

The evaluation of hemodynamic status through the ketamine premedication

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ABSTRACT

Background : Any drug given in the period before induction of anesthesia refers to premedication. In addition to those normally taken by the patient, some drugs are given with specific intentions. The aim of the current study was to evaluate the impact of premedication with Ketamine on heart rate (H.R) during and after induction .

Methods : The dose-dependent direct stimulation of the central nervous system (CNS) is caused by Ketamine, which results in increasing the outflow of the sympathetic nervous system. Forty nine (49) patients were enrolled in this study, females were (27) and males were (22) patients with the American Society of Anesthesiologist (ASA) physical status (I and II).

Results : During the induction of ketamine, no significant differences at ($p \leq 0.05$) were observed in all groups. In term of heart rate parameter, the heart rate was (94.24 beat/min) at baseline, while the H.R readings were (97.10, 98.45, 98.22, and 97.12 beat /min) after 1min, 3min, 5min, and 10min after administration of ketamine respectively..

Conclusion : It was concluded that ketamine causes no significant increase in heart rate due to effect of propofol which has opposite effect to ketamine on heart rate, as well as the use of low dose of ketamine at induction.

Keywords: Heart rate, hemodynamic status, Ketamine.

INTRODUCTION

The heart rate may differ in accordance with the body's physical requirements, such as oxygen absorption and carbon dioxide excretion. It is often close to or equal to the pulse measured at any peripheral points. The activities which may provoke changes involve physical exercises, anxieties, sleep, illness, stress and drug ingestions [1]. The american heart association stated that the normal resting adult's human heart rate is (60–100) beat /min. Tachycardia is defined as the fast heart rate above 100 beat/min at rest, while bradycardia is defined as the slow heart rate below 60 beat/min at rest [2]. The slow heart beat around 40–50 beat/min is commonly observed during sleep and is regarded as normal, and arrhythmia occurs when the heart does not beat in regularly. Diseases are sometimes indicated when the heart beats are abnormal [3] .

In 1965, ketamine which is a phencyclidine derivative was used for clinical purposes. It differs from most other intravenous anesthetic in producing significant analgesia. The characteristic cataleptic states seen following ketamine induction dose is called “dissociative

anesthesia” where the eyes of patients are open with slow nystagmic gazing (cataleptic state) [4]. Ketamine is supplied as aqueous solutions in many concentrations, and formulated as weak acid with a (3.5- 5.5) pH. Ketamine contains a single chiral carbon atom [5]. Ketamine may be used as useful induction factor in asthmatic children, because it causes smooth muscle relaxation in the air ways [6].

Ketamine is different from the majority of anesthetics in its stimulation of the cardiovascular system, making heart rate alteration, cardiac output and blood pressure. Probably, reuptake inhibition of circulating catecholamines can participate in such phenomenon. However, cardio depression impacts were observed in severely ill patients, which may be attributed to chronic catecholamine depletions leading to prevent any sympathomimetic ketamine effect and unmasking the negative inotropic effects that are often overshadowed by sympathetic stimulations [7]. The ketamine's cardiovascular impacts often cause no problems, but using it is contraindicated in those who develop significant ischemic cardiac diseases and must be avoided in individuals with a history of hypertension or cerebrovascular accident. Among people who use recreational ketamine, and presented to emergency departments, tachycardia was the major common finding during physical examinations [8].

Patients and methods:

This study was conducted in Baghdad teaching hospital / Iraq from October 2018 to April 2019 under the supervision of the department of Anesthesia technology, College of Health and Medical Technology- Baghdad. Forty nine (49) patients, were involved in this study, (27) were females & (22) were males with American Society of Anesthesiologist (ASA) physical status (I and II). Their ages ranged from 20-60 years. Patients who were hypersensitive to Ketamine have been excluded from the present study.

All patients received dexamethasone (0.11 mg/kg) (I.V.), metoclopramide (0.14mg/kg), ranitidine (0.71mg/kg) and ketamine (0.5mg/kg) as premedication. Induction of anesthesia performed with (2.14mg/kg) propofol as hypnotic drugs. After that, intubation was done with the aid of (0.6mg/kg) Rocuronium non-depolarizing muscle relaxant. Intra-operative monitoring involved continuous ECG, pulse rates, non-invasive blood pressures and oxygen saturations. Prior to proposed surgery time, all patients fasted for 8 hours. Endotracheal tube was inserted with direct laryngoscopy and maintained with inhalational agent and O₂. Then pulse rate was recorded at baseline before ketamine induction and 1min, 3min, 5min, and 10min after ketamine induction.

RESULTS

Table (1): Distribution of ages and gender of study groups

Ages/years	No. %	p. value
20 -30	10 (20.4 %)	0.31 (N.S)
31 - 40	14 (28.5%)	
41 - 50	11 (22.4 %)	
51 - 60	14 (28.5%)	
Gender	No. %	0.41 (N.S)
Males	22 (44.9 %)	
Females	27 (55.1 %)	

***N.S= non-significant**

Table (1) showed that the age groups of this study (31-40) years& (51–60) years were the mostly age group (14,14, 28.5%, 28.5%) respectively, while the last frequencies of age group (20-30) was (10,20.4 %). Also the table demonstrated that the female patients were more than male group (27) patients (55.1%) from the total study group. These findings were non significant at (P<0.05).

Table (2):Mean (±) SD of heart rates at baseline, 1min, 3min, 5min, and 10min after ketamine administration

Test	Age (years)	H.R at Baseline (Beat/min.)	H.R at 1min after ketamine	H.R at 3min after ketamine	H.R at 5min after ketamine	H.R at 10min after ketamine
M±SD	41.5±11.3	94.3± 6.9	97.1±7.0	98.5±6.9	98.2±6.4	97.1 ± 5.8
Minimum	20	80	82	83	84	85
Maximum	61	112	114	115	112	111

***M±SD = Mean±Std.Deviation**

In table (2), the heart rate was (94.3 b/min.) at baseline, while the H.R readings were (97.10, 98.45, 98.22, and 97.12 b/min.) after 1min, 3min, 5min, and 10min after ketamine administration, respectively. Higher readings were observed after 3min and 5min after ketamine administration, and this may be due to use of propofol, which counteracts the increases in the heart rate produced by ketamine. Hence, propofol causes slow heart rate. On the other hand, the low increase of heart rate may be due to use of low dose ketamine.

Table (3): Comparison between the baseline heart rate reading with other readings of heart rate at different time intervals

Paired samples correlations

Time intervals	No.	(r)	p.value
H.R at Baseline & H.R at 1min after kitamine	49	.974	.000 (H.S)
H.R at Baseline & H.R at 3min after kitamine	49	.958	.000(H.S)
H.R at Baseline & H.R at 5min after kitamine	49	.914	.000(H.S)
H.R at Baseline & H.R at 10min after kitamine	49	.858	.000(H.S)

*(r) = Pearson correlation; *H. S=highly significant.

In table (3), the results revealed that the H.R at baseline & H.R at 1min after ketamine were correlated positively with ($r=0.974$, $p=0.000$), while this rate was decreased gradually at 3, 5 and 10 minutes with ($r=0.958$, $p=0.00$, $r=0.91$, $p=0.000$, $r=0.85$, $p=0.00$), and these differences were statistically highly significant at ($P<0.05$).

DISCUSSION

Ketamine is a common anesthetic agent for surgical procedures in both pediatric and adult populations in our environment [9]. The increased usage of ketamine as a possible rapid onset anti depressant necessitates a better understanding of its impact on heart rates and blood pressures and well known side effects at high doses [10].

Our findings disagreed with the findings of Munro HM, *et. al* [11], who reported a significant increase in heart rate after induction with ketamine at 5min (dose ketamine 2mg/kg) as compared to baseline. On the other hand, in the study of Basagan-Mogol E.*et.al.* [12] there were no significant changes in heart rate (HR) and mean pulmonary artery pressure (PMP) at any group during measure time. Also, our results disagreed with ZA Suleiman.*et.al.* [9] who reported a significant elevation above the baseline in heart rate (HR) after induction of anesthesia with intravenous ketamine (2mg/kg) at 5min. from induction. Other researches found that some patients recorded tachycardia in premedication of ketamine [13] .

CONCLUSION

Depending on the present study, we conclude that ketamine causes insignificant inducing of heart rate, and this is due to physiological antagonism between ketamine which causes tachycardia, and propofol which causes bradycardia. Other causes were the use of low dose of ketamine at induction, which was an insufficient effect on heart rate (H.R).

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