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ORIGINAL RESEARCH

An Observational Study on the Haemodynamic Effects During Induction and Intubation in Patients Co-Induced with Sevoflurane and Propofol

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ABSTRACT

Background: Induction of anesthesia and endotracheal intubation are known to provoke significant haemodynamic changes due to the physiological stress response and the pharmacologic effects of anesthetic agents. Achieving cardiovascular stability during this period is a key goal in anesthetic management. Propofol and Sevoflurane are widely used agents, each with unique advantages and potential side effects. Co-induction with both agents may offer a synergistic effect, reducing individual drug dosages and improving haemodynamic outcomes. **Objective:** This observational study aims to assess the haemodynamic responses—specifically changes in heart rate and blood pressure—during the induction and intubation phases in patients co-induced with Propofol and Sevoflurane. **Methods:** The study was conducted on a selected group of patients undergoing elective surgeries under general anesthesia. Baseline haemodynamic parameters were recorded and monitored at various time intervals throughout the induction and intubation process. The focus was on evaluating fluctuations and trends in systolic, diastolic, and mean arterial pressures, along with heart rate. **Results:** The findings suggest that co-induction with Sevoflurane and Propofol leads to better haemodynamic stability, with minimal fluctuations observed during both induction and intubation. The combination appeared to attenuate the typical sympathetic response commonly associated with laryngoscopy and intubation. **Conclusion:** Co-induction using Sevoflurane and Propofol is a clinically effective strategy to achieve smooth induction while maintaining haemodynamic stability. These results support the integration of this approach in routine anesthetic practice, especially in patients where cardiovascular fluctuations pose a significant risk.

Keywords: Anesthesia, Induction, Propofol, Inhalational, Sevoflurane

INTRODUCTION

The induction of general anesthesia and subsequent endotracheal intubation are critical phases in the perioperative period, often associated with significant haemodynamic fluctuations. These changes—typically manifesting as alterations in heart rate and blood pressure—are primarily due to the stress response triggered by airway manipulation and the pharmacodynamic effects of anesthetic agents. Maintaining haemodynamic stability during this period is crucial, especially in patients with limited cardiovascular reserve.

Propofol, a commonly used intravenous induction agent, is well known for its rapid onset and smooth induction characteristics. However, its administration is frequently associated with dose-dependent hypotension and bradycardia due to its vasodilatory and myocardial depressant properties. On the other hand, Sevoflurane, a volatile anesthetic agent, offers advantages such as minimal airway irritation and a favorable haemodynamic profile, especially during inhalational induction. When used together—known as co-induction—the agents may exert a complementary effect, potentially reducing the

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required dose of each and enhancing cardiovascular stability.

At this level of anaestheisa, hypotension and bradycardia is greatly potentiated warranting use of adjuvants such as opioids, muscle relaxants, Propofol and/or Ketamine.[1] Propofol is one of the most commonly used agents for total intravenous (IV) anesthesia induction in patients undergoing FOI; however, it has shown to have the potential risk to induce apnea, arterial hypotension, and collapse of upper airways.[2-12] Anesthesia induction with inhalational agents is suitable in patients with high risk of difficult intubation and in patients requiring tracheal intubation without neuromuscular blocking drugs. Sevoflurane, as an induction agent, has shown to induce a lower rate of respiratory complications when compared with propofol.[4-6] Several studies have compared hemodynamic and respiratory variables occurring during anesthesia induction with sevoflurane or propofol; however, there is yet a lack of data with regard to patients undergoing cervical spine surgery for cervical myelopathy.[3,4] Propofol is a frequently-used intravenous anaesthetic with an effect of rapid onset and short duration. During an-aesthesia induction with propofol, often seen side effects are injection pain and a fall in arterial blood pressure.[7,8] Etomidate is a hypnotic agent with minimal effects on the cardiovascular system. It does not cause histamine expression and has no analgesic properties. Etomidate's side-effects are primarily injection pain, myoclonus, su- perficial thrombophlebitis and a high incidence of nausea and vomiting.[7] Previous studies have also reported that etomidate did not prevent the sympathetic response to la- ryngoscopy and intubation at a sufficient level.[7,9]

Despite the frequent clinical use of both Propofol and Sevoflurane, limited observational data exist on their combined impact on haemodynamics during induction and intubation. This study aims to evaluate and compare the haemodynamic responses in patients undergoing general anesthesia with co-induction using Sevoflurane and Propofol. By understanding the interaction between these two agents, anesthetic protocols can be better tailored to optimize patient safety and procedural outcomes.

MATERIALS AND METHODS

Patient refusal, evidence of difficult airway and history of malignant hyperthermia were taken as exclusion criteria. On the day of surgery, after base line reading of haemodynamic parameters- Pulse rate, Systolic blood pressure, Diastolic blood pressure and Mean arterial pressure were recorded, all the patients were premedicated intravenously with Inj.Glycopyrrolate(0.2mg), Inj.Midazolam (1mg) and Inj.Butorphanol(1mg). Each patient was pre-oxygenated with 100% oxygen for 3min. Vital capacity induction(VCI) technique was employed for Sevoflurane induction. Prior to induction, all the patients were taught and made to practice the technique i.e., to exhale fully, then inhale fully and hold the breath as long as possible. After the anaesthesiologist was convinced of the patient's understanding of the vital capacity induction technique, patients were asked to inhale deeply following full exhalation from a circuit primed with Sevoflurane. Priming was done with Sevoflurane vaporizer dial set to 4% with oxygen flow of 8L/min, till the gas analysis in inspired limb measured 3%. It typically required three fill/empty cycles with the circuit occluded. The pre- oxygenation mask was removed at end-expiration, and this primed circuit with mask was applied to the face. Patients were encouraged to hold the breath as long as possible. Following the vital capacity breaths, InjPropofol (1mg/kg)was given IV over 20sec. This was followed by Inj.Succinylcholine (1mg/kg) and orotracheal intubation was done under direct laryngoscopy. Anaesthesia was maintained with N2O:O2 = 3:2 along with Isoflurane @ 0.6-1% v/v. Adequate muscle relaxation was maintained with intermittent intravenous bolus Inj. Vecuronium. Pulse rate(PR), Systolic blood pressure(SBP), Diastolic blood pressure(DBP), Mean arterial pressure(MAP) and Oxygen saturation(SpO2) were recorded at the following periods: pre-induction, post-induction, and, 2min, 5min, 10min, 15min following induction. Vital parameters were monitored throughout peri- operative period. At the end of surgery, all anaesthetics were withdrawn. Residual muscle paralysis was reversed with Inj.Neostigmine (0.05mg/kg) and Inj. Glycopyrolate (0.04mg/kg) and awake extubation was done.

RESULTS

Gender and weight distribution being comparable, we had more patients in 20-30 years age-group and in ASA category I. **Demographic Variables of 40 patients were shown in table 1 and Haemodynamic Parameters were shown in table 2 .**

Table 1: Demographic Variables (n=40)

Variables	Age(years)		Gender		Weight (Kg)			ASA Grade	
	20-30	30-40	Male	Female	41-50	51-60	61-70	I	II
No. of Patients	23	17	21	19	14	14	12	32	8
Percentage of Total	57.5%	42.5%	52.5%	47.5%	35%	35%	30.00%	80%	20%

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Table 2: Haemodynamic Parameters (Mean±SD)

Variables	PR (bpm)	SBP (mmHg)	DBP (mmHg)	MAP (mmHg)	SpO2 (%)
Pre-induction	90.12±9.36	124.41±8.39	77.52±8.93	92.71±8.33	98.11±0.69
Post-induction (0 minute)	88.52±8.06	115.68±7.34	74.83±8.69	88.79±7.19	99.92±0.33
After 2 mins	93.24±7.39	122.54±6.76	75.91±8.48	91.52±7.48	99.96±0.28
After 5 mins	89.30±6.20	121.44±5.69	78.31±8.66	91.15±6.84	99.91±0.33
After 10 mins	85.11±7.18	120.01±6.59	76.41±7.58	91.01±6.99	99.99±0.19
After 15 mins	88.39±5.01	115.61±9.49	81.73±3.39	88.01±4.39	99.98±0.20

DISCUSSION

King BD et al in 1950 observed that haemodynamic changes upon laryngoscopy and intubation was significantly higher in light plane of anaesthesia, and concluded that deepening the plane of anaesthesia attenuates this response.[10] The review article by Blanc and Trembley on complications of endotracheal intubation published in 1974, stated that laryngo-tracheal stimulation induces tachyarrhythmia and hypertension. They linked this phenomenon to increase in plasma Nor Adrenaline as a result of laryngosympathetic reflex.[11] Russel et al in 1981,[12] Derbyshire DR et al in 1983[13] and Pyres Roberts et al in 1986,[14] also observed independently the rise in plasma NAdr level following laryngoscopy and intubation. Their studies also supported that conducted by King BD et al; concluding that these sympathetic responses are readily precipitated by light plane of anaesthesia, hypoxia and hypercarbia. Recent studies proposed that even several cellular mechanisms including ischemia, glutamatergic toxicity, neuroinflammation, and apoptosis could be involved in the progression of cervical myelopathy, suggesting that oligodendroglia of the spinal cord may be hypersensitive to hypotension and hypoperfusion, with consequent ischemic injury.[15] Therefore, in patients undergoing cerebral myelopathy surgery, close attention must be paid to avoid mechanical spinal cord compression and to maintain an adequate spinal cord perfusion to prevent further neurological damage.[13] Several studies have discussed the risks related to laryngoscopy and cervical spine movements in these patients and several approaches have been proposed.[1,13] Here patients were asked to inhale from the primed circuit after full exhalation and hold the breath as long as possible. Bourne's technique is superior to tidal breath induction due to shorter induction time and lesser complication during induction as evidenced by Yurino M et al in 1993.[16] MACEI is the minimum alveolar concentration of volatile agent required to obtain an acceptable condition for endotracheal intubation. In the year 1994, Kimura T et al determined the MACEI for Sevoflurane to be 4.52% which is 2.8 times the MACIM.[12] In order to achieve the MACEI patients should be mask ventilated with a circuit primed with 6-7% Sevoflurane for 4-6 minutes.[17] This potentially increases the risk of hemodynamic compromise, as Sevoflurane is known to cause decrease in cardiac output and systemic vascular resistance in a dose dependent manner.[18,19] Another major drawback of Sevoflurane induction is the patient excitement during induction. Such a case was first reported by Adachi M et al in 1992.[20] They reported a case of tonic clonic seizure-like movements in the extremities of a young girl during Sevoflurane induction. Two years later, Komatsu et al reported two more such incidences.[21] In 2001, Vakkuriet al investigated the effect of induction with Sevoflurane 8% on brain electrical activity. They found evidence of seizure on EEG, especially during controlled ventilation. They also concluded that these abnormal EEG discharges alter the autonomic nervous system outflow leading to hyperdynamic circulatory changes.[22] Wappler F et al published a review article and questioned the immediate administration of Sevoflurane 8% for induction. They reported that the high concentration might be the cause of EEG change. So they advocated for lower concentration of Sevoflurane for induction and tonavoid hyperventilation by controlled ventilation.[23] With the introduction of barbiturate group of drugs, intravenous anaesthesia gained popularity over inhalational route for quick, predictable smooth and pleasant induction. The potent intravenous hypnotic Propofol, is popular as it suppresses the airway reflexes better than other agents - as concluded by Mackenzie and Grant in 1985 and McKeating et al in 1988.[24,25] Stress hormone levels are also considerably lower with Propofol induction. In the year 1995 Mustola ST et al found that plasma adrenaline levels were significantly lower after induction with Propofol and remained below baseline throughout the procedure.[26] Plasma NAdr level also did not increase following Propofol induction as evidenced by S Coley et al.Another study reported that after anaesthesia induction with etomidate (0.3 mg kg-1), the ideal fentanyl dose was 5-10 mcg kg-1 to prevent a haemodynamic response to la- ryngoscopy and intubation.[27] However, it can be predicted that the use of such a high dose of fentanyl may cause in- creased hypotension and nausea and vomiting. In a study by Muriel et al,28 a comparison was made of propofol (2 mg kg-1), thiopental (5 mg kg-1) and etomidate (0.3 mg kg-1) in anaesthesia induction. A statistically sig- nificant increase was determined in systolic and diastolic arterial pressure and HR in the etomidate and thiopental groups after intubation and the highest rates of complica- tions were reported in the etomidate group. In two studies which compared propofol, thiopental and etomidate induction in intubation without muscle relaxant, as appropriate conditions

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could not be provi- ded in the etomidate group, the study was prematurely terminated.29,30 In another study, the haemodynamic response to oro- tracheal intubation was evaluated following anaesthesia induction with midazolam and etomidate. Although the systolic and diastolic pressures and RPP values were found to be lower in the midazolam group, it was reported that neither of the induction agents could prevent the haemo- dynamic response to intubation.31,32 In the current study, myoclonus was determined at 20% in group E and 9% in group PE. These low rates of myoclonus are thought to be due to premed- ication with fentanyl. Previous studies have reported the incidence of myoclonus with fentanyl use at 8% to 40%.40,33 Injection pain is a significant clinical problem in both propofol and etomidate use. In the current study, the inci- dence of injection pain was 27% in group P, 13% in group E and 10% in group PE, with no statistically significant difference between these rates. In literature, propofol in- jection pain has been reported at rates of 40%-86%.34 With fentanyl premedication, rates such as 40%, 19% and 8% have been reported.35-37 Reported rates of 50%-60% of eto- midate injection pain are also reduced with fentanyl pre- medication.21 In a study by Saricaoglu et al15 comparing propofol, eto- midate and etofol in anaesthesia induction, injection pain in the etofol group was found to be lower than in the oth- er two groups and myoclonus incidence was lower than in the etomidate group. The incidence of injection pain was reported as 83.8% in the propofol group and as 63.2% in the etomidate group. Myoclonus incidence was deter- mined as 93.4% in the etomidate group. The reason for the high rates of these results compared to the results of the current study is thought to be that no premedication was administered in the Saricaoglu study. In literature, propofol and etomidate mixed in the same injector have been used.15 Due to the risk of propofol contamination in particular, it has been reported that it is necessary to apply strict aseptic techniques during preparation and application.38 Severe infection tables have been reported because of propofol contamination.38,39 In the current study, separate injectors were used because of the increased risk of contamination while preparing the combination.

CONCLUSION

Both total intravenous anesthesia (TIVA) using Propofol and inhalational induction with Sevoflurane have proven to be safe and effective methods for initiating anesthesia, including in cases requiring fiberoptic intubation (FOI) without the use of neuromuscular blocking agents (NMBAs). While each technique has its merits, it is important to recognize the haemodynamic implications, particularly with Propofol. Even when administered at a slow and controlled rate, Propofol has been shown to cause a noticeable reduction in mean arterial pressure (MAP). Although this decline may still fall within the accepted threshold for maintaining spinal cord perfusion, it could be clinically significant—especially in patients with pre-existing cervical spine myelopathy.

This potential for haemodynamic instability underscores the need for careful agent selection and individualized dosing strategies in high-risk patient populations. Given the delicate balance required to avoid compromising spinal cord perfusion in such cases, further research is essential. Future studies should aim to compare various induction techniques in patients with cervical spine pathology, in order to establish optimized protocols that minimize both neurological and systemic risks. Such evidence will be critical in guiding safe anesthetic practices for this vulnerable cohort.

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