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Original Research Article

TO STUDY THE HAEMODYNAMIC CHANGES IN RESPONSE TO INDUCTION OF GENERAL ANAESTHESIA IN KETAMINE & CONTROL GROUPS.

Dr. Kannam Peddulu[1] (Associate Professor)

Dept. Anaesthesiology, Amaltas Institute of Medical Sciences, Dewas[1]

Corresponding Author: Dr. Kannam Peddulu

Abstract

Background & Methods: The aim of the study is to study the haemodynamic changes in response to induction of general anaesthesia in ketamine & control groups. All patients were premeditated with Inj. Glycopyrolate 0.004 mg/kg body weight intravenously. Patients were randomly divided in four groups of 25 patients each by a separate anaesthesiologist.

Results: Heart rate with standard deviation at different time intervals in group A & B. There was initially rise in heart rate after premedication. After induction heart rate decreased in all the groups and was statistically significant. Change in heart rate was least in group B among all the groups.

Conclusion: There was greater change in heart rate in group A as compared to group B in which the heart rate remained more stable throughout the period of anaesthesia. The fall in MAP pressure just after induction was more in group A as compared to group B. The blood pressure in ketamine group remained more stable among all the groups.

Keywords: haemodynamic, induction, anaesthesia & ketamine.

Study Design: Observational Study.

1. Introduction

Ketamine, a phencyclidine derivative, is an N-Methyl D-Aspartate receptor antagonist that also acts at numerous other sites[1]. It inhibits opioid mu receptors while stimulating delta and kappa receptors. Ketamine is in wide use as an anaesthetic, sedative and analgesic agent. Intravenous induction doses for anaesthesia range from 1-2mg/kg (with an intramuscular induction dose of 5-10mg/kg.) Ketamine is widely available yet has scarcely been soundly studied as an agent for the attenuation of haemodynamic response to LTI. Indeed, it is known to cause hypertension and tachycardia at anaesthetic doses[2].

Ketamine is a phencyclidine derivative that produces "dissociative anaesthesia" which is characterized by evidence on the EEG of dissociation between the thalamocortical and limbic system. Dissociative anaesthesia resembles a cataleptic state in which the eyes remain open

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with a slow nystagmic gaze[3]. The patient is noncommunicative, although wakefulness may appear to be present. Varying degrees of hypertonus and purposeful skeletal muscle movements often occur independently of surgical stimulation. The patient is amnesic and analgesia is intense[4].

This cardio-stimulatory effect is however minimal to nil at sub-anaesthetic/analgesic doses. This study was designed to compare the overall occurrence of hypertension in response to laryngoscopy and endotracheal intubation between low-dose ketamine versus fentanyl in patients undergoing general anaesthesia. Considering the enormous effect of anaesthetic drugs on the patients' haemodynamics during and after intubation, this study intends to explore a possible alternative regimen to reduce this morbidity[5-6].

2. Material and Methods

This study was conducted in patients undergoing elective surgery at Osmania M. C. & MNJ Cancer Hospital, Hyderabad for 01 year on 50 patients of ASA grade I and II aged 20-40 years of either sex undergoing elective surgery were randomly selected. Each patient underwent a pre-anaesthetic checkup including investigations for major surgery.

All patients were premeditated with Inj. Glycopyrolate 0.004 mg/kg body weight intravenously. Patients were randomly divided in four groups of 25 patients each by a separate anaesthesiologist. Patients were preoxygenated with 100% oxygen (8L/MIN) using face mask and bains cirucuit for three minutes followed by co-induction agent which was either 10 ml saline (control group A), 0.3 mg/kg ketamine (group B).

Exclusion Criteria:

- Patients those taking benzodiazepines.
- Patients on concurrent drug therapy with beta blockers, beta agonists, alpha blockers, digitalis and ant arrhythmic drugs,
- Patients with history of allergic reaction to any of the drug used in the study.

3. Result

Table 1: Patients Characteristic (Mean +_SD)

Characteristic	Group A	Group B
No. of patients	25	25
Age (years)	27.24+_4.49	27.44+_5.41
Wt.(kg)	58.04+_5.15	58.8+_5.16
M/F	21/4	11/14

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The patients with respect to their age, weight and sex in all the four groups. There was statistically no significant difference in the demographic data between the groups. The preoperative history, examination, biochemical value, ASA grading in all the groups were comparable.

Table 2: Induction dose and prop up in number of patients

Groups	No. of patients	Induction Dose (mg/kg)	Prop up in no. of patients	Total dose of propofol (in mg)
Group A	25	2.71 +-0.05	16	157.22+-13.39
Group B	25	1.21+_0.04	Nil	71.55+-6.26

The table shows number of patients, induction dose, prop up in number of patents and total dose of propofol. Induction dose and total dose of propofol was least in group B and prop up dose is also not required in group B. The P value for induction dose, prop up in number of patient and total dose of propofol is <0.001 that is highly significant.

Table 3: Heart rate at different time intervals (Mean ±SD)

Group	Pre-operative	After premedication	1 min after co-induction	1 min after induction	5 min after co-induction	10 min after co-induction	15 min after co-induction	20min after co-induction
Group A	87.4±9.97	90.12±9.95	90.8±9.96	74.88±10.13	75.36±10.19	75.04±10.34	74.84±10.45	74.84±10.5
Group B	85.76±8.66	88.48±8.7	95.68±8.42	79.2±7.96	91.72±7.99	80.68±8.09	83.76±8.66	83.76±8.66

Heart rate with standard deviation at different time intervals in group A & B. There was initially rise in heart rate after premedication. After induction heart rate decreased in all the groups and was statistically significant. Change in heart rate was least in group B among all the groups.

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Table 4: Mean arterial pressure at Different Time intervals (Mean +SD)

Group	Pre-operative	After pre-medication	1 min after co-induction	1 min after induction	5 min after co-induction	10 min after co-induction	15 min after co-induction	20min after co-induction
Group A	93.03±6.27	93.4±5.94	94.27±6.03	69.78±4.79	79.95±5.21	73.84±5.24	73.31±5.03	74.08±4.8
Group B	93.67±5.30	93.91±4.87	97.65±5.45	86.88±4.86	91.68±5.14	90.96±4.78	90.4±4.74	91.79±5.31

Mean arterial pressure with standard deviation at different time intervals in group A & B. After induction means arterial pressure decreased in all the groups that were statistically highly significant. Change in mean arterial pressure was least in group B among all the groups.

4. Discussion

Anderson and Robb proposed a pharmacokinetic theory that part of the mechanism of action of co-induction drugs is to reduce anxiety and the associated sympathetic response. Both propofol and midazolam produces anxiolysis when administered before induction and this mechanism reduces cardiac output, helps in preventing rapid distribution of propofol[7].

No improvements in cardiovascular stability associated with either propofol or midazolam predosing. Infact the tendency for hypotension on induction of anaesthesia was greatest in the midazolam group. The synergistic actions of midazolam and propofol and found that synergism extended the hypotension which occurred at induction of anaesthesia, investigated the sympathy- adrenergic, haemodynamic and stress response to co-induction in the elderly and found that in spite of a reducing the dose of propofol to half required for induction did not confer any cardiovascular benefit even with prior administration of midazolam[8].

The three co-induction agents were effective in reducing the induction dose of propofol considerably compared to placebo (Saline).Dose reduction following midazolam is probably due to synergistic interactions between the two drugs. Synergism has been reported between agents with known functional link in the central nervous system viz. midazolam and propofol acting on a common receptor site the GABA receptors. Reduced dose requirement of propofol following ketamine cannot be explained by this mechanism as these agents act via distinctly different receptors, ketamine acts by antagonism of NMDA receptors while propofol acts on GABA receptors[9].

Ketamine in Sub-Anaesthetic doses with propofol has gained attention in total intravenous anaesthetic technique because of its powerful analgesic action in a small dose without causing myocardial and respiratory depression. Ketamine also causes some degree of sympathetic stimulation which tends to counterbalance the cardiovascular effects of propofol.

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One of the major drawbacks with ketamine anaesthesia has been emergence deliriums, which propofol seems to be effective in eliminating[10].

5. Conclusion

There was greater change in heart rate in group A as compared to group B in which the heart rate remained more stable throughout the period of anaesthesia. The fall in MAP pressure just after induction was more in group A as compared to group B. The blood pressure in ketamine group remained more stable among all the groups.

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