39.93±2.89 years in group 3 and 38.80±2.45 in group 4.).Of the total 60 cases, twenty one were female and thirty-nine were male. All of them were in good nutritional status and free from systemic diseases. The patients in Group 1 had a mean weight 55.73 kg with a range from 40 kg to 70 kg. and in Group 2 was 52.67 kg. from 40 kg to 68 kg. The average weight in group 3 was 56.27 kg (range 44 kg to 70 kg), in Group 4 was 53.40 kg (range 45 kg to 68 kg).

There was an increase of pulse rate in all the groups of patient just after laryngoscopy and endotracheal intubation and 1 minute after intubation. At time 3, pulse rate was highest in group 1 (20.79%) and lowest in Esmolol group. When compared to basal value this increase was highly significant in all the groups ($p<0.01$). At time 4, the increase of pulse rate was highest in control group (15.73%) and lowest in esmolol group (6.28%) thereafter, the pulse rate decreases gradually. In control group, pulse rate returns to baseline value at 10 minute after intubation (Time 7). In lignocaine (Group 2) pulse rate returns to base line value at 5 minute after intubation and in group 4, pulse rate returns to base line value at 2 minutes after intubation and at 10 minute it goes below the basal level which is statistically significant ($p<0.01$).

Just after laryngoscopy and intubation, there was a peak increase of systolic bood pressure in all the groups. This increase was highest in group 1 (13.22%) and lowest in group 4 (6.12%). When compared to the control group of patients, lignocaine produce no significant change but nifedipine and esmolol produced a significant decrease of systolic blood pressure at 1 minute after intubation. With nifedipine and esmolol, the systolic blood pressure returns to the basal value within 1 minute after intubation and at 5, 10 minutes it comes down below the basal level which is statistically significant ($p<0.01$). In group 1, the systolic blood pressure comes down to the basal level at 10 minute and in lignocaine group at 5 minutes after intubation.

There was a peak increase of diastolic pressure just after laryngoscopy and intubation (Time 3). This Peak increase was highest in control group (15.63%) and lowest in esmolol group (6.10%). This increase was highly significant in all the groups when compared to basal value. When compared to the control group of patients Nifedipine and esmolol produce no significant change at 1 and 2 minutes after intubation. But there is significant decrease of diastolic pressure at 5 and 10 minutes after intubation. In group 2, these is no significant change of diastolic pressure at Time 2, Time 5 and Time 6 but there is significant decrease of pressure at 10 minutes after intubation ($p<0.01$).

There was peak increase of mean arterial pressure just after laryngoscopy and intubation (Time 3). This increase was highest in control group (14.54%) and lowest in esmolol group (6.11%). This increase was 10.23% in lignocaine group and 7.43% in Nifedipine group. This increase was highly significant in all the groups when compared to basal value ($p<0.01$).When compared to the control group of patients, lignocaine produced no significant change at 2 and 5 minutes after intubation. But at 5 and 10 minutes both Nifedipine and esmolol produced a significant decrease of mean arterial pressure (MAP) ($p<0.01$).

There was peak increase of Rate-pressure product just after laryngoscopy and intubation (Time 3) in all the groups. This increase was highest in control group (36.87%) and lowest in esmolol group (16.54%). This increase was highly significant in all the group ($p<0.01$). When compared to the control group of patients lignocaine produced no change at 5 minutes but in Nifedipine and esmolol group, no significant change found at 2 minutes after intubation. At 10 minutes after intubation, there is statistically significant decrease of rate-pressure product in all the groups except control groups. Sublingual Nifedipine was unable to attenuate the pulse rate but it produced a significant attenuation of systolic, diastolic and mean arterial pressure. More-over it produced statistically significant fall of pressure throughout the study period which is unwanted.

Intravenous lignocaine produced significant rise of pulse rate and blood pressure just after laryngoscopy and intubation (Time 3), 1 and 2 minutes after intubation (Time 4 and Time 5). Pulse rate and blood pressure comes to the basal level at 5 minutes after intubation. Significant decrease of blood pressure occurs at 10 minutes after intubation (Time 7, $p<0.01$).Intravenous esmolol was able to attenuate the pulse rate, systolic, diastolic and mean arterial pressure throughout the study period. Though there was significant drop of pressure at 5 and 10 minutes after intubation but it was not to the extent of Nifedipine.

Discussion

Endotracheal intubation has become the mainstay of modern anaesthesia due to various reasons like maintenance of good airway, prevention of aspiration, better oxygenation and laryngoscopy and tracheal intubation leads to reflex cardioacceleration stimulation, leading to an increase in systemic arterial pressure and heart rate.In modern anaesthesia, endotracheal intubation is essential for balance anaesthesia and for respiratory resuscitation measures in intensive care unit.

Bursstein (1950) suggested that these changes are due to an increase in sympathetic discharge via cardioaccelerator fibres.

The reflex cardioacceleration during laryngoscopy occurs due to laryngoscope-blade pressing on the base of the tongue and raising the epiglottis. The afferent path of the reflex is through the sensory fibres of the vagus and efferent is traveling through the cervical sympathetic nerves.

The increased sympathetic activity caused by stimulation of the upper respiratory tract has been supported by the observation that increase in arterial pressure during endotracheal intubation is associated with an increase of plasma nor-adrenaline level (Russel, W J 81)[3]. The initial rise of blood pressure and pulse rate is due to laryngoscopy and later an slight more increase is due to intubation. After intubation there is gradual return of blood pressure and pulse rate to pre-laryngoscopic value. This is probably due to the fatigue of the receptor.

The overall effect of inhaled anaesthetic is decrease in cardiac output and systemic vascular resistance. The inhaled anaesthetics are direct and potent depressants of myocardial contraction (Miller RD, '90). [Anaesthesia,4th edition, 1994][4].

Various methods have been used to attenuate the cardiovascular responses due to endotracheal intubation. These methods include deepening of the plane of anaesthesia (King BD '51. Prys – Roberts, '71), topical anaesthesia of laryngopharynx and Epiglottis (Delinger JK, '74, Stoelting RK '77, '78)[5,6,7]. Fentanyl and alfentanyl (Martin DE, '82, Black TE, '84). produce significant attenuation of cardiovascular response during intubation[8,9].

The effect of intravenous and oral practolol in hypertensive patients showed a significant attenuation of cardiovascular responses following laryngoscopy and intubation (Prys-Roberts '73).

The effect of esmolol, a new ultrashort acting beta blocker with an elimination half life only nine minutes, on the attenuation of cardiovascular responses to laryngoscopy was found to be much satisfactory (Achola KJ '88)[10].

Intravenous lignocaine 1.5 mg/kg 90 seconds before laryngoscopy and viscous lidocaine 25 ml (2%) given as mouth wash 10 minutes before laryngoscopy were equally protective but former seemed to be more logic choice (Stoelting RK, '77). But viscous or IV lignocaine were of no value when laryngoscopy is of very short duration (less than 15 seconds) (Stoelting RK '78)[11].

In 1979, Stoelting RK concluded that a single rapid intravenous injection of sodium nitroprusside (1 or 2 microgram/kg) is a practical pharmacological method to attenuate the blood pressure increase during direct laryngoscopy and tracheal intubation[12].

For attenuating the hypertensive and tachycardia response during endotracheal intubation, various other agents have been used such as metoprolol (Magnusson J '88), labetolol (Roelopse JA, '87), Magnesium sulphate (Allen RW, '91), captopril blocks the pressure response and tachycardia. The methods may them selves carry some additional risks and the drugs used may be long acting or have undesirable side effects[13,14]. The present study was carried out to observe the changes in arterial pressure, heart rate, Mean arterial pressure and Rate trachea and to compare the ability of IV lignocaine, sublingual Nifedipine and IV esmolol to obtend these responses.

Lignocaine causes peripheral vasodilation and myocardial depression. Lignocaine in plasma concentration 2-5 microgram/ml causes mild peripheral vasodilation with slight or no changes in myocardial contraction, diastolic filling and cardiac output. Lignocaine can accelerate the ventricular response during atrial flutter. It should be used cautiously in the treatment of supraventricular tachycardia during anaesthesia. Lignocaine can also cause seizures. Rapid IV Injection of lignocaine may cause cardiovascular collapse in susceptible patients. Following an concentration declines rapidly with a redistribution half life of about 10 minutes and elimination half life of about 2 hours (Roelepse, JA '87)[15].

Nifedipine inhibits the passage of calcium through the voltage-gated membrane channel of vascular smooth muscle and cardiac muscle. It reduces the available intracellular calcium and the muscle to relax.

It is the most potent vasodilator and can cause hypotension and faintness. Conventional Nifedipine has been used together with antihypertensive drugs that attenuates adrenergic responses. Reflex cardiac stimulation precipitating angina or myocardial infarction is rare because of the concomitant coronary vasodilatation of the drug. It is effective both in angina and hypertension.

Esmolol is a B_1 selective antagonist with a very short duration of action. It's elimination half life being 8 minutes. It's peak of action within 8-9 minutes and its activity ceases within 15-20 minutes after stoppage of the drug. It is metabolized by red cell esterase [DR Laurense, PN Bennett: Clinical Pharmacology, 1994][16].

In the present study sixty adult patients from both the sexes were divided into four groups. Group 1 served as control, Group II, Group III and Group IV were pretreated with intravenous.

Lignocaine (1.5 mg/kg), sublingual nifedipine (10 mg) and Intravenous esmolol (2 mg / kg) respectively.Increse in arterial pressure and heart rate caused by laryngoscopy and ETI was first described by King *et al*[5]. Rise of pulse rate after laryngoscopy and ETI was highest among control group in our study Prior administration of esmolol was most effective in blunting this rise of pulse rate. Intravenous esmolol was able to attenuate he pulse rate, SBP, DBP, and MAP through the study period.Though there was significant drop of pressure at 5 and 10 minutes after intubation but it was not to the extent of nifedipine. This observation was similar to the previous studies[11]. Other also reported that various beta blockers (like metoprolol,

labetelol) have got the similar effects on pulse rate following laryngoscopy and ETI[13,15]. Sublingual nifedipine and esmolol effectively blunted the rise of SBP, DBP and MAP following laryngoscopy and ETI in our study. Sublingual nifedipine was unable to attenuate the pulse rate but it produced a significant attenuation of SBP, DBP and MAP. Morever it produced statistically significant fall of pressure throughout the study period which is unwanted. This was similar to the study results reported by Korpriva *et al* who used for reduction of heart rate but showed no effect on cardiac output and MAP[17].

Intravenous lignocaine produced significant rise of pulse rate and blood pressure just after laryngoscopy and intubation (Time4 and Time5) Pulse rate and blood pressure comes to the basal level at 5 minutes after intubation. Significant decrease of blood pressure occurs at 10 minutes after intubation ($p<0.01$, Time7). This findind is similar to other previous studies[7,18].

Conclusion

Laryngoscopy and intubation of trachea often evokes cardiovascular responses characterized by an increase of arterial pressure and heart rate and disturbance of cardiac rhythm. Usually these transient change have no deleterious effect in healthy patients, but in patients with altered tone in cardiovascular system, these changes may provoke life threatening consequences. The present study compares the efficacy of intravenous lignocaine, sublingual Nifedipine and intravenous esmolol for attenuation of cardiovascular responses during laryngoscopy and endotracheal intubation.

Analyzing the different data obtained from this study it was found that intravenous lignocaine was not so much effective in attenuating the cardiovascular responses to larygoscopy and intubation.

Sublingual Nifedipine produce a significant attenuation of systolic, diastolic and mean arterial pressure but it was unable to attenuate the pulse rate satisfactorily. At the same time, there is significant fail of pressure during the study period which is unwanted.

In the esmolol group, both pulse rate and arterial pressure showed a significant rise just after laryngoscopy and intubation. But two minutes after intubation the rise was not statistically significant in comparison to control group. Esmolol could check the rise both pulse rate and blood pressure at 2 minutes after intubation.The action of the easily controllable and reversible. It seems that esmolol is a very selective and appropriate answer to the problem of short time pressure response to laryngoscopy and endotracheal intubation.

From the present study, it is concluded that intravenous esmolol is the best attenuator amongst the three drugs studied over here to the cardiovascular responses during laryngoscopy and intubation.

Acknowledgements

We are thankful to all the staff members and interns & residents of department of anaesthesia, Surgery and department of orthopedics for their support. We are also thankful to our patients to be part of our study.

References

1 Takeshima K, Noda K, Higachi M — Cardiovascular response to rapid anaesthesia induction and endotracheal intubation. *Anesth Analg* 1964; **43:** 201-8.
2 Forbes A, Dally FG — Acute hypertension during induction of anaesthesia and endotracheal intubation in normotensive man. *Br J Anaesth* 1970; **42:** 618-24.
3 Russell WJ, Morris DB, Frewin, Drew SE — Changes in plasma catecholamine concentration during endotracheal intubation. *Br J Anaesth* 1981; **53:** 837-9.
4 Miller RD — Anaesthesia,4th edition,1994.
5 King BD, Harris LC Jr, Griefenstein FE, Elder JD Jr, Dripps RD — Reflex Circulatory responses to direct laryngoscopy and tracheal intubation performed during general anaesthesia. *Anaesthesiology* 1951; **12:** 56-66.
6 Prys Roberts C, Foex P, Biro GP, Roberts JG — Studies of anaesthesia in relation to hypertension. V Adrenergic beta-receptor blockade. *Br J Anaesth* 1973; **45:** 671-81.
7 Denlinger JK, Ellison N, Ominsky AJ — Effects of intratracheal lidocaine on circulatory responses to tracheal intubation. *Anesthesiology* 1974; **41:** 409-12.
8 Martin DE, Rosenberg H, Aukburg SJ, Bartkowski RR, Edwards MW Jr, Greenhow DE, Klineberg P — Low-dose fentanyl blunts circulatory responses to tracheal intubation. *Anesth Analg* 1982; **61:** 680-4.
9 Black TE, Kay B, Healy TE — Reducing the haemodynamic responses to laryngoscopy and intubation. A comparison of alfentanil with fentanyl. *Anaesthesia* 1984; **39:** 883-7.
10 Achola KJ, Jones MJ, Mitchell RW, Smith G — Effects of beta-adrenoceptor antagonism on the cardiovascular and catecholamine responses to tracheal intubation. *Anaesthesia* 1988; **43:** 433-6.
11 Stoelting RK — Blood pressure and heart rate changes during short-duration laryngoscopy for tracheal intubation: influence of viscous or intravenous lidocaine. *Anasth Analg* 1978; **57:** 197-9.
12 Stoelting RK — Attenuation of blood pressure response to laryngoscopy and tracheal intubation with sodium nitroprusside. *Anesth Analg* 1979; **58:** 116-9.
13 Magnusson J, Thullin T, Werner T — Haemodynamic effects of pretreatment with metoprolol in hypertensive patients undergoing surgery. *Br J Anaesth* 1986, **58:** 251-60.
14 Allen RW, James MF, Uys PC — Attenuation of the pressor response to tracheal intubation in hypertensive proteinuric pregnant patients by lignocaine, alfentanil and magnesium sulphate. *Br J Anaesth* 1991; **66:** 216-23.
15 Roelofse JA, Shipton EA, Joubert JJ,Grotepass FW — A comparison of labetalol, acebutolol, and lidocaine for controlling the cardiovascular responses to endotracheal intubation for oral surgical procedures. *J Oral Maxillofac Surg* 1987; **45:** 835-41.
16 Laurense DR, PN Bennett: Clinical Pharmacology,1994
17 Korpiva CJ, Brown ACD, Pappas G — Haemodynamics during general anaesthesia in patients receiving propranolol. *Anesthesiology* 1978; **48:** 28-33.
18 Hamill JF, Bedford RF, Weaver DC, Colohan AR — Lidocaine before endotracheal intubation: intravenous or laryngotracheal? *Anaesthesiology* 1981; **55:** 578-81.