

**“VARIATION IN INDUCTION DOSE AND TIME OF PROPOFOL ON
ADMINISTERING INHALED NITROUS OXIDE DURING
INDUCTION: A PROSPECTIVE, RANDOMISED CONTROLLED
STUDY”**

By

Dr. DANDAMUDI SIRI CHANDANA



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IN
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Under the Guidance of

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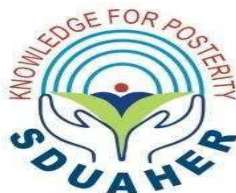
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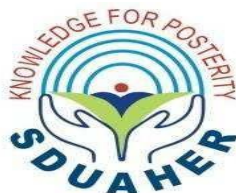
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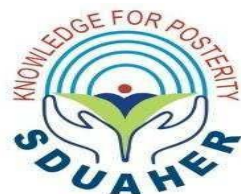
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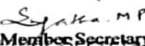

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VARIATION IN INDUCTION DOSE AND TIME OF PROPOFOL ON ADMINISTERING INHALED NITROUS OXIDE DURING INDUCTION: A PROSPECTIVE, RANDOMISED CONTROLLED STUDY I ABSTRACT Background: Commonly used intravenous induction agent, propofol is in contemporary anesthesia practice, valued for its rapid onset, amnesic properties, and ability to provide optimal conditions for intubation. Nitrous oxide, though a weak anesthetic, offers analgesic and anxiolytic benefits and is often used as an adjuvant to reduce the requirement for intravenous agents. The present study explores the role of inhaled nitrous oxide in optimizing propofol induction by reducing its dose and onset time while maintaining hemodynamic stability. Aim: To evaluate the variation in induction dose and induction time of propofol when combined with inhaled nitrous oxide during general anesthesia. Materials and Methods: This study was conducted on 96 patients (ASA I and II, aged 18–60 years) undergoing elective surgeries under general anesthesia at R.L. Jalappa Hospital. Participants were randomly divided into two groups (n=48 each). Group A received 70% nitrous oxide with 30% oxygen and propofol infusion; Group B received 100% oxygen with propofol infusion. Induction time, propofol dose, and hemodynamic parameters were recorded and analyzed using standard statistical tests. Results: Group A demonstrated significantly shorter induction times ($p < 0.05$), with 85.4% achieving induction in <200 seconds compared to 12.5% in Group B. Propofol dose was also significantly lower in Group A. The systolic, diastolic, and mean arterial pressures, was better maintained in Group A. Heart rate differences were not statistically significant. Conclusion: The co-administration of inhaled nitrous oxide significantly reduces propofol induction time and dose while enhancing hemodynamic stability. This combination can be a safer and more efficient approach to anesthesia induction. II Keywords: Propofol, Nitrous oxide, General anesthesia, Induction dose, Induction time, Hemodynamic stability III INTRODUCTION Propofol has been the most often utilized drug for intravenous induction of anesthesia in recent years. It blunts the airway reflexes in hyperreactive airway. Propofol as an induction agent is safe and effective hypnotic, it provides rapid awakening, amnesic effect and better intubating conditions. However it results in significant post-induction hypotension, bradycardia, pain on injection, apnea hemodynamic and respiratory depression, hypertriglyceridemia.1 Nitrous Oxide is safe for majority of the patients due to its analgesic effect and anxiolytic effect. Nitrous oxide due to its weak anesthetic properties is used as an adjuvant. The use has decreased the necessity of intravenous anaesthetics.1–3 By inducing with nitrous oxide, we are trying to maximize the benefits of using propofol and also minimizing the side effects which are dose related. According to the studies done so far, there is a 44% reduction in Propofol dose requirement and induction time with 66% concentration of Nitrous oxide inhalation.4,5 Though Nitrous oxide has drawbacks like second gas effect, gas filled cavities expansion, nausea and vomiting postoperatively, it reduces propofol requirement. It is unknown, yet, how both medications affect general hemodynamics. REVIEW OF LITERATURE A medically induced condition of unconsciousness known as general anesthesia causes patients to lose their defensive reflexes and become totally unresponsive to painful, tactile, or spoken stimuli. It is accomplished by administering a variety of anesthetics that, when taken together, result in amnesia, analgesia, muscle relaxation, unconsciousness, and autonomic reflex suppression. To maintain airway patency, a laryngeal mask airway or endotracheal tube is often required due to the risk of upper airway obstruction.6 Historically, before the development of systematic monitoring techniques, determining the depth of anesthesia relied solely on physical examination, leading to frequent anesthetic overdoses, particularly by less experienced anesthetists. It was not until the 20th century that a structured approach to monitoring anesthesia depth emerged. Despite advancements in anesthetic agents and delivery methods that allow for quicker induction and recovery—sometimes bypassing certain stages—Guedel's classification remains a relevant reference in modern anesthesia practice.7 Guedel's Classification - Anesthesia Stages Stage 1 – Disorientation or Analgesia: often beginning in a preoperative anesthesia holding area, is referred to as the induction stage. Patients receive medication that induces sedation while remaining conscious and conversational. Breathing remains slow and steady. The patient moves from analgesia without amnesia to analgesia with amnesia at the same time during this phase.8 The stage concludes when the patient loses consciousness. Stage 2 – Delirium or Excitement: Characterized by disinhibition, uncontrolled movements, delirium, and the loss of the eyelash reflex, this stage is associated with higher heart rate and blood pressure. Airway reflexes remain intact but become hypersensitive, increasing the risk of laryngospasm if manipulated. Any airway procedures, such as endotracheal tube placement or deep suctioning, should be avoided to prevent complications. Spastic movements, vomiting, and irregular breathing patterns may further endanger the airway. To minimize time spent in this phase, fast-acting anesthetic agents are often used to facilitate a smoother transition to the next stage.9 Stage 3 – Surgical Anesthesia: This level of anesthesia is the goal for most surgical procedures, marked by the absence of eye movements and significant respiratory depression, making airway manipulation safe. Surgical anesthesia is further classified into four planes.9,10 Stage 4 – Overdose: This dangerous phase results from excessive anesthetic administration relative to surgical stimulation, leading to profound brain and medullary suppression. It begins with respiratory cessation and, if not managed promptly, can result in death. Due to cardiac depression and severe vasodilation, patients at this stage exhibit flaccid skeletal muscles, locked and dilated pupils, and dangerously low blood pressure with weak, thready pulses. This stage poses a serious risk to life if cardiovascular and respiratory measures are not taken quickly. The primary goal of the anesthetist is to quickly induce Stage 3 anesthesia in the patient and maintain that level of anesthesia through the procedure.9,11 The General anesthesia can trigger physiological responses that, if not managed properly, may lead to morbidity or even mortality. As a high-risk medical intervention, its benefits must outweigh potential harms.12 Although deaths from anesthesia are rare, they can nonetheless happen as a result of complications such as severe allergic responses, airway blockage, or pulmonary aspiration of stomach contents. The primary cause is still human mistake, even though equipment failure might play a role. However, because to better safety procedures, better monitoring and detection tools, and the adoption of standardized norms, the death rate from anesthesia has significantly decreased over the last 20 years. Patient safety results have improved and errors have been drastically decreased as a result of these quality initiatives.13,14. NITRIC OXIDE It is a colorless, odorless, non-flammable gas that can support combustion similarly to oxygen. Commonly known as laughing gas due to its euphoric effects, it is the least potent inhalational anesthetic. Nitrous oxide is usually paired with a more powerful volatile agent because it cannot be employed as a stand-alone anesthetic due to its minimum alveolar concentration (MAC) of 104percent. It contributes to the second gas effect, which increases the concentration of other volatile anesthetics in the lungs, because of its rapid onset and short offset caused by its limited blood solubility (blood-gas partition coefficient of 0.47).2 Nitrous gas finds extensive application in dental treatments, general anesthesia, procedural sedation, and pain management, especially in emergency and obstetric contexts. In these situations, it is frequently given as a 50percent oxygen mixture. =Overall minute ventilation stays constant even as the tidal volume drops and the respiratory rate increases. Although it has the potential to directly cause cardiac depression, sympathetic stimulation balances this effect, thus the overall effect is minimal.15,16 Mechanism of action Nitrous oxide exerts its effects through multiple targets in the CNS, acting as a non-competitive NMDA receptor inhibitor to produce anesthesia. Mediated through GABA-A activation. 3 According to pharmacokinetics, nitrous oxide enters the body quickly through the alveoli and



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ABBREVIATIONS

ACOG – American College of Obstetricians and Gynecologists

ASA – American Society of Anesthesiologists

BIS – Bispectral Index

CI – Confidence Interval

EDTA – Ethylenediaminetetraacetic Acid

EKG – Electrocardiogram

ENIGMA – Evaluation of Nitrous Oxide in the Gas Mixture for Anaesthesia

FO – Propofol only (Group FO in Jain K et al. study)

FN – Propofol + Nitrous oxide (Group FN in Jain K et al. study)

GABA – Gamma-Aminobutyric Acid

ICU – Intensive Care Unit

IV – Intravenous

MAC – Minimum Alveolar Concentration

MAP – Mean Arterial Pressure

mg/kg – Milligrams per kilogram

min – Minutes

N₂O – Nitrous Oxide

NMDA – N-Methyl-D-Aspartate

O₂ – Oxygen

PACU – Post-Anesthesia Care Unit

PN – Propofol + Nitrous oxide (Group in Singh S et al. study)

PO – Propofol only (Group in Singh S et al. study)

PONV – Postoperative Nausea and Vomiting

QT – Time interval in EKG representing ventricular depolarization and repolarization

SpO₂ – Peripheral capillary oxygen saturation

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ABSTRACT

Background: Commonly used intravenous induction agent, propofol is in contemporary anesthesia practice, valued for its rapid onset, amnestic properties, and ability to provide optimal conditions for intubation. However, its use is associated with dose-dependent adverse effects such as hypotension, bradycardia, and respiratory depression. Nitrous oxide, though a weak anesthetic, offers analgesic and anxiolytic benefits and is often used as an adjuvant to reduce the requirement for intravenous agents. The present study explores the role of inhaled nitrous oxide in optimizing propofol induction by reducing its dose and onset time while maintaining hemodynamic stability.

Aim: To evaluate the variation in induction dose and induction time of propofol when combined with inhaled nitrous oxide during general anesthesia.

Materials and Methods: This study was conducted on 96 patients (ASA I and II, aged 18–60 years) undergoing elective surgeries under general anesthesia at R.L. Jalappa Hospital. Participants were randomly divided into two groups (n=48 each). Group A received 70% nitrous oxide with 30% oxygen and propofol infusion; Group B received 100% oxygen with propofol infusion. Induction time, propofol dose, and hemodynamic parameters were recorded and analyzed using standard statistical tests.

Results: Group A demonstrated significantly shorter induction times ($p < 0.05$), with 85.4% achieving induction in <200 seconds compared to 12.5% in Group B. Propofol dose was also significantly lower in Group A. The systolic, diastolic, and mean arterial pressures, was better maintained in Group A. Heart rate differences were not statistically significant.

Conclusion: The co-administration of inhaled nitrous oxide significantly reduces propofol induction time and dose while enhancing hemodynamic stability. This combination can be a safer and more efficient approach to anesthesia induction.

Keywords: Propofol, Nitrous oxide, General anesthesia, Induction dose, Induction time, Hemodynamic stability

INTRODUCTION



INTRODUCTION

Propofol has been the most often utilized drug for intravenous induction of anesthesia in recent years. It blunts the airway reflexes in hyperreactive airway. Propofol as an induction agent is safe and effective hypnotic, it provides rapid awakening, amnesic effect and better intubating conditions. However it results in significant post-induction hypotension, bradycardia, pain on injection, apnea hemodynamic and respiratory depression, hypertriglyceridemia.¹

Nitrous Oxide is safe for majority of the patients due to its analgesic effect and anxiolytic effect. Nitrous oxide due to its weak anesthetic properties is used as an adjuvant. The use has decreased the necessity of intravenous anaesthetics.¹⁻³

By inducing with nitrous oxide, we are trying to maximize the benefits of using propofol and also minimizing the side effects which are dose related. According to the studies done so far, there is a 44% reduction in Propofol dose requirement and induction time with 66% concentration of Nitrous oxide inhalation.^{4,5} Though Nitrous oxide has drawbacks like second gas effect, gas filled cavities expansion, nausea and vomiting postoperatively, it reduces propofol requirement. It is unknown, yet, how both medications affect general hemodynamics.

AIMS & OBJECTIVES

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AIMS & OBJECTIVES

Objectives

1. To evaluate the variation in induction dose of propofol on administering inhaled nitrous oxide
2. To evaluate the variation in induction time of propofol on administering inhaled nitrous oxide

REVIEW OF LITERATURE

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REVIEW OF LITERATURE

A medically induced condition of unconsciousness known as general anesthesia causes patients to lose their defensive reflexes and become totally unresponsive to painful, tactile, or spoken stimuli. It is accomplished by administering a variety of anesthetics that, when taken together, result in amnesia, analgesia, muscle relaxation, unconsciousness, and autonomic reflex suppression. To maintain airway patency, a laryngeal mask airway or endotracheal tube is often required due to the risk of upper airway obstruction.⁶

Historically, before the development of systematic monitoring techniques, determining the depth of anesthesia relied solely on physical examination, leading to frequent anesthetic overdoses, particularly by less experienced anesthetists. It was not until the 20th century that a structured approach to monitoring anesthesia depth emerged. Despite advancements in anesthetic agents and delivery methods that allow for quicker induction and recovery—sometimes bypassing certain stages—Guedel's classification remains a relevant reference in modern anesthesia practice.⁷

Guedel's Classification - Anesthesia Stages

Stage 1 – Disorientation or Analgesia: often beginning in a preoperative anesthesiology holding area, is referred to as the induction stage. Patients receive medication that induces sedation while remaining conscious and conversational. Breathing remains slow and steady. The patient moves from analgesia without amnesia to analgesia with amnesia at the same time during this phase.⁸ The stage concludes when the patient loses consciousness.

Stage 2 – Delirium or Excitement: Characterized by disinhibition, uncontrolled movements, delirium, and the loss of the eyelash reflex, this stage is associated with higher heart rate and blood pressure. Airway reflexes remain intact but become hypersensitive, increasing the risk of laryngospasm if manipulated. Any airway procedures, such as endotracheal tube placement or deep suctioning, should be avoided to prevent complications. Spastic movements, vomiting, and irregular breathing patterns may further endanger the airway. To minimize time spent in this phase, fast-acting anesthetic agents are often used to facilitate a smoother transition to the next stage.⁹

Stage 3 – Surgical Anesthesia: This level of anesthesia is the goal for most surgical procedures, marked by the absence of eye movements and significant respiratory depression, making airway manipulation safe. Surgical anesthesia is further classified into four planes.^{9,10}

Stage 4 – Overdose: This dangerous phase results from excessive anesthetic administration relative to surgical stimulation, leading to profound brain and medullary suppression. It begins with respiratory cessation and, if not managed promptly, can result in death. Due to cardiac depression and severe vasodilation, patients at this stage exhibit flaccid skeletal muscles, locked and dilated pupils, and dangerously low blood pressure with weak, thready pulses. This stage poses a serious risk to life if cardiovascular and respiratory measures are not taken quickly. The primary goal of the anesthetist is to quickly induce Stage 3 anesthesia in the patient and maintain that level of anesthesia during the procedure.^{9,11}

The General anesthesia can trigger physiological responses that, if not managed properly, may lead to morbidity or even mortality. As a high-risk medical intervention, its benefits must outweigh potential harms.¹² Although deaths from anesthesia are rare, they can

nonetheless happen as a result of complications such as severe allergic responses, airway blockage, or pulmonary aspiration of stomach contents. The primary cause is still human mistake, even though equipment failure might play a role. However, because to better safety procedures, better monitoring and detection tools, and the adoption of standardized norms, the death rate from anesthesia has significantly decreased over the last 20 years. Patient safety results have improved and errors have been drastically decreased as a result of these quality initiatives^{13,14}.

NITROUS OXIDE

A colorless, odorless, non-flammable gas that can support combustion similarly to oxygen. Commonly known as laughing gas due to its euphoric effects, it is the least potent inhalational anesthetic. Nitrous oxide is usually paired with a more powerful volatile agent because it cannot be employed as a stand-alone anesthetic due to its minimum alveolar concentration (MAC) of 104percent. It contributes to the second gas effect, which increases the concentration of other volatile anesthetics in the lungs, because of its rapid onset and short offset caused by its limited blood solubility (blood-gas partition coefficient of 0.47).²

Nitrous Oxide finds extensive application in dental treatments, general anesthesia, procedural sedation, and pain management, especially in emergency and obstetric contexts. In these situations, it is frequently given as a 50percent oxygen mixture. =Overall minute ventilation stays constant even as the tidal volume drops and the respiratory rate increases. Although it has the potential to directly cause cardiac depression, sympathetic stimulation balances this effect, thus the overall effect is minimal.^{15,16}

Mechanism of action

Nitrous oxide exerts its effects through multiple targets in the CNS, acting as a non-competitive NMDA receptor inhibitor to produce anesthesia. Mediated through GABA-A activation.³

According to pharmacokinetics, nitrous oxide enters the body quickly through the alveoli and starts working in two to five minutes.^{17,18} Nitrous oxide's fast diffusion across alveolar membranes causes the second-gas effect, which concentrates residual gases and improves anesthetic absorption. Nitrous oxide is a mild anesthetic with a Minimum Alveolar Concentration (MAC) of 105percent, however it effectively relieves pain. Rapid diffusion back into the alveoli after anesthesia might lower oxygen levels and possibly result in diffusion hypoxia.¹⁹ Nitrous oxide undergoes minimal metabolism, with only trace amounts reduced by anaerobic gut bacteria. Lung is primary route of, where it is exhaled unchanged.

Administration

It is delivered through inhalation using a laryngeal mask airway, face mask, or endotracheal tube.^{19,20}

It is typically administered in concentrations of 30percent to 50percent mixed with oxygen while sedating for surgical or dental procedures. When used for general anesthesia, nitrous oxide is provided at 50percent to 70percent concentration, but due to its low potency, it must be combined with more potent anesthetic agents. Specially designed equipment is required to ensure a 50:50 nitrous oxide-to-oxygen ratio. Unlike

dental delivery systems, devices designed for obstetric use do not allow for manual adjustments of gas proportions.²¹

Induction

Nitrous oxide is one of the quickest anesthetics accessible because of its quick start of action and limited solubility in both blood and tissues. Its absorption in the lungs improves the absorption of oxygen and anesthetics that are breathed simultaneously, resulting in a faster induction and better arterial oxygenation.

Emergence

It facilitates rapid emergence due to its short elimination half-life. Its low lipid solubility ensures a swift washout from the brain, leading to quick postoperative recovery.²⁰

Use in specific conditions;

Renal and Hepatic Impairment: There are no specific manufacturer recommendations regarding dosage adjustments.

Pregnancy Considerations: due to the risk of maternal sedation and hypoxemia, ACOG and the ASA advise against combining nitrous oxide with systemic opioids, sedatives, or hypnotics.²²

Breastfeeding Considerations: Since nitrous oxide has a short half-life in the maternal system, it is unlikely to be transferred to the infant in significant amounts. Breastfeeding can be safely resumed once the mother has fully recovered from anesthesia.²³

Adverse effect

Respiratory Depression: Nitrous oxide alone has minimal impact on respiratory function. However, when combined with sedatives, hypnotics, or opioids, it can amplify their respiratory depressant effects, increasing the risk of hypoventilation or apnea.

Diffusion Hypoxia: After discontinuing, the rapid reversal of the gas concentration gradient in the lungs leads to oxygen dilution in the alveoli, which can cause hypoxia. To prevent this, 100percent oxygen should be administered immediately following nitrous oxide cessation.

Postoperative Nausea and Vomiting (PONV): ENIGMA II study discovered that operations longer than two hours are associated with a higher prevalence of severe PONV. Nitrous oxide has not, however, been connected to increased mortality, cardiovascular problems, or surgical wound infections. PONV can be managed with the use of preventative antiemetic drugs.^{24,25}

Hyperhomocysteinemia²⁶

Subacute Myeloneuropathy: Subacute myeloneuropathy is a serious but perhaps treatable neurological condition that can be brought on by prolonged or excessive usage. This condition presents as an axonal sensorimotor neuropathy, leading to symptoms such as limb weakness, sensory deficits, and impaired coordination.²⁷

Contraindications for Nitrous Oxide Use

While many contraindications to nitrous oxide are relative and depend on the provider's judgment, several conditions warrant caution or avoidance.

Patients who are Critically Ill

cardiovascular Disease

Pregnancy (First Trimester)

Conditions Involving Closed Gas Spaces: Pneumothorax

- Small bowel obstruction
- Retinal surgeries involving intraocular gas bubbles
- Middle ear surgery

Psychiatric Disorders

Impaired Consciousness

Pulmonary Hypertension²⁸

Head and Neck Procedures Involving Cautery Use²⁹

PROPOFOL³⁰

Propofol is an IV anesthetic that can be used for procedural sedation, general anesthesia induction, or anesthesia therapy monitoring. A bolus, an infusion, or a mix of the two are the routes of administration. The lipid emulsion in which propofol is manufactured gives it its distinctive milky white look and the nickname "milk of amnesia."³¹⁻³³

Mechanism of action³⁰

Propofol's precise mechanism of action is unknown, like that of other general anesthetics, although it is believed to be connected to its effects on GABA-mediated chloride

channels in the brain. Propofol may increase GABA's inhibitory effects by reducing its dissociation from the brain's GABA receptors.³⁴

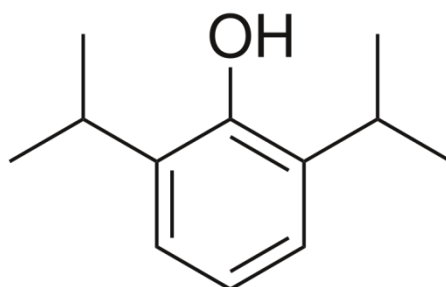


Figure 1: Structure of propofol

Adverse effect

- The most frequent side effect is temporary local injection site pain, that can be reduced by administering intravenous lidocaine prior to the propofol bolus.
- Hypotension is a common hemodynamic response.
- Myoclonic movements may occur.
- In rare cases, propofol has been associated with electrocardiographic changes, such as QT interval prolongation, though these are typically not clinically significant.
- Exceptionally rare cases of green-tinted urine discoloration have also been reported.^{35–37}

Contraindications

Patients having a history of medication hypersensitivity reactions should not use it. Any patient with excessively low blood pressure has to be treated with caution.³⁸

Various articles;

In a research by Kumar AA et al. (2006), the impact of priming on the propofol induction dosage needed was evaluated. Group II saw a 27.48percent reduction in the induction dosage needed for propofol, with an average reduction of 29.72 mg. Stable hemodynamics were maintained at the time of intubation and the required induction dosage was significantly reduced by 27.48percent by applying the Priming Principle during propofol induction. However, this approach was linked to a higher incidence of post-suxamethonium fasciculations.³⁹

42.5percent of patients in the PN group were induced in less than 100 seconds, 57.5percent in less than 200 seconds, and no patient needed more than 200 seconds, according to a research by Singh S et al. (2014). In contrast, only 22.5percent of patients in the PO group were induced within 200 seconds, none were induced in less than 100 seconds, and 77.5percent of patients needed more than 200 seconds. These results imply that while maintaining stable hemodynamics, co-administration of nitrous oxide during induction considerably shortens the induction period and propofol dosage.⁴⁰

According to a research by Sunil R et al. (2016), Group A needed a substantially lower dosage of propofol for induction than Group. The induction time of Group B was 5.09 ± 1.33 minutes, while that of Group A was 1.52 ± 1.31 minutes. The groups' heart rates were consistent throughout the study. Crucially, there were no instances of desaturation ($\text{SpO}_2 < 90\text{percent}$) found. Three minutes of 66percent nitrous oxide inhalation greatly decreased the amount of time needed for induction, decreased the amount of propofol needed, and preserved MAP stability during induction. Furthermore, it prevented oxygen desaturation while reducing the stress reaction to laryngoscopy and intubation.⁴¹

In a prospective randomized research, Jain K et al. (2017) discovered that Group FN had a considerably reduced induction time and dosage than Group FO. In particular, the average induction time in Group FO was significantly greater at 242 ± 43 seconds ($P < 0.001$) than in Group FN, which was 172 ± 32 seconds. Both the necessary induction dose and the duration to induction were successfully reduced when nitrous oxide (N_2O) was administered in addition to propofol during the induction of anesthesia. Additionally, it helped to keep hemodynamics steady with no known side effects.⁵

In Biyani C et al., (2022) study to evaluate the effect of nitrous oxide on consumption of propofol. It is noteworthy that none of the patients in Group D needed extra analgesics or suffered from nausea or vomiting. Both dexmedetomidine and nitrous oxide successfully decreased the overall amount of propofol used, making them suitable anesthetic adjuvants to lessen adverse effects associated with propofol. Dexmedetomidine could be a better and safer substitute for nitrous oxide because it doesn't have any negative long-term or environmental impacts.⁴²

In Jain N et al., (2022) study included 50 patients, with group A, 76percent of patients achieved induction in under 200 seconds, while only 24percent required more than 200 seconds. In contrast, Group B had a significantly lower proportion of patients (32percent) induced within 200 seconds, with 68percent requiring a longer induction time. Group A's mean induction time (183 seconds) was much less than Group B's (256 seconds). These results show that the induction dose and induction duration were dramatically decreased when nitrous oxide (N_2O) and propofol were administered together.⁴³

MATERIALS &

METHODS

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MATERIAL & METHOD

This study was conducted on 96 patients undergoing general anaesthesia with 48 patients in each group at “R.L.Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar during the period from April 2023 to May 2025.”

Study Design: Double blinded randomized control trial.

Sample Size: 48 in each group.

Duration of study: From May 2023 to October 2024.

Sampling Method: Computerized random sampling.

Sample size estimation:

The maximum sample size of 48 in each group can be taken for the present study which will satisfy both for induction time and induction dose.^{5,44}

Formula :

$$n = \frac{2 \times (z_{1-\alpha/2} - z_{1-\beta})^2 \times \sigma^2}{d^2}$$

Where n = minimum required sample size

$z_{1-\alpha/2}$ = The critical value (Table value) from a standard normal distribution that the test statistic must exceed in order to show a statistically significant result at ' α ' level of significance.

$z_{1-\beta}$ = Standard normal table value for the power of the test ($1 - \beta$)

σ = Standard deviation of the response variable (obtained from previous study)

d = the effect size = the minimum clinically important difference that the investigator wishes to detect.

In the present case, $z_{1-\alpha/2} = 2.58$ at 1 % level of significance

$z_{1-\beta} = 1.28$ at 90 % power

Inclusion criteria

- Age 18 - 60 years.
- Patients proposed for elective surgical procedures under general anaesthesia.
- ASA 1 and 2

Exclusion criteria

- Pregnant women.
- Patients with allergy to anesthetic agents.
- Morbidly obese patients.
- Patients with difficult airway.
- Patients with contraindications to the utilisation of N₂O like increased intracranial pressure, obstruction of the bowel, pneumothorax, surgeries of the middle ear.

Method of collection of data:

- Patients undergoing general anaesthesia was randomly selected.
- Patients provided Informed consent.

-
- Result were recorded in proforma.

SAMPLING PROCEDURE

- History in detail was taken.
- Complete physical examination was done, followed by routine investigation.
- IV line was secured for fluids.
- General anesthesia procedure was clearly explained, consent was taken.
- Patients were asked to be on NPO overnight and Tab. Anxit 0.5mg was given on the night before the surgery to decrease anxiety.
- In the operation theatre, multipara monitors was connected to the patient and baseline heart rate, SBP, DBP, MAP and SpO2 were noted.
- Glycopyrrolate 0.2mg, Fentanyl 2mcg/kg was given as premedication.
- All patients were provided with 3 min of preoxygenation with 100% oxygen.
- Patients were divided into two groups randomly.

Group A: received a mixture of 70% N2O and 30% oxygen via Bains Circuit with 30mg/min(3ml/min) infusion of propofol.

Group B: received a mixture of 100% oxygen via Bains Circuit with 30mg/min(3ml/min) infusion of propofol.

“In both groups A and B, propofol infusion was initiated at a rate of 30 mg/min (equivalent to 3 ml/min). The infusion was discontinued once apnea was achieved. Induction time was defined as the interval between the start of propofol administration

and the onset of apnea, while the induction dose referred to the total amount of drug given during this period. Following this, patients received succinylcholine at a dose of 2 mg/kg and were intubated with a suitably sized cuffed endotracheal tube. Anesthesia was then maintained using oxygen, nitrous oxide, and other inhalational agents.”

“The various hemodynamic parameters like HR, SBP, DBP & MAP were monitored and recorded at predefined time intervals: preoperatively (T1); immediately before initiating propofol infusion (T2); 1 minute (T3), 2 minutes (T4), 3 minutes (T5), and 4 minutes (T6) after starting the infusion; at the onset of apnea (T7); just before intubation (T8); and immediately following intubation (T9).”

PARAMETERS TO BE OBSERVED

“Heart rate, Mean Arterial Pressure, Systolic Blood Pressure, Diastolic Blood Pressure and SpO₂ were monitored”

- Preoperatively - T1
- Before the start of propofol infusion - T2
- 1 minutes after the start of infusion - T3
- 2 minutes after the start of infusion - T4
- 3 minutes after the start of infusion - T5
- 4 minutes after the start of infusion - T6
- Apneic state - T7
- Before intubation - T8
- After intubation - T9

STATISTICAL ANALYSIS

The Collected data were coded and entered into an excel data base. All the quantitative measures were presented by (Mean+/-SD), Confidence interval (CI), qualitative measures like, ASA Physical status etc....by proportions and CI. Independent sample t-test, Chi -square test were used to interpret the results. Data were analysed using SPSS v26.0 with significance value of $p < 0.05$ as statistically significant.

RESULTS



RESULTS

Present study included total of 96 patients, with 48 cases in group A and 48 cases in group B.

Table 1: Showing mean age between the groups

	Group A		Group B		p-value
	Mean.	SD	Mean.	SD	
Age in yrs	39.9	12.6	39.3	11.9	0.83

The mean age between the groups were comparable with no significant difference noted.

The mean age was 39.9yrs in group A and 39.3yrs in group B patients,

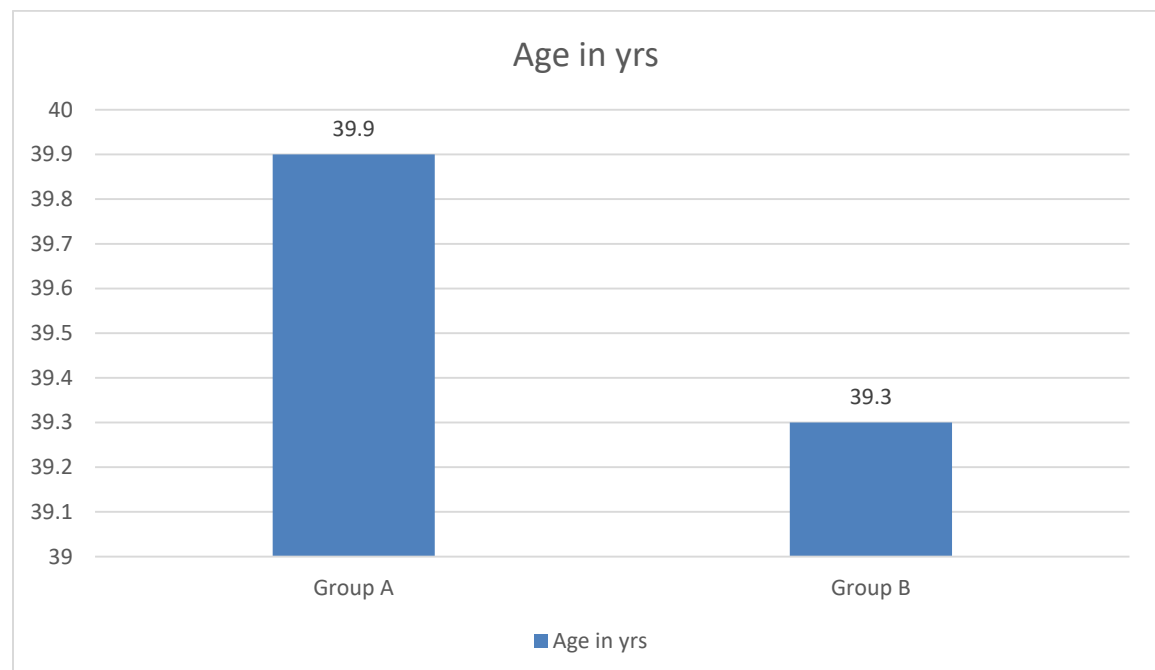


Figure 2: Showing mean age between the groups

Table 2: Gender distribution between the groups

		Group A		Group B		Chi-square (p-value)
		N	N %	N	N %	
Gender	Female	29	60.4%	25	52.1%	1.21 (0.21)
	Male	19	39.6%	23	47.9%	

Gender distribution is comparable with marginal female preponderance in both the groups.

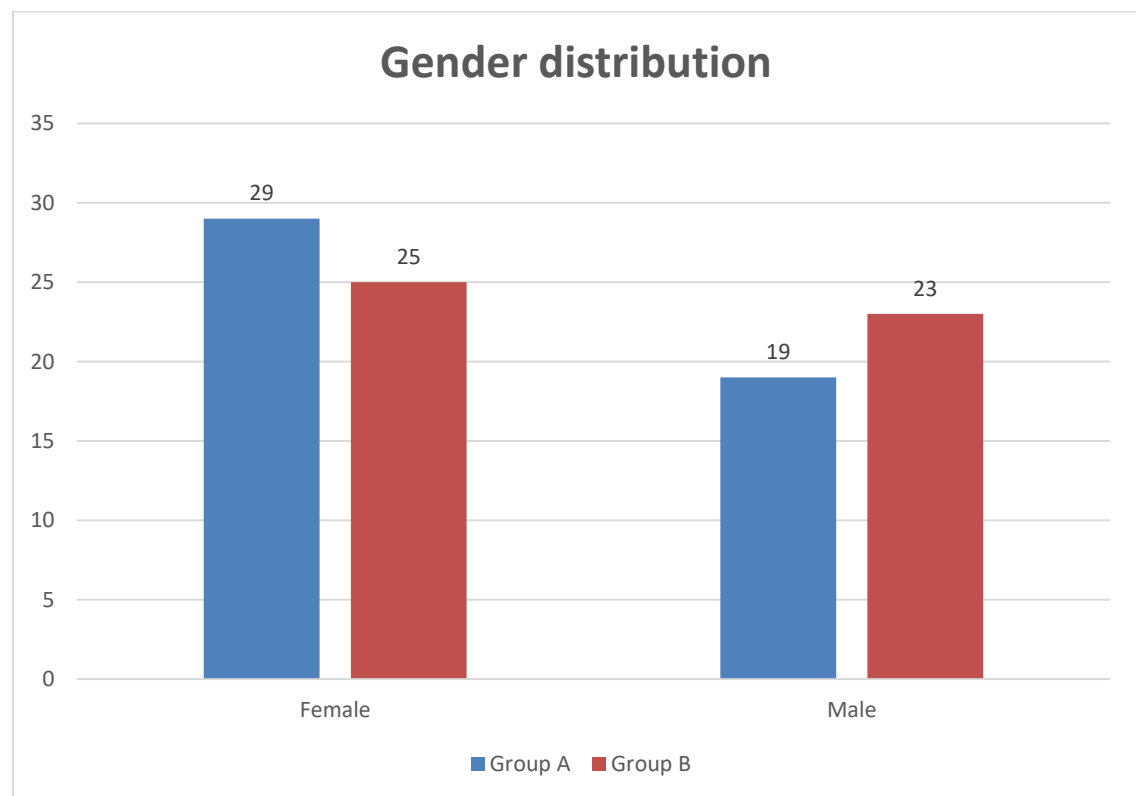


Figure 3: Gender distribution between the groups

Table 3: Distribution according to ASA grade

		Group A		Group B		Chi-square (p-value)
		N	N %	N	N %	
ASA Grade	1	31	64.6%	32	66.7%	0.52 (0.85)
	2	17	35.4%	16	33.3%	

The ASA grade distribution was found to be comparable between the groups, with majority of patients in ASA grade 1. ($p>0.05$)

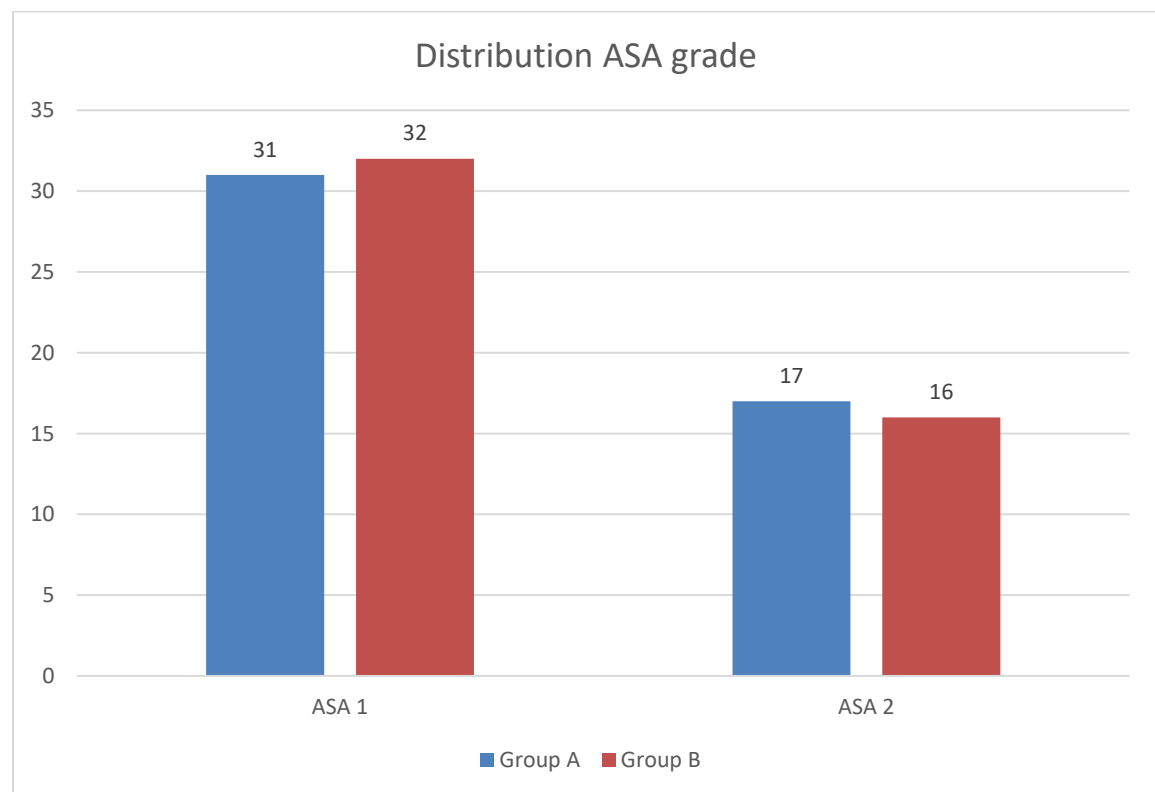


Figure 4: Distribution according to ASA grade

Table 4: Showing induction time between the groups

		Group A		Group B		Chi-square (p-value)
		N	N %	N	N %	
Induction time	<200sec	41	85.4%	6	12.5%	13.347 (0.05)*
	>200sec	7	14.6%	42	87.5%	

There is significant higher incidence of shortest induction time in group A compared to group B. 85.4% of the cases with induction time <200sec and 12.5 in group B. ($p < 0.05$).

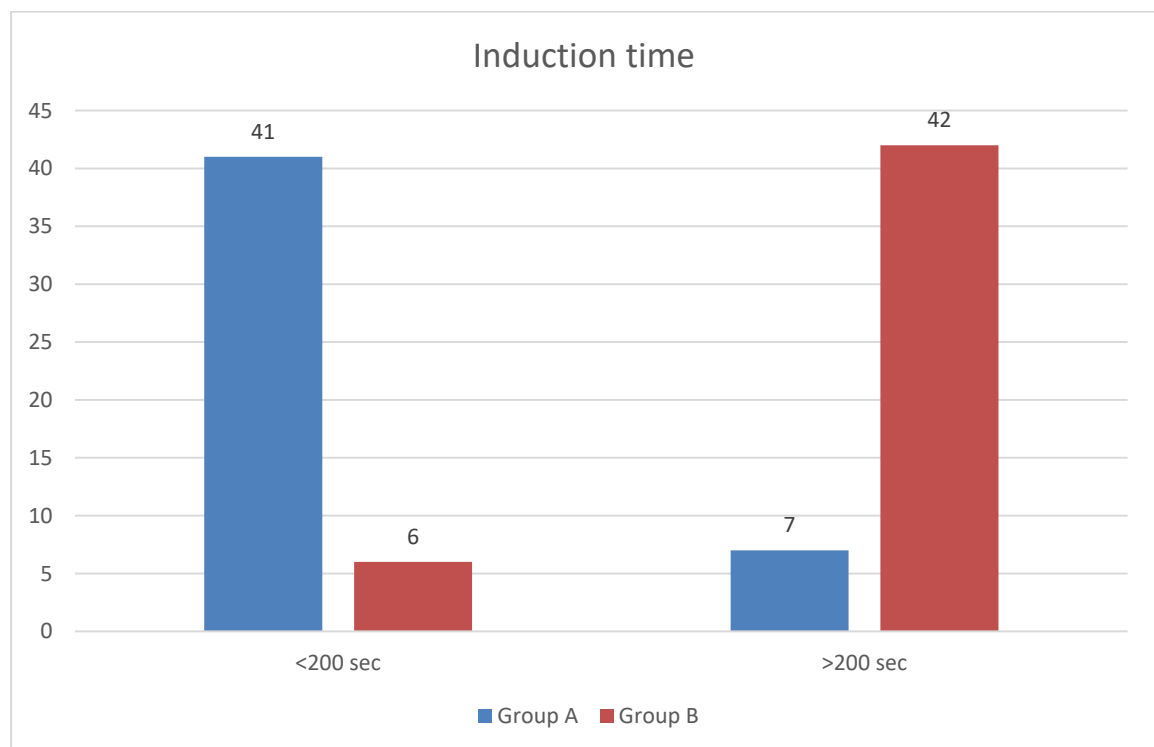


Figure 5: Showing induction time between the groups

Table 5: Showing the induction dose between the groups

		Group A		Group B		Chi-square (p-value)
		N	N %	N	N %	
Induction dose (mg/kg)	>1.5	0	0.0%	37	77.1%	73.11 (0.01)*
	0.5-1.0	36	75.0%	0	0.0%	
	1.0-1.5	12	25.0%	11	22.9%	

There is significant lower dose of induction seen in group A patients compared to group B. majority of patients in group A needed induction dose of 0.5-1mg/kg in 75% of the cases. Also, 25% cases in group A needed 1-1.5,g/kg, compared to group B (22.9%).
(p<0.05)

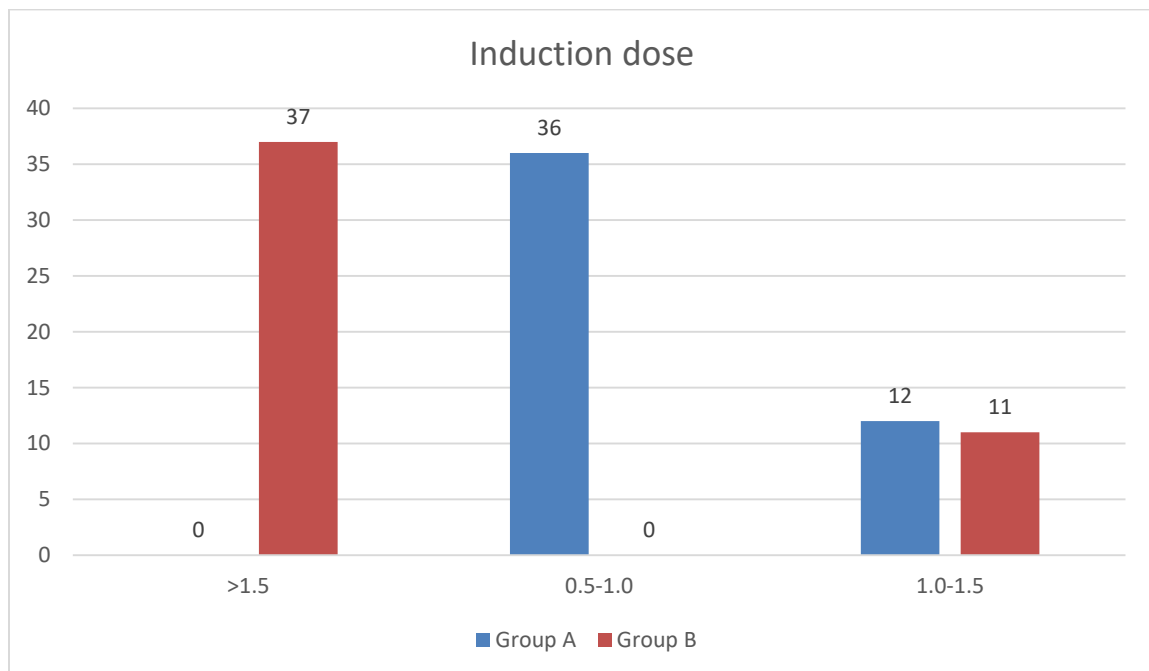


Figure 6: Showing the induction dose between the groups

Table 6: Comparison of systolic blood pressure between the groups

Systolic blood pressure	Group A		Group B		p-value
	Mean	SD	Mean	SD	
T1	123.5	8.0	124.8	11.3	0.52
T2	123.6	7.6	126.1	12.4	0.25
T3	122.3	8.0	125.5	13.1	0.14
T4	119.9	7.7	124.1	12.6	0.051
T5	118.3	8.3	122.7	12.3	0.04*
T6	116.3	8.1	121.2	12.7	0.02*
T7	116.4	7.8	120.0	12.7	0.09
T8	115.7	8.0	119.3	12.3	0.120
T9	118.3	8.1	124.9	11.4	0.02*

There is significant higher mean systolic blood pressure at T5, T6 and T9 in group B compared to group A. overall the group B systolic blood pressure was higher compared to patients in group A.

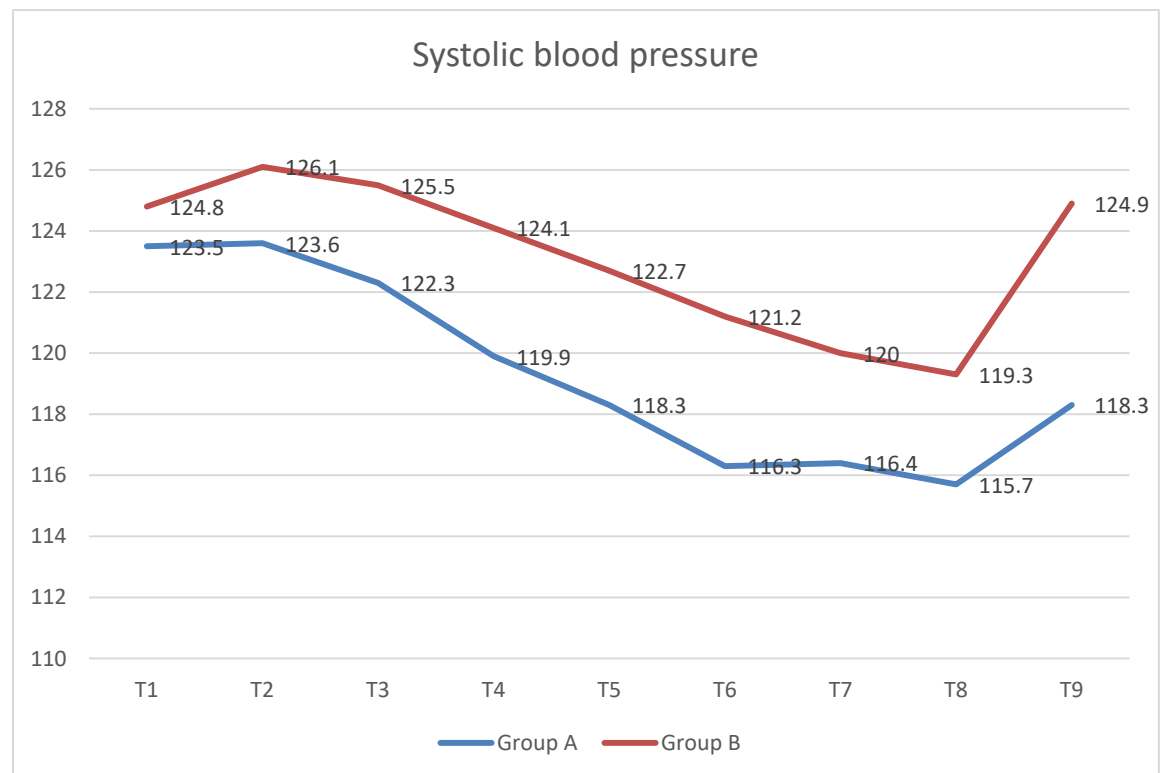


Figure 7: Comparison of systolic blood pressure between the groups

Table 7: Comparison of diastolic blood pressure between the groups

Diastolic blood pressure	Group A		Group B		p-value
	Mean	SD	Mean	SD	
T1	82.0	6.9	82.9	8.5	0.58
T2	81.2	7.5	83.5	8.2	0.15
T3	80.0	7.4	82.8	8.1	0.08
T4	78.7	7.4	81.8	7.9	0.052
T5	77.1	7.7	80.9	8.3	0.02*
T6	76.0	7.5	79.1	8.7	0.06
T7	76.0	7.8	78.3	8.4	0.159
T8	76.1	8.0	77.7	8.3	0.339
T9	78.4	8.3	82.4	8.4	0.02*

There is significant higher mean diastolic blood pressure at T5, and T9 in group B compared to group A. overall the group B diastolic blood pressure was higher compared to patients in group A.

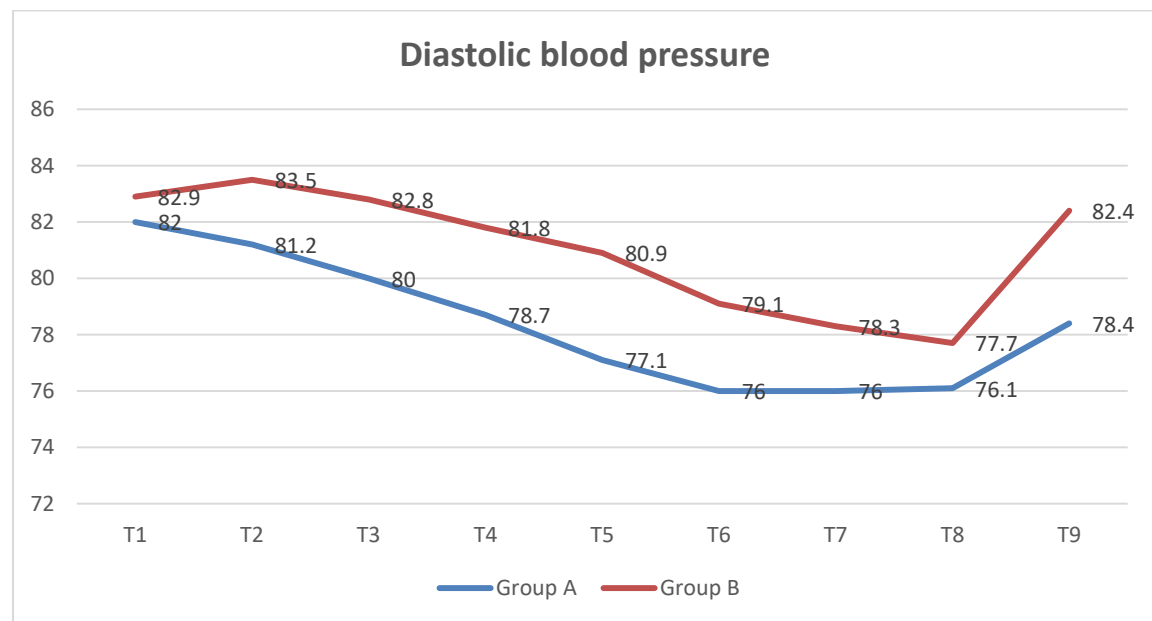


Figure 8: Comparison of diastolic blood pressure between the groups

Table 8: Comparison of mean arterial pressure between the groups

Mean arterial pressure	Group A		Group B		p-value
	Mean	SD	Mean	SD	
T1	95.9	6.4	96.9	8.2	0.515
T2	95.4	6.5	97.8	8.3	0.122
T3	94.1	6.6	96.9	8.1	0.07
T4	92.4	6.7	95.9	7.7	0.02*
T5	90.8	7.1	94.8	8.1	0.01*
T6	89.4	6.8	93.7	8.2	0.01*
T7	89.5	7.0	92.6	8.1	0.04*
T8	89.3	7.3	92.0	8.0	0.079
T9	91.5	7.3	95.1	8.1	0.02*

There is significant higher mean arterial pressure at T4, T5, T6, T7 and T9 in group B compared to group A. Overall the group B mean arterial pressure was higher compared to patients in group A.

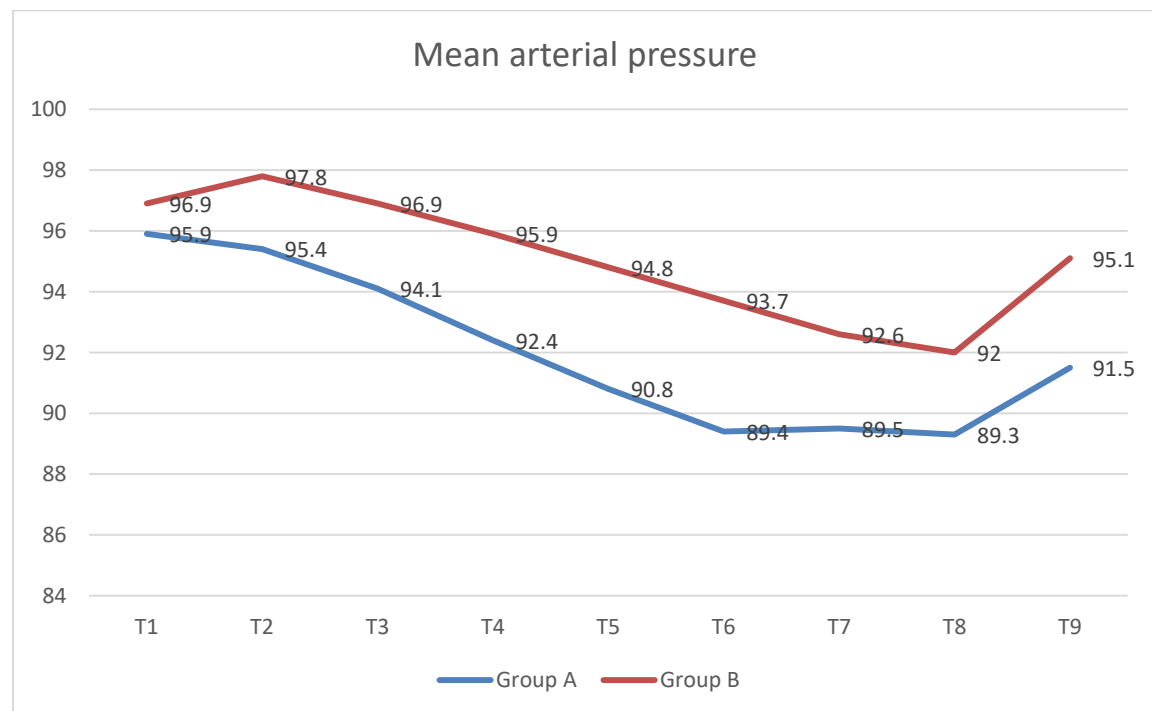


Figure 9: Comparison of mean arterial pressure between the groups

Table 9: Comparison of mean Heart Rate between the groups

Heart rate	Group A		Group B		p-value
	Mean	SD	Mean	SD	
T1	85.9	9.4	86.5	8.2	0.74
T2	87.2	9.9	87.6	8.9	0.81
T3	86.0	10.3	88.0	8.8	0.31
T4	85.3	9.3	87.1	8.6	0.38
T5	84.0	8.9	85.6	8.4	0.39
T6	83.3	8.5	84.3	8.3	0.57
T7	82.7	8.3	83.1	8.2	0.80
T8	82.9	8.9	82.6	7.7	0.86
T9	87.4	8.8	89.2	8.0	0.30

There is no significant difference in mean heart rate between the groups at different time interval. However, overall the mean heart rate was higher in group B patients compared to group A patients.($p>0.05$)

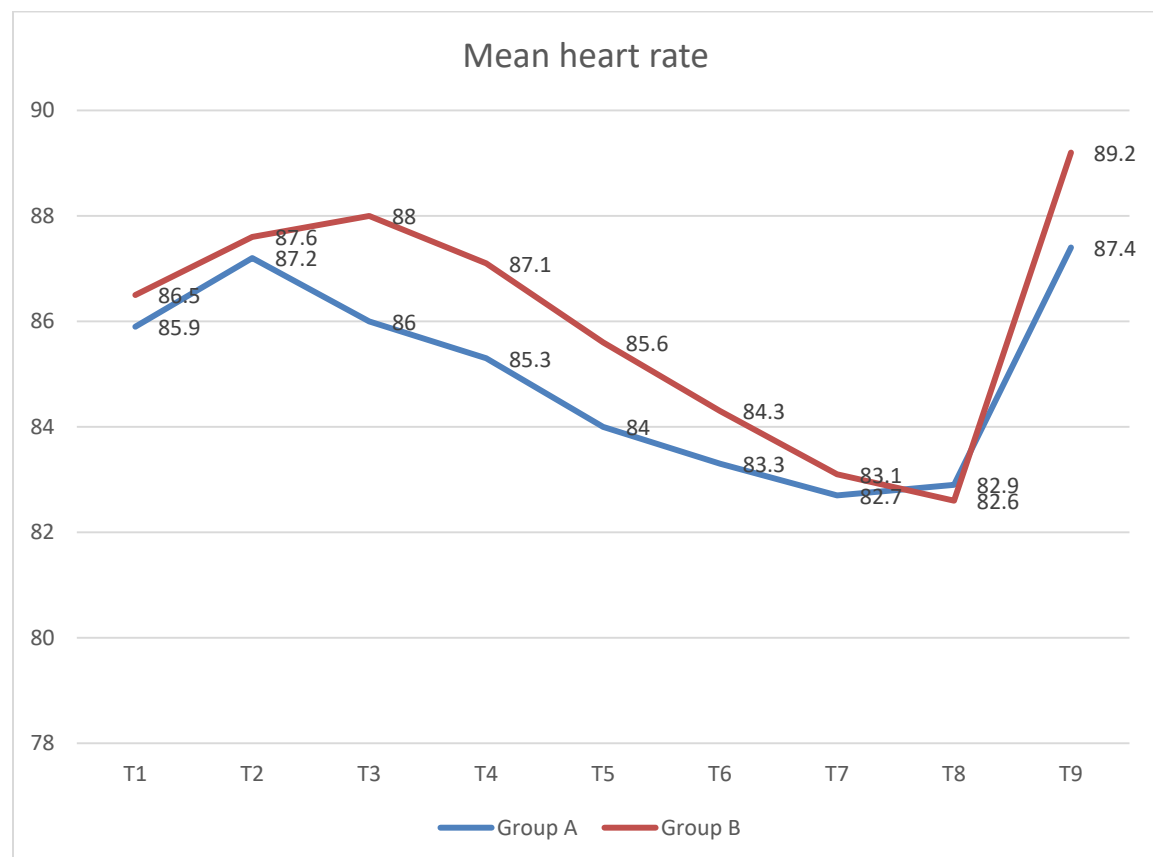


Figure 10: Comparison of Mean Heart Rate between the groups

DISCUSSION



DISCUSSION

Propofol's fast onset, easy induction, and quick recovery profile have made it the most popular drug for intravenous induction of general anesthesia in recent years. It is a highly effective hypnotic agent that offers excellent intubating conditions, amnesic effects, and suppression of airway reflexes, particularly in patients with hyperreactive airways. It is a recommended option for induction in a variety of therapeutic situations because of these qualities. Propofol usage is not without its problems, though. Adverse symptoms include bradycardia, apnea, respiratory depression, hypotension, injection discomfort, and hypertriglyceridemia are commonly linked to it. These side effects are often dose-dependent and can pose significant challenges, especially in hemodynamically unstable patients.

On the other hand, N₂O is a commonly used inhalational agent known for its analgesic and anxiolytic properties. Although it is a weak anesthetic on its own, nitrous oxide is often used as an adjuvant to reduce the dosage requirement of more potent anesthetic agents like propofol. Its use as a gaseous vehicle for volatile anesthetics and its potential to decrease the need for intravenous agents have been well documented. According to studies, giving 66% nitrous oxide before propofol induction can cut the amount of propofol needed by up to 44% and minimize the induction period, which helps to mitigate many of the negative effects of propofol's dosage.

Despite the benefits, nitrous oxide has some limitations, including the potential for gas expansion in closed cavities, the second gas effect, and an increased risk of postoperative nausea and vomiting. Nonetheless, the combined use of nitrous oxide and propofol offers a promising approach to balancing efficacy and safety in anesthesia induction. The

present study aims to further evaluate the impact of this combination on induction characteristics and overall hemodynamic stability.

96 patients in all, 48 of whom were in group A and 48 of whom were in group B. “There was no discernible difference in the mean age across the groups. There is a little female preponderance in both groups, but there is no discernible difference in the gender distribution. The mean age of the patients in groups A and B was 39.9 and 39.3 years, respectively. The majority of patients were in ASA grade 1, and the distribution of ASA grades was found to be similar across the groups.” ($p>0.05$)

In a related research by Jain K et al., demographic traits including age, gender, and weight did not significantly change across the groups. In a similar vein, the groups' distribution of surgical operation types and ASA physical status classifications was similar.⁵ Participants in the Singh S et al. research were between the ages of 18 and 60 in both the PN and PO groups. The PN and PO groups had mean ages of 37.05 ± 10.70 and 37.90 ± 11.35 years, respectively. The proportion of participants by gender was very similar, with 55% of the participants in both groups being female and 45% being male. Additionally, both groups' ASA physical status grade distributions were comparable, suggesting that the patients' initial clinical statuses were well matched.⁴⁰

Compared to group B, group A had a noticeably greater incidence of the lowest induction time. In group B, induction times were 12.5 and less than 200 seconds in 85.4% of instances. ($p<0.05$) Additionally, group A patients had a noticeably lower induction dosage than group B patients. In 75% of instances, the majority of patients in group A required an induction dosage of 0.5–1 mg/kg. Additionally, compared to group B (22.9%), 25% of patients in group A required 1-1.5 g/kg. ($p<0.05$)

In study by Kumar AA et al., the Group II, the induction dose requirement for propofol was reduced by an average of 29.72 mg, representing a 27.48% decrease.³⁹ In study by Singh S et al., the PN group, 42.5% of patients achieved induction in less than 100 seconds, while 57.5% were induced within 200 seconds, with no patients requiring more than 200 seconds. Conversely, in the PO group, 77.5% of patients required more than 200 seconds, while only 22.5% were induced within 200 seconds, and none achieved induction in under 100 seconds. The PN group, which received 67% nitrous oxide in oxygen, had a significantly shorter induction time (113.38 seconds) compared to the PO group (258.00 seconds). Additionally, patients in the PN group required a lower induction dose of propofol (average 0.58 mg/kg) than those in the PO group (average 1.43 mg/kg), indicating that nitrous oxide effectively reduced both the time and dose needed for induction.⁴⁰

In similar study by Sunil R et al., the required propofol dose for induction was significantly lower in Group A compared to Group B (30.4 ± 26.17 mg vs. 101.87 ± 26.19 mg). Similarly, induction time was notably shorter in Group A (1.52 ± 1.31 min) than in Group B (5.09 ± 1.33 min).⁴¹ Also in study y Jain K et al., found that the mean induction time in Group FN was 172 ± 32 seconds, whereas it was notably longer in Group FO at 242 ± 43 seconds ($P < 0.001$). Additionally, the mean induction dose of propofol was reduced in Group FN, averaging 56.10 ± 13.92 mg, compared to 81.67 ± 17.64 mg in Group FO ($P < 0.05$).⁵

Group P (139.02 ± 65.24 µg) consumed considerably more propofol overall than Group N (94.72 ± 48.04 µg) and Group D (98.31 ± 39.45 µg) in the research by Biyani C et al. The mean differences were 44.3 µg between Groups P and N, 40.71 µg between Groups P and

D, and 3.59 µg between Groups N and D. The differences between Group P and the other two groups were statistically significant ($P = 0.015$).⁴²

In Jain N et al., study found that Group A, 76% of patients achieved induction in under 200 seconds, while only 24% required more than 200 seconds. On the other hand, 68% of patients in Group B needed a longer induction period, and only 32% of patients were induced in less than 200 seconds. Group A's mean induction time (183 seconds) was much less than Group B's (256 seconds). These results show that the induction dose and induction duration were dramatically decreased when nitrous oxide (N₂O) and propofol were administered together.⁴³

At T5, T6, and T9, group B's mean systolic blood pressure is noticeably higher than group A's. In general, patients in group B had greater systolic blood pressure than those in group A. There is significant higher mean diastolic blood pressure at T5, and T9 in group B compared to group A. overall the group B diastolic blood pressure was higher compared to patients in group A. Overall the group B mean arterial pressure was higher compared to patients in group A. The groups' mean heart rates at various time intervals do not differ significantly. Overall, nevertheless, group B patients had a greater mean heart rate than group A patients. ($p > 0.05$)

One minute after induction, the control group (Group I) in the study by Kumar AA et al. showed a higher mean heart rate. The systolic, diastolic, and mean blood pressures of Group II were significantly higher at one minute after induction, immediately following intubation, and five minutes after induction. Furthermore, fasciculations occurred in 16% of Group II patients while none were seen in Group I. Propofol induction using the Priming Principle successfully reduces the induction dosage by 27.48% while preserving

stable hemodynamics during peri-intubation. Nevertheless, there is a higher risk of post-suxamethonium fasciculations with this method.³⁹

The heart rate rose by 16.38% in the PN group and 6.42% in the PO group in the Singh S et al. research. These results imply that while maintaining stable hemodynamics, co-administration of nitrous oxide during induction considerably shortens the induction period and propofol dosage.⁴⁰ In a related study, Sunil R. et al. reported Throughout the trial, the heart rates of the groups were similar. At induction, however, Group A's mean arterial pressure (MAP) was much higher (94.51 ± 16.21 vs. 86.57 ± 15.47). Group B had considerably higher MAP at 5 and 10 minutes, but Group A had marginally greater oxygen saturation at induction (99.81 ± 0.46 vs. 99.96 ± 0.26). Crucially, there were no instances of desaturation ($SpO_2 < 90\%$) found. Three minutes of 66% nitrous oxide inhalation greatly decreased the amount of time needed for induction, decreased the amount of propofol needed, and preserved MAP stability during induction. Furthermore, it prevented oxygen desaturation while reducing the stress reaction to laryngoscopy and intubation.⁴¹

Hemodynamic parameters were constant during the treatment, and no problems were noted in a related research by Jain K et al. Both the necessary induction dose and the duration to induction were successfully reduced when nitrous oxide (N_2O) was administered in addition to propofol during the induction of anesthesia. Additionally, it helped to keep hemodynamics steady with no known side effects.⁵

LIMITATIONS

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LIMITATIONS OF THE STUDY

The study are with some limitations, which include first being a single centre study, which may limits its generalizability of the findings to other institutions or broader population. Secondly, the smaller sample size of 48 in each group, however it was statistically estimated and appropriate for the study objectives, still it may be insufficient to detect the rare adverse events or allow for robust subgroup analysis. The short-term observation as study mainly focused on induction parameters and immediate hemodynamic responses. To strengthen the present study findings, a larger sample size study conducted at different health centres is needed to generalise the findings to a larger population.

SUMMARY

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SUMMARY

- There were 96 patients in the current study, 48 in both the groups.
- There was no discernible difference in the mean age across the groups. • There is a little female preponderance in both groups, but there is no discernible difference in the gender distribution. The mean age of the patients in groups A and B was 39.9 and 39.3 years, respectively.
- It was discovered that the groups' ASA grade distributions were similar, with the majority of patients falling into ASA grade 1. ($p>0.05$)
- Compared to group B, group A had a noticeably greater incidence of the lowest induction time. In group B, induction times were 12.5 and less than 200 seconds in 85.4percent of instances. ($p<0.05$)
- Patients in group A received a noticeably lower induction dosage than those in group B. In 75percent of instances, the majority of patients in group A required an induction dosage of 0.5–1 mg/kg. Additionally, compared to group B (22.9percent), 25percent of patients in group A required 1-1.5 g/kg. ($p<0.05$)
- Group B's mean SBP is noticeably greater than group A's at T5, T6, and T9. In general, patients in group B had greater systolic blood pressure than those in group A. Group B's mean DBP is significantly greater than group A's at T5 and T9. In general, patients in group B had greater diastolic blood pressure than those in group A.
- At T4, T5, T6, T7, and T9, group B's mean arterial pressure is noticeably greater than group A's. In general, the MAP of patients in group B was greater than that of patients in group A.

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- The groups' mean heart rates at various time intervals do not differ significantly.

Overall, nevertheless, group B patients had a greater mean heart rate than group A patients. ($p>0.05$)

CONCLUSION

CONCLUSION

In conclusion, the study affirms that the co-administration of inhaled nitrous oxide during induction with propofol is both effective and beneficial. It offers clear advantages in terms of faster induction, reduced drug requirement, and better hemodynamic control. Incorporating nitrous oxide into induction protocols can therefore enhance safety and efficiency in anaesthetic practice, particularly in patients where minimizing anaesthetic dosage and preserving cardiovascular stability are clinical priorities.

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ANNEXURES

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ANNEXURE

PROFORMA

STUDY: VARIATION IN INDUCTION DOSE AND TIME OF PROPOFOL ON ADMINISTERING INHALED NITROUS OXIDE DURING INDUCTION : A PROSPECTIVE, RANDOMISED CONTROLLED STUDY.

Investigators: Dr Dandamudi Siri Chandana / Dr Kiran.N

1. Name of the patient: 2. Age/Sex: 3. Ward:

4. IP No: 5. ASA grade:

• **General physical examination:**

Pulse rate: respiratory rate: BP: Temperature:

Pallor/icterus/cyanosis/clubbing/lymphadenopathy/edema

• **Systemic examination:**

RS -

CVS -

CNS -

P/A -

• **Investigations:**

Blood group: Hb: WBC: Platelets:

RBS: Blood urea: Sr. Creatinine: Sodium:

Potassium:

ECG:

• **Diagnosis:**

• **Surgery:**

INDUCTION TIME	GROUP A	GROUP B
Less than 200 seconds		
More than 200 seconds		

Group A: Will Receive a mixture of 70% N₂O and 30% oxygen via Bains Circuit with 30mg/min infusion of propofol.

Group B: Will Receive a mixture of 100% oxygen with 30mg/min infusion of propofol
.

INDUCTION DOSE	GROUP A	GROUP B
0.5-1.0mg/kg		
1.0-1.5mg/kg		
More than 1.5mg/kg		

HEART RATE	GROUP A	GROUP B
Preoperatively - T1		
Before the start of propofol infusion - T2		
1 minutes after the start of infusion - T3		
2 minutes after the start of infusion - T4		
3 minutes after the start of infusion - T5		
4 minutes after the start of infusion - T6		
Apneic state - T7		
Before intubation - T8		
After intubation - T9		

MEAN ARTERIAL PRESSURE	GROUP A	GROUP B
Preoperatively - T1		
Before the start of propofol infusion - T2		
1 minutes after the start of infusion - T3		
2 minutes after the start of infusion - T4		
3 minutes after the start of infusion - T5		
4 minutes after the start of infusion - T6		
Apneic state - T7		
Before intubation - T8		
After intubation - T9		

SYSTOLIC BLOOD PRESSURE	GROUP A	GROUP B
Preoperatively - T1		
Before the start of propofol infusion - T2		
1 minutes after the start of infusion - T3		
2 minutes after the start of infusion - T4		
3 minutes after the start of infusion - T5		
4 minutes after the start of infusion - T6		
Apneic state - T7		
Before intubation - T8		
After intubation - T9		

DIASTOLIC BLOOD PRESSURE	GROUP A	GROUP B
Preoperatively - T1		
Before the start of propofol infusion - T2		
1 minute after the start of infusion - T3		
2 minutes after the start of infusion - T4		
3 minutes after the start of infusion - T5		
4 minutes after the start of infusion - T6		
Apneic state - T7		
Before intubation - T8		
After intubation - T9		

PATIENT INFORMATION SHEET

Study: VARIATION IN INDUCTION DOSE AND TIME OF PROPOFOL ON ADMINISTERING INHALED NITROUS OXIDE DURING INDUCTION : A PROSPECTIVE, RANDOMISED CONTROLLED STUDY

Investigators: Dr. D. Siri Chandana / Dr Kiran.N

Study location: R.L.Jalappa Hospital and Research Centre attached to Sri Devaraj Medical College, Tamaka, Kolar.

Details -

In this study patients undergoing general anaesthesia will be divided into two groups; Group A will receive a mixture of 67% N₂O and 33% oxygen and Group B will receive a mixture of 100% oxygen. Both the groups will receive a 30mg/min infusion of propofol. The infusion will be stopped on temporary cessation of breathing. The induction dose and time will be calculated consequently. Patients will be given succinylcholine of 2ml/kg and will be intubated using cuffed endotracheal tube of appropriate size and then maintained with O₂, N₂O and other inhalational agents.

This study aims to reduce the disadvantages caused by propofol on the patients when used in higher doses, i.e decreased arterial blood pressure and heart rate. Patient and the patient's attenders will be completely explained about the procedure being done.

Please read the information above and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study, then relevant information and history will be taken. This information collected will be used only for dissertation and publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. All the expenses required for the study will be endured upon by the candidate. There will not be any monetary benefits/incentives for taking part in this study. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

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ರೋಗಿಯ ವಿಷಯ ಸೂಚಿ ಪಟ್ಟಿ

ಅಧ್ಯಯನ: ಮೊದಲು ಪ್ರವೇಶಿಸಲ್ಪಡುವ ಪ್ರಮಾಣದಲ್ಲಿ ಬದಲಾವಣೆ ಮತ್ತು ಮೊದಲ ಸಲ ನೈಟ್ರಸ್ ಆಕ್ಸೈಡ್ ಉಸಿರಳಿತದ ನಿರ್ವಹಣೆಯಲ್ಲಿ ಪ್ರೋಪೋಫಾಲ್ ನ ವ್ಯವಧಿ : ನಿರೀಕ್ಷಿತ ಕ್ರಮರಹಿತ ನಿಯಂತ್ರಣದ ಅಧ್ಯಯನ

ಪರೀಕ್ಷಕರು : ಡಾ. ಡಿ ಸಿರಿಚಂದನ /ಡಾ.ಕಿರಣ್.ಏನ್

ಅಧ್ಯಯನ ಸ್ಥಳ: ಆರ್ ಎಲ್ ಜಾಲಪ್ಪ ಆಸ್ಪತ್ರೆ ಮತ್ತು ಸಂಶೋಧನಾ ಕೇಂದ್ರ ಸಹಯೋಗ ಶ್ರೀ ದೇವರಾಜ ಅರಸ್ ಮೆಡಿಕಲ್ ಕಾಲೇಜ್ ಟಿ. ಮ. ಕೆ. ಕೋಲಾರ

ವಿವರಗಳು: ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಸಾಮಾನ್ಯ ಅನಸ್ಥೆಸಿಯಾಕೊಳ್ಳಪಡುವ ರೋಗಿಗಳ ಎರಡು ಗುಂಪುಗಳನ್ನಾಗಿ ಮಾಡಿ : ಗುಂಪು 'ಎ' ಗೆ ೬೭% ನೈಟ್ರಸ್ ಆಕ್ಸೈಡ್ ಮತ್ತು ೩೩% ಆಮ್ಲ ಜನಕ ಮಿಶ್ರಣವನ್ನು ಗುಂಪು 'ಬಿ' ಗೆ ೧೦೦% ಆಮ್ಲಜನಕ. ಎರಡು ಗುಂಪಿನವರಿಗೂ ಕನಿಷ್ಠ ೩೦ ಎಂ.ಜಿ ಪ್ರೋಪೋಫಾಲ್ ತೂರಿಸುವುದು. ತಾತ್ಕಾಲಿಕ ಉಸಿರಾಟದ ಪೂರ್ಣ ನಿಲುಗಡೆಯಲ್ಲಿ ಒಳಗೆ ತೂರುವುದನ್ನು ಸುವುದನ್ನು ನಿಲ್ಲಿಸುವುದು. ಮೊದಲ ಪ್ರಮಾಣ ಮತ್ತು ಕಾಲಾವಧಿಯ ಪರಿಣಾಮವನ್ನು ಲೆಕ್ಕಮಾಡುವುದು. ರೋಗಿಗಳಿಗೆ ಸಕ್ರಿಯನೋಲಿನ್ ಒಂದು ಕೆ.ಜಿ ಗೆ ೨ ಎಂ.ಎಲ್ ಮುಂಗೈ ಪಟ್ಟಿಯೊಂದಿಗೆ ಇಂಡೋ ಟ್ರಾಕ್ಸಲ್ ನಳಿಕೆಯನ್ನು ಅದಕ್ಕೆ ತಕ್ಕದಾದ ಆಕರವನ್ನು ತೂರಿಸುವುದು ಅಲ್ಲದೆ ಆಕ್ಸಿಜನ್, ನೈಟ್ರಸ್ ಆಕ್ಸೈಡ್ ಇತರೆ ಉಸಿರಳಿತದ ಸಾಧನಗಳಿಂದ ಕಾಪಾಡಿಕೊಳ್ಳುವುದು.

ಈ ಅಧ್ಯಯನದ ಗುರಿ ರೋಗಿಗಳ ಮೇಲೆ ಪ್ರೋಪೋಫಾಲ್ ಹೆಚ್ಚಿನ ಪ್ರಮಾಣದಲ್ಲಿ ಉಪಯೋಗಿಸಿದಾಗ ಆರಟೀ ರಯಾಲ್ ರಕ್ತದೊತ್ತಡ ವನ್ನು ಮತ್ತು ಹೃದಯ ತೀವ್ರತೆಯಿಂದೊದಗುವ ಪ್ರತಿಕೂಲತೆಯನ್ನು ಕಡಿಮೆಮಾಡುವುದು ರೋಗಿಗಳಿಗೆ ಮತ್ತು ರೋಗಿಗಳ ಮೇಲ್ವಿಚಾರಕರಿಗೆ ಈ ವಿಧಾನವನ್ನು ಪೂರ್ತಿಯಾಗಿ ವಿವರಿಸುವುದು.

ದಯವಿಟ್ಟು ಇದನ್ನು ಓದಿ ನಿಮ್ಮ ಕುಟುಂಬದ ಸದಸ್ಯರೊಡನೆ ವಿವೇಚಿಸಿ. ಈ ಅಧ್ಯಯನದ ಬಗ್ಗೆ ಯಾವುದೇ ಪ್ರಶ್ನೆ ಕೇಳಬಹುದು ನೀವು ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ಒಪ್ಪಿದ್ದೇ ಆದರೆ ನಾವು ಇಲ್ಲಿ ಕೊಟ್ಟಿರುವ ಪ್ರೋಫಾರ್ಮ್ ದಂತೆ ನಿಮ್ಮಿಂದ ಅಥವಾ ಈ ಪರಿಸ್ಥಿತಿಗೆ ಹೊಣೆ ಅಥವಾ ಇಬ್ಬರ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಪಡೆಯುತ್ತೇವೆ. ಸಂಬಂಧ ಪಟ್ಟ ಪೂರ್ವಾಪರಗಳನ್ನು ಪಡೆದುಕೊಳ್ಳುತ್ತೇವೆ.

ನಿಮ್ಮಿಂದ ಪಡೆದ ಈ ವಿವರಗಳನ್ನು ನಮ್ಮ ಪ್ರಕಟಣೆಗೆ ಮತ್ತು ಉಪನ್ಯಾಸಗಳಿಗೆ ಉಪಯೋಗಿಸಿ ಕೊಳ್ಳುತ್ತೇವೆ. ಇದಕ್ಕೆ ವೂದುಗುವ ಪೂರ್ತಿ ವೆಚ್ಚವನ್ನು ಇದಕ್ಕೆ ಸಂಬಂಧ ಪಟ್ಟ ವೈದ್ಯರು ವಹಿಸಿಕೊಳ್ಳುತ್ತಾರೆ.

ನಿಮ್ಮಿಂದ ಪಡೆದ ವಿವರಗಳನ್ನು ಗೌಪ್ಯ ವಾಗಿ ಇಟ್ಟಿರುತ್ತಾರೆ. ಅಲ್ಲದೆ ಇದನ್ನು ಬೇರೆ ಯಾರೇ ಹೊರಗಿನವರಿಗೆ ಕೊಡುವುದಿಲ್ಲ. ನಿಮ್ಮ ವೈಯಕ್ತಿಕ ಗುರುತನ್ನು ತಿಳಿಸುವುದಿಲ್ಲ. ಇದನ್ನು ಈ ಸಂಸ್ಥೆಯ ನೈತಿಕ ಹೊಣೆ ಹೊತ್ತಿರುವವರು ಆಗಾಗ ವಿಮರ್ಶಿಸುತ್ತಾರೆ. ನೀವು ಅವರನ್ನು ಸುಲಭವಾಗಿ ಭೇಟಿಯಾಗಬಹುದು. ಈ ಅಧ್ಯಯನಕ್ಕೆ ಒಪ್ಪಿಗೆಗೆ ಯಾವುದೇ

ಶೈಕ್ಷಣಿಕ ವ್ಯಾಸಂಗಕ್ಕೆ ಮತ್ತು ಸಂಶೋಧನಾ ಉದ್ದೇಶದ ಶಸ್ತ್ರ ಚಿಕಿತ್ಸೆ ಮುಂತಾದವುಗಳಿಗೆ
ಇದು ವಿನಿಯೋಗವಾಗಲಿ. ವಿಡಿಯೋ ಅಥವಾ ಛಾಯಾ ಚಿತ್ರ ತೆಗೆದುಕೊಳ್ಳುವುದಕ್ಕೆ
ಒಪ್ಪಿಕೊಳ್ಳುತ್ತೇನೆ

ಎಲ್ಲಾ ಮಾಹಿತಿಗಳನ್ನು ಪ್ರಕಟಿಸಬಹುದು ಅಥವಾ ಯಾವುದೇ ಶೈಕ್ಷಣಿಕ ಕಾರ್ಯಕ್ರಮಕ್ಕೆ
ಉಪಯೋಗಿಸಬಹುದು. ಈ ಅಧ್ಯಯನದ ಮಾರ್ಗದಲ್ಲಿ ನಾನು ವೈದ್ಯರನ್ನಾಗಲೀ ಅಥವಾ
ಸಂಸ್ಥೆ ಯನ್ನು ಯಾವುದೇ ರೀತಿಯ ಅನುಚಿತ ದುಸ್ಪರಿಣಾಮ ದಿಂದ ಅವರು ಹೊಣೆ
ಎಂದು ಹೇಳುವುದಿಲ್ಲ.

ನನ್ನ ಒಪ್ಪಿಗೆ ತೆಗೆದೊಕೊಂಡ ಮಾಹಿತಿ/ವರದಿಯನ್ನು ಮತ್ತು ರೋಗಿಯ ವಿಷಯ
ಸೂಚಿಯನ್ನು ಇಲ್ಲಿ ಭಾಗವಹಿಸುವವರೆಲ್ಲರಿಗೂ ಅದರ ಪ್ರತಿಯನ್ನು ಕೊಟ್ಟಿರುತ್ತೇನೆ.

ಸಹಿ/ಹೆಬ್ಬರಳ ಗುರುತು
ರೋಗಿಯ ಪರಿಚಾರಕ ಹೆಸರು

ಸಹಿ/ಹೆಬ್ಬರಳ ಗುರುತು
ರೋಗಿ/ಮೇಲ್ವಿಚಾರಕರು
ರೋಗಿಯೊಂದಿಗೆ ಸಂಬಂಧ

ಸಾಕ್ಷಿಗಳು -೧
ಸಾಕ್ಷಿಯ ಹೆಸರು

ರೋಗಿಯ ಸಂಬಂಧ

ಅಧ್ಯಯನ / ಸಂಶೋಧನೆ ಪರೀಕ್ಷಕ ವೈದ್ಯರ ಸಹಿ

INFORMED CONSENT FORM

Name of the institution: SRI DEVARAJ ACADEMY OF HIGHER EDUCATION
AND RESEARCH.

Name of the principal investigator: Dr. Dandamudi Siri Chandana

Name of the guide: Dr. Kiran.N

Name of the subject/participant:

**STUDY: VARIATION IN INDUCTION DOSE AND TIME REQUIREMENT OF
PROPOFOL ON ADMINISTERING INHALED NITROUS OXIDE DURING
INDUCTION : A PROSPECTIVE, RANDOMISED CONTROLLED STUDY.**

Date:

I, _____ aged _____
,after being explained in understandable language about the purpose of the study and the risks and complications of the procedure, hereby give my valid written informed consent without any force or prejudice for studying variation in induction dose and time requirement of propofol on administering inhaled nitrous oxide. The nature and risks involved have been explained to me to my satisfaction. I have been explained in detail about the study being conducted. I have read the patient information sheet and I have had the opportunity to ask any question. Any question that I have asked, have been answered to my satisfaction. I consent voluntarily to participate as a participant in this research. I hereby give consent to provide my history, undergo physical examination, undergo the procedure, undergo investigations and provide its results and documents etc. to the doctor / institute etc. All the data can be used for any published, dissertation or study. I will not hold the doctors / institute etc. responsible for any untoward consequences during the procedure / study. I am aware that there wont be any monetary benefits for taking part in this study.

A copy of this Informed Consent Form and Patient Information Sheet has been provided to the participant.

(Signature & Name of Pt. Attendant)
(Relation with patient)

(Signature/Thumb impression & Name of
Patient/Guardian)

Witness 1:

Witness 2:

(Signature & Name of Research person /doctor)

ಒಪ್ಪಿಗೆ ಕೊಟ್ಟು ಪತ್ರ

ಸಂಸ್ಥೆಯ ಹೆಸರು : ಶ್ರೀ ದೇವರಾಜ ಅರಸ್ ಮೆಡಿಕಲ್ ಕಾಲೇಜ್, ಉನ್ನತ ವಿದ್ಯಾ ಮತ್ತು
ಸಂಶೋಧನಾ ಅಕಾಡೆಮಿ ಟಿ. ಮ. ಕೆ. ಕೋಲಾರ
ಮುಖ್ಯ ಪರಿೀಕ್ಷಕರ ಹೆಸರು ಡಾ. ದಂದಾಮುಡಿ ಸಿರಿ ಹಂದನ
ಮಾರ್ಗದರ್ಶಿಯ ಹೆಸರು ಡಾ. ಕಿರಣ್ ಏನ್.
ವಿಷದ ಹೆಸರು /ಭಾಗವವಿಸುವವರ
ಮೊದಲು ಪ್ರವೇಶಿಸಲ್ಪಡುವ ಪ್ರಮಾಣದಲ್ಲಿ ಬದಲಾವಣೆ ಮತ್ತು ಮೊದಲ ಸಲ
ನ್ಯೂಟ್ರಿಸ್ ಆಕ್ಸೆಡ್ ಉಸಿರಳಿತದ ನಿರ್ವಹಣೆಯಲ್ಲಿ ಪ್ರೋಪೋಫಾಲ್ ನ ವ್ಯವಧಿ :
ನಿರೀಕ್ಷಿತ ಕ್ರಮರಹಿತ ನಿಯಂತ್ರಣದ ಅಧ್ಯಯನ
ದಿನಾಂಕ:

ನಾನು ವಯಸ್ಸು.....

ನಾನು ,ವರ್ಷ ದವನಾದ ನನಗೆ ಅರ್ಥವಾಗುವ ಭಾಷೆಯಲ್ಲಿ ಸಂಪೂರ್ಣ
ವಾಗಿ ಇದರ ಉದ್ದೇಶವನ್ನು ತಿಳಿಸಿರುತ್ತಾರೆ. ಇನ್ನು ಬೇಶನ್ ಆರ್. ಎ. ಎಂ. ಪಿ. ಸ್ನಾನಕ್ಕೂ
ಮತ್ತು ಎಲೆಕ್ಟಿವ್ ಸ್ನಿಫಿಂಗ್ ನಲ್ಲಿರುವ ಇನ್ನು ಬೇಶನ್ ಸ್ನಾನಕ್ಕೂ ಇರುವ ಸ್ವಾಸ್ಥ್ಯದ
ಒಂದು ತುಲನಾತ್ಮಕ ಧೃಷ್ಟಾಂತದ ಅಧ್ಯಯನ

ಆದ್ದರಿಂದ ಈ ಅಧ್ಯಯನಕ್ಕೆ ನಾನು ಲಿಖಿತ ರೂಪದಲ್ಲಿ ನನ್ನ ಒಪ್ಪಿಗೆಯನ್ನು ಯಾವುದೇ
ಒತ್ತಡಕ್ಕೊಳಗಾಗದೆ ಪೂರ್ವಗ್ರಹ ಪೀಡಿತನಾಗದೆ ಒಪ್ಪಿಕೊಂಡಿರುತ್ತೇನೆ.

ಇಂಡೋ ಟ್ರಾಕ್ ಹೀಲ್ ಇನ್ನು ಬೇಶನ್ ಉದಾ ಲರೆನ್ಸ್ ಗಾಯ, ಇಂಡೋ ಟ್ರಾಕ್
ಹೀಲ್, ನಳಿಕೆಯ ಸ್ನಾನ ಪಲ್ಲಟ, ರಕ್ತ ಸ್ರಾವ, ಹಲ್ಲು ಮತ್ತು ತುಟಿಗಳ ಹಾನಿ, ಕರ್ಕಶತೆ
ಯನ್ನು ರೋಗಿಗೆ ಶಸ್ತ್ರ ಚಿಕಿತ್ಸೆಗೆ ಮುನ್ನ ವಿವರವಾಗಿ ತಿಳಿಸಿದ್ದಾರೆ. ಇದರ ಸ್ವಭಾವ
ಮತ್ತು ಉಂಟಾಗುವ ಅಪಾಯ ಮತ್ತು ದುಸ್ಪರಿಣಾಮಗಳನ್ನು ವಿವರವಾಗಿ ನನಗೆ
ಅರ್ಥವಾಗುವ ಹಾಗೆ ಸಮಂಜಸವಾಗಿ ತಿಳಿಸಿದ್ದಾರೆ.

ಇದರ ಬಗ್ಗೆ ನಡೆಸಿದ ಅಧ್ಯಯನವನ್ನು ವಿವರಿಸಿದ್ದಾರೆ. ನನಗೆ ರೋಗಿಯ ವಿವರಣಾ
ಪತ್ರವನ್ನು ಓದಿ ನನಗೆ ಪ್ರಶ್ನಿಸುವುದಕ್ಕೆ ಅವಕಾಶವನ್ನು ಕೊಟ್ಟರು. ಯಾವುದೇ ಪ್ರಶ್ನೆ
ಕೇಳಿದರೂ ನನಗೆ ಸಮಂಜಸ ಉತ್ತರ ಸಿಗುತ್ತಿತ್ತು. ಈ ಎಲ್ಲಾ ಅಧ್ಯಯನದಲ್ಲಿ ಉಂಟಾಗುವ
ಖರ್ಚನ್ನು ಪರಿೀಕ್ಷಕರು ವಹಿಸುತ್ತಾರೆ. ಈ ಅಧ್ಯಯನ ಪೂರ್ತಿಯಾಗುವವರೆಗೂ ನನಗೆ
ಖರ್ಚು ಬರುವುದಿಲ್ಲ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ನಾನು ಮನಃ ಪೂರ್ತಿಯಾಗಿ ನನ್ನ
ಒಪ್ಪಿಗೆಯನ್ನು ಮತ್ತು ಅವರಿಗೆ ಬೇಕಾದ ನನ್ನ ಎಲ್ಲಾ ವಿವರಗಳನ್ನು ಕೊಟ್ಟಿರುತ್ತೇನೆ.
ಭೌತಿಕ ಪರಿೀಕ್ಷೆ ಪದ್ಧತಿ ಪ್ರಕಾರ ಅವರ ತನಿಖೆಗೆ ಮತ್ತು ಅಲ್ಲಿಂದ ದೊರೆತ ಫಲಿತಾಂಶ
ಮತ್ತು ಎಲ್ಲಾ ಮಾಹಿತಿಯನ್ನು ವೈದ್ಯರಿಗಾಗಲೀ ಮತ್ತು ಸಂಸ್ಥೆಗಾಗಲೀ ಕೊಡುತ್ತೇನೆ.

ರೀತಿ ಒತ್ತಡವಿಲ್ಲ. ನೀವು ಪಾಲ್ಗೊಳ್ಳಲು ಇಷ್ಟವಿಲ್ಲದ ಪಕ್ಷದಲ್ಲಿ ನಿಮ್ಮ ಉಪಚಾರದಲ್ಲಿ
ಯಾವುದೇ ರೀತಿ ವ್ಯತ್ಯಾಸ ವಿರುವುದಿಲ್ಲ . ನೀವು ನಿಮ್ಮ ಸಹಿ ಅಥವಾ ಹೆಬ್ಬೆಟ್ಟಿನ
ಗುರುತನ್ನು ಕೊಡುವುದು. ವೈಯಕ್ತಿಕವಾಗಿ ಒಪ್ಪಿ ಮಾತ್ರ ಸಹಿ ಮಾಡಬಹುದು.

ಮಿಕ್ಕ ವಿವರಗಳಿಗೆ ಸಂಪರ್ಕಿಸಿ :

ಡಾ. ದಂದಾಮುಡಿ ಸಿರಿ ಚಂದನ . ಡಾ. ಕಿರಣ್ . ಏನ್.

ಅನೆಸ್ಸಯಾಸ್ತಿಯಾಲಜಿ ಸ್ನಾತಕೋತ್ತರ ಪದವಿ . ಎಸ್.ಡಿ.ಯು.ಎಂ.ಸಿ ಕೋಲಾರ .

ಅನೆಸ್ಸಯಾಸ್ತಿಯಾಲಜಿ, ಪ್ರಾಂಶುಪಾಲರು.

ಮೊ: 7330874979 ಅನೆಸ್ಸಯಾಸ್ತಿಯಾಲಜಿ, ವಿಭಾಗ Mo: 9740468460

MASTER CHART

A decorative graphic consisting of a thick horizontal line and a thick vertical line intersecting at the right end of the horizontal line. The lines are black with a slight gray shadow or offset.

KEY TO MASTER CHART

ASA : American Society of Anesthesiologists Physical Status Classification.

SBP : Systolic Blood Pressure

DBP: Diastolic Blood Pressure

MAP: Mean Arterial Pressure

HR : Heart Rate

MASTERCHART

SL.NO	Group	UHID NO.	AGE[YRS]	GENDER	ASA Grade	INDUCTIO N TIME	INDUCTIO N DOSE(mg	SBP_T1	SBP_T2	SBP_T3	SBP_T4	SBP_T5	SBP_T6	SBP_T7	SBP_T8	SBP_T9
1	Group A	340125	39	F	1	<200sec	1.0-1.5	128	126	126	122	120	118	118	120	122
2	Group A	340331	56	M	1	<200sec	0.5-1.0	130	128	124	120	120	118	116	116	118
3	Group A	333730	41	F	1	<200sec	0.5-1.0	130	126	124	118	116	114	116	118	120
4	Group A	329381	61	F	2	<200sec	0.5-1.0	130	126	126	122	120	118	120	122	124
5	Group A	331839	51	F	1	<200sec	1.0-1.5	110	110	106	104	104	100	121	120	114
6	Group A	330210	50	M	2	<200sec	0.5-1.0	120	124	120	118	118	116	122	120	128
7	Group A	315236	30	F	2	<200sec	1.0-1.5	130	127	130	119	114	110	110	114	122
8	Group A	340607	42	F	1	<200sec	1.0-1.5	122	128	124	120	120	118	118	120	122
9	Group A	365997	57	M	1	<200sec	1.0-1.5	128	130	132	130	128	124	124	122	128
10	Group A	367833	54	F	2	<200sec	0.5-1.0	130	126	126	122	120	118	120	122	124
11	Group A	367439	21	F	1	>200sec	1.0-1.5	120	122	116	114	112	110	110	110	114
12	Group A	361182	38	M	1	<200sec	0.5-1.0	140	140	136	134	132	132	128	126	130
13	Group A	361472	42	F	2	>200sec	1.0-1.5	128	126	126	122	120	118	118	120	122
14	Group A	366182	58	F	1	>200sec	0.5-1.0	110	108	108	106	104	104	101	101	106
15	Group A	366812	22	F	1	<200sec	0.5-1.0	120	118	116	116	114	114	114	112	114
16	Group A	58275	27	M	1	<200sec	0.5-1.0	124	126	124	124	122	120	118	118	122
17	Group A	348199	18	M	2	<200sec	0.5-1.0	122	120	120	118	118	116	116	114	118
18	Group A	327376	50	F	1	<200sec	0.5-1.0	112	116	114	114	112	110	110	108	112
19	Group A	293575	22	M	1	>200sec	1.0-1.5	116	118	118	116	116	112	112	110	114
20	Group A	387732	27	F	2	<200sec	0.5-1.0	132	134	134	134	132	130	130	130	132
21	Group A	59697	23	F	1	<200sec	1.0-1.5	128	126	126	124	124	122	120	118	122
22	Group A	347646	43	F	2	<200sec	0.5-1.0	120	127	130	130	126	124	124	122	124

23	Group A	379126	23	M	1	<200sec	0.5-1.0	130	127	130	122	120	118	118	120	122
24	Group A	377379	38	F	1	<200sec	0.5-1.0	122	126	124	122	122	116	116	114	118
25	Group A	377489	19	F	1	<200sec	0.5-1.0	124	126	124	122	122	120	118	116	116
26	Group A	383821	53	M	1	<200sec	0.5-1.0	130	130	128	126	126	124	120	120	122
27	Group A	408552	40	M	2	<200sec	0.5-1.0	112	112	110	108	106	104	104	102	104
28	Group A	366698	58	M	2	<200sec	0.5-1.0	120	118	118	116	114	112	112	112	116
29	Group A	379523	53	F	1	<200sec	0.5-1.0	140	140	138	136	134	132	132	130	132
30	Group A	386116	51	F	1	<200sec	1.0-1.5	122	120	118	118	116	114	114	112	114
31	Group A	416691	57	M	2	<200sec	0.5-1.0	118	118	116	114	114	114	112	110	114
32	Group A	393975	34	M	1	>200sec	0.5-1.0	116	116	114	112	112	110	108	106	108
33	Group A	408076	44	F	1	>200sec	0.5-1.0	110	110	108	104	102	102	100	98	96
34	Group A	410501	39	M	1	<200sec	1.0-1.5	112	112	110	110	96	96	96	94	98
35	Group A	422018	35	M	1	<200sec	0.5-1.0	122	122	124	122	120	118	116	114	114
36	Group A	411026	38	F	1	<200sec	0.5-1.0	132	132	130	128	128	126	126	124	122
37	Group A	410968	38	F	2	<200sec	0.5-1.0	120	120	118	116	114	114	112	112	116
38	Group A	556587	35	M	1	<200sec	0.5-1.0	124	126	126	124	122	120	118	118	122
39	Group A	436651	33	M	2	<200sec	0.5-1.0	118	118	116	116	114	112	112	110	108
40	Group A	542760	49	F	1	<200sec	0.5-1.0	120	124	120	118	118	116	122	120	128
41	Group A	562001	38	F	1	<200sec	0.5-1.0	140	140	136	134	132	132	130	128	126
42	Group A	562201	25	F	2	>200sec	1.0-1.5	120	122	116	114	112	110	110	110	114
43	Group A	556106	29	M	1	<200sec	0.5-1.0	118	118	116	116	114	112	110	110	116
44	Group A	555423	56	F	2	<200sec	0.5-1.0	124	126	124	122	120	118	118	116	114
45	Group A	560010	52	F	1	<200sec	0.5-1.0	140	138	138	136	136	132	132	132	134
46	Group A	554840	37	F	2	<200sec	0.5-1.0	130	126	126	122	120	118	120	122	124
47	Group A	348199	18	M	2	<200sec	0.5-1.0	122	120	120	118	118	116	116	114	118
48	Group A	327376	50	F	1	<200sec	0.5-1.0	112	116	114	114	112	110	110	108	112
49	GROUP B	379579	19	M	1	<200sec	>1.5	106	110	108	104	101	90	90	94	101

50	GROUP B	366727	30	F	1	>200sec	>1.5	128	130	126	128	126	124	124	124	135
51	GROUP B	363744	45	M	2	>200sec	1.0-1.5	148	144	146	144	142	140	140	138	142
52	GROUP B	379148	62	M	1	>200sec	1.0-1.5	130	130	126	128	126	124	122	120	128
53	GROUP B	372350	48	F	2	>200sec	>1.5	150	155	160	153	150	148	144	140	148
54	GROUP B	363744	40	M	1	>200sec	>1.5	128	130	126	128	126	124	124	120	135
55	GROUP B	388376	30	F	2	>200sec	1.0-1.5	140	144	142	140	138	136	134	134	138
56	GROUP B	371517	50	F	2	>200sec	>1.5	142	144	144	142	140	138	136	136	138
57	GROUP B	378038	49	F	1	>200sec	>1.5	130	130	128	126	124	124	122	122	120
58	GROUP B	377095	55	M	1	>200sec	>1.5	105	105	102	100	98	96	94	94	98
59	GROUP B	375454	58	M	2	>200sec	>1.5	130	132	132	130	128	126	123	124	128
60	GROUP B	374865	20	M	1	<200sec	1.0-1.5	120	120	118	116	114	112	110	110	116
61	GROUP B	374345	45	F	2	>200sec	>1.5	140	140	138	136	132	130	128	128	130
62	GROUP B	369252	50	F	2	>200sec	>1.5	134	138	138	134	134	130	128	128	124
63	GROUP B	371879	45	F	2	>200sec	>1.5	126	126	124	124	120	118	118	120	124
64	GROUP B	374842	46	F	1	>200sec	>1.5	128	128	128	126	126	126	124	124	120
65	GROUP B	394804	36	F	1	>200sec	>1.5	130	134	134	132	132	132	130	130	134
66	GROUP B	381310	39	M	1	>200sec	>1.5	110	108	108	108	106	106	104	104	122
67	GROUP B	387559	50	F	2	>200sec	>1.5	124	126	126	122	122	120	120	118	125
68	GROUP B	377533	36	F	2	>200sec	1.0-1.5	126	128	128	126	124	124	122	122	128
69	GROUP B	390646	31	M	1	<200sec	>1.5	110	110	106	104	104	102	102	98	112
70	GROUP B	105237	50	M	2	>200sec	>1.5	124	124	128	126	126	124	124	122	126
71	GROUP B	253649	30	F	1	>200sec	1.0-1.5	110	112	112	110	112	110	110	108	114
72	GROUP B	419632	57	M	1	>200sec	>1.5	116	114	114	116	118	118	116	116	122
73	GROUP B	177530	42	F	1	>200sec	>1.5	122	124	126	126	124	124	122	122	124
74	GROUP B	418105	23	F	1	>200sec	>1.5	132	132	134	132	130	130	132	132	138
75	GROUP B	414693	46	M	1	<200sec	>1.5	126	126	126	124	123	123	119	119	121
76	GROUP B	413744	34	M	1	>200sec	1.0-1.5	119	117	117	117	115	115	113	115	122

77	GROUP B	365143	20	M	1	>200sec	1.0-1.5	117	115	115	115	113	113	111	111	118
78	GROUP B	404648	43	M	1	>200sec	>1.5	126	128	126	126	124	124	124	122	128
79	GROUP B	407004	36	F	1	>200sec	>1.5	110	110	106	106	104	104	102	101	112
80	GROUP B	395726	34	F	1	>200sec	>1.5	124	126	128	126	126	124	124	124	126
81	GROUP B	467455	43	M	2	>200sec	>1.5	124	124	126	126	124	122	122	120	124
82	GROUP B	418942	31	M	1	>200sec	1.0-1.5	110	114	114	112	110	108	106	104	108
83	GROUP B	381381	22	M	1	>200sec	>1.5	108	106	104	104	104	102	102	100	108
84	GROUP B	374716	49	F	2	<200sec	>1.5	122	120	124	122	122	120	120	118	126
85	GROUP B	362296	40	F	1	>200sec	1.0-1.5	110	110	106	104	104	102	102	102	110
86	GROUP B	421071	33	F	1	>200sec	1.0-1.5	130	134	134	132	132	130	130	130	134
87	GROUP B	252487	20	F	1	>200sec	>1.5	126	126	126	124	124	122	122	120	126
88	GROUP B	435303	24	F	1	<200sec	>1.5	110	110	108	108	106	106	104	104	110
89	GROUP B	522216	26	M	1	>200sec	>1.5	130	134	132	132	130	130	128	126	132
90	GROUP B	517913	49	F	2	>200sec	>1.5	120	124	126	126	124	122	120	118	126
91	GROUP B	524993	58	M	1	>200sec	>1.5	130	136	136	134	132	132	130	128	132
92	GROUP B	526064	22	M	1	>200sec	>1.5	130	134	136	134	132	130	130	128	134
93	GROUP B	194784	45	M	2	>200sec	>1.5	140	148	144	142	140	140	138	138	144
94	GROUP B	525471	30	F	1	>200sec	>1.5	110	108	106	106	104	102	100	100	106
95	GROUP B	437041	39	F	2	>200sec	>1.5	140	148	146	144	142	142	142	138	142
96	GROUP B	371855	58	M	1	>200sec	>1.5	138	136	136	134	132	130	130	130	136

DBP_T1	DBP_T2	DBP_T3	DBP_T4	DBP_T5	DBP_T6	DBP_T7	DBP_T8	DBP_T9	MAP_T1	MAP_T2	MAP_T3	MAP_T4	MAP_T5	MAP_T6	MAP_T7	MAP_T8	MAP_T9	HR_T1	HR_T2	HR_T3	HR_T4	HR_T5	HR_T6	HR_T7	HR_T8	HR_T9
80	82	82	80	78	78	80	82	84	96	97	97	94	92	91	93	95	97	76	80	82	84	82	80	82	84	86
90	86	86	84	84	82	80	82	84	103	100	99	96	96	94	92	93	95	82	84	84	82	84	86	86	90	96
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10 0	10 0	96	94	94	92	90	90	94	11 3	11 6	11 2	11 0	11 0	10 8	10 7	10 6	11 0	98	10 0	10 6	10 6	10 4	10 4	10 2	10 0	10 2
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