Indian Journal of Anesthesia and Analgesia

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Pulse Oximetry Revisited

K.K. Mubarak

Oximetry is the measurement of transmitted light through a translucent measuring site to determine the oxygen status. Pulse oximeter is used for arterial hemoglobin oxygen saturation and pulse rate monitoring. Under normal physiological conditions arterial blood is 97% saturated, and venous blood 75% saturated.

History

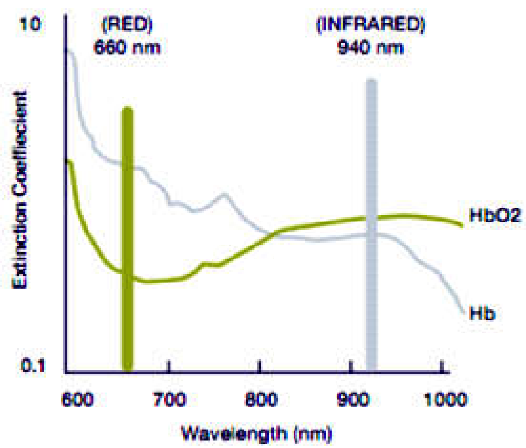
In 1930's German investigators used spectrophotometers to research light transmission through human skin. Kramer (1934) measured oxygen saturation in blood flowing through vessels in animals using photocell. In 1939, German researchers used "ear oxygen meter" that used red and infrared light. British researcher Millikan in 1940 used two wavelengths of light to produce ear oxygen meter for which he coined the word "oximeter". In 1964, Robert Shaw, San Francisco surgeon developed a self-calibrating ear oximeter. In 1972, Takuo Aoyagi, Japanese bioengineer, found that pulsating changes in the light transmission through the ear could measure arterial oxygen saturation and invented the first pulse oximeter in 1975. It was first clinically used by Japanese anaesthesiologist Yoshiya (1980) to monitor oxygen saturation during surgery. In 1980's, there were advances in size reduction, cost, and development of multiple site probes. During 1990's 'new generation' pulse oximeters with better accuracy of readings became popular.

In 1994, American Association of Respiratory Care released the guidelines for the use of pulse oximetry as the 5th vital sign, along with heart rate, respiratory rate, temperature and blood pressure.

Principle

Pulsatile signal generated by arterial blood, is relatively independent of non-pulsatile arterial, venous and capillary blood, and other tissues. Oxyhemoglobin (O_2Hb) and reduced hemoglobin (Hb) have different absorption spectra. Oxygen saturation is the amount of oxygen dissolved in blood, based on the detection of Hemoglobin and Deoxyhemoglobin. Bloodstream is affected by the concentration of O_2Hb and Hb, and their absorption coefficients are measured using wavelengths 660 nm (red light spectra) and 940 nm (infrared light spectra). Deoxygenated hemoglobin has a higher absorption at 660 nm and oxygenated hemoglobin has a higher absorption at 940 nm. Isobestic point is the wavelength at which the absorption by the two forms of the molecule is the same.

Absorption Ppectra of Hb and HbO2



Beer-Lambert law relates the concentration of a solute to the intensity of light transmitted through a solution. It estimates the concentration of a light absorbing substance in a clear solution from the intensity of light transmitted through it. The concentration of a solute in clear solution can be calculated from the intensity of transmitted and incident light of known wave lengths. If there is one solute, the absorption is a product of the path length, the concentration and the extinction coefficient (a constant for a given solute at a specified wave length). If more than one solute is present, the absorption is the sum of for each solute. The absorbance of different wavelengths is dependent on the different solute concentrations (reduced and oxygenated hemoglobin), and is detected by transmitting light of specific wavelengths across thesolution and measuring the intensity.

Conventional pulse oximetry uses two

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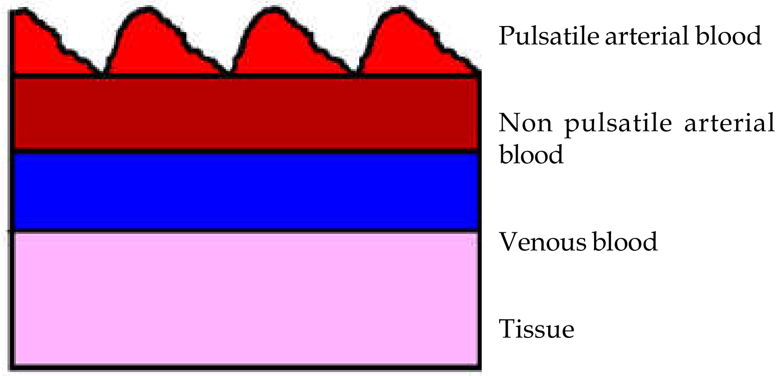

wavelengths of light (red and infrared) transmitted through the finger which is sensed by a photodetector. The pulse oximeter uses the red-to-infrared signal ratio and proprietary calibration tables to calculate SpO_2. The photodetector in the sensor perceives the non-absorbed light from the LEDs. This signal is inverted using an inverting operational amplifier.

This signal represents the light that has been absorbed by the finger and is divided into DC and AC components. DC component represents the light absorption of the tissue, venous blood, and non-pulsatile arterial blood and AC component, the pulsatile arterial blood. Pulse oximeter analyzes the light absorption of two wavelengths from the pulsatile-added volume of oxygenated arterial blood (AC/DC) and calculates the absorption ratio using the equation.

(AC660)/(DC660)

(AC940)/(DC940)

SpO2 is taken out from a table stored on the memory calculated with empirical formulas.

For more reliability, the table is based on measurements on healthy persons. The photoplethy smographic waveform is used to determine the pulse rate.

Errors in Pulse Oximetry

As pulse oximeters are dual-wavelength devices, presence of Hb species other than Hb and O_2Hb result in false reading.

Carboxyhemoglobin

At 920 nm, COHb has low absorbance and does not contribute to total absorbance. At 660 nm, COHb has an absorbance similar to that of O_2 Hb; therefore, SpO_2 will be falsely high. Each % increase in COHb results in 1 % increase in SpO_2 reading.

Methemoglobin (MetHb) has larger absorbance than either of the two major species of Hb at 940 nm, but simulates Hb at 660 nm. Therefore, at high SaO_2 levels (>85%) the reading underestimates the true value; at low SaO_2 levels (<85%) the value is falsely high. In high MetHb concentrations, SpO_2 approaches 85 percent, independently of the actual arterial oxygenation.

Other Hemoglobin Species

Sufhemoglobinemia can produce errors in oximetry, usually with a false reading of methemoglobin. Hemoglobin F in neonates has almost the same absorption spectrum as hemoglobin A, hence no measurable effect on SpO_2. During sickle cell crisis SpO_2 overestimates SaO_2 by 6.9 percent. Patients with sickle cell disease have rightward shift of the O_2 dissociation curve and therefore at any given PaO_2 value, SpO_2 is lower than the normal Hb-O_2 dissociation curve would predict.

Hemoglobin Concentration

At normal oxygenation levels, over a range of hemoglobin 2.3 to 8.7 g/dL, SpO_2 accurately reflects SaO_2. During hypoxia, SpO_2 underestimate SaO_2 to a degree that increases linearly as Hb concentration falls. Polycythemia has no apparent effect on the reading.

Dyes

Methylene blue results in decrease in measured SpO_2. Indocyanine green causes less decrease than methylene blue. Fluorescein injection has no measurable effect. Isosulfan blue, dye used to visualize lymphatics for surgical procedures is associated with prolonged reduction in SpO_2.

Nail Polish and Nail Pigments

Blue nail polish, with absorbance near 660 nm, has the greatest effect, an artifactual decrease. Other colors have smaller effects. Red henna has no significant effect, but black henna can block the light to prevent a correct reading. *Bilirubin* have no significant effect on pulse oximeter readings.

Skin Pigment

Deeply pigmented skin can result in inability to pick up arterial pulsations.

Ambient Light

particularly fluorescent light can falsely elevate SpO_2 reading. Poor contact of the sensor with the skin can result in direct "optical shunting" of light from source to detector, either directly or by reflection from the skin, resulting in a falsely low SpO_2 reading.

Electrical interference from electrosurgical unit can give incorrect pulse rate or decrease in oxygen saturation. This is more in patients with weak pulse signals. Steps to minimize electrical interference include locating the grounding plate close to the oximeter sensor and far from the surgical field as possible.

Motion Artifacts

Motion may produce prolonged detection time for hypoxaemia. It can be significant in shivering or during patient transport.

Evoked potential monitors and nerve stimulators can produce motion artifacts if the pulse oximeter sensor is on the same extremity.

Pressure on the sensor may result in inaccurate SpO2 readings without affecting pulse rate. *Reduced blood flow* results in a diminished signal and can cause inability to obtain SpO_2 reading or low reading, due to greater fractional tissue consumption of arterial oxygen. This results in lower saturation in the pulsatile blood component. There may be a slight reduction in measured SpO_2 during the reactive hyperemia after ischemia in the arms. *Vasoconstriction or hypotension* can cause loss of SpO_2 signal. Topical nitroglycerin ointment has been used to restore the signal, but can worsen hypotension. Digital block is often successful. Topical Emla cream (lidocaine 2.5%, prilocaine 2.5%) to the earlobe covered by an occlusive plastic dressing for 30 minutes may facilitate the signal.

Irregular Heart Rhythms

During aortic balloon pulsation, the augmentation of diastolic pressure exceeds that of systolic pressure. This leads to a double or triple-packed arterial pressure waveform that confuses the pulse oximeter so that it may not provide a reading. Pulse oximetry is unreliable in atrial fibrillation and fast cardiac rhythms.

Neonates

Pulse oximeters are unreliable in the newborn, as minor changes in skin temperature, and minor adjustments in contact can cause motion artifacts and poor signal.

Delayed Detection of Hypoxia

There may be a delay between change in alveolar oxygen tension and change in the oximeter reading. Desaturation is detected earlier when the sensor is more central. Lag time will be increased with poor perfusion and decrease in blood flow. Neural block may cause the lag time to decrease while venous obstruction, vasoconstriction, hypothermia and motion artifacts delay detection of hypoxaemia.

Loss of Accuracy at Low Values

SpO2 is less accurate at low values. 70% saturation is generally taken as the lowest accurate reading.

Recent advances include analysis of photo plethysmographic waveform (respiratory variations, perfusion index, and venous pulse) use of multiple wavelengths of light to quantify methemoglobin, carboxyhemoglobin and total hemoglobin in blood and use of electronic processes to improve pulse oximeter signal processing during low signal-to-noise ratio.

Morphological Analysis of Photo Plethysmographic Waveform

The waveform is based on a signal proportional to infrared light absorption between the emitter and photo detector in the probe. The raw waveform has DC and AC components. Photo plethysmography measures changes in volume of the finger. The larger the blood volume in the finger, the more light is absorbed by the finger. Thus, less light passes through the finger and current generated by the photodetector is smaller. So, during systole the amount of light transmitted is less than diastole, and the original plethysmograph signal resembles a mirror image of an arterial blood pressure waveform. To make it easier for interpretation, the plethysmograph waveform is inverted and auto-scaled to fit the display area.

Changes in the AC and DC components are related to the vasomotor tone. The DC component is also influenced by respiration and is related to the fluid status.

Perfusion index(PI) is defined as AC/DC × 100% of the plethysmograph waveform. It reflects the peripheral vasomotor tone. Low PI suggests peripheral vasoconstriction (severe hypovolemia) and high PI suggests vasodilation. PI is also sensitive to temperature of the finger, vasoactive drugs, sympathetic activity (pain, anxiety) and stroke volume.

Pleth Variability Index (PVI)

It quantifies the variability in plethysmograph waveform due to respiration and is a measure of intravascular volume. It is defined as $(PI_{max} - PI_{min}) / PI_{max} \times 100\%$.

Monitoring Intravascular Volume Status

In 2005, the respiratory variation in pulse oximeter waveform amplitude (ÄPOP) was described. ÄPOP was shown to be sensitive to venous return in mechanically ventilated patients and an accurate predictor of fluid responsiveness by noninvasive method. PVI is a clinically available continuous measurement of the respiratory variations of the plethysmographic waveform amplitude. Future studies will define the utility of PVI to guide intravascular volume management.

Pulse Oximetery for Regional Anaesthesia

Local anesthetic-induced sympathectomy during regional anesthesia causes peripheral vasodilation. This can be quantified by the plethysmograph waveform (PI). PI has been evaluated as a predictor of the success of regional anesthesia. Studies have shown that PI was a good indicator of intravascular epinephrine injection which induced significant decrease in PI.

Other morphological Analysis

Morphological analysis of the waveform can provide information regarding venous pulsation or analgesia (analysis of the slope of the plethysmographic waveform ascending portion). Use of waveform amplitude and pulse rate can be used to estimate the nociception-antinociception balance of the anesthetized patient. This reflects the sympathetic nervous system activation and level of analgesia, which can be used to guide the intra-operative analgesic requirements.

Carboxyhemoglobin and Methemoglobin Measurements

Conventional pulse oximeter measure only oxyhemoglobin and reduced hemoglobin in the blood. Other hemoglobins such as methemoglobin, carboxyhemoglobin, also absorb light in the blood. Recently developed pulse oximeter uses multiple wavelengths of light to analyze several different hemoglobins, which can also measure carboxyhemoglobin and methemoglobin concentrations.

Motion Artifacts, Low Perfusion

Motion artifacts result in a low signal-to-noise ratio, with SpO_2 lower due to venous motion. This venous component is exacerbated by low perfusion. Technological advances are being made to overcome these defects.

Conclusion

Despite problems and limitations, pulse oximetry remains the standard of care in most clinical situations and patients under anaesthesia. As with all monitors one must be familiar

with its performance, advantages and limitations. Intelligent use of pulse oximetry can save lives and prevent hypoxic events.

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Comparison of Intubating Condition between Injection Rocuronium Bromide and Injection Suxamethonium Chloride

Deepa Jadav*, Heena Chhanwal, Paresh Patel*****

Abstract

Objective: Endotracheal intubation is an integral part of administration of anesthesia during surgical procedure. The time interval from suppression of protective reflexes by induction, to accomplishment of intubation is a critical period, during which regurgitation and aspiration of acid gastric contents can occur. Rapid sequence intubation is proved to be the technique of choice in these situations. ***Method:*** We studied 100 patients of either sex aged 18-70 years of ASA grade-1 and 2, with mallampati grade 1-2 undergoing elective surgery. They were randomly allocated in two groups – 50 each to compare the intubating condition of inj. Rocuronium 0.9mg/kg in Group 1 to inj. Suxamethonium 2mg/kg in Group 2. Anaesthesia was induced with fentanyl 2 µg/kg and Propofol 2 mg/kg and intubating conditions were assessed 60s after the administration of the neuromuscular blocking drug. Intubating conditions were graded on a three-point scale as excellent, good or poor, the first two being considered clinically acceptable. **Result:** All patients were successfully intubated in both groups. Overall intubating conditions in group 1 & 2 were excellent in 94% and 100% patients while good in 06% and 00% patients respectively with not quite statistically significant difference (p>0.05). **Conclusion:** It is concluded that rocuronium 0.9 mg/kg can be used as an alternative to suxamethonium 2.0 mg/kg as part of a rapid sequence induction provided there is no anticipated difficulty in intubation.

Keywords: Intubating Condition; Rocuronium; Suxamethonium; Rapid Sequence Induction.

Introduction

Endotracheal intubation is an integral part of administration of anesthesia during surgical procedure [1]. Intubation is one of the common procedures performed during general anaesthesia. Neuromuscular blocking agents are used to achieve muscle relaxation to facilitate endotracheal intubation and to provide surgical relaxation[2]. Rapid sequence induction (RSI) is a technique used to facilitate endotracheal intubation in patients at high risk of aspiration and for those who require rapid securing of the airway [3].

The ideal neuro-muscular blocking agent for rapid sequence intubation should have a fast onset, brief duration of action, provide profound relaxation and free from hemodynamic changes.

Suxamethonium(succinylcholine) - depolarizing neuromuscular blocker (NMB) with its rapid onset and short duration of action is still relaxant of choice to facilitate tracheal intubation [4]. In addition to fasciculation it has got many side effects such as bradycardia and other dysrhythmias [5], rise in serum potassium [6], post operative myalgia, rise in intraocular, intragastric and intracranial pressure [7], incidences of prolonged recovery in patients with pseudo-cholinesterase deficiency and triggering of malignant hyper-thermia.

Because most of the side effects of Suxamethonium reflect its depolarizing mechanism of action therefore search for ideal neuromuscular blocking agent focused on nondepolarizing type of relaxants which has rapid onset time and offers good to excellent intubating condition, as rapidly as Suxamethonium and which lacks the above mentioned adverse effects.

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Rocuronium bromide, a newer amino-steroidal compound, is a derivative of vecuronium; Rocuronium has a rapid onset time, an intermediate duration of action and rapid recovery with cardiovascular stability [8]. Dose : 0.6 -1.2 mg/kg This study was undertaken to evaluate, whether Rocuronium onset time was sufficiently short to permit its use for rapid sequence induction of anesthesia and whether intubating conditions achieved by Rocuronium were similar to those achieved by Suxamethonium.

Materials and Methods

100 patients ranging from 18-70 years of age and from both sex having mallampati grade 1&2 undergoing elective surgery. Patients were assessed pre operatively through history and clinical examination. Investigations were carried out and analysed. Only patients belonging to ASA 1 and 2 were selected for the study. We exclude the patients having following features. (1)Neuromuscular disease (2)H/O or anticipated difficult airway (3)Medications that influence neuromuscular function (4)Allergic to study drugs (5)Pregnancy. After obtaining institutional board approval, written informed consent was obtained from 100 patients and were randomly placed in two different groups (50 in each group)

Group 1: Receives IV inj. Rocuronium 0.9 mg/kg

Group 2: Receives IV inj. Suxamethonium 2 mg/kg

Patients were kept NBM for 6 hours. On arrival of patient in the operative room, patient's baseline heart rate, blood pressure and oxygen saturation was recorded. After securing venous access inj. Glycopyrrolate 0.004mg/kg and inj. Midazolam 0.05 mg/kg was given IV slowly over 1-2 minutes in both groups. Intravenous fentanyl 2 μg/kg was given to all patients at the start of preoxygenation with O2 for 3 min. Anesthesia was induced with intravenous propofol 2 mg/kg this was followed by IV Rocuronium 0.9 mg/kg in group 1, while IV Suxamethonium 2 mg/kg in group 2. Laryngoscopy was performed at 60s after administration of the neuromuscular blocker and intubation done with proper sized endotracheal tube. The duration of laryngoscopy and intubation was restricted to 30s. If diaphragmatic or limb movement occurred at the time of endotracheal intubation, then this was recorded and propofol administered as a rescue drug in a dose of 1 mg/kg body weight.

Neuromuscular monitoring, end-tidal carbon dioxide (ETCO2) and anesthetic agent monitoring was established following induction of anesthesia. Surface electrodes of a peripheral nerve stimulator were placed over the ulnar nerve at the wrist on the hand with the intravenous access. The NIBP cuff was applied over the otherarm. Heart rate, systolic blood pressure, diastolic blood pressure, mean blood pressure, and pulse oximetric saturation were recorded for the first 10 min after intubation. Neuromuscular monitoring was done using a peripheral nerve stimulator to stimulate the ulnar nerve at the wrist using surface electrodes placed along the course of the nerve. Anesthesia was maintained with 50% oxygen, 50% N2O and sevoflurane with controlled ventilation. In Group 1 maintenace dose of Rocuronium was used while in Group 2 Inj. Atracurium was used for maintenance of muscle relaxation. After completion of surgery, reversal of muscle relaxant was achieved with inj. Neostigmine 0.05 mg/kg and inj. Glycopyrollate 0.008 mg/kg iv. After satisfactory recovery, patients were extubated.

Intubating condition was assessed using scoring system of Sluga et al [9].

	Score 3	Score 2	Score 1
Laryngoscopy			
Mandibular muscle relaxation	Relaxed	Acceptable relaxation	Poor relaxation
Resistance to blade insertion	None	Slight resistance	Active resistance
Vocal cords			
Position	Abducted	Intermediate	Closed
Movement	None	Moving	Closing
Intubation response			
Limb movement	None	Slight	Vigorous
Coughing	None	Diaphragmatic	Sever coughing

Intubation Condition Defined as Follows

Excellent – All three factors were rated 3

Good – All three factors were rated 3 or 2

Poor – Presence of one score of 1

Excellent and good intubating condition taken to be "clinically acceptable".

The onset time from the time of administration of muscle relaxant to the complete abolition of all four twitches on train-of-four (TOF) stimulation was noted. The assessment of neuromuscular blockade was done by the appearance of first twitch in the TOF response in the rocuronium groups (nondepolarizingblockers). In the case of succinylcholine group, the return of all twitches with the lowest amplitude (depolarizing blocker). A repeat TOF stimulation was used once every 12s to follow the time course of neuromuscular blockade.

Observation and Results

Data calculation and p value calculation is done by unpaired t-test using SPSS software. The present study includes 100 patients belonging to ASA group 1 and 2 undergoing elective surgery. They were randomly assigned into two groups of 50 each. All the patients were given the drug according to methodology of our study.

Table 1: Demographic Data: age, sex and weight distribution

Group	Age(Years) Mean± S.D.	Weight(Kg) Mean ± S.D.	Sex F:M
GROUP 1 (n=50)	46.52±12.004	55.54±9.389	27:23
GROUP 2 (n=50)	47.06±10.436	53.64±6.548	29:21

Table 1 shows the two groups were comparable with regard to the demographic data.

Table 2: Comparison of Intubating Condition in both groups

Intubating Condition	Group 1 (n=50)	Group 2 (n=50)
Excellent	94%	100%
Good	06%	00%
Poor	00%	00%

Table 3: Comparison of Intubating Score in both groups

	Intubating Score (at 60 sec.)MEAN± S.D.
Group 1 (n=50)	8.94±0.239
Group 2 (n=50)	9.00±0.000

There is not quite statistically significant difference in the intubation score at 60 sec. in both groups

Discussion

We used rocuronium in dose of 0.9 mg/kg in this study. The extra anaesthetic depth needed, coupled with these laryngeal movements are two drawbacks that cannot make the low dose rocuronium as a desirable technique for rapid sequence intubation. Use of higher dose of rocuronium to improve intubating conditions during rapid sequence intubation and to cut short the onset time below 60 secs has been advocated by various workers [10,11,12,13] but doses larger than 0.6 mg/kg would be associated with a long duration of action which may be inappropriate in many situations.

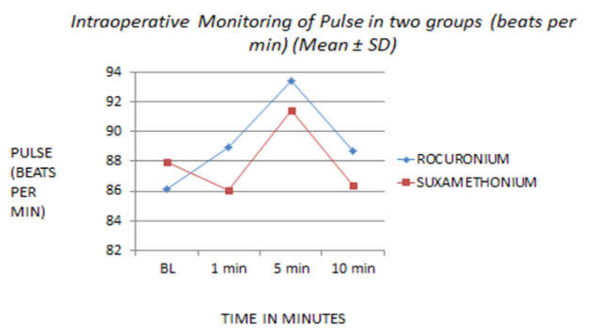
Table 1 shows comparable demographic data among two groups. The endotracheal intubation was commenced at 60 seconds and intubating conditions noted. Similar clinically acceptable (excellent or good) intubating conditions were found in both Rocuronium 0.9 mg/kg and Suxamethonium 2 mg/kg groups. After Rocuronium administration, the response of diaphragm to intubation was more pronounced than that after administration of Suxamethonium. But the overall intubating conditions were similar to those after Suxamethonium administration. Intubation conditions were rated as excellent in 94% and good in 06% of the patients who received Rocuronium, and excellent in 100% of the patients who received Suxmethonium [Table 2]. In our study, intubation score at 60 sec. in both groups shows not quite statistically significant difference[Table 3].

Bhardwaj et al. [14], in his study concluded that clinically acceptable intubation conditions were obtained in all patients after rocuronium 0.9 mg/kg and succinylcholine 1.5 mg/kg as compared to only 37% in rocuronium 0.6 mg/kg group (p<0.0001). Jaw relaxation was significantly better in rocuronium 0.9 mg/kg and succinylcholine 1.5 mg/kg. The intubation time was significantly longer in rocuronium 0.6 mg/kg. They concluded, rocuronium in a dose of 0.9 mg/kg may be a valuable alternative to succinylcholine for rapid tracheal intubation in emergency situations where succinylcholine is contraindicated and surgery if of intermediate duration.

Similar results about onset time and intubating conditions were found in other studies of Dubois *et al* [15] and Huizinga *et al [16]* in these studies, no difference was observed in the frequency distribution of clinically acceptable intubating conditions at 60 and 90 seconds after the administration of Suxamethonium or Rocuronium.

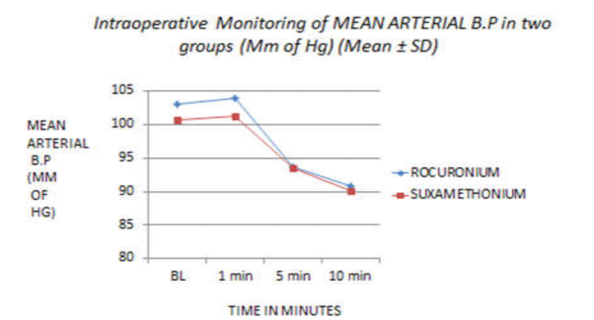
The study of Maria et al., aimed at comparing intubation conditions after 0.6 mg/kg rocuronium at 60 seconds in children, adults and elderly patients and concluded that 0.6 mg/kg rocuronium was sufficient for tracheal intubation in 60 seconds in adult and elderly patients. It was however, insufficient for clinically acceptable tracheal intubation conditions in 60 seconds in 100% of children [17].

The heart rate and mean arterial blood pressure changes upto 10 minutes after intubation in both the groups were statistically non-significant (*P*>0.0001), [Figure1&2]



There is no significant difference in the intra operative pulse in both groups as shown in the graph.

Fig. 1:



There is no significant difference in the post operative mean arterial B.P. in both groups as shown in the graph.

Fig. 2:

Conclusion

It is concluded from this study that Rocuronium 0.9 mg/kg can be used as an alternative to Suxamethonium 2 mg/kg as part of rapid sequence induction at 60 sec., provided there is no anticipated difficulty in intubation.

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An Observational Study to Compare Dexmedetomidine and Esmolol for Induced Hypotension in Nasal Surgeries

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Abstract

Aims &Objectives: To Compare the effectof IV infusion of Dexmedetomidine and Esmolol for induced hypotension during nasal surgeries under general anaesthesia.

To Compare

- Hemodynamic changes
- Intraoperative surgical field
- Sedation and analgesia in post-operative period between the groups.

Materials and Methods: 60 patients for nasal surgeries under ASA I/II were allocated in 2 groups. All the patients were premedicated, induced and maintained inusual manner. *Group D*: received Inj. Dexmedetomidine1 μg/kg as a loading dose over 20 minutes followed by an infusion of 0.2-0.6 μg/kg/hr IV. *Group E*: received Inj. Esmolol 1mg/kg as a loading dose over 1 minute followed by an infusion of0.4-0.8 mg/kg/hr IV. Intra operative Heart rate, Mean arterial pressure, surgical field ,post-operative sedation and analgesia were evaluated. *Results and Summary*: There was no significant difference of MAP and Heart rate in both groups intraoperatively , but there was significant difference at the end of surgery.There was no significant difference in the amount of blood loss in both groups. Mean postoperative sedation score was significantly higherin D than in E group. The duration of first analgesic request was significantly longer in D than E group. No side effects were observed. *Conclusion*: Dexmedetomidine or Esmolol is effective in providing ideal surgical field during nasal Surgeries, but compared with Esmolol, Dexmedetomidine offers the advantage of sedation and analgesia.

Keywords: Nasal Surgeries; Dexmedetomidine; Esmolol.

Introduction

Endoscopic nasal surgeries have several advantages over conventional techniques [1]. Complication like haemorrhage was reported most commonly after Endoscopic Intranasal Ethmoidectomy [2]. Induced hypotension helps to limit intraoperative blood loss.

Esmolol is an ultrashort acting selective β_1 adrenergic antagonist. It has rapid onset of action without development of rebound hypertension [3].

Dexmedetomidine is potent α_2 adrenergic receptor agonist. It acts on peripheral and central nervous system [4]. It has sedative, analgesic effects [5].

Observational study to compare efficacy and safety of dexmedetomidine and esmolol as a hypotensive agent in nasal surgeries with regard to amount of blood loss, quality of the surgical field, sedation, and analgesia.

Method

This is observational prospective study conducted at Sumandeep Vidyapeeth University, and Dhiraj Hospital, Piparia, Dist.-Vadodara. After approval from the local ethical committee, 60 ASA physical status I or II patients aging 20-60 years scheduled for elective nasal surgery were selected for the study. Patients with coronary artery disease, renal, hepatic or cerebral insufficiency, patients with coagulopathies or receiving drugs influencing blood coagulation and chronic hypertension were excluded from the study. The patients were assessed clinically in addition to ECG, chest X ray and basic laboratory tests and written informed consent was obtained. Patients included in this study were assigned according to computer generated to receive either dexmedetomidine (D-group n=30) or esmolol (E-group n=30).

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In the operating room, two cannulae were inserted, one for infusion of dexmedetomidine or esmolol and the second for administration of fluids and other drugs. In D-group, patients received loading dose of 1 μg/kg dexmedetomidine diluted in 0.9% saline infused over 20 minutes before induction of anaesthesia, followed by continuous infusion in the range of 0.2- 0.6 μg/kg/hr. In E group, patients received esmolol as a loading dose of 1 mg/kg over 1 minute followed by continuous infusion in the range of 0.4-0.8 mg/kg/hr. In both groups infusion rate was titrated to maintain Mean Arterial Pressure (MAP) within 55-75 mmHg. All patients were premedicated with IV Inj. glycopyrrolate 0.004 mg/kg, Inj. ondansetron 0.1 mg/kg and Inj. midazolam 0.05 mg/kg. Patients were induced with propofol 2-2.5 mg/kg. Endotracheal intubation was facilitated with succinylcholine 1.5 - 2mg/kg with suitable sized cuffed tube. Anaesthesia was maintained with O_2, N_2O, isoflurane & Inj. atracurium with mechanical ventilation. Oropharyngeal pack was used. Nitroglycerine was to infused if these target limits could not be achieved with upper most doses. The drug infusion rate was then decreased when targeted MAP was achieved. Respiratory rate (RR) and tidal volume (TV) were adjusted according to body weight. Patients were placed in a 10° reverse Trendlenburg position to improve venous drainage. In both groups cotton soaked with epinephrine in a concentration of 1:2,00,000 was inserted into the nasal cavity to minimize blood loss.When MAP reached the desired range (55-75 mmHg) and maintained forabout 10 minutes, surgeon estimated the quality of the surgical field using Frommes' Bleeding Scale [6].

Frommes' Bleeding scale for assessment of intraoperative surgical field:

0 - No bleeding, Ideal Surgical Field

1 - Mild bleeding – Aspiration not required

2 - Mild bleeding –occasional requirement of aspiration.

3 - Significant bleeding–frequent aspiration required. If aspiration is discontinued for 5 seconds surgical field is impaired

4 - Diffused bleeding – continuous aspiration required.

5 - Abundant Bleeding, even with continuous aspiration surgical field is obscured and surgery not possible

Surgeon was blinded to the hypotensive agent used. The ideal scale value for surgical condition was predetermined to be two or three. The total blood loss was measured from the suction apparatus. Infusion of the study drugs was stopped five minutes before the anticipated end of surgery, and isoflurane was stopped at the end of the surgery and the residual neuromuscular blockade was antagonized with neostigmine (0.05 mg/kg) and glycopyrrolate (0.008 mg/kg).

Monitoring included non-invasive blood pressure measurement, pulse rate and arterial oxygen saturation. These parameters were recorded preoperatively (baseline), after administration of hypotensive agents, intraoperatively at regular intervalsand after stoppage of hypotensive agents. Requirement for additional hypotensive agent (nitroglycerine) was recorded. After extubation and full recovery, patients were transferred to the postanesthesia care unit (PACU) for observation. Postoperative pain was evaluated using a Visual Analogue Scale (0-10), and first analgesic rescue was given when it was ≥ 4. Sedation score [7] was measured using the Ramsay scale at 5,15,30 and 60 minutes after tracheal extubation:

Ramsay Sedation scorefor assessment of post-operative sedation:

1 - anxious, agitated, or restless.

2 - cooperative, oriented, and tranquil.

3 - responsive to commands.

4 - asleep, but with brisk response to light, glabellar tap, or loud auditory stimulus.

5 - asleep, sluggish response to glabellar tap, or auditory stimulus.

6 - asleep, no response.

Results

60 patients who fulfilled the criteria were included in this study. Demographic data regarding age, sex and ASA (I/II) were comparable between the groups.

Out of 60 patients :

10 patients posted for FESS

21 patients posted for septoplasty

29 patients posted for septoplasty with FESS,

Baseline values of MAP were comparable in both groups. There was a significant reduction of MAP intraoperatively in both groups compared to baseline value. In both groups the desired MAP (55-75 mmHg) was observed with no intergroup significant differences either after induction or during hypotensive period. In both groups, an additional hypotensive agent (nitroglycerine) was not required intraoperatively. At 5 and 10 minutes after stoppage of hypotensive agents, at end of surgery and after recovery, MAP was significantly lower in D group than E group(P<0.001). [Table 1, Graph 1]

Baseline values of pulse rate were comparable in both groups.Pulse rate decreased significantly compared to baseline after administration of loading dose in both groups.There were no intergroup significant differences in pulse rate after induction or during the hypotensive period. Pulse rate showed significant increase in E group at 5 and 10 minutes after stoppage of hypotensive agent, at end of surgery and after recovery compared to D group(P<0.001). [Table 2, Graph 2].

Table 1: Mean arterial blood pressure (mm of Hg) of the studied groups

Time	D group		E group		
	Mean	**SD**	**Mean**	**SD**	**P value**
Base line	86.83333	4.503511	84.86667	4.15836	0.0841
5 min	75.66667	2.48212	76.53333	4.614321	0.3687
10 min	73.36667	1.920548	74.23333	2.955805	0.1833
15 min	68.9	2.294671	69.7	1.932481	0.1494
30 min	61.36667	1.49674	61.83333	2.118609	0.3265
60 min	58.7	2.394678	59.6	1.693802	0.0982
90 min	56.43333	1.568732	56.33333	1.561019	0.8054
120 min	62.9	2.218263	84.3	2.246069	0.0001

Table 2: Mean Pulse rate (bpm) of the studied groups

Time	D Group		E Group		
	Mean	**Sd**	**Mean**	**Sd**	**P Value**
Base line	86.26667	4.084735	85.76667	4.980537	0.6723
5 min	73.2	2.441029	74.8	5.579519	0.1555
10 min	70.86207	1.626293	71	5.626293	0.8978
15 min	68.86667	2.080009	67.76667	4.264394	0.2092
30 min	62.53333	2.515241	62.86667	3.339764	0.6640
60 min	61.4	1.544735	61.06667	3.236999	0.6127
90 min	59.86667	2.41737	58.4	3.783813	0.0788
120 min	65.06667	2.517981	93.93333	2.490441	0.0001

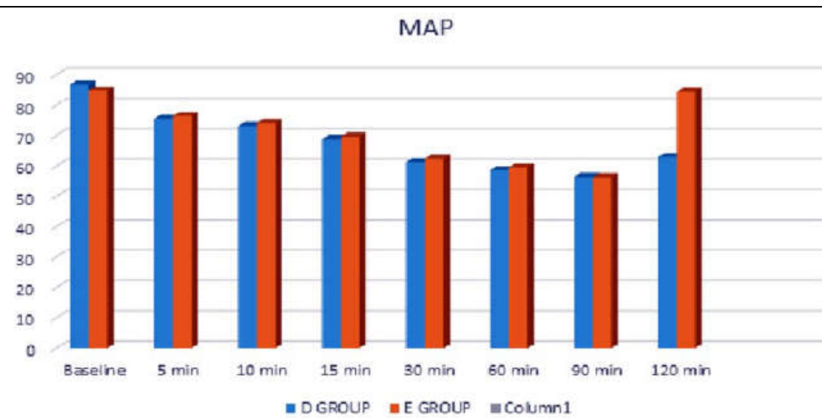

Fig. 1: Mean arterial blood pressure (mm of Hg) of the studied groups

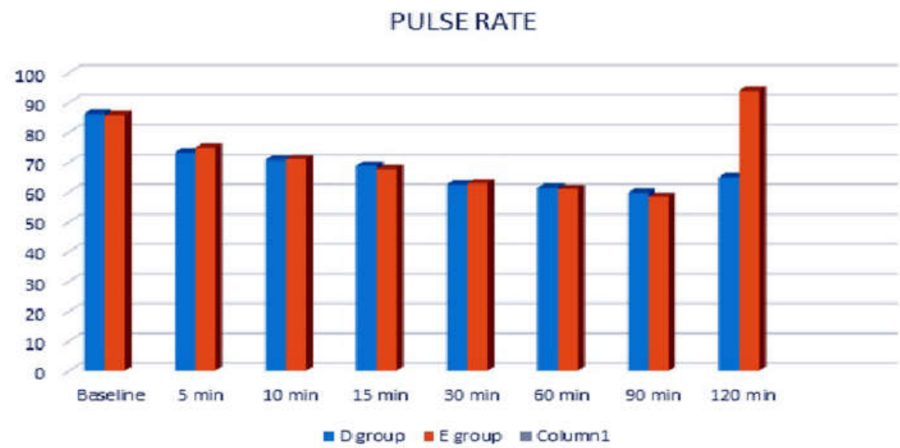

Fig. 2: Mean Pulse rate (bpm) of the studied groups

Table 3: Frommes' Bleeding Scale (0-5) during hypotensive anaesthesia periods [Median (Range)] and Blood loss during surgeries

Time	D Group		E Group		
	Mean	**SD**	**Mean**	**SD**	**P value**
5 min	2.3	0.466092	3.066667	0.449776	0.0001
10 min	2.333333	0.479463	2.233333	0.430183	0.3987
15 min	2.166667	0.379049	2.133333	0.345746	0.7232
30 min	2.233333	0.430183	2.3	0.466092	0.5670
60 min	2.3	0.466092	2.266667	0.449776	0.7790
90 min	2.233333	0.430183	2.166667	0.379049	0.5267
120 min	2.133333	0.345746	2.033333	0.182574	0.1666
Blood Loss During Surgeries					
	Mean	**SD**	**Mean**	**SD**	**P value**
Blood Loss	88.66667	12.24276	87.03333	11.49058	0.5962

Table 4: Ramsay Sedation scores and time to first analgesic request

Time	D group		E group		
	Mean	**SD**	**Mean**	**SD**	**P value**
5 min	4.033333	0.718395	3.166667	0.461133	0.0001
15 min	3.9	0.661764	2.366667	0.490133	0.0001
30 min	3.166667	0.530669	2.133333	0.345746	0.0001
60 min	2.366667	0.490133	2.066667	0.253708	0.0042
Time recorded to first analgesic request	**D group**		**E group**		
	Mean	**Sd**	**Mean**	**Sd**	**P value**
	46.33333	5.862407	25.23333	3.74795	0.0001

The Frommes' Bleeding scale for quality of surgical field was comparable in both groups. Scores for the bloodless surgical field were low in both groups; the median range of scores was 2.2 in both groups.There was no significant difference in the surgical field in both groups ($P>0.05$), but at 5 minutes after induction there was significant difference in the surgical field in both groups ($P<0.001$). There was no significant difference in the blood loss in both groups (*P*>0.05). [Table 3].

The mean postoperative sedation score was significantly higher in D group than in E group at 5,15, 30 and 60 minutes (*P*<0.005).Time recorded to first analgesic request was significantly longer in D group than E group (46.33±5.86 minutesversus 25.23±3.74 minutes) respectively (*P*<0.001) [Table 4]

No postoperative nausea or vomiting observed in both groups.

Discussion

Intraoperative bleeding is one of the major problems in endoscopic surgery of sinuses. Controlled arterial hypotension significantly reduced intraoperative hemorrhage and improved the visibility of the operative field in endoscopic rhinosurgery [8]. In our study dexmedetomidine a nd esmolol, both were effective in achieving MAP of 55 to 75 mmHg, lowering the heart rate and providing bloodless surgical field during FESS and septoplasty.

Patients who were treated with dexmedetomidine 20 minutes before induction of anaesthesia had significant decrease in MAP and pulse rate after administration of loading dose. Dexmedetomidine induced hemodynamic profile can be attributed to the known sympatholytic effect of α_2 agonists. The α_2-receptors are involved in regulating the autonomic and cardiovascular systems. α_2 receptors are located on blood vessels, where they mediate vasoconstriction, and on sympathetic terminal, where they inhibit, norepinephrine release [9].

The efficacy of dexmedetomidine in providing better surgical and less blood loss during controlled hypotension was previously reported during tympanoplasty, septoplasty and maxillofacial surgery [10,11,12].

Esmolol lowers arterial blood pressure through a decrease in cardiac output secondary to negative chronotropic and ionotropic effects of β adrenergic antagonism. It provided a stable course of controlled hypotension and produces beneficial effects in the surgical field and in blood conservation during tympanoplasty [13].

Dexmedetomidine and Esmolol both are effective in providing good surgical field, with an additional effect of analgesia and sedation by dexmedetomidine as compared to Esmolol during FESS surgeries [14].

Dexmedetomidine has sedative effect via central actions in the locus ceruleus and in the dorsal horn of the spinal cord [15].

Conclusion

This study demonstrated that dexmedetomidine or esmolol is safer agent for controlled hypotension and both are effective in providing ideal surgical field during Nasal Surgeries. Compared with esmolol, dexmedetomidine offers the advantage of analgesia and sedation.

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Original Article

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A Double Blind Comparative Study of Effect of Intravenous Magnesium Sulphate with Lignocaine and Intravenous Clonidine with Lignocaine on Heart Rate in Response to Laryngoscopy and Tracheal Intubation during General Anesthesia

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Abstract

Background and Objectives: Laryngoscopy and tracheal intubation is invariably associated with a reflexsympathetic pressor response resulting in elevated heart rate and blood pressures. Thismay prove detrimental in high risk patients. The main objectives of the present study was To study the effect of intravenous magnesium sulphate 30 mg/kg with intravenous lignocaine 1.5mg/kg, and intravenous clonidine 3mcg/kg with intravenous lignocaine 1.5mg/kg on changes in the heart rate (HR) during laryngoscopy and intubation under general anesthesia. *Methods*: 60 ASA I and II status normotensive patients scheduled for elective surgicalprocedures were selected randomly and divided into three groups of 20 each. Allpatients received premedicationwith studydrug magnesiumsulphate 30mg/kg or clonidine 3μgm / kg or normal saline (as per double blind study protocol) prepared by anaesthesia staff and glyopyrrolate 0.2mg i.v., tramadol 3mg /kg 3min before induction . Induction of anesthesia was standardized for all patients who received,thiopentone 5 mg/kg i.v. and preservative free lignoaine 1.5mg i.v and were relaxed with succinylcholine 2mg/kg i.v. and laryngoscopy and intubation is done with appropriate sized endotracheal tube. HR, systolic, diastolic blood pressure were recorded noninvasively before induction, postintubation, 1,3,5, 7 and 10 minutes from the onset of laryngoscopy. 'z' test was used for statistical analysis. *Results*: In CL group, mean heart rate at 5 min and 10 min were 96.25 and 9.83 respectively. In ML group, mean heart rate at 5 min and 10 min were 100.95 and 97.45 respectively. *Conclusion*: There was a statistically significant attenuation of heart rate response was observed after giving study drug in CL group

Keywords: Attenuation; Pressor Response; Laryngoscopy; Intubation; Lignocaine; Magnesiumsulphate; Clonidines.

Introduction

Hypertension and tachycardia during intubation under general anesthesia have been reported since1950 [1.2]. Increase in blood pressure and heart rate occurs most commonly from reflex sympathetic discharge in response to laryngotracheal stimulation, which in turn leads to increased plasma norepinephrine concentration [3]. These changes may be associated with morbidity and mortality in patients with heart disease and hypertension, provoking complications like bleeding, increased intracranial and intraocular pressure.

There are various techniques by which this intubation-related stress response can be attenuated, all of which depend on reduction in input stimuli or the blockade of adrenergic responses e.g. deep anesthesia, topical anesthesia, use of ganglionic blockers, beta blockers [4], antihypertensive agents like phentolamine [5], Sodium nitroprusside, nitroglycerine [6] and calcium channel blockers [7,8].

Intravenous magnesium sulphate inhibits catecholamine release associated with tracheal intubation and produces vasodilation by directly acting on blood vessels [9].

Clonidine, α_2 adrenoreceptor agonist attenuates adrenergic hemodynamic stress response [10]. It is effective in attenuating increase in heart rate and mean arterial pressure during endotracheal intubation [11].

Intravenous preservative free lignocaine with its well established centrally depressant and anti-arrhythmic effect is a more popular method to minimize this pressor response [12,13]. This drug is used routinely for

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generalanesthesia cases in our institution.

In spite of so many studies, so far not many studies have been published for comparing the efficacy of combination of drugs. Hence the present study was undertaken to compare advantages and efficacy of combining intravenous magnesium sulphate with intravenous lignocaine, and intravenous clonidine with intravenous lignocaine on blunting heart rate response in endotracheal intubation during general anesthesia in our institution.

Methodology

A clinical doubleblind comparative study of attenuation of hemodynamic response to laryngoscopy and intubation was done in 60 patients of 15-50years of age scheduled to undergo elective surgery under general anesthesia in Medical College Hospital (MCH), VIMS, Bellary, Karnataka. The patients are included in the study by applying the following inclusion and exclusion criteria.

Inclusion Criteria

a. Patients aged between 15 to 50 years of age posted for elective surgeries under general anesthesia
b. ASA grade I and II patients
c. Patients with Mallampatti airway grade I and II

Exclusion Criteria

a. Patients refusal
b. Patients with medical comorbidities like Hypertension, ischemic heart diseases and arrythmias
c. Patients with Mallampatti III and IV
d. Expected difficult intubation
e. If patient is allergic to any of these drugs

Methods of Collection of Data

Specially designed proforma are used to collect the data which includes patient's particulars, indication for surgery, the anaesthetic details, intra-operative monitoring, observation for side effects etc. 60 patients are randomly allocated to three different groups of 20 each as using block randomization method of randomization as described below (group ML,CL, and NL)

Group ML –will receive magnesium sulphate 30mg/kg iv 3 minutes before induction

Group CL –will receive clonidine 3mcg/kg iv bolus 3 minutes before induction

Group NL –will receive normal saline 4ml iv 3 minutes before induction.

All three groups are coded as A, B, and C by co-ordinator. These are again randomized in all possible permutations and combinations e.g., BAC, CBA, ABC etc and a list of 20 such blocks are prepared by co-ordinator. Such blocks are selected randomly by chit method and given to the researcher ensuring adequate randomization

All the patients were visited the day before surgery and pre-anestheticcounseling was done. All patients received Diazepam 10mg orally at night on theday before surgery

Patients are explained the procedure and informed/written consent obtained.

Anestheticprocedure

- On arrival in the operating room, patient's basal parameters- B.P, heart rate and ECG are recordre using pulse oximetry, NIBP and ECG monitor.
- Intravenous access will be established and an IV infusion of Ringer lactate started
- All the patients are premedicated with Glycopyrrolate 0.2mg iv.
- Patients in each group receive respective drugs as per timing and dose mentioned earlier. The study drug will be prepared by anesthesia staff and the observer will be blind for study drug.
- After preoxygenation, Patients in each group is induced by Thiopentone sodium 5mg/kg iv. Then intravenous lignocaine 1.5mg/kg given. After this Succinyl choline 2mg/kg will be given followed by laryngoscopy and intubation with appropriate sized cuffed endotracheal tube.
- Anesthesiais maintained with Oxygen 33%. Nitrous Oxide 66% and Halothane 0.5 to 1% through Bain's circuit on controlled ventilation.
- Muscle relaxation is done with intermittent doses of Vecuronium Bromide and for analgesia iv tramadol 3mg/kg will be given.
- At the end of surgery reversal is done with Glycopyrrolate and Neostigmine 0.05mg/kg and patient will be extubated.
- The recovery time(the time between injection of reversal agent and extubation) will be noted.

- Patients recovery is monitered by aldretes score after extubation.
- All the parameters of the study will be recorded at following stages –preoperative
  - After giving the study drug
  - Immediately after intubation
  - at 1 minute, 3 minutes, 5 minutes and 10 minutes after intubation.
- All groups are decoded at the end of study by taking information from co-ordunator. Group ML and Group CL will be studied for effects of combination of drugs and Group NL will be for study of effect of single drug and all three are compared in the end.

Results obtained are analyzed statistically.

Results

Table 1: Age wise distribution of study subjects

Age group	Clonidine with lignocaine	Magnesium sulphate with lignocaine	Lignocaine only
< 25 yrs	05 (25%)	05 (25%)	02 (10%)
26-35 yrs	05 (25%)	08 (40%)	10 (50%)
36-45 yrs	05 (25%)	05 (25%)	05 (25%)
> 45 yrs	05 (25%)	02 (10%)	03 (15%)
Total	20 (100%)	20 (100%)	20 (100%)

The table1shows the age distribution in control and the two study groups. The age range was 20 – 50 years for control and study groups

Table 2: Sex wise distribution of study subjects

Sex	Clonidine with lignocaine (CL)	Magnesium sulphate with lignocaine (ML)	Lignocaine only
Male	11 (55%)	09 (45%)	12 (60%)
Female	09 (45%)	11 (55%)	08 (40%)
Total	20 (100%)	20 (100%)	20 (100%)

In CL group, 55% were males and 45% were females
In ML group, 45% were males and 55% were females
In control, 60% were males and 40% were females

Table 3: Comparison of heart rate (bpm) between three groups

Heart rate	Clonidine with lignocaine (A)	Magnesium sulphate with lignocaine (B)	Lignocaine only (C)	P value *	P value #		
					A-B	A-C	B-C
Baseline	89.85 +/- 13.9	97.80 +/- 21.73	91.95 +/- 10.83	0.28	0.27	0.91	0.49
Pre laryngeal	85.00 +/- 16.21	99.55 +/- 16.90	93.35 +/- 11.28	0.01	0.00	0.19	0.39
One min	98.25 +/- 12.34	109.50 +/- 14.54	115.15 +/- 11.33	0.00	0.02	0.00	0.35
Three min	98.00 +/- 11.37	104.10 +/- 15.17	109.75 +/ - 12.34	0.02	0.30	0.01	0.36
Five min	96.25 +/- 11.18	100.95 +/- 15.71	103.20 +/ - 14.56	0.28	0.53	0.26	0.86
Ten min	94.10 +/- 9.83	97.45 +/- 16.50	97.85 +/- 15.06	0.42	0.73	0.67	0.99

* ANOVA test # Post-hoc tukey test
All values are in mean +/- sd

Statistical analysis of changes in heart rate prelaryngoscopy, post intubation at different (1 5, 10,) time intervals from onset of laryngoscopy and intubation in all the 3 study group is presented.

Group CL(A): The basal and prelaryngoscopy mean heart rate and standard deviations in this group were 89.85 +/- 13.9 and 85.00—: 6.21 respectively. After 1min of intubation 8.4bpm (9.34%) increase in the value of heart rate was observed with values of 98.25 +1- 12.3.4 and remained higher with a mean heart rate of 98.00 +/- 11.37 at 3 minutes. Subsequently a decreasing trend in the heart rate was noted starting from 5 minutes to 10 minutes after laryngoscopy. Mean heart rate at 5minutes and 10 minutes were 96.25 +/- 11.18 and 94.10 +/- 9.83 respectively.

Group ML(B): The basal and pre laryngoscopy mean heart rate and standard deviations in this group were 97.80 +/- 21.73 and 99.55 +/- 16.90 respectively. After 1 min of intubation 11.7bpm (11.93%) increase in mean heart rate was observed with mean heart rate and standard deviations of 109.50 +/- 14.54. Subsequently decreasing trend in the heart rate was noted starting from 3 minutes to 10 minutes after laryngoscopy. Mean heart rate at 3, 5 minutes and 10 min were. 104.10 +/- 15.17, 100.95 +1- 15.71, and 97.45 +/- 16.50 respectively.

Group NL(C):The basal and pre laryngoscopy mean heart rate and standard deviations in this group were 91.95 +/- 10.83and 93.35 +/- 11.28 respectively. After l min of intubation23 bpm (25.01%) increase in heart rate was observed with mean heart rate 115.15.

No significant variations noted in all groups in heart rate basal recording. There was attenuation of heart rate response was observed after giving study drug in CL group.

Discussion

The sequence of induction anaesthesia, laryngoscopy and tracheal intubation areassociated with marked haemodynamic changes and autonomic reflex activity which may be a cause of concern in many high risk patients [14].

Laryngoscopy and intubation is associated with rise in heart rate, blood pressureand incidence of cardiac arrhythmias. These potentially dangerous changes disappearwithin 5 minutes of onset of laryngoscopy [15].

Although these responses of blood pressure and heart rate are transient and short lived they may prove to be detrimental in high risk patients especially in those with cardiovascular disease, increased intracranial pressure or anomalies of the cerebral blood vessels [16].

In group NL inj lignocaine 1.5mg/kg i.v. 3 minutes before laryngoscopy and intubation was used to blunt the pressor response, the base line value of heart rate was 91.95. bpm .One minute following laryngoscopy and intubation, the heart rate increased to 115.50 bpm, representing a rise of 23.25 bpm above the base line value.

By 3 minutes, it was 109.75 bpm, representing a rise of 18.25 bpm above the base line value. By 5 minutes it was 103.20 bpm, representing a rise of 11.5 bpm above the base line it was seen that the elevated heart rate started settling down towards base line value by 10 minutes at 97.85.

CD Miller [17] et al employed a dose of 1.5 mg lignocaine and noticed a rise in the heart 72 rate (HR) of 25 bpm and Splinter et al noticed it to be 19 bpm. Hence the results of the present study with regards to increase in the heart rate observed following laryngoscopyand intubation concurs with the observation made by Splinter et al. and CD Milller.

Group ML: where Inj. $MgSO_4$ 30mg/kg i.v. + lignocaine 1.5mg/kg i.v 3minutes before laryngoscopy and intubation was used to blunt the pressor response, the base line value of Heart rate was 97.80. bprn . One minute following laryngoscopy and intubation, the heart rate increased to 109.50 bpm, representing a rise of 11.70 bpm above the base line value.

By 3 minutes, it was 104.10 bpm, representing a rise of 6.30 bpm above the base line value. By 5 minutes it was 100.95 bpm, representing a rise of 3.15 bpm above the base line . It was seen that the elevated heart rate started settling down towards base line value by 5 minutes rached baseline value at 10 mill 97.45.

Drsanthosh kumar [18] employed study using $MGSO_4$ 60mg /kg and noticed that maximum heart rate increased after intubation is 15. Hence the results of the present study with regards to increase in the heart rate observed following laryngoscopy and intubation concurs with the observation made by Drsanthoshkumar et al and Michael FM et al

In group CL Inj. Clonidine 3µg/kg i.v.+lignocaine 1.5mg/kg i.v 3 minutes before laryngoscopy and intubation was used to blunt the pressor response, the base line value of mean heart rate was 89.85 bpm One minute following laryngoscopy and intubation, the heart rate increased to 98.25 bpm, representing a rise of 8.40 bpm above the base line value.

By 3 minutes, it was 98.10 bpm, representing a rise of 8 bpm above the base line value. By 5 minutes it was 96.25. bpm, representing a rise of 6.15 bpm above the base line. It was seen that the elevated heart rate started settling down towards base line value by 5 minutes and at 10 min HR was 94.10 a difference 4bpm compared to baseline was observed

In Marco P. Zalunardo [19] et al employed Clonidine 311g/kg i.v. 3 minutes before laryngoscopy and intubation to blunt the, pressor response and found that difference between base line and 10min post intubation heart rate was 2.4 bpm.inPeter J. Kulka et al [10] study mean heart rate after intubation was (67+/-12) bpm.

Conclusions

In group clonidine and lignocaine, there was statistically significant attenuation of heart rate responses at one minute as compared to mgso4 and lignocaine.

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Role of Intraoperative Dexamethasone with Propofol to Improve Outcome after Laparoscopic Surgeries

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Abstract

Despite advances in anesthetic drugs and techniques, post-operative nausea and vomiting (PONV) remain the second most common post-operative complaint after surgery. Despite the increasing attention to postoperative pain control, PONV is still considered a minor complication. In the absence of antiemetic treatment, the estimate puts the incidence of PONV at 25-30% for all surgical interventions and patient populations. Pharmacological approaches based on anticholinergics, antihistamines, phenothiazines, butyrophenones, benzamides, corticosteroids and serotonin receptor antagonists have been investigated in the prevention and treatment of PONV, with various results. Dexamethasone may offer additional benefits over traditional antiemetics in improving the surgical outcomes. Compared with placebo, 8 mg of Dexamethasone given intravenously 90 minutes before laparoscopic cholecystectomy (LC) has been demonstrated to reduce PONV significantly. Combination of both Propofol and Dexamethasone may reduce the chances of PONV and additionally decreases incidence of sore throat in patients operated for laparoscopic cholecystectomy. The limitation of this study was that we could not measure the cuff pressure and did not use fibre optic bronchoscope to assess the amount of tissue damage. Our study was not designed for extended follow up beyond 24 hours, as the process of acute inflammation usually peaks by 24 hours. Sore throat, hoarseness and cough cannot be assessed objectively and there are inter individual variations and hence, a chance of bias always exists. The BMI of our patients in the two groups were comparable and we used standard sized tubes of the same manufacturer to ameliorate the possible error due to different tube size and quality.

Keywords: Dexamethasone; Propofol; Laparoscopy; Metoclop Ramide.

Introduction

Despite advances in anesthetic drugs and techniques, post-operative nausea and vomiting (PONV) remain the second most common postoperative complaint after surgery. Despite the increasing attention to postoperative pain control,PONV is still considered a minor complication [1].

The determination of true incidence of PONV is difficult due to the lack of a single stimulus of onset and multiple etiologies (medical, surgical and those related to patient and anesthesia). In the absence of antiemetic treatment, the estimate puts the incidence of PONV at 25-30 % for all surgical interventions and patient populations. Of these, 0.18 % of cases are resistant to PONV [2]. Nevertheless, when compared to other operations, the incidence rate of PONV after laparoscopic cholecycystectomy is higher owing to indirect stimulation of vestibular afferent fibers. Based on records, the PONV rate in patients who received no antiemetic treatment after laparoscopic cholecystectomy ranges from 62 % to 80 %.

Pharmacological approaches based on anticholinergics, antihistamines, phenothiazines, butyrophenones, benzamides, corticosteroids, and serotonin receptor antagonists have been investigated in the prevention and treatment of PONV, with various results [3,4].

Dexamethasone may offer additional benefits over traditional antiemetics in improving the surgical outcomes. Compared with placebo 8 mg of Dexamethasone given intravenously 90 minutes before laparoscopic cholecystectomy (LC) has been demonstrated

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to reduce PONV significantly. Combination of both Propofol and Dexamethasone may reduce the chances of PONV and additionally decreases incidence of sore throat in patients operated for laparoscopic cholecystectomy. This prospective, randomized, double-blind study aimed to evaluate the efficacy and safety of a small dose of Propofol and 8 mg of Dexamethasone for the prevention of PONV in patients undergoing laparoscopic cholecystectomy [5].

Material and Methods

Study included 100 adult patients scheduled for laparoscopic cholecystectomy operation with American Society of Anesthesiologist (ASA) risk I or II. The patients were randomly assigned into two groups. The patients in Group 1 received Propofol plus 8 mg Dexamethasone. The Group II served as placebo control group. The patients were assigned to one of the two study groups using a computer-generated random number table. Those with a history of hepatic, renal or cardiovascular diseases, chronic obstructive pulmonary disease, hematological and/ or gastrointestinal disorders, hypersensitivity to Propofol or any other drugs, as well as patients with history of vertigo and motion sickness, pregnant or menstruating patients, and patients who had taken antiemetics 24 hours before the surgery were excluded from the study.

The patients fasted for eight hours before the operation and were not premedicated. At the operation room, the heartbeat rates (HBR), systolic (SAP), diastolic (DAP), and mean (MAP) arterial pressures and peripheral oxygen saturations of the patients were monitored.

All patients in the two groups were induced with Propofol 2.5 mg/kg (intravenous, bolus dose) followed by Fentanyl at a dose of 2 mcg/Kg and Sevoflurane inspiration at 2 % concentration. After receiving Vecuronium (0.1 mg/kg), the patients were orally intubated and mechanically ventilated with O_2/air (30% ,70%), 3 L/min in closed circuit with $EtCO_2$ maintained at 35-40 mmHg. The maintenance doses of Fentanyl and Sevoflurane were adjusted for hemodynamic stability. Throughout the operation, hydration was maintained with infusion of isotonic glucose saline or ringer lactate solution at a rate of 3-5 ml/ kg/hr.

Towards the end of surgery (skin incision closure), the patients in Group I were given Propofol in an intravenous dose of 0.5 mg/kg (bolus) plus 8 mg of Dexamethasone while Group II (control) was given intravenous N.S (bolus) in the same volume. All syringes with Propofol, Dexamethasone or placebo were prepared by the same investigator. Administration of anesthesia and drugs used in the study and intraoperative data collection were made by other investigators blinded to the study drugs.

At the time of the last surgical suture, all anesthetic agents were terminated and the time was recorded. The lungs were manually ventilated with 100 % oxygen (3 L/min) until achieving spontaneous respiration. Residual muscle relaxation was antagonized with Neostigmine in a dose of 0.05 mg/kg and Atropine in a dose of 0.02 mg/kg (1).The patients were appropriately extubated. Sedation was assessed after cholecystectomy operation and for the following six postoperative hours as per the modified Ramsay Sedation Score (where 1 = anxious, agitated, restless, 2 = awake, cooperative, oriented, tranquil, 3 = semi sleep but responds to commands, 4 = asleep but responds briskly to glabellar tap or loud auditory stimulus, 5 = asleep with sluggish or decreased response to glabellar tap or loud auditory stimulus, 6 = no response can be elicited).

All patients were removed to postoperative recovery room and monitored after extubation. They were kept in this room for evaluation of potential postoperative complications and recovery for a minimum of one hour.

The degree of postoperative nausea and vomiting were scored using Nausea Vomiting Scale (NVS) and for sore throat (Table 1) at 0-4, 4-12, 12-24 hours. Additional antiemetics (10mg of Metoclopramide) were administered intravenously when NVS score was = 3. The patients were observed for 24 hours postoperatively, while the time of the occurrence of nausea and vomiting, as well as that of additional administration of antiemetics and analgesics were recorded.

Statistical analysis was performed with SPSS statistical package for windows. Parametric values were evaluated by means.-

Table 1: Nausea vomiting score

Nausea vomiting scale	Nausea vomiting severity
0	No complaints
1	Mild nausea
2	Moderate nausea
3	Frequent vomiting (4 times)
4	Severe vomiting (continuous vomit).

Table 2: Differences in demographic parameters in the two groups [mean ± SD, (n)]

Results

Table 2: Differences in demographic parameters in the two groups [mean ± SD, (n)]

	Group I (n=50)	Group II (n=50)
Age (year)	41.9±11.4	44.6±11.3
Weight (kg)	69.9±11.3	70.2±10.3
Height (cm)	166.2±7.5	165.6±6.6
ASA (I/II)	35/15	37/13
Gender (F/M)	29/21	28/22
Sedation scores	2	2
Duration of operation (min)	178.5±35.7	183.8±45.2

Table 3: Number of patients experiencing nausea vomiting [n(%)]

	Group I (n=50)	Group II (n=50)
Nausea		
0-4 hour	16 (31.4)	32(71.4)
4-12hour	10(20)	20 (40)
12-24 hour	4 (5.7)	7(14.3)
Vomiting		
0-4 hour	14 (28.6)	32 (65.7)
4-12 hour	8 (17.1)	17 (34.3)
12-24 hour	3 (5.7)	6 (11.4)

Table 4: The number of patients subjected to NVS and additional antiemetics [median(25-75%), n (%)]

	Group I (n=50)	Group II (n=50)
Additional antiemetics	17 (28.6)	32 (65.7)
NVS	0(1.75)	0 (01.75)
Sore throat	3(5)	9 (19.3)

There was no significant difference among the study groups in terms of age, body weight, height, ASA group, gender, duration of operation and anesthesia (Table 2) (p>0.05).

The comparisons of groups for the number of patients with nausea showed a significant difference at 0-4 hours, while there were no statistically significant differences at 4-12 and 12-24 hours. The incidence of nausea at 0-4 hours was 31.4 % in Group I and 71.4 % in Group II (p<0.0001). The incidence rates of nausea in Group I and Group II were statistically significant (p=0.002).

The comparisons of groups as to the incidence of vomiting at 0- 4 hours showed a rate of 28.6 % in Group I, and 65.7 % in Group II, (p<0.0001). The incidence rates of vomiting in Group I and Group II were significant at 0-4 hours (p=0.004).

It was found out that there was a significant difference at 0-4 hours, while no significant differences were found between the values at 4-12 and 12-24 hours. There were significant differences between groups in the need for additional antiemetics. Seventeen patients in Group I, and 32 patients in Group II received Metoclopramide. Three patients in group I had sore throat post operatively compared to 9 patients in group II.

Discussion

To evaluate the efficiency of these agents, various parameters such as nausea and vomiting scores for four hours in the early postoperative period or for 24 postoperative hours, number and severity of vomiting, number of antiemetics required, amount of antiemetics used, hospitalization time, and problems caused by nausea and vomiting are studied[6,7].

In our study, we recorded and compared the severity of nausea and vomiting measured with NVS during 24 postoperative hours and number of patients with nausea, vomiting, and need for additional antiemetics during 0-4, 4-12, and 12-24 hours of the postoperative period. The results were expressed in percentages. Our results of PONV control provided during the first four hours of postoperative period show that immediate postoperative bolus of Propofol in dose of 0.5 mg/kg plus 8 mg of Dexamethasone were more effective than N.S. However, there were no significant differences in postoperative recovery[7,8,9].

PONV develops as a complication after anesthesia, and if not prevented, surgical recovery and hospitalization time are prolonged. This not only leads to patients' unpleasant hospital experience, it also increases their health care costs. Prolonged vomiting may result in electrolyte imbalance (hypokalemia, hypochloremia, hyponatremic metabolic alkalosis) and dehydration, Mallory-Weiss tear, esophageal rupture, wound opening, and hematoma formation under skin flaps associated with abdominal, vascular, ocular or plastic surgery [10,11].

In this study, however, the treatment groups were similar as to patient demography, type of operation, anesthetics administered, and analgesics used postoperatively. Patients with a history of motion sickness and/or previous PONV and those menstruating were excluded from the study because they were at a remarkably high risk of PONV [12, 13].

In one study it was observed that 2.5 mg of Dexamethasone to be the minimum effective dose for preventing PONV in patients undergoing major gynecological surgery [14], while subsequent studies found 5 mg to be the minimum effective dose in patients undergoing thyroidectomy [15]. Dexamethasone is most effective in preventing PONV when it is administered immediately before the induction of anesthesia rather than near the end of anesthesia [15,16].

Longterm administration of Dexamethasone causes undesirable adverse effects such as an increased risk for infection, glucose intolerance, delayed wound healing, superficial ulceration of gastric mucosa, and adrenal suppression. In this study, however, these adverse effects were not related to a single dose of Dexamethasone. Adverse effects observed in the present study were not clinically important in any of the groups [17,18].

In earlier studies, the incidence rate of PONV associated with nitrous oxide (N2O) was high, while the incidence rate of PONV from combined administration of Sevoflurane and Fentanyl was lower[19]. Nitrous oxide is known to cause nausea and vomiting when administered as a sole anesthetic agent. Nitrous oxide also causes PONV due to changes in middle ear pressure or bowel distension due to diffusion into closed cavities [20]. Gan et al have recently reported consensus guidelines for managing PONV. Their conclusion is that the use of N_2O during maintenance of anesthesia should be avoided. Therefore, in the present study, we did not use this anesthetic gas.

It is known that positive pressure ventilation, full stomach, opioids, and anticholinergics used in premedication are among the factors increasing PONV in anesthesia induction [20, 21].

In the present study, premedication was not carried out in our cases. We tried to avoid strong positive pressure ventilation. Before extubation, we performed gastric aspiration in order to decrease the effect of factors increasing nausea and vomiting in the post-operative period. In order to neutralize patient dependent and anesthesia dependent factors, we tried to homogenize the study groups in terms of age, body weight, height, ASA group, gender, duration of operation and anesthesia. Such differences may be the cause of differences observed in above mentioned studies.

Conclusion

In conclusion, subhypnotic bolus doses of Propofol plus 8mg of Dexamethasone used at the end of laparoscopic cholecystectomy are effective than placebo and it also decreases incidence of sore throat in patients post extubation. Further studies are needed to compare the efficacy of subhypnotic doses of Propofol plus Dexamethasone with other commonly used and well established antiemetics.

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A Study of Effect of Intravenous Magnesium Sulphate with Lignocaine and Intravenous Clonidine with Lignocaine on Blood Pressure in Response to Laryngoscopy and Tracheal Intubation during General Anaesthesia

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Abstract

Background and Objectives: Laryngoscopy and tracheal intubation is invariably associated with a reflexsympathetic pressor response resulting in elevated heart rate and blood pressures. Thismay prove detrimental in high risk patients. The main objectives of the present study are:

1. To study the effect of intravenous magnesium sulphate 30 mg/kg with intravenous lignocaine 1.5mg/kg, and intravenous clonidine 3mcg/kg with intravenous lignocaine 1.5mg/kg on changes in the Systolic blood pressure (SBP), Diastolic blood pressure (DBP) and Mean arterial blood pressure (MAP) during laryngoscopy and intubation under general anesthesia. *Methods:* 60 ASA I and II status normotensive patients scheduled for elective surgical Procedures were selected randomly and divided into three groups of 20 each. Allpatients received premedicationwith studydrug. Induction of anesthesia was standardized for all patients who received, thiopentone 5 mg/kg i.v.and preservative free lignoaine 1.5mg i.v and were relaxed with succinylcholine 2mg/kg i.v. and laryngoscopy and intubation is done with appropriate sized endotracheal tube. HR, systolic, diastolic blood pressure were recorded noninvasively before induction, postintubation, 1,3,5, 7 and 10 minutes from the onset of laryngoscopy. ' z' test was used for statistical analysis. *Results:* The basal and pre laryngoscopy mean SBP and standard deviations in CL group were 122.15 +/- 8.12 and 114.50 +/- 7.30 respectively. In ML group basal systolic blood pressure was 117.30 -+-/- 9.78. After giving study drug prelaryngoscopy SBP decreased by 3mm of mm Hg to 114.15 +1- 15.26. *Conclusion:* Both study drugs were more effective in attenuation of pressor response to intubation than when lignocaine alone was used

Keywords: Attenuation; Pressor Response; Laryngoscopy; Intubation; Lignocaine; Magnesium-sulphate; Clonidines.

Introduction

Laryngoscopy and endotracheal intubation are associated with significant hypertension, tachycardia and arrhythmias. These hemodynamic responses were first recognised as early as in 1940 by Reid and Bruce et al [1].

In 1950 Burstein et al [2] studied the effects of laryngoscopy and tracheal intubation on ECG changes and suggested the pressor response as consequences of an increase in sympathetic and sympathoadrenal activity. These facts were further confirmed by various investigators[3].

These responses are transitory, variable and unpredictable and are much more pronounced in hypertensive patients than in normotensive individuals [4]. These transient changes can result in potentially deleterious effects like, left ventricular failure, pulmonary edema, Myocardialischaemia, ventricular dysrhythmias and cerebral hemorrhage.

Various attempts [5-9]were made in the past to reduce the pressor response to laryngoscopy and intubation using inhalational anesthetics, á adrenergic drugs, â blockers, calcium channel blockers, vasodilators, low dose opioids, all with one or other adverse effects.

Robert K Stoelting [10] noted that the best way to prevent laryngoscope reaction was to minimize the duration of laryngoscopy and intubation to 15seconds. He also suggested that, Intravenous Lignocaine given in the doses of 1.5 mg/kg 3 minutes before laryngoscopy and intubation sufficiently attenuates

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the laryngoscopic reactions. The advantages of intravenous lignocaine claimed by this author was intravenous lignocaine depressed the autonomic nervous system and in addition had antiarrhythmic properties.

Many studies have shown that magnesium sulphate can attenuate cardiovascular response to endotracheal intubation. Jame MF et al [11] in 1989 showed that pretreatment with magnesium sulphate 60mg/kg body weight attenuates catecholamine mediated responses to tracheal intubation. K Montazeri MD and M Fallah MD [12] in 2005 showed that intravenous magnesium sulphate in the dose of 30mg/kg body weight was equally effective.

In 1991, Wright PMC et al and CarabineUA et al [13] showed that intravenous clonidine sufficiently attenuates pressor response to intubation. Ishiyama T et al [14] in 2006 demonstrated that clonidine produced stable hemodynamic condition during induction sequence.

Hence the main objectives of the present study is to study the effect of intravenous magnesium sulphate 30 mg/kg with intravenous lignocaine 1.5mg/kg, and intravenous clonidine 2mcg/kg with intravenous lignocaine 1.5mg/kg on changes Systolic blood pressure (SBP), Diastolic blood pressure (DBP) and Mean arterial blood pressure (MAP) during laryngoscopy and intubation under general anesthesia.

Methodology

60 ASA I and II status normotensive patients scheduled for elective surgicalprocedures were selected randomly and divided into three groups of 20 each. Allpatients received premedication with study drug magnesium sulphate 30mg/kg or clonidine 3µgm / kg or normal saline (as per double blind study protocol) prepared by anaesthesia staff and glyopyrrolate 0.2mg i.v., tramadol 3mg /kg 3min before induction . Induction of anesthesia was standardized for all patients who received, thiopentone 5 mg/kg i.v.and preservative free lignoaine 1.5mg i.v and were relaxed with succinylcholine 2mg/kg i.v. and laryngoscopy and intubation is done with appropriate sized endotracheal tube. HR, systolic, diastolic blood pressure were recorded noninvasively before induction, postintubation, 1, 3, 5, 7 and 10 minutes from the onset of laryngoscopy. 'z' test was used for statistical analysis.

Results

SBP	Clonidine with lignocaine (A)	Magnesium sulphate with lignocaine (B)	Lignocaine only (C)	P value *	P value #		
					A-B	A-C	B-C
Baseline	122.15 +/- 8.12	117.30 +/- 9.78	117.50 +/- 8.34	0.15	0.19	0.22	0.99
Pre laryngeal	114.50 +/- 7.30	114.15 +/- 15.26	113.75 +/- 15.07	0.98	0.99	0.98	0.99
One min	138.50 +/- 10.51	134.33 +/- 14.16	155.80 +/- 13.03	0.00	0.55	0.00	0.00
Three min	127.35 +/- 11..60	121.85 +/- 11.63	140.65 +/- 15.06	0.00	0.37	0.00	0.00
Five min	119.65 +/- 11.20	117.65 +/- 13.83	126.65 +/- 12.17	0.06	0.86	0.18	0.06
Ten min	116.20 +/- 9.71	119.95 +/- 12.28	119.80 +/- 9.57	0.06	0.40	0.52	0.05

* ANOVA test
# Post-hoc tukey test
All values are in mean +/- sd

Analysis of systolic blood pressure Statistical analysis of changes in systolic blood pressure at basal, prelaryngoscopy ,nost intubation at different (1 ,3, 5, 10,) time intervals from the onset of laryngoscopy and intubation in all the 3 study group is presented.

Group CL (A)

The basal and pre laryngoscopy mean SBP and standard deviations in this group were 122.15 +/- 8.12 and114.50 +/- 7.30 respectively . After lmin of intubation 16.35 mm Hg (13.38%) increase in mean SBP was observed with mean SBP and standard deviations of 138.50 +/- 10.51. Subsequently a decreasing trend in the SBP was noted starting from 3 minutes to 10 minutes after laryngoscopy. Mean SBP at 3, 5 minutes and 10 minutes were 127.35 +/- 11.60, 119.65 +/- 11.20 and 116.20 +/-9.71. At 10 minutes post laryngoscopy the SBP almost returned to base line with a mean value of 119.95 +1- 12.28.

Group ML(C)

In this group basal systolic blood pressure was 117.30 -+-/- 9.78. After giving study drug prelaryngoscopy SBP decreased by 3mm of mm Hg to 114.15 +1- 15.26. Increase in systolic blood pressure Of 17.03 mmHg (14.51%) with a mean of 134.33 +/-

14.16 was observed at 1 minute following laryngoscopy. After 3 min SBP fell by 12mmhg with a mean of 121.85 +/- 11.63, from there on a gradual fall in SBP was observed at 5 minutes mean SBP was 117.65 +/- 13.83. At 10 minutes postlaryngoscopy the SBP almost returned to base line with a mean value of 119.95 +1- 12.28.

Group NL (C)

In this group basal systolic blood pressure was 117.50 +/- 8.34. After giving study drug prelaryngoscopy SBP decreased by 4 mm Hg toll 3.75 +/- 15.07. increase in systolic blood pressure Of 38.3 mm Hg(32.59%) with a mean of 155.80 +/- 13.03 was noted at 1 minute following laryngoscopy. After 3 min SBP fell by 23.15 mmHg (19.70%) with a mean of 140.65 +/- 15.06, from there on a gradual fall in SBP was noted. At 5 minutes it was 126.65 +/- 12.17. At 10 minutes post laryngoscopy the systolic blood pressure almost returned to base line with a mean value of 119.80 +1-9.57.

No significant variations were noted in all groups in SBP at basal and after giving study drug. The increase of systolic blood pressure at one and three minutes after intubation was significantly less in ML group and CL group compared to NL group, But there was no significant reduction in increase of SBP at five and ten minutes of recording. there was no significant changes in attenuation of SBP response between ML group and CL group at any time of recording.

Table 2: Comparison of DBP (mmHg) between three groups

DBP	Clonidine with lignocaine (A)	Magnesium sulphate with lignocaine (B)	Lignocaine only (C)	P value *	P value #		
					A-B	A-C	B-C
Baseline	87.90 +/- 5.55	78.95 +/- 7.60	77.35 +/- 7.48	0.11	0.37	0.10	0.74
Pre laryngeal	77.15 +/- 9.04	77.50 +/- 13.87	117.30 +/- 18.72	0.39	1.00	0.45	0.46
One min	90.60 +/- 7.68	90.25 +/- 13.75	101.75 +/- 8.14	0.00	0.99	0.00	0.00
Three min	84.65 +/- 7.80	79.90 +/- 11.93	92.85 +/- 11.73	0.00	0.34	0.04	0.00
Five min	80.35 +/- 10.27	79.60 +/- 15.73	82.55 +/- 13.49	0.76	0.98	0.86	0.76
Ten min	76.10 +/- 7.65	72.40 +/- 13.78	77.15 +/- 9.80	0.34	0.52	0.94	0.34

* ANOVA test
\# Post-hoc tukey test
All values are in mean +/- sd

Analysis of diastolic blood pressure Statistical analysis of changes in systolic blood pressure at basal, prelaryngoscopy ,post intubation at different (1 ,3, 5, 10,) time intervals from the onset of laryngoscopy and intubation in all the 3 study group is presented .

Group CL (A): In this group pre-induction diastolic blood pressure was83.90 +1- 5.55 . After giving study drug prelaryngoscopy DBP decreased by 11 mm Hg to mean of 77.15 +/- 9.04 . Increase in diastolic blood pressure Of 6.70mmHg (7.9%) with a mean of 90.60 +7-7.68 was noted at 1 minute following laryngoscopy. After 3 min DBP fell by 6 mmhg(6.8%) with a mean of 84.65 +/- 7.80 , from there on a gradual fall in DBP was noted. At 5 minutes it was 80.35 +/- 10.27. At 10 minutes post laryngoscopy the DBP decreased to less than base line with a mean value of 76.10 +/- 7.65.

Group ML (B): In this group pre-induction diastolic blood pressure was 79.95 +/- 7.60. After giving study drug prelaryngoscopy DBP decreased by 1 mm Hg to mean of 77.50 +/- 13.87. Increase in diastolic blood pressure Of 10.30 mm Hg(12.8%) with a mean of 90.25 +/- 13.75 was noted at 1 minute following laryngoscopy. After 3 min DBP was 79.90 +/- 11.93, from there on a gradual fall in DBP was noted. At 5 minutes it was 79.60 +/- 15.7,. At 10 minutes post laryngoscopy the DBP decreased to less than base line with a mean value of 72.40 +/- 13.78.

Group NL (C): In this group pre-induction diastolic blood pressure was77.35 +/- 7.48. After giving study drug prelaryngoscopy DBP decreased to mean of 75 .30 +1..18.72. Increase in diastolic blood pressure Of 24.40 mm Hg(31.54%) with a mean of 101.75 +/- 8.14 was noted at 1 minute following laryngoscopy. After 3 min DBP fell by 15.5 mmHg (20.0%) with a mean of 92.85 +/- 11.7, from there on a gradual fall in DBP was noted at 5 minutes it was 82.55 +/- 13.49. At 10 minutes post laryngoscopy the DBP decreased to less than base line with a mean value of 77.15 +/- 9.80. No significant variations were noted in all groups in diastolic blood pressure at basal and after giving study drug. There was significant attenuation of DBP response at one minute and three minutes after intubation in CL group and ML group compared to NL group however there was no significant difference in attenuation of DBP at five and ten minutes of recording. There was no significant difference in attenuation of DBP response between ML group and CL group at any time of recording.

Table 3: Comparison of MAP (mmHg) between three groups

Heart rate	Clonidine with lignocaine (A)	Magnesium sulphate with lignocaine (B)	Lignocaine only (C)	P value *	P value #		
					A-B	A-C	B-C
Baseline	94.55 +/- 7.01	91.45 +/- 8.58	134.55 +/- 20.07	0.43	0.99	0.52	0.47
Pre laryngeal	87.50 +/- 6.49	87.50 +/- 11.90	88.35 +/- 13.08	0.96	1.00	0.96	0.96
One min	107.30 +/- 13.01	103.35 +/- 13.22	116.80 +/- 10.66	0.00	0.57	0.04	0.00
Three min	97.25 +/- 8.61	95.35 +/- 11.16	107.75 +/- 14.36	0.00	0.86	0.01	0.04
Five min	93.25 +/- 10.31	91.70 +/- 15.00	97.25 +/- 12.25	0.36	0.92	0.58	0.35
Ten min	90.25 +/- 7.55	84.70 +/- 11.64	95.65 +/- 13.61	0.37	1.00	0.44	0.44

* ANOVA test
# Post-hoc tukey test
All values are in mean +/- sd

Analysis of MAPStatistical analysis of changes in MAP at basal.prelaryngoscopy ,post intubation at different (1, 3, 5, 10,) time intervals from the onset of laryngoscopy and intubation in all the 3 study group is presented .

Group CL (A): In this group basal MAP was 94.55 +/- 7.01 . After giving study drug prelaryngoscopy MAP decreased by 7.06 mm Hg to mean of 87.50 +/- 6.49. Increase in MAP of 10.30 mm Hg(11.13%) with a mean of 107.30 +1- 13.01 was noted at 1 minute following laryngoscopy. After 3 minutes mean MAP was 97.25 +/- 8.61, from there on a gradual fall in MAP was noted. At 5 minutes it was 9325 +/- 10.31. At 10 minutes post laryngoscopy the MAP almost returned to base line with a mean value of 90.25 +/- 7.55.

Group ML (B): In this group pre-induction MAP was 91.45 +/- 8.58. After giving study drug prelaryngoscopy MAP decreased by 4mm hg to mean of 87.50 +/- 11.90. Increase in MAP Of 11.9(13.01%) mm hg with a mean Of 103.35 +/- 13.22 was noted at 1 minute following laryngoscopy. After 3 min mean MAP was 95.35 +/- 11.16, from there on a gradual fall in MAP was noted as at 5 minutes it was 91.70 +/- 15.00. At 10 minutes post laryngoscopy the DBP almost returned to base line with a mean value of 84.70 +/- 11.64.

Group NL (C): In this group pre-induction MAP was 94.55 +/- 20.07. After giving study drug prelaryngoscopy MAP decreased by 6 mm Hg to mean of 88.35 +/- 13.08. Increase in MAP Of 22.2.5 mm Hg (24.35%) with a mean of 116.80 +1- 10.66 was noted at 1 minute following laryngoscopy. After 3 min mean MAP was107.75 +/- 14.36 , from there on a gradual fall in MAP was noted. At 5 minutes it was of 97.70 +/- 12.25. At 10 minutes post laryngoscopy the systolic blood pressure almost returned to base line with a mean value of 95.65 +/- 1.3.61. No significant variations were noted in all groups in mean arterial pressure at basal and after giving study drug. There was significant reduction of increase in MAP at one minute and three minutes after intubation in CL group and ML group compared to NL group, however there was no significant difference in attenuation of MAP between CL group and ML group at any time of recording.

Discussions

Many factors influence the cardiovascular changes associated with laryngoscopyand intubation. Age, drugs, type and duration of procedures, depth of anesthesia,hypoxia, hypercarbia etc., influence the pressor responseVariations of heart rate changes decrease with increasing age. Young patientsshow more extreme changes [18]. Marked fluctuations in hemodynamic responses areoften seen in geriatric patients[15,16].

In our study we selected the optimal age range of 18 to 50 years. Patients on antihypertensive drugs may exhibit a decrease in pressor response. Weexcluded the patients on antihypertensive medications from our study Thiopentone was selected for induction since it still continues to be the mostpopular agent for induction. In normovolemic patients thiopentone 5mg/kg i.v cantransiently decrease 10-20mm Hg of blood pressure and increase the heart rate by 15-20 beats/min. There is increase in catecholamine levels, both noradernaline andadrenaline [17].

Succinyl choline has negative inotropic and chronotrpic effect. It acts on themuscarinic receptors of SA node. A marked noradrenergic response was noted whenintubation was performed under succinylcholine[18].

Nitrous oxide may increase the tone of sympathetic nervous system. The directaction of Nitrous oxide is negative inotropism which is offset by increased sympathetictone. Halothane has potency to decrease the heart rate but at concentration used formaintenance it does not appreciably change the heart rate [19].

Nasotracheal intubation comprises of three distinct phases a) nasopharyngealintubation b) direct laryngoscopy to identify the vocal cords and c) Passage of tracheal tube into the trachea. Nasopharyngeal intubation causes significant pressor response. This response in heightened by the passage of tracheal tube in the larynx and trachea in our study we included only direct laryngoscopy andorotracheal intubation.

Laryngoscopy alone may produce most of the cardiovascular responses reported after laryngoscopy and tracheal intubationduringanaesthesiaThe most significant laryngoscopic factor influencing cardiovascular responses is found to be the duration of laryngoscopy. A linear increase in heart rate and mean arterial pressure during the first 45 seconds has been observed. Further prolongation has little effect. As the duration of laryngoscopy is normally less than 30 seconds, the results of studies in which it takes longer than this have less clinical relevance. The force applied during laryngoscopy has only minor effect. In our study the duration of laryngoscopy and intubation was limited to 20 seconds.

Adequate care was taken to achieve the required depth of anesthesia avoidinghypoxia and hypercarbia which can influence e the hemodynamic variations. Excluding hypoxia and hypercarbia other contributory causes of hypertension and tachycardia could be continued manifestation of anxiety concerning anesthesia and surgery, glycopyrrolate premedication, reflex baroreceptor effect after thiopentone and possible effect of suxamethonium.But they seem to be less important than laryngotracheal stimulation during laryngoscopy and intubation.

Attenuation of sympathetic responses during laryngoscopy and intubation is ofprime concern to the anaesthetist more so in high risk subjects as mentioned earlier.Many strategies have been recommended which include minimising the duration oflaryngoscopy to less than 20 seconds, topical application of local anaesthetics, iv?-blockers, calcium channel blockers, Clonidine, Sodium Nitroprusside, lignocaine. No single drug or technique is satisfactory. Each technique has advantages and disadvantages, the most obvious being that the prevention often outlasts the stimulus.

In group NL the basal value of Systolic blood pressure, Diastolic blood pressure, and Mean arterial pressure was 1117.50 mm Hg, mm 77.35 Hg, and 94.55 mm Hg respectively. Following laryngoscopy and intubation, the maximal rise in Systolic blood pressure (SBP) was found to be 38.30 mm Hg, that of Diastolic blood pressure (DBP) was 20.45 mm Hg and that of Mean arterial pressure (MAP) was 22.33 mm Hg. These elevated pressure readings started coming down by 3 minutes. However they remained above the baseline value even at the end of 5 minutes and reached baseline at 10 mins.

Stanley Tam employing inj. Lignocaine 1.5 mg/kg i.v 3 minutes before laryngoscopy and intubation to blunt the pressor responses, found out the maximal increase in SBP was(15)12 mm Hg, DBP was 9 mm Hg and MAP to be 11 mm Hg. Mounir Abou- Madi et al [18] noticed the change in SBP by 30 mm Hg, change in the DBP was 22 mm Hg. CD Miller37noticed a increase in SBP to be 33 mm Hg, DBP to be 37 mm Hg. Splinter et al. noticeda change in SBP to be 26 mm Hg, DBP to be 41 mm Hg and change in MAP to be 44 mmHg.

In group ML Inj. $MgSO_4$ 30mg/kg i.v. 3 minutes before laryngoscopy and intubation was used to blunt the pressor response, the basal value of Systolic blood pressure (SBP), Diastolic blood pressure (DBP), and Mean arterial pressure (MAP) was 117.30 mm Hg, mm 78.95 Hg, and 91.45 mm Hg respectively. Following laryngoscopy and intubation, the maximal rise in Systolic blood pressure (SBP) was found to be 17.03mm Hg, that of Diastolic blood pressure (DBP) was 11.30 mm Hg and that of Mean arterial pressure (MAP) was 11.9 mm Hg. These elevated pressure readings started coming down by 3 minutes. However they remained above the baseline value even at the end of 5 minutes and reached baseline at 10 mins

In the study of Dr. Santosh Kumar using $MgSO_4$i.v 30mg/kg 3 minutes before laryngoscopy and intubation to blunt the pressor responses, found out the maximal increase in SBP was 5 mm Hg, DBP was 4 mm Hg and MAP to be 4mm Hg. In K.Montazeri et al study employing $MgSO_4$ i.v 30mg/kg 3 minutes before laryngoscopy and intubation to blunt the pressor responses, found out the maximal increase in MAP to be 20 mm Hg

In group CL lnj clonidine 3μg/kg i.v. 3 minutes before laryngoscopy and intubation was used to blunt the pressor response, the basal value of Systolic blood pressure (SBP), Diastolic blood pressure (DBP), and Mean arterial pressure (MAP) was 122.15 mm Hg, mm 83.90 Hg, and 96.5d5 mm Hg respectively. Following laryngoscopy and intubation, the maximal rise in Systolic blood pressure (SBP) was found to be 16.35mm Hg, that of Diastolic blood pressure (DBP) was6.70 mm Hg and that of Mean arterial pressure

(MAP) was 12.75 mm Hg. These elevated pressure readings started coming down by 3 minutes. However they remained above the baseline value even at the end of 5 minutesand at 10minutes difference Of MAP between basal and 10 min after intubation was 4 mm hg

In study done by In Marco P Zalunardo [20] with clonidine 3 u/kg the difference of MAP between basal and 10 min after intubation was 21.1.

Conclusion

With respect to attenuation of blood pressure responses, there were no significant differences between clonidine & lignocaine group and $MgSO_4$ & lignocaine group during at different times after laryngoscopy and intubation.

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Original Article

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A Study on Complications of I - Gel Versus Cuffed Tracheal Tube in Elective Laparoscopic Cholecystectomy

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Abstract

Introduction: In spite of tremendous advances in contemporary anesthesia practice, airway management continues to be of paramount importance to anesthesiologist. *Methodology*: 60 patients of ASA 1 and 2 with BMI <35kg/m2 ,between 18 and 60 yrs. were enrolled into the study. They were randomized into two groups of equal number using the chit-in-a-box method for the use of either i-gel or endotracheal tube for the maintenance of airway during the anesthesia. Both groups were comparable with respect to age, sex,BMI, ASA grading, airway characteristics and anesthesia technique used was same in both groups. *Results:* No gastric distension, regurgitation, aspiration, laryngospasm or bronchospasm seen while using the i-gel or endotracheal tube during our study. 4 cases reported of having hoarseness of voice and 2 cases reported of having sore throat in endotracheal group but there were no cases of sore throat or hoarseness of voice in i- gel group, which are well known advantages of laryngeal mask air way.*Conclusion:*Our study supports the use of i-gel during VCV in elective laparoscopic cholecystectomy using low to moderate tidal volumes provided that peak airway pressure is not more than device leak pressure.

Keywords: I-Gel; Leak Fraction; Pneumoperitoneum; Cholecystectomy; Complications.

Introduction

Different supra-glottic devices are being used since last many years for maintaining airway and positive pressure ventilation during general anesthesia. Laryngeal mask airway and perilaryngeal airway (cobra tube) are among them and "I gel" is new addition to these devices. TheI-gel is made of soft thermoplastic elastomer, gel like and transparent as well. It doesn't have an inflatable cuff but effectively covers the perilaryngeal area for ventilation. Different studies have shown that its insertion is easier and faster than conventional laryngeal mask airways [1,2].

Studies on Cadaver showed that i-gels effectively conformed to the perilaryngeal anatomy and consistently achieved proper positioning for supraglottic ventilation [3]. Manikins studies and patients have shown that the insertion of the i-gel was significantly easier when compared with insertion of other SADs [4,5]. Few studies had been done to evaluate the use of i-gel during controlled ventilation but they did not evaluate its use during procedures with airway pressure more than 25 cm H_2O [6].

This study aims to test and compare cuffed endotracheal tube and i-gel in terms of their: Incidence of intra-operative and post-operative complications.

Methodology

60 patients satisfying the above criteria were enrolled into the study. They were randomized into two groups of equal number using the chit-in-a-box method for the use of either i-gel or endotracheal tube for the maintenance of airway during the anesthesia. All patients were pre-medicated with oral Ranitidine 150 mg, Metoclopramide 10 mg and Alprazolam 0.5mg the night before surgery and with oral Ranitidine150mg and Metaclopramide 10mg two hour prior to the induction of anesthesia.

We used Datex-Ohmeda; Aestiva 5 anesthesia machine with standard monitor. Anesthesia protocol was made the same for all patients.

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Preoxygenation then induction of anesthesia was done by propofol 1.5 to 2 mg kg_1, fentanyl 2 mcg kg_1 and neuromuscular relaxation achieved by atracurium 0.5 mg kg_1 with increments of 5 mg boluses every 30 min. Anesthesia was maintained by oxygen, nitrousoxide,isoflorane mixture. In i-gel group, Size 3 i- gel used in all female patients and size 4 is used in male patients. Proper preparation, lubrication with water soluble lubricant was done. Senior anesthetist inserted the i-gel as recommended by the manufacturer. In endotracheal group the trachea of the participant was intubated with an appropriate size tracheal tube: size 8.5 was used for the male participants and size 7.5 was used for the female participants.

Inclusion Criteria

1. Patients between 18 and 60 years of age.
2. ASA status of either I or II
3. BMI below 35 kg/m^2

Exclusion Criteria

1. Patients with a known or predicted difficult airway.
2. Patients at increased risk for aspiration.
3. Patients with an active respiratory tract infections or a reactive airway.
4. Patients with any pathology of the neck or cervical spine.
5. Edentulous patients.

After induction of anesthesia by a suitable intravenous induction agent and after achieving adequate anesthetic depth, the randomly chosen, appropriately sized airway device was inserted and connected to the breathing circuit. The following parameters were then studied:

Intraoperative complications: such as airway Loss, laryngospasm and coughing

Postoperative complications: Blood on the device, laryngospasm, coughing, sore throat, hoarseness of voice.

The data was analysed using the Statistical Package for Social Sciences software for Windows.

Results

Age, height, weight, BMI and ASA status were comparable in both groups. There were no incidents of gastric distension,bronchospasm, laryngospasm gastric regurgitation, in both groups.

Blood staining is noted in one case of i-gel and oral trauma is noted in two cases, but this was not found to be significant statistically as shown below.

Table 1: Comparison of sex based on group

Sex	I gel		Endotracheal tube	
	Number	Percent	Number	Percent
Male	13	43.3	14	46.7
Female	17	56.7	16	53.3

x^2 = 0.07, p = 0.795(significant if p< 0.05)

Table 2: Comparison of blood staining of air way device based on group

Blood Staining of air way device	I- gel		Endotracheal tube	
	Number	Percent	Number	Percent
Yes	1	3.3	0	0.0
No	29	96.7	30	100.0

p = 0.500 (Fisher Exact test) (significant if p< 0.05)

Distribution According to Blood Staining of Airway Device

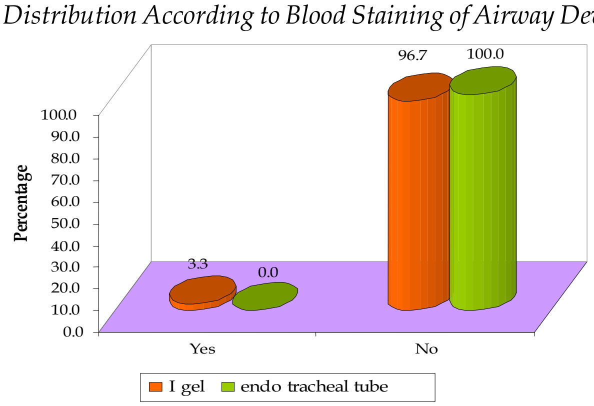
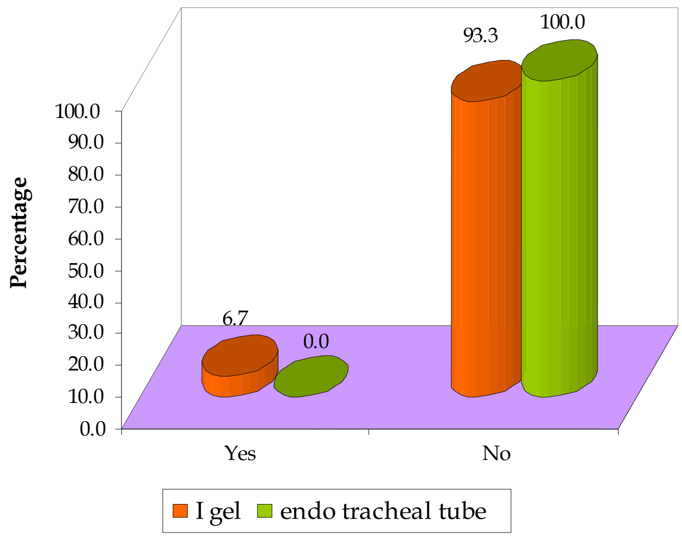


Table 3: Comparison of oral trauma based on group

Oral Trauma	I gel Number	I gel Percent	Endotracheal tube Number	Endotracheal tube Percent
Yes	2	6.7	0	0.0
No	28	93.3	30	100.0

p = 0.246 (Fisher Exact test) (significant if p< 0.05)

Distribution According to Oral Trauma



Hoarseness of voice and sore throat noted post operatively in endotracheal group in 4 and 2 cases respectively, but no such complications were noted in I gel group.And this was not found to be significant statistically as shown below

Table 4: Comparison of hoarseness of voice based on group

Hoarseness of voice	I-gel Number	I-gel Percent	Endotracheal tube Number	Endotracheal tube Percent
Yes	0	0.0	4	13.3
No	30	100.0	26	86.7

p = 0.056 (Fisher Exact test) (significant if p< 0.05)

Distribution According to Hoarseness of Voice

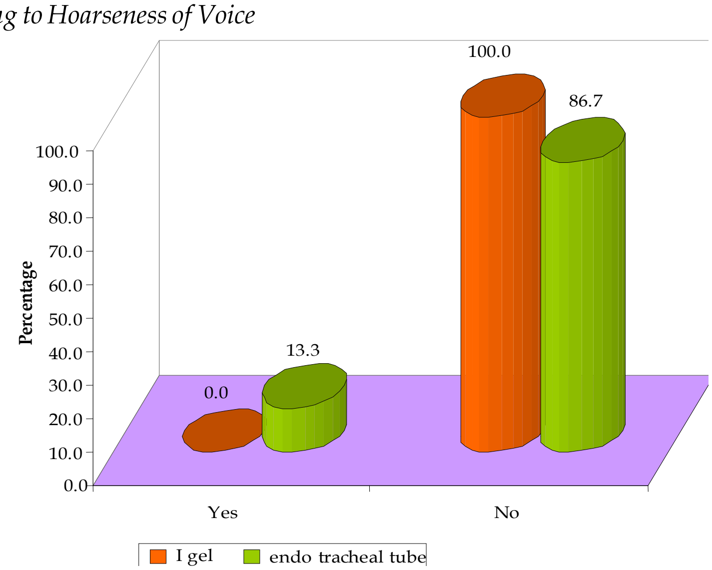
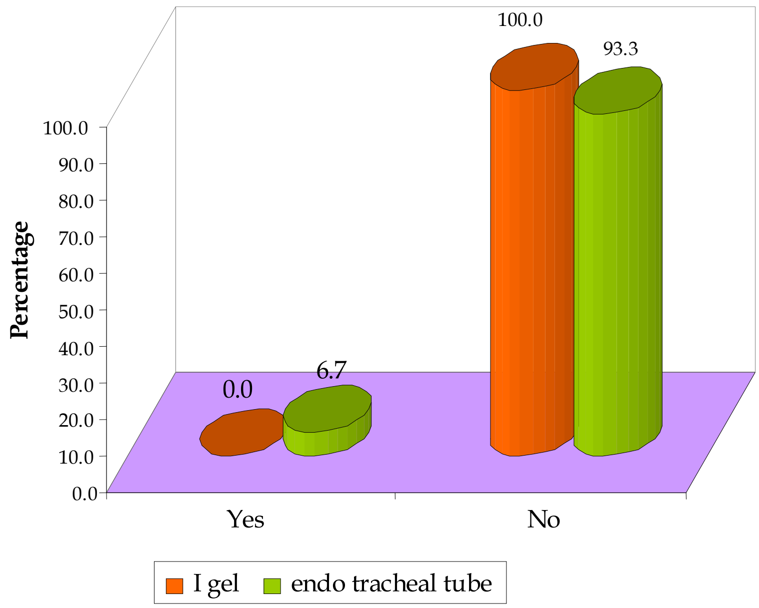


Table 5: Comparison of sore throat based on group

Sore throat	I gel		Endotracheal tube	
	Number	Percent	Number	Percent
Yes	0	0.0	2	6.7
No	30	100.0	28	93.3

p = 0.246 (Fisher Exact test) (significant if p< 0.05)

Distribution According to Sore Throat



Discussion

No gastric distension, regurgitation, aspiration, laryngospasm or broncho spasm, while using the i-gel or endotracheal tube for VCV during our study. There were no cases of failed insertions. Visible blood was observed on the i-gel after removal in our study was 3.3% (1/30). This is similar to those reported with other SAD. The occurrence of visible blood with the use of other SAD has been reported from 12% to 18%, depending upon the type of SAD, technique of insertion, and ease of insertion [7,8].

4 cases reported of having hoarseness of voice and 2 cases reported of having sore throat in endotracheal group but there were no cases of sore throat or hoarseness of voice in I gel group, which are well known advantages of laryngeal mask air way.

There are some limitations of the present study. Firstly, we studied only low risk patients (ASA I and II) who had normal airways and were mostly not obese. Secondly, we included only elective cases and excluded the patients having risk of gastric aspiration.

In our study we found that i-gel airway can be used safely and effectively during volume controlled ventilation with low and moderate tidal volumes. Our study supports the use of i-gel during VCV in elective laparoscopic cholecystectomy using low to moderate tidal volumes provided that peak airway pressure not more than device leak pressure. Although leak volume was significant, ventilation and oxygenation were optimal in most cases. Tracheal tube should be inserted if failed ventilation and oxygenation. Gastric tube insertion with I-gel helps in preventing gastric insufflation and decreasing air leak and regurgitation.

Conclusions

Gastric tube insertion with I-gel helps in preventing gastric insufflation and decreasing air leak and regurgitation.

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Current Role of Dexmedetomidine in Pediatric Cardiac Anesthesia

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Abstract

Dexmedetomidine is a novel drug which is a selective α2-adrenoceptor agonist. It has unique properties of producing sedation, anxiolysis, amnesia and analgesia but at the same time without respiratory depression.

It provides a unique "conscious sedation" (patients appear to be asleep, but are readily roused), analgesia, without respiratory depression. It decreases central nervous system (CNS) sympathetic outflow in a dose dependent manner and has analgesic effects best described as opioid-sparing. There is increasing evidence of its organ protective effects against ischemic and hypoxic injury, including cardioprotection, neuroprotection and renoprotection.

This article is intended to highlight special situations in cardiac anesthesia (especially paediatric) where dexmedetomidine plays a crucial role.

Keywords: Dexmedetomidine; Cardiac Anesthesia; Pediatric.

Dexmedetomidine is a novel drug which is a selective α2-adrenoceptor agonist. It has unique properties of producing sedation, anxiolysis, amnesia and analgesia but at the same time without respiratory depression [1,2].

It provides a unique "conscious sedation" (patients appear to be asleep, but are readily roused), analgesia, without respiratory depression. It decreases central nervous system (CNS) sympathetic outflow in a dose dependent manner and has analgesic effects best described as opioid-sparing. There is increasing evidence of its organ protective effects against ischemic and hypoxic injury, including cardioprotection, neuroprotection and renoprotection [3].

History

Dexmedetomidine was approved by the United States Food and Drug Administration (FDA) in 1999 only for short-term sedation/analgesia (< 24 hours) in the intensive care unit (ICU). Its unique properties make it as an attractive agent during perioperative period. In 2008 it was again additionally recommended in non-intubated patients as premedication during surgery or other medical procedures for sedation [4]. It is also approved in Europe for conscious sedation in adult ICU patients [5]. However, it is still not approved for use in pediatric patients but several case reports suggest its successful use without any evidence of adverse effects. Its use in pediatric population has expanded in preventing emergence delirium, to facilitate radiological and cardiac catheterization procedural sedation as well as in opioid withdrawal management [6].

Pharmacology

Dexmedetomidine is chemically described as (+)-4-(S)-[1-(2,3-dimethylphenyl)ethyl]-1 H-imidazole monohydrochloride. Dexmedetomidine is an active D-enantiomer of medetomidine, the methylated derivative of etomidine. It incorporates an imidazoline structure, thus having an agonist effect on imidazoline receptors. Dexmedetomidine is chemically related to clonidine, but is approximately eight times more specific for α-2 adrenoceptors with α-2: α-1 selectivity ratio of 1620:1, compared with 200:1 for clonidine, especially for the 2a subtype, which makes dexmedetomidine more effective than clonidine for sedation and analgesia [7]. It has a pH in the range of 4.5-7. It is water soluble with pKa of 7.1. Its effects are dose-dependently reversed by administration of a selective á-2 antagonist, such as atipamezole [3].

Systemic Effects and Mechanism of Action

Hypnotic effect : It is mediated

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by the hyperpolarization of noradrenergic neurons in the locus ceruleus of the brain stem. α-2 adrenergic receptor activation inhibits adenylyl cyclase reducing cyclic AMP (cAMP). At the same time, efflux of potassium through calcium-activated potassium channels occurs and inhibition of calcium entry into nerve terminals occurs [8]. This hyperpolarization of the neuronal membrane suppresses neuronal firing in the locus ceruleus as well as activity in the ascending noradrenergic pathway [9].

Dexmedetomidine inhibits norepinephrine release from the neurons in locus ceruleus. Loss of inhibitory control over the ventrolateral preoptic nucleus (VLPO) releases GABA and galanin, which further inhibits locus ceruleus. This decreases release of histamine, and the reduced occupancy of the histamine receptors on the cells of the subcortical areas induces a hypnotic state [10. Suppression of locus ceruleus results in decreased heart rate (HR) and systemic vascular resistance (SVR) [4].

Analgesic Effects

Activation of α2B-adrenoceptors in the dorsal horn of the spinal cord inhibits substance P release producing analgesia [11,12].

Cardiovascular Effects

Activation of peripheral α2b receptors results in vasoconstriction and the initial increase in systolic blood pressure, whereas the eventual decrease in blood pressure and heart rate results from central presynaptic α 2a receptor stimulated sympatholysis in the central nervous system, causing a decrease in norepinephrine release. Bradycardia is caused by both a reflex response at the sinus node to peripheral vasoconstriction and the decrease in sympathetic outflow from the central nervous system [8,13].

Respiratory Effects

Some studies have demonstrated respiratory depression with mild increases of PaCO2 (4-5 mm Hg), decreased minute ventilation, decreased response to carbon dioxide challenge using carbon dioxide response curves, or upper airway obstruction following bolus doses [14].

In contrast to infusions of opioids, benzodiazepines, or propofol, dexmedetomidine can safely be infused through tracheal extubation and beyond [3].

No adverse effects on the pulmonary vasculature have been reported including patients with preexisting pulmonary hypertension [15].

Organ Protection

Dexmedetomidine has been shown to exhibit myocardial protection, neuroprotection and renal protective properties. Perioperative infusion appears to benefit the perioperative hemodynamic management of surgical patients undergoing vascular surgery [16].

In one study, dexmedetomidine attenuated hypoxic-ischemic brain injury in developing brain and significant improvement in functional neurological outcomes after brain injury was also demonstrated [17].

Dexmedetomidine decreases renal cortical release of norepinephrine and exerts a diuretic effect too. There is experimental evidence that dexmedetomidine attenuates murine radiocontrast nephropathy by preserving cortical blood flow [18.19].

DOSAGE [12]

ICU Sedation

Adult Patients

Loading dose of 1 ìg/kg over 10 min

Maintenance infusion generally initiated at 0.4 μg/kg/h

Titrate to desired clinical effect with doses ranging from 0.2 to 0.7 μg/kg/h

More than 65 years old/impaired hepatic or renal function:

A dose reduction should be considered

Sedation for Surgical or other Procedures

Adult Patients

Loading dose of 1 μg/kg over 10 min

Maintenance infusion generally initiated at 0.6 ìg/kg/h

Titrate to desired clinical effect with doses ranging from 0.2 to 1 μg/kg/h

Adult patients undergoing less invasive procedures:

Loading infusion of 0.5 μg/kg given over 10 min may be suitable

More than 65 years old/impaired hepatic or renal function:

A dose reduction should be considered

Awake fiberoptic intubation:

Loading infusion of 1 μg/kg over 10 min

Maintenance infusion of 0.7 μg/kg/h until the endotracheal tube is secured.

Literature Review of Dexmedetomidine Use in Paediatric Cardiac Anesthesia

As an adjunct: Dexmedetomidine has been proved as a useful adjunct in cardiac anesthesia. It is shown that an infusion of dexmedetomidine @ 0.2-0.4 μg/kg/hr during perioperative period decreases extubation time and the length of ICU stay [20]. Recent metaanalysis in 2003 concluded that the use of α-2 adrenergic agonists reduced mortality and incidence of myocardial infarction following vascular surgery. It also reduced ischemic episodes during cardiac surgery [21].

Decreased heart rate, SVR and antiarrhythmic properties of dexmedetomidine contribute to better management of pediatric cardiac surgical patients postoperatively.

Attenuation of Sympathoadrenal Stress Response

A study conducted at our institute [22] in 60 pediatric patients between five to seven years of age undergoing cardiac surgery, dexmedetomidine when used as an adjunct to general anesthesia, we found that it considerably reduces anesthetic requirement and attenuates surgical stress response in the form of reduced incidence of hyperglycemia.

Pulmonary Hypertension

Regarding pulmonary hypertension, dexmedetomidine has been successfully used in patients undergoing mitral valve replacement in which it decreased fentanyl requirements, attenuated the increase in systemic vascular resistance index and pulmonary vascular resistance index after sternotomy and also decreased mean arterial pressure, mean pulmonary arterial pressure, and pulmonary capillary wedge pressure, in comparison with the values in the placebo group [23].

Few centres in India are using dexmedetomidine along with fentanyl in order to prevent episodes of pulmonary hypertensive crisis intraoperatively.

Tobias et al. [24], in a prospective, randomized trial, found that dexmedetomidine at a dose of 0.5 μg/ kg/ hr provided more effective sedation than midazolam. This was demonstrated by the need for fewer bolus doses of morphine, a decrease in the 24h requirements for supplemental morphine, as well as a decrease in the total number of assessment points with a Ramsay sedation score of 1 (inadequate sedation) and the number of patients who had a Ramsay score of 1.

Chrysostomou et al [25], in a retrospective study of 38 spontaneously breathing and mechanically ventilated children undergoing cardiothoracic surgery, found that dexmedetomidine provided adequate sedation 93% of the time and adequate analgesia 83% of the time. Side effects included hypotension (15%) and transient bradycardia in one patient.

Antiarrythmic Actions

Numerous case reports and research work have demonstrated anti arrhythmic properties of dexmedetomidine. High dose of dexmedetomidine upto 3 μg/kg/hr successfully reverted junctional ectopic tachycardia (JET) to sinus rhythm (SR) in an infant undergoing intracardiac repair of Tetralogy of Fallot (TOF) [26]. In a recent randomized trial, dexmedetomidine exerts its effectiveness in preventing occurrence of postoperative JET following complete surgical repair of TOF [27]. Chrysostomou C et al concluded that dexmedetomidine decreased incidence of atrial, junctional, ventricular and supraventricular tachyarrhythmias after congenital cardiac surgery[28].

Refractory Arrhythmia

Few case reports have demonstrated successful use of dexmedetomidine in restoring sinus rhythm when other antiarrhythmic drugs were not effective [29,30).

Electrophysiological Studies and Intervention

Dexmedetomidine depressed sinus and atrioventricular nodefunction resulting into bradycardia during electrophysiological study in paediatric patients [31]. But, one should keep in mind that the changes in PR interval, QRS interval etc were related to changes in heart rate only [32].

Prevention of Delirium

Dexmedetomidine has been also used to provide sedation in the postanesthesia care unit following sevoflurane anesthesia to decrease the incidence of agitation in the pediatric population, and to allow intubation in a sedated pediatric patient.

Cardiac Catheterization

It is successfully used for procedural sedation in cardiac catheterization laboratory[33].

Robert Mester et al [34]suggested that a combination of ketamine and dexmedetomidine provides effective sedation for cardiac catheterization in infants and children without significant effects on cardiovascular or ventilatory function.

Neuroprotection

Neurological injury is a common and frequent problem encountered in paediatric cardiac population. Dexmedetomidine has generated a lot of enthusiasm because of its neuroprotective properties. Various animal studies have shown neuroprotection exhibited by dexmeddetomidine [35,36]. Sato et al [37] found improved short-term neurologic outcome with combination of hypothermia and dexmedetomidine therapy.

Overall, dexmedetomidine is an attractive agent both during perioperative and ICU settings. Its opioid-sparing properties, minimal respiratory depression; preserved gut motility; prevention of postoperative nausea, vomiting and shivering; and potential neuroprotection, cardioprotection and renoprotection make it as an invaluable anesthetic agent in pediatric cardiac and also during general anesthesia.

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Awake Parathyroidectomy in a Patient with Aortic Stenosis with Renal Failure: Role of Regional Plexus Block, A Viable Alternative to General Anaesthesia

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Abstract

Introduction: Primary hyperparathyroidism is a hypercalcemic state due to excessive secretion of parathyroid hormone (PTH). The most common cause being single parathyroid adenoma. Majority of these cases detected by screening of serum calcium concentration, are older individuals who present with associated comorbidities. Few patients may present with Hyperparathyroid crisis a serious and potentially life threatening complication ofsevere primary Hyperparathyroidism. Parathyroidectomy leads to the cure of the disease. Parathy-roidectomy for localized adenomas are usually performed under general anaesthesia, however cervical plexus block is also a good alternative. Case Report: We describe the anaesthetic management of a 72 years old, ASA III MP IIImale patient who underwent parathyroid adenoma (right) excision under combined superficial and deep cervical plexus block. Patient was a diagnosed case of primary hyperparathyroidism. Other comorbidities beingmoderate aortic stenosis,ischaemic heart disease, chronic kidney disease, hypertension and cervical spondylosis. Patient was having regular episodes of parathyroid crisis and had to be operated upon, but the patients' medical condition contradicted general anaesthesia so patient was operated under right sided combined superficial and deep cervical plexus block with awake sedation, resulting in excellent anaesthesia of the operation site, stable haemodynamics, adequate intraoperative analgesia withreduced postoperative analgesic requirement. *Discussion:* Cervical plexus blocks the motor and sensory nerves originating from C2 to C4 nerve roots. It has been shown to be safe for thyroid and parathyroid surgeries, with cure rates equivalent to general anaesthesia.Parathyroidectomies done under regional anaesthesia significantly reduced post-operative pain, nausea, and vomiting.However the studies available till now on regional versus general anaesthetic approach for parathyroidectomies do not yield definitive information, regarding which technique is superior. Hypercalcemia along with associated muscular weakness was another reason why cervical plexus block was preferred in our patient. *Conclusion:* Thus cervical plexus block may be used as an alternative to general anaesthesia for thyroid and parathyroid surgeries, especially in patients with multiple co morbidities in whom administering a general anaesthesia may be problematic.

Keywords: Hyperparathy-roidism; Aortic Stenosis; Hyperparathyroid Crisis; Cervical Plexus Block.

Introduction

Primary Hyperparathy roidism (PHPT) is a hypercalcemic state due to excessive secretion of Parathyroid Hormone (PTH) [1]. Causes being single parathyroid adenoma (most common) in 90% of the cases, multiple adenomas or hyperplasia and rarely carcinoma 1-2% of the cases [2]. Primary hyperparathyroidism due to parathyroid hyperplasia is less common and may occur in the context of the syndrome of Multiple Endocrine Neoplasia (MEN) [3]. Parathyroid crisis is a rare manifestation of Primary Hyperparathyroidism for which Parathyroidectomy is the only curative therapy [4]. Usually parathyroidectomies are performed under General

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Anaesthesia (GA) using a supraglottic airway devices or routine endotracheal tube. The presence of comorbid conditions like symptomatic heart disease, airway and cervical spine abnormalities, renal derangement etc. makes the administration of general anaesthesia risky in these patients. Regional anaesthetic techniques with or without conscious sedation has also been shown to be safe in these patients and allowsfor cure rates equivalent to GA [5]. Regional anesthetic block of the cervical plexus is a safe and useful alternative to general endotracheal anesthesia for surgery of the neck, upper shoulder and occipital scalp area. The sensory component of the cervical plexus can be blocked separately and easily by a superficial cervical plexus block. Both motor and sensory block can be obtained by deep cervical plexus block. Minor transient side effects are common to deep cervical plexus blocks, but they are rarely of any consequence [6].

Case Report

A 72 year old American Society of Anaesthesiology (ASA) physical status III malepatient weighing 65 kgs was posted for parathyroidectomy. Patient was a diagnosed case of parathyroid adenoma with primary hyperparathyroidism. He have had two episodes of Hyperparathyroid Crisis within last 30 days needing hospitalisation. His other comorbidities being vulvular heart disease (moderate aortic stenosis)NYHA Class III at present diagnosed at the age of 60years, cervical spondylosis for last 20 yrs, hypertension for last 15yrs, ischaemic heart disease for last 5 years and recently diagnosed chronic kidney disease. He was on the following medications Tab. Metoprolol 25 mg, Tab. Furosemide 40mg, Tab. Aspirin 75 mg. On Preanesthetic Check-up, Pulse Rate (PR)was 86/min, Blood Pressure (BP)150/ 80mmHg , Respiratory Rate (RR) was 20/min and MET (Metabolic Equivalent of Physical Activity) 4. He had a Mallampati grade of III with moderately restricted neck movement, edentulous tooth, a large tongue and absent buccal pad of fat. Systemic examination revealed an ejection systolic murmur, heard loudest at the upper right sternal border & at the right 2nd intercostal space which radiated to the carotid arteries bilaterally. All routine investigations were within normal limit except pre-operative Haemoglobin being 8.4 gm% after one unit whole blood transfusion, Urea 70 mg/dl, Creatinine 2.85 mg/dl, Calcium (Ca^{++})14.6 mg/dl, Phosphate 2.5 mg/dl, Parathyroid hormone (PTH) level 878 pg/ ml, Alkaline Phosphatasewas 830 IU/L, coagulation profile normal. Chest X-Ray showed cardiomegaly. Electro Cardiogram had a left axis deviation with T wave inversion in V_{3-5} leads. Transthoracic Echocardiography showed moderate ASwith thickened calcific aortic valve (AVA=1.4 cm^2, mean gradient of 40 mm Hg), concentric LVH, Grade-I diastolic dysfunction and LVEF 50%.Ultrasound scan neck revealed a 2.5 cm well-defined mass inferoposterior to right thyroid lobe. Parathyroid scintigraphy confirmed hyperfunctional parathyroid tissue in the posteroinferior aspect of right thyroid gland with brown tumour involving right upper humerus.The patient, relatives and medical specialists decided go ahead with surgery, inspite ofthe risks as he was having regular episodes of hyperparathyroid crisis. He was counselled on the previous day about the merits and demerits of both general and regional anesthesia techniques. Anaesthetic plan formulated was cervical plexus block. Cardiac risk evaluation was done.On arrival at the operation room, an intravenous line was secured with 18G cannula and isotonic saline was started. All anesthetic equipments were prechecked and standard ASA monitors were attached to the patient. Electrocardiogram (ECG), Oxygen Saturation (SpO2), Noninvasive Blood Pressure (NIBP), and Heart Rate (HR) were monitored continuously throughout the intraoperative period.Patient was premedicated with Inj. Midazolam 1mg IV, Inj. Fentanyl 50 mcg IV. Under strict aseptic and antiseptic precaution, landmark based right sided combined superficial and deep cervical plexus block was performed. A 22G needle connected via an extension 3 way to a 20ml syringe containing local anaesthetic, wasused forsuperficial cervical plexus block (SCPB)and three injections of 5 mL of 0.5% ropivacaine were injected at the SCPB needle insertion site subcutaneously in perpendicular, cephalad and caudad directions in a 'fan' shaped fashion after negative aspiration for blood. For deep cervical plexus block (DCPB), a single-injection technique was followed. A 5 cm 22 gauge needle was inserted at the second needle entry site,at 2cm depth when the transverse process of C3 vertebra was approached, the needle was withdrawn slightly and 15 ml of 0.5% ropivacaine was injected after negative aspiration for blood and CSF.

Supplemental oxygen was delivered @4 L/min through face mask. After confirming the adequacy of the block, surgery was started. Whole surgical procedure lasted for 2 hours 15 minutes. Throughout the procedure patient was verbally responsive with a RAMSAY sedation score of <3 and a VAS score of ≤3. No intraoperative complications noted& throughout the procedure patient was haemodynamically stable. Patient was shifted to ICU for observation and was closely monitored for signs and symptoms of hypocalcemia. Patient's VAS score recorded 2 hours post operatively was ≥4 so rescue analgesia in the form of Fentanyl patch 25 mcg/hourwas provided for the next 24 hours. No other supplemental analgesic were administered. Serum Ca^{++} and PTH levels were monitored postoperatively. Patient's postoperative course was uneventful, he was started on oral calcium on the second postoperative day. He was subsequently discharged home after a week.

Discussion

Primary hyperparathyroidism often remains asymptomatic in many patients. Hypercalcemia is the underlaying cause for the signs and symptoms in a hyperparathyroid patient. In these patientstress or illness may precipitate a Hyperparathyroid Crisis[7]. An acute hyperparathyroid crisis raises the serum calcium level acutely above 14 mg% with, nearly 4 times higher PTH levels. Clinical manifestations include weakness, lethargy, hypertension, palpitations, polyuria, polydypsia, anorexia, nausea, vomiting, dehydration, altered mental state or even coma. The best treatment for hypercalcemic crisis is early diagnosis and proper medical management followed by removal of hyperfunctional glandular tissue at a later stage [8]. Medical management comprises of adequate intravenous hydration, diuresis, Bisphosphonates and dialysis in cases refractory to medical management. Criteria for surgery in these patientsinclude significant hypercalcemia (> 1 mg/dl above upper limit of normal), marked hypercalciuria (>400mg/day), low bone density, unexplained renal insufficiency and episode of acute primary hyperparathyroidism [9].Consideration for parathyroidectomy should also be given to elderly patients with primary hyperparathyroidism who are vitamin D deficient. Our patient presented with generalized muscular weakness, lethargy and gave history of 2 episodes of hypercalcaemic crisis within a span of last 30 days, requiring hospital admission. Parathyroidectomy was indicated in our patient because of the persistently elevated serum calcium levels, regular episodes of life threatening hyperparathyroid crisis.

In patients of Parathyroid adenoma presenting with hyperparathyroid crisis, emergency parathyroidectomy has a success rate of 92%with excellent long term outcome [10,8]. Usually parathyroid gland surgeries are performed under GA. However the cervical plexus block/local anaesthesia techniques has been shown to be safe, with cure rates equivalent to GA[11,12]. However the studies available till now on local versus general anaesthetic approach for parathyroidectomies do not yield definitive information, regarding which technique is superior. However operating under local anaesthesia keeps patient fully conscious, hence surgeon can easily communicate as well as assess the phonation of patient. Our patient was operated using regional anaesthesia technique combining superficial with deep cervical plexus block and excellent analgesia and anaesthesia of the operative site was achieved.

Ziegler R et al [13] noted that there is a risk of life threatening cardiac arrhythmias when severe hypercalcemia is not treated preoperatively in these patients. As such there are no guidelines regarding the preoperative serum calcium levels in these patients. Hypercalcemia along with associated muscular weakness was another reason why we preferred regional anaesthesia technique in our patient.

Cervical plexus block anaesthesises the motor and sensory nerves originating from C2 to C4 nerve roots [14].The SCPBis used for superficial cutaneous surgeries of the head and neck whereas the DCPB is used for deeper surgeries of the neck, such as carotid artery or thyroid surgery. This block is also useful to supplement other regional techniques of the upper torso [14]. Studies have shown that superficial cervical plexus block with local infiltration is as effective as a combined superficial and deep cervical plexus block. Still many clinicians perform a SCPB to complement the DCPB [14,15] to achieve complete anaesthesia and avoid possible failure of the block. We performed a combined superficial and deep cervical plexus block to ensure complete anaesthesia. We used a single injection technique for DCPB in our patient as he was on Tab Aspirin .

Black M J et al [16] showed that parathyroidectomies done under regional anaesthesia significantly reduced post-operative pain, nausea, and vomiting in patients. Sang Yoon et al [17] reported successful management of parathyroidectomy under superficial cervical plexus block in a patient with severe kyphoscoliosis. Our patient had no episodes of hypocalcaemia, nausea and vomiting postoperatively with reduced postoperative analgesic requirement.

Perioperative management of symptomatic heart disease in patients undergoing non cardiac surgery requires careful team work and communication between patient, primary care physician, anesthesiologist and surgeon. Several cardiac indices are present to predict postoperative cardiac complications in these patients. Our patient was to undergo an intermediate risk surgery, he had major clinical predictors positive for post-operative cardiac complications with GOLDMAN MULTI FACTORIAL CARDIAC RISK INDEX score [18] revealing CLASS 2 with 7 to 11% post-operative risk. Goldman first reported aortic stenosis as an independent predictor for life threatening cardiac complications during non-cardiac surgery [18] and this was confirmed by Detsky [19]. Perioperative mortality and nonfatal myocardial infarction was higher in patients with Aortic stenosis [20]. Anaesthetic plan in our patient was formulated keeping in mind the associated comorbidities like ischaemic heart disease, aortic stenosis, chronic kidney disease, cervical spondylosis along with hyperparathyroidism. Generally these patients are in a state of low fixed cardiac output state, so haemodynamic goals in them includes maintaining a normal sinus rhythm, avoiding tachycardia, adequate volume loading, avoiding hypervolaemia and maintaining a normal systemic vascular resistance and afterload. Taking general anaesthesia for consideration, nearly all induction agents as well as inhalational anaesthetics cause generalized vasodilatation to a variable extent, causing decompensation, therefore titrating dose of all inhalational and induction agents is mandatory. Maintainance of intraoperative blood pressure at pre-induction levels prevents intraoperative cardiac ischaemia. Adequate analgesia maintaining a proper plan of anaesthesia intraoperatively specially during the time of intubation prevents catecholamine induced tachycardia, hypertension and the risk of cardiac ischaemia.Taking into consideration the problems related with general anaesthesia for such cases, performing the operation under regional nerve blocks with or without sedation is a good alternative. It has the following advantages of offering excellent hemodynamic stability in terms of cardiac output and systemic vascular resistance, adequate sensory and motor blockade of the operative site, decreased incidence of intraoperative tachycardia and arrhythmias, avoiding problems related to difficult airway and intubation with decreased requirement of supplemental analgesics. All these advantages prompted the use of cervical plexus block in our patient. Anesthetic toxicity, intravascular injection, phrenic nerve block etc being the main complications of cervical plexus blocks which can be detected by closely monitoring.

Intraoperative invasive arterial blood pressure monitoring is helpful in patients with multiple comorbidityespecially for long surgical procedures. Adequate fluid balance is essential, especially CVP, Oesophageal Doppler, Trans-thoracic/oesophageal Echocardiography guided fluid therapy may be helpful. Treating hypotension using directly acting alpha-agonists such as phenylephrine may improve systolic and diastolic LV function. Arrhythmias must be treated promptly. Postoperative ICU care is mandatory for these patients. We did not use invasive monitoringas the operative duration in our patientwas short, no episodes of arrhythmia, ischaemia or hypotension occurred intraoperatively. Post-operatively he was shifted to ICU for observation.

Adequate preoperative assessment and proper anaesthesia technique selection followed by adequate postoperative care monitoring for hypocalcemia helped us in successful management of this patient. Serum calcium was monitored at regular postoperative intervals. Oral calcium therapy was started on 2nd postoperative day.

Conclusion

Elderly patients of hyperfunctional parathyroid adenoma who have associated comorbidities pose a challenge to the anaesthesist. Setting up anaesthetic goals along with a detailed anaesthetic plan which could be curtailed as per patient's requirement is mandatory for them. Preoperative optimization of patient, maintenance of adequate hydration status and keeping ionized calcium within normal limits during perioperative period can reduce potential complications. Parathyroidectomy under combined superficial and deep cervical plexus block is an acceptable regional anaesthetic technique and provides effective analgesia with limitation of side-effects, but such technique can be extremely hazardous in light of inadequate anaesthesia. As it requires considerable practice and skill in recognition of anatomical structures and placement of the needle.

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[9] National Statistics Online—Trends in suicide by method in England and Wales, 1979-2001. www.statistics.gov.uk/downloads/theme_health/ HSQ 20.pdf (accessed Jan 24, 2005): 7-18. Only verified references against the original documents should be cited. Authors are responsible for the accuracy and completeness of their references and for correct text citation. The number of reference should be kept limited to 20 in case of major communications and 10 for short communications.

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