

OLD BUT NOT FORGOTTEN: INTRAVEINOUS KETAMINE HYDROCHLORIDE FOR
SHORT SURGICAL PROCEDURES

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ABSTRACT

Background: Ketamine is still considered a valuable drug. A drug with special properties that make it the only agent that presently serves as an anesthetic, sedative, amnesic and analgesic. Although it is recently somewhat forgotten, once was regarded as complete anesthetic drug. Water soluble, stable and non-irritant when administered intravenously, Ketamine has rapid onset and provides acceptable anaesthesia, minimal depression of cardiovascular and respiratory system, rapid degradation to inactive non-toxic metabolites and rapid and smooth emergence with minimal side effects with no hypersensitivity reaction. These properties make Ketamine useful for total intravenous (IV) anaesthesia. In selected patients where intense analgesia is needed without any muscular relaxation, Ketamine comes almost to the ideal. **Aim:** To evaluate the efficacy of administration of Ketamine for total intravenous (IV) anaesthesia in short term procedures. **Methods:** After taking informed consent, 60 patients of (ASA) physical status I-II of both sexes, aged between 19 and 70 years, categorized into three groups, Group-A (n=20) for general surgery, Group-B (n=20) for orthopedic surgery, Group-C (n=20) for Gynecological surgery, for various short surgical procedures. Note was being made of quality of anesthesia, analgesia and any complications. **Results:** It was concluded by evaluation and clinical trial that Ketamine can be used as a sole anaesthetic agent with minimum adverse effects and maximum safety. Regarding the intraoperative quality of anesthesia, 17 patient in group A had an excellent and 3 patients had good anesthesia. In group B, 18 patients had excellent and 2 patients had good anaesthesia. In group C 18 patients had an excellent and 2 patients had good anesthesia. The statistical difference between the groups was not significant. There was a insignificant difference regarding time to rescue analgesia and the total dose of rescue analgesics required during the postoperative 24 hours. **Conclusion:** Ketamine can be used as a sole anaesthetic agent with considerable amount of success in most of the short surgical procedures. It has the advantage of rapid and smooth onset of action, minimal depression of respiratory system and intense analgesia during intra-operative and post-operative period.

KEYWORDS: Ketamine, sedative, amnesic and analgesic.

INTRODUCTION

Ketamine hydrochloride, a non-barbiturate general anaesthetic agent which may be given intravenously or intramuscularly. A phencyclidine derivative, was introduced into clinical anaesthetic practice by Domino, Chodoff and Corssen in 1965. It was proved to be an excellent analgesic.^[1] A drug with special properties that make it the only agent that presently serves as an anesthetic, sedative, amnesic and analgesic. Although it is sometimes forgotten, Ketamine is still considered a valuable drug.^[2] Water soluble, stable and non-irritant

when administered intravenously, Ketamine has rapid onset and provides acceptable anaesthesia, minimal depression of cardiovascular and respiratory system, rapid degradation to inactive non-toxic metabolites and rapid and smooth emergence with minimal side effects with no hypersensitivity reaction. These properties make Ketamine useful for total intravenous (IV) anaesthesia.^[3] The drug acts rapidly and produces marked analgesia without cardiovascular depression or significant respiratory depression; laryngeal and pharyngeal reflexes are said to be well maintained. The anaesthetic state

produced by ketamine differs from that achieved with conventional anaesthetic agents. It is suggested that the drug produces somesthetic sensory blockade by selectively interrupting association pathways of the brain and this has led to the introduction of the term 'dissociative' anaesthesia.^[4] Ketamine produces a so-called dissociative anesthetic state in which the patient is dissociated from their surroundings, and although it also causes an emergence reaction, the symptoms are less severe than those produced by phencyclidine. Both drugs have effects at multiple receptor systems, but their main effect is blockade of the *N*-methyl-D-aspartate receptor (NMDAR), an excitatory ionotropic glutamate receptor present in the spinal cord and brain.^[5] Ketamine binds to the so-called PCP-binding site of the *N*-methyl-D-aspartate (NMDA)-receptor complex, located within the ion channel, thereby blocking the transmembranous ion flux. This makes ketamine a non-competitive NMDA-receptor antagonist. NMDA-receptors are calcium-gated channel receptors. The endogenous agonists of this receptor are the excitatory amino acids glutamic acid, aspartic acid, and glycine. Activation of the receptor results in opening of the ion channel and the depolarisation of the neurone. The NMDA-receptor is involved in sensory input at the spinal, thalamic, limbic and cortical levels. Ketamine would be expected to block (or interfere with) sensory input to higher centres of the CNS, with the emotional response to these stimuli, and with the process of learning and memory.^[6] The commercially available ketamine is a racemic mixture of two enantiomers. The *S*-enantiomer is shown to be the more potent one with an approximately 3-4 fold anaesthetic potency compared to the *R*-enantiomer. This correlates with the higher binding affinity for the PCP-site of the NMDA-receptor. The psychotomimetic properties of ketamine are mainly caused by the *S*-enantiomer, although subanaesthetic doses of *R*-ketamine may induce a state of relaxation.^[7,8] Several studies indicate that opioid receptors are also involved in the pharmacological effects of ketamine, and that the analgesic effect of ketamine may largely be attributed to the activation of these central and spinal receptors.^[9,10] The plasma levels at which analgesia is achieved are 0.15 µg/ml following intramuscular administration and 0.04 µg/ml after oral administration. This difference may be explained by a higher norketamine concentration due to first-pass metabolism. This main metabolite apparently contributes to the antinociceptive effect of ketamine.^[11] Ketamine differs from most anaesthetic agents in that it appears to stimulate the cardiovascular system, producing changes in heart rate, cardiac output, and blood pressure.^[12] Possibly, re-uptake inhibition of circulating catecholamines may contribute to this phenomenon. On the other hand, cardiodepressant effects have been noted in critically ill patients. This may be due to chronic catecholamine depletion preventing any sympathomimetic effects of ketamine and unmasking a negative inotropic

effect, which is usually overshadowed by sympathetic stimulation.^[13,14] The cardiovascular effects of ketamine usually do not pose a problem, but its use is contraindicated in patients with significant ischaemic heart disease and should be avoided in patients with a history of high blood pressure or cerebrovascular accidents.^[15] In recreational ketamine users, presenting to an emergency department, tachycardia was the most common finding upon physical examination.^[16] Ketamine is a mild respiratory depressant. It causes a shift of the CO₂ dose-response curve to the right, in a dose-related manner, but does not change the slope of the curve. Respiratory drive to CO₂ may be depressed as much as 15 to 22%. This effect is similar to that of opioids, but dissimilar from most sedative hypnotics and anaesthetics, suggesting that opioid receptors may play a role in the respiratory depressant effect. In clinical studies, the effects were observed only at high doses. Some case reports describe respiratory depression after rapid intravenous injection, but also after routine paediatric use of ketamine administered intramuscularly.^[17,18] Ketamine is considered to be an anaesthetic with a good safety profile.^[19] Its major drawback, limiting its clinical use, is the occurrence of emergence reactions. Emergence phenomena in patients awakening from a ketamine narcosis have been described following early clinical experience, and include hallucinations, vivid dreams, floating sensations and delirium. These symptoms were found to be reduced by concurrent use of benzodiazepines, putting the patient in a low stimulus environment and by providing information on the possible emergence reactions preoperatively. These emergence phenomena appear to occur more frequently in adults (30-50%) than in children (5-15%).^[20,21]

Methods

The present study was conducted in the department of anesthesiology in Govt; medical collage Srinagar from 2015 to 2017 for 60 patients of (ASA) physical status I-II of both sexes, aged between 19 and 70 years. **Exclusion criteria:** Patients with history or clinical evidence of Ischemic heart disease, Hypertension, Diabetes mellitus, Raised intraocular pressure, Epilepsy, Cerebrovascular disorders, Personality disorders, Old age, Surgery on head and neck, Psychiatric disorders.

Patients were categorized into three groups, Group-A (n=20) for general surgery, Group-B (n=20) for orthopedic surgery, Group-C (n=20) for Gynecological surgery, for various short surgical procedures. The patients which were not meeting the inclusion criteria were excluded from the study. Sixty patients underwent randomization (see Fig. 1 for patients' flowchart).

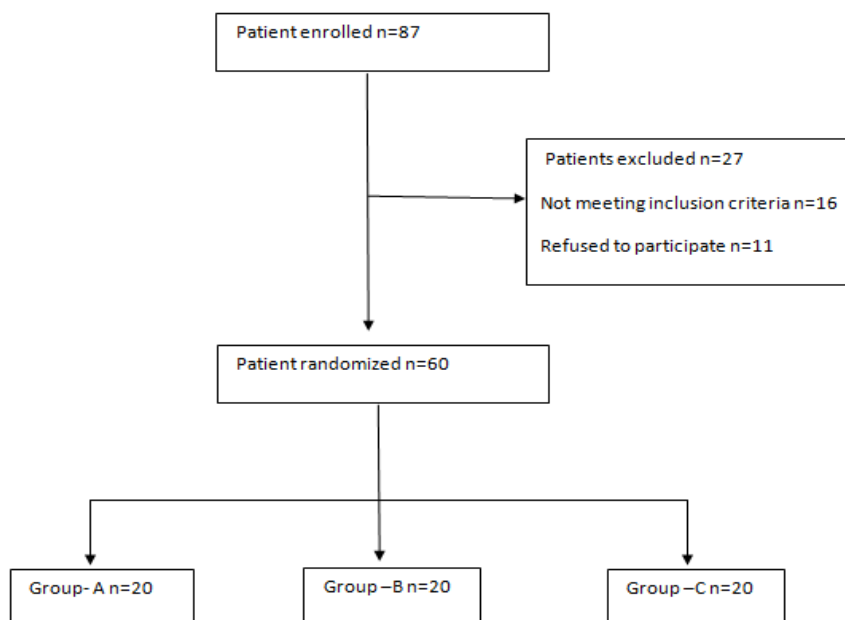


Fig. 1: Patients' flowchart. Eighty-seven patients were considered for participation in the study; 16 were excluded because they did not fulfill the study criteria, while 11 others declined to participate, after which 60 patients underwent randomization. A few patients were lost for determination of secondary end-points but not for primary endpoints.

All patients were transported to the operating room, premedication with midazolam. On arrival to operating room, an 18-gauge intravenous (IV) catheter was inserted and 6ml/kg/h crystalloid was infused intraoperatively, monitoring of electrocardiography, non-invasive blood pressure, oxygen saturation (SpO₂) was started and baseline values were recorded. All patients were premedicated with 1gm midazolam and Ketamine 2mg/kg body weight was given intravenously slowly over a period of 30-40seconds. After injecting Ketamine, time taken to obtain the surgical anaesthesia was noted. Patient was closely monitored for vital signs at 2, 5 and every 5 minutes thereafter including airway maintenance. Patient was also observed for side effects. The time taken by the patient to recover from the initial dose was noted. A subsequent dose of Ketamine was given if the procedure was continued further. The duration of action of the subsequent dose was also noted.

Once the procedure was over, the patient was allowed to recover in a recovery room. The patients were said to be completely recovered from anaesthesia when they started responding to oral commands. They were then shifted to the ward.

Patients characteristics like, age, sex, height, ASA class were recorded. Vital parameters including heart, blood pressure, respiratory rate and oxygen saturation were recorded prior to injection of ketamine. Intraoperative anaesthesia were recorded. Intraoperative adverse affects viz. nausea, vomiting, hypotension, bradycardia and respiratory depression were recorded. Duration of surgery was also recorded.

At the end of operation, the quality of anaesthetic conditions produced was graded as shown in table 1:

Table 1: Quality of anaesthetic conditions.

Excellent	Patients remained calm. There were no movements, no interference with surgery.
Good	Patients remained calm. Minor movements not interfering with surgery.
Fair	Moderate movements interfered with surgery but no supplementary anaesthesia was required.
Poor	Patients required conventional general anesthesia.

The patients were interviewed the next day morning for any unpleasant dreams or untoward experience during recovery or during their sleep over night.

Statistical analysis was performed using Microsoft (MS) Office Excel Software (Microsoft Microsoft Excel, Redmond, Washington: Microsoft 2003, Computer software). Results were expressed as mean \pm standard

deviation, number and percentage (%). Data were analysed using *post hoc* analysis method. Normally distributed data were assessed using unpaired Student's *t*-test (for comparison of parameters among groups). Comparison was carried out using Chi-square (χ^2) test with a *P* value reported at 95% confidence level. Level of significance used was *P* = 0.05.

RESULTS

Table 1: Age.

Age	N	Mean	SD	Range	P-value	Remarks
Group A	20	42.5	16.259	19-69	0.649	Not sig.
Group B	20	38.4	11.315	20-60		
Group c	20	49.8	14.896	22-68		

In group A, age ranged from 19 to 69 years with a mean age of 42 ± 16.259 years. In group B, age ranged from 20 to 60 years with a mean age of 38.4 ± 11.315 years and in group C, age ranged from 22 to 68 years with a mean age of 39.8 ± 14.896 years. The statistical analysis between three groups was not significant ($p = 0.649$).

All the patients in all three groups were comparable regarding the gender of the patients and the variation in gender distribution between groups was statistically insignificant ($p = 0.720$).

Table 2: Sex Distribution.

Sex		Group		
		A	B	C
Male	Count	13	05	15
	%age	65	75	75
Female	Count	07	05	05
	%age	35	25	25
Total	Count	20	20	20
	%age	100	100	100
P- Value =0.720				

Table 3: Distribution of Height.

Height	N	Mean	SD	Range	P-Value	Remarks
Group A	20	166.3	4.610	157-175	0.264	Not sig.
Group B	20	168.4	5.547	160-179		
Group C	20	166.1	4.407	158-174		

In group A, height ranged between 157-175cm with a mean height of 166.3 ± 4.61 cm. In group B, height ranged between 160-179cm with a mean height of 168.4 ± 5.547 cm and in group C, height ranged between 158-174cm with a mean height of 166.1 ± 4.407 cm. When the values were compared statistically the difference was found to be insignificant ($p = 0.264$).

Majority of patients in the study population belonged to ASA class-I in all the three groups. The variation in ASA class distribution of patients among different groups was statistically insignificant ($p = 0.431$).

Table-4: ASA Class of patients.

ASA		Group		
		A	B	C
ASA-I	Count	15	17	18
	%age	75	85	90
ASA-II	Count	5	3	2
	%age	25	15	10
Total	Count	20	20	20
	%age	100	100	100

P-Value =0.431

Table 5: Duration of Surgery.

Group	N	Mean	SD	Range	P-Value	Remarks
A	20	35.0	2.5	30-40 (min)	0.12	Not Sig
B	20	33.5	2.2	27-40 (min)	0.11	Not Sig
C	20	34.0	2.0	28-40 (min)	0.10	Not Sig

Duration of surgery ranged between 30-40 minutes with a mean duration of 35.0 ± 2.5 minutes in group A, 27-40 minutes with a mean duration of 33.5 ± 2.2 minutes in

group B and 28-40 minutes with a mean duration of 34.0 ± 2.0 minutes in group C. The statistical difference between the groups was insignificant ($p > 0.001$).

Table 6: list of surgeries.

G Groups	Type of surgery	No. of cases
Group A n= 20	General surgery	20
I	Secondary suturing	2
II	Stunt removal	2
III	Skin grafting	3
IV	Fibro adenoma	2
V	Lipoma	1
VI	Wound debridement	2
VII	Lymph node biopsy	3
VIII	Cystoscopy	2
IX	Sebaceous cyst	3
Group B n= 20	Orthopedic surgery	20
I	External fixator removal	3
II	Closed reduction	4
III	Femoral pinning	2
IV	Amputations	3
V	Disarticulation	2
VI	K- wire removal	1
VII	Biopsies	5
Group C n= 20	Gynecological surgeries	20
I	Abdominal tubectomy	5
II	I and D	3
III	Perineal tear repair	4
IV	D and C	8

Table 7: Time of onset of anesthesia.

Group	N	Mean	SD	Range	P-value	Remarks
A	20	42.5	5.5	25-60 sec	0.27	Not Sig
B	20	40.0	5.7	22-58 sec	0.28	Not Sig
C	20	39.0	5.7	21-57 sec	0.28	Not Sig

The time of onset of anaesthesia was taken from the time of intravenous administration of Ketamine to surgical anaesthesia. The time of onset of anaesthesia ranged from 25 to 56 sec with a mean of 42.5 ± 5.5 in group A,

22 to 58 sec with a mean of 40.0 ± 5.7 in group B and 21 to 57 sec with a mean of 39.0 ± 5.7 in group C. The statistical difference among the study groups was insignificant with a p value of (>0.001).

Table 8: Duration of onset of Anesthesia.

Group	N	Mean	SD	Range	P-value	Remarks
A	20	32.2 ± 7.9 sec	7.9	20-40 (min)	0.39	Not Sig
B	20	33.3 ± 8.0 sec	8.0	20-41 (min)	0.40	Not Sig
C	20	34.3 ± 8.4 sec	8.5	21-42 (min)	0.42	Not Sig

The mean duration of onset ranged from 20 to 40 minutes with a mean of 32.2 ± 7.9 sec in group A, 20 to 41 minutes with a mean of 33.3 ± 8.0 sec in a group B

and 21 to 42 minutes with a mean of 34.3 ± 8.4 sec in group C. There was no static difference among the study groups with a p value of (>0.001).

Table 9: Duration of action of initial dose.

Group	N	Mean	SD	Range	P-value	Remarks
A	20	15.90 ± 3.0	3.0	9-18 (min)	0.15	Not Sig
B	20	16.00 ± 3.5	3.5	8-20 (min)	0.17	Not Sig
C	20	16.9 ± 2.9	2.9	9-20 (min)	0.14	Not Sig

The duration of action of initial dose of Ketamine after intravenous administration was noted. The least duration was 9 min, and maximum was 18 min with a mean of 15.90 ± 3.0 in group A, 8 to 20 minutes with a mean of

16.00 ± 3.5 in group B and 9 to 20 minutes with a mean of 16.9 ± 2.9 in group C. The statistical difference was insignificant among the study groups ($p > 0.001$).

Table 10: Duration of action of subsequent dose.

Group	N	Mean	SD	Range	P-value	Remarks
A	20	10.5±2.00	2.00	8-13 (min)	0.50	Not Sig
B	20	11.5±2.50	2.50	9-14 (min)	0.57	Not Sig
C	20	9.6±1.5	1.5	7-12 (min)	0.48	Not Sig

45 patients required only the initial bolus dose while the other 15 patients required a top up dose. The subsequent dose repeated was half the initial dose. The duration of action of the subsequent dose ranged from 8 to 13 minutes with a mean of 10.5±2.00 minutes in a group A, 9 to 14 minutes with a mean of 11.5±2.50 minutes in a group B and 7 to 12 minutes with a mean of 9.6±1.5 minutes in a group C. The statistical difference was insignificant among the study groups ($p>0.001$).

Table 11: Quality of Intra operative anesthesia.

Quality of Intra operative anesthesia	Group		
	A	B	C
Excellent	17	18	18
Good	03	02	02
Fair	0	0	0
Poor	0	0	0
Total	20	20	20

Above table shows 17 patient in group A had an excellent and 3 patients had good anesthesia. In group B, 18 patients had excellent and 2 patients had good anaesthesia. In group C 18 patients had an excellent and 2 patients had good anesthesia. The statistical difference between the groups was not significant ($p=0.851$).

Table 12: Intraoperative pulse rate.

Group	N	Mean	SD	Range	P-value	Remarks
A	20	15.0±5.0	5.0	10-20	0.002	Sig
B	20	15.5±5.5	5.5	9-21	0.001	Sig
C	20	14.5±4.7	4.7	8-19	0.01	Sig

In the study, all the patients showed rise in pulse rate. The mean rise in pulse rate was 15.0 ± 5.0 beats per min in group A, 15.5 ± 5.5 mean rise in pulse rate in group B

and 14.5 ± 4.7 mean rise in pulse rate in group C. The rise was statistical significant ($P < 0.001$) at 5 min and the pulse rate came to preanaesthetic levels at 30-35 min.

Table 13: Intraoperative systolic blood pressure.

Group	N	Mean	SD	Range	P-value	Remarks
A	20	18	3.8	6-30(mmHg)	0.009	Sig
B	20	21	4.4	7-35(mmHg)	0.002	Sig
C	20	24	5.02	8-40(mmHg)	0.005	Sig

Changes in systolic blood pressure: Maximum rise in systolic pressure was 30 mm Hg and minimum was 6 mmHg with a mean of rise 18.0 ± 3.8 in group A, 35 to 7 mmHg with a mean of rise 21.0 ± 4.4 in group B and 40 to

8 mmHg with a mean of rise 24.0 ± 5.02 in group C. There was a statistically significant rise ($P < 0.001$) in systolic blood pressure at 5 min and rise in systolic blood pressure came to its preanaesthetic level at 30 min.

Table 14: Intraoperative diastolic blood pressure.

Group	N	Mean	SD	Range	P-value	Remarks
A	20	12.1±8.8	8.8	0-30(mmHg)	0.022	Sig
B	20	11.2±7.8	7.8	0-29(mmHg)	0.019	Sig
C	20	12.0±8.0	8.0	0-28(mmHg)	0.02	Sig

Changes in diastolic blood pressure: The maximum rise in the diastolic blood pressure observed was 30 mm Hg and the minimum was 0 mm Hg with the mean rise being 12.1 ± 8.8 mm Hg in group A, 29mmHg and 0mmHg with a mean rise of 11.2 ± 7.8 mmHg in group B and 28mmHg

and 0 mmHg with a mean rise of 12.0 ± 8.0 in group C. There was a statistically significant rise at 5 min and the rise in diastolic blood pressure came to preanaesthetic value at 30min.

Table 15: Intra operative respiratory rate.

Group	N	Rise	Decrease/Apnea	No change	P-value	Remarks
A	20	05	02	13	0.006	Sig
B	20	06	03	11	0.003	Sig
C	20	07	02	11	0.002	Sig

There was increase in respiratory rate in 05 patients and decrease (apnea) in 02 patients in group A, increase in respiratory rate in 06 and decrease in 03 patients in group

B and rise in respiratory rate in 07 patients and decrease in 02 patients in group C. In 35 patients there was no significant change in all the study groups.

Table 16:

Variables	Group A	Group B	Group C	P-value	Remarks
Vomiting	03	04	02	0.45	Not Sig
Hallucination	04	03	02	0.40	Not Sig
Dreams	05	04	03	0.55	Not Sig

Postoperative side effects

Vomiting was seen in 03, 04 and 02 patients in group A, B and C respectively, Hallucinations was present in 04, 03 and 02 patients in group A, B and C respectively and dreams was present in 05, 04 and 03 patients in group A, B and C respectively. Among them, some were pleasant and some were horrifying dreams.

DISCUSSION

Ketamine, recently introduced by Parke, Davis and Co, is a non-barbiturate general anaesthetic agent which may be given intravenously or intramuscularly. The drug acts rapidly and produces marked analgesia without cardiovascular depression or significant respiratory depression; laryngeal and pharyngeal reflexes are said to be well maintained. The anaesthetic state produced by ketamine differs from that achieved with conventional anaesthetic agents. It is suggested that the drug produces somesthetic sensory blockade by selectively interrupting association pathways of the brain and this has led to the introduction of the term 'dissociative' anaesthesia.^[22] An increasing interest in intravenous anaesthetic techniques has resulted from the availability of more efficacious intravenous agents, the rising cost of traditional volatile agents, and the concern over anaesthetic gas pollution in the operating room. Infact, Ketamine is the only available agent which can function as a sole anaesthetic because of its unique sedative, amnesic, analgesic, and anaesthetic properties and thus Ketamine may prove to be a useful addition to the armamentarium of an anaesthetist.^[23,24] In a study conducted by Dhar et al on 110 patients undergoing minor surgical procedures using Ketamine as sole anaesthetic agent, it was observed that the incidence and severity of the unwanted side effects of Ketamine hydrochloride was significantly reduced by prior administration of diazepam intravenously.^[25] So, we have taken midazolam 0.2mg/kg IV as premedication in our study.

A study by White P F et al stated that Ketamine 2mg/kg IV, given as a rapid bolus injection produced significant reduction in PaO₂. In contrast, premedicated patients with Diazepam, Who received Ketamine 2mg/kg IV over 60 seconds showed no significant change in PaO₂.^[26] So, in our study we have injected Ketamine 2 mg/kg body weight IV, over 30 to 40 seconds.

In the present study, the time of onset of anaesthesia ranged from 25 to 60 sec with a mean of 42.5±5.5 in group A, 22 to 58 sec with a mean of 40.0±5.7 in group B

and 21 to 57 sec with a mean of 39.0±5.7 in group C. The statistical difference among the study groups was insignificant with a p value of (>0.001). Which is in acceptance to the studies conducted by Diwale et al 60 sec.^[27] In our study, the mean duration of onset ranged from 20 to 40 minutes with a mean of 32.2±7.9 sec in group A, 20 to 41 minutes with a mean of 33.3 ± 8.0 sec in a group B and 21 to 42 minutes with a mean of 34.3±8.4 sec in group C. There was no statically difference among the study groups with a p value of (>0.001). correlates with Knox et al 13.2 ± 1.25 and Diwale et al^[42] 5-17.^[27,28]

A study by Gudi et al stated that when the surgery was prolonged or when patients were coming out of the initial dose, subsequent dose of 1mg/kg of Ketamine was given.^[29]

Even in our study, when the time of surgery was prolonged or when the patient came out of the initial dose, subsequent dose of 1mg/kg of Ketamine was given in 15 patients required a top up dose. The subsequent dose repeated was half the initial dose. The duration of action of the subsequent dose ranged from 8 to 13 minutes with a mean of 10.5±2.00 minutes in a group A, 9 to 14 minutes with a mean of 11.5±2.50 minutes in a group B and 7 to 12 minutes with a mean of 9.6±1.5 minutes in a group C. The statistical difference was insignificant among the study groups (p>0.001).

Our study has similar observations of 74% excellent, 19% good, 7% fair and no case had poor anaesthetic condition. 17 patient in group A had an excellent and 3 patients had good anaesthesia. In group B, 18 patients had excellent and 2 patients had good anaesthesia. In group C 18 patients had an excellent and 2 patients had good anaesthesia. The statistical difference between the groups was not significant (p=0.851).

Airway and Jaw tone were well maintained during anaesthesia in our study. Patients were able to swallow during the course of anaesthesia. This is in correlation with Morgan M et al.^[30]

Vomiting was seen in 03, 04 and 02 patients in group A, B and C respectively, Hallucinations was present in 04, 03 and 02 patients in group A, B and C respectively and dreams was present in 05, 04 and 03 patients in group A, B and C respectively. Among them, some were pleasant and some were horrifying dreams. In the present

study, dreams were present in 5% and this is not in correlation with any authors. This is because dreams depends on the racial, geographical, nutritional and psychological makeup of patient in study.^[31] As the above studies were done in patients of different race, different geographical area, the finding were different. Ketamine has certain absolute contraindications like recent myocardial infarction and uncontrolled hypertension. Nevertheless, its cardiostimulatory properties can be exploited in hypovolemic shock and critically ill patients where blood pressure is low and life threatening. Remarkable preservation of pharyngeal and laryngeal reflexes was an asset in Ketamine anaesthesia, as it can be used where difficult intubation is anticipated. As far as the cost of anaesthesia is concerned, it is economical for routine use particularly for procedures of shorter duration. However it also plays a very useful role in mass casualties and places where sophisticated anaesthetic equipment is not found as in warzones and high altitudes.

Although the present study is not to rewrite the indications of Ketamine it is only to reiterate its place in the anaesthetic armamentarium. Studies on Ketamine have established the usefulness of the anaesthetic agent in a variety of clinical conditions and have confirmed the safety, reliability and effectiveness of Ketamine as sole anaesthetic agent in short surgical procedures.

Conflict of Interests

The authors declare that there is no conflict of interests regarding publication of this paper.

BIBLIOGRAPHY

- Dhar CL et al. Ketamine as a sole anaesthetic agent in minor surgical procedures. *Ind J Anesth*, 1983; 31: 20-24.
- Morales JA, Miranda GF. Ketamina (Ketamine). *Rev-Esp-Anesthesiol-Reanim*, 1999; 46(3): 111-22.
- Budavari S, O'Neil MJ, Smith A, Heckelman PE (eds.). *The Merck index. An encyclopedia of chemicals, drugs and biologicals*. 11th Edition. Merck & Co. Inc.: Rahway, New Yersey, USA, 1989.
- Kim EM, Lee JS, Choi SK, Lim MA, Chung HS. Analysis of ketamine and norketamine in urine by automatic solid-phase extraction (SPE) and positive ion chemical ionization-gas chromatography-mass spectrometry (PCI-GC-MS). *Forensic Sci Int.*, 2008 Jan 30; 174(2-3): 197-202.
- Domino EF, Chodoff P, Corssen G. Pharmacological effects of CI-581, a new dissociative anaesthetic, in man. *Clin Pharmacol Ther.*, 1966; 6: 279-291.
- Bergman SA. Ketamine: review of its pharmacology and its use in pediatric anesthesia. *Anesth Prog*, 1999; 46: 10-20.
- Vollenweider FX, Leenders KL, Øye I, Hell D, Angst J. Differential psychopathology and patterns of cerebral glucose utilisation produced by (S)- and (R)-ketamine in healthy volunteers using positron emission tomography (PET). *Eur Neuropsychopharmacology*, 1997; 7: 25-38.
- Engelhardt W. Recovery and psychomimetic reactions following S-(+)-ketamine. *Anaesthesist*, 1997; 46(1): S38-42.
- Freo U, Ori C. Opioid pharmacology of ketamine. *Acta Anaesthesiologica Italica/Anaesthesia & Intensive Care in Italy*, 2002; 53(3): 149-163.
- Crisp T, Perrotti JM, Smith DL, Stafinsky JL, Smith DJ. The local monoaminergic dependency of spinal ketamine. *Eur. J. Pharmacol*, 1991; **194**: 167-72.
- Shimoyama M, Shimoyama N, Gorman AL, Elliott KJ, Inturrisi CE. Oral ketamine is antinociceptive in the rat formalin test: role of the metabolite, norketamine. *Pain.*, 1999; 81: 85-93.
- Haas DA, Harper DG. Ketamine: a review of its pharmacologic properties and use in ambulatory anesthesia. *Anesth Prog*, 1992; 39: 61-8.
- White JM, Ryan CF. Pharmacological properties of ketamine. *Drug Alc Review*, 1996; 15: 145-155.
- Reich DL, Silvay G. Ketamine: an update on the first twenty-five years of clinical experience. *Can J Anaesth*, 1989; 36: 186-97.
- Haas DA, Harper DG. Ketamine: a review of its pharmacologic properties and use in ambulatory anesthesia. *Anesth Prog*, 1992; 39: 61-8.
- Weiner AL, Vieira L, McKay CA, Bayer MJ. Ketamine abusers presenting to the emergency department: a case series. *J Emerg Med.*, 2000; 18: 447-51.
- Reich DL, Silvay G. Ketamine: an update on the first twenty-five years of clinical experience. *Can J Anaesth*, 1989; 36: 186-97.
- White JM, Ryan CF. Pharmacological properties of ketamine. *Drug Alc Review*, 1996; 15: 145-155.
- Reich DL, Silvay G. Ketamine: an update on the first twenty-five years of clinical experience. *Can J Anaesth*, 1989; 36: 186-97.
- White JM, Ryan CF. Pharmacological properties of ketamine. *Drug Alc Review*, 1996; 15: 145-155.
- Bergman SA. Ketamine: review of its pharmacology and its use in pediatric anesthesia. *Anesth Prog*, 1999; 46: 10-20.
- Coarsen, O. and Domino, E. P. Dissociative anesthesia: Further pharmacologic studies and first clinical experience with the phencyclidine derivative CI-581. *Anesthesia and Analgesia, Current Researches*, 1966; 45,29.
- White PF, Way WL, Trevor AJ. Ketamine – its pharmacology and therapeutic uses. *Anesthesiology*, 1982; 56: 119-136.
- Dhruva AJ, Dasgupta D, Modi KJ. Dissociative anaesthesia - preliminary experience with Ketamine hydrochloride. *Ind J Anaesth*, 1971; 50-57.
- Dhar CL, Charak D, Hashia AM, Magazine C. Clinical studies on Ketamine hydrochloride-II : Ketamine-Diazepam Combination in Minor Surgical Procedures. *Ind J Anaesth*, 1983; 31(1): 26-30.

26. White PF, Ham J, Way WL, Trevor AJ. Pharmacology of Ketamine isomers in surgical patients. *Anesthesiology*, 1980; 52: 231-239.
27. Diwale DB, Moulick NB, Bhatt PN, Matta JS, Bhalla SK. Comparative evaluation of Ketamine and ketamine-Diazepam in cardiac catheterization. *Ind J Anaesth*, 1983; 31(2): 132-139.
28. Knox JWD, Bovill JG, Clarke RSJ, Dundee JW. Clinical studies of induction agents XXXVI: KETAMINE. *Br J Anaesth*, 1970; 42: 875.
29. Gudi AR, Bhide VV, Dabir SA. Clinical experiences of Ketamine anaesthesia. *Ind J Anaesth*, 1973; 150-161.
30. Morgan M, Loh L, Singer L, Moore PH. Ketamine as the sole anaesthetic agent for minor surgical procedures. *Anaesthesia*, 1971; 62(2): 158-165.