DETERMINATION OF DOSE AND EFFICACY OF TRACURIUM FOR RAPID SEQUENCE INDUCTION OF ANESTHESIA: ANDOMIZED PROSPECTIVE STUDY

By

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“DETERMINATION OF DOSE AND EFFICACY OF ATRACURIUM FOR RAPID SEQUENCE INDUCTION OF ANAESTHESIA: A RANDOMIZED PROSPECTIVE STUDY"

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IN

ANAESTHESIOLOGY

ABBREVATIONS

ASA- American Society of Anaesthesiologists

ECG- Electrocardiogram

HR- Heart rate

BP- Blood Pressure

I.V- Intravenous

Inj - Injection

NIBP- Non-invasive Blood Pressure

SPO_2- Oxygen Saturation

S.D- Standard Deviation

PNS-Peripheral Nerve Stimulator

mcg- Microgram

mg- Milligram

kg- Kilogram

mL- Millilitre

hrs- Hours

mins- Minutes

P - 'p' value

Sl. No.- Serial number

SBP:- Systolic blood pressure

DBP:- Diastolic blood pressure

TOF- Train of four

PTC- Post tetanic contractions

ABSTRACT

INTRODUCTION: Rapid sequence induction and intubation (RSII) is used to induce anesthesia quickly in individuals who are at high risk of aspiration.[1] The major goal of the approach is to shorten the interval between the loss of protective airway reflexes and tracheal intubation using a cuffed endotracheal tube. Succinylcholine and high dose rocuronium were recognised as traditional neuromuscular blocking drugs because of their rapid and reliable onset of action (NMBAs). Atracurium, an intermediate-looking Neuromuscular Blocking Drug (NMBD), is used to create the ideal intubating conditions. The effectiveness of NMBAs is inversely correlated with the rate at which they start to function. A bigger dose of atracurium—roughly three to four times its ED95—would therefore make tracheal intubation go more quickly than it would with a high dose of rocuronium. Present study is designed the current study to evaluate the dosage and effectiveness of atracurium for rapid sequence anesthesia induction.

AIMS & OBJECTIVES

Aim: To determine the dosage and efficacy of atracurium without priming for rapid sequence intubation of anesthesia.

Objectives:

- To compare different doses of atracurium [0.75mg/kg, 1mg/kg] without priming dose.
- To determine difficulty in the passage of tube .

- To estimate the complications associated with different doses.

MATERIALS AND METHODS: This study included 112 patients who were scheduled to undergo elective surgery under general anaesthesia with endotracheal intubation and required intraoperative neuromuscular blockade. The study subjects were randomly split into 2 groups of 56 each.

Group A: 56 patients receiving 0.75mg/kg [3ED95]

Group B: 56 patients receiving 1 mg/kg [4ED95].

Atracurium was given in varying doses—0.75 mg/kg (3ED95) for group A patients and 1 mg/kg (4ED95) for group B patients. The circulatory strain, pulse, and heartbeat oximeter were recorded each moment for 10 minutes, trailed by observing like clockwork after the enlistment of sedation.

RESULTS: Compared to the lower doses, the mean arterial pressure significantly decreased at 1, 3, and 5 minutes after the onset of anaesthesia. In comparison to the lower doses, the mean heart rate, significantly increased at 1-, 3-, and 5-minutes following anaesthesia induction. The SBP and DBP were significantly lower at 1, 3, and 5 minutes following the induction of anaesthesia as compared to the lower dose. Intubating conditions had been exceptional in 55.4% of the patients in 1mg/kg atracurium group, compared to 32.1% in 0.75mg/kg group. More number of patients in 0.75mg group had excellent intubation conditions (53.6%). Only one affected person in higher dose group showed bad intubating conditions. The time of onset and spontaneous recovery have been 65.4±10.6 and85.0±11.40 min for 1mg/kg atracurium in comparison to 132.3±14.65 and 60.8 ±9.70 min in 0.75mg/kg.

CONCLUSION: Intubation had a high dose-dependent success rate. Atracurium provided excellent intubating circumstances with a shorter onset time at 1 mg/kg

Keywords: Rapid sequence induction, intubation, atracurium, neuro muscular blockade

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INTRODUCTION

A crucial component of managing general anesthesia during surgical procedures is endotracheal intubation. A tracheal intubation procedure called rapid sequence induction and intubation (RSII) is used to induce anesthesia quickly in individuals who are at high risk of aspiration.[1] The major goal of the approach is to shorten the interval between the loss of protective airway reflexes and tracheal intubation using a cuffed endotracheal tube.

Control of balanced general anaesthesia has been revolutionized by neuromuscular blocking medications (NMBDs). In the field of neuromuscular blockade, significant progress has been made since the introduction of d-tubocurarine and succinylcholine.[2] At some point during the administration of general anaesthesia, these medications offer various advantages, including the ability to perform lengthy procedures without the patient making voluntary or reflex movements. They cannot cross the blood-brain barrier, hence succinylcholine is the only substance that causes an increase in intracranial pressure. These medications allow for prolonged muscular relaxation, aid in controlling respiration, circulation, and airway management, and maintain steady hemodynamic. These medications only have little side effects, a quick conversion to inactive metabolites, and movements limited to the neuromuscular junction.[3]

The ideal neuromuscular blockading agent should have a quick onset, a short duration of action, be free of hemodynamic adjustments, leave no residual paralysis, and provide exceptional intubating conditions like a fully fixed jaw, a widely opened vocal

cord, and little response to intubation, which reduces the time for intubation and, as a result, lowers the risk of an unfavourable hemodynamic stress reaction.[4]

Due to their quick and consistent onset of action, succinylcholine and high dose rocuronium were established as classic neuromuscular blocking agents (NMBAs). Succinylcholine is not recommended for people who are hyperkalemic, as well as those who have elevated intracranial pressure (ICP) or intraocular pressure. Due to possible extended excretion and lingering neuromuscular inhibition, rocuronium must be avoided in patients with renal impairment.3,5

A non-depolarizing muscle relaxant (NDMR) with a quick onset time and excellent intubating conditions has been the focus of research.

Atracurium is a Neuromuscular Blocking Drug (NMBD) that has an intermediate appearance and is utilized to present the best intubating circumstances. The recommended dose of atracurium for tracheal intubation takes around 3 minutes to start working. The rate at which NMBAs begin to work is inversely related to how well they work. Atracurium's effectiveness was virtually as effective as rocuronium, according to prior studies. Therefore, compared to a high dose of rocuronium, a higher dose of atracurium—roughly three to four times its ED95—would facilitate quick tracheal intubation.[5] According to a report by Chalermkitpanit et al[6], the injection of an obviously high dose of atracurium without priming may be employed in some circumstances as a chance neuromuscular blockading agent for swift sequence induction of anaesthesia.

Due to the limited number of research that have been done and the paucity of available literature, we designed the current study to evaluate the dosage and effectiveness of atracurium for rapid sequence anaesthesia induction.

AIMS & OBJECTIVES

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REVIEW OF LITERATURE

PHYSIOLOGY OF NEUROMUSCULAR JUNCTION[7]

Skeletal muscle tissues are delivered by motor neurons, whose cell bodies are found in the spinal cord. The motor neurons' axons help carry information from the central nervous system (CNS) from the spinal cord to distant parts of the body where they supply numerous muscle cells (or fibers). Acetylcholine is produced and released with the assistance of a unique structure called a synapse, which is located at the terminal regions of the axon. The synaptic cleft, or thin space between the endplate of the muscle fiber and the synapse, measures about 50 nm in width.

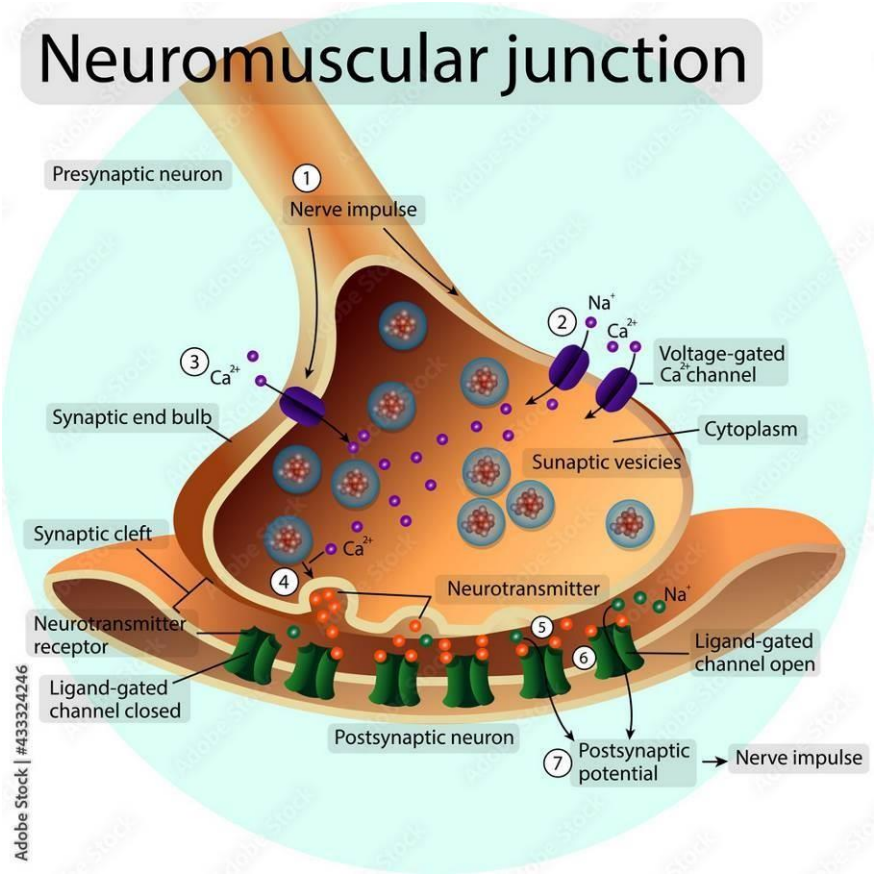


Fig 1: Neuromuscular Junction

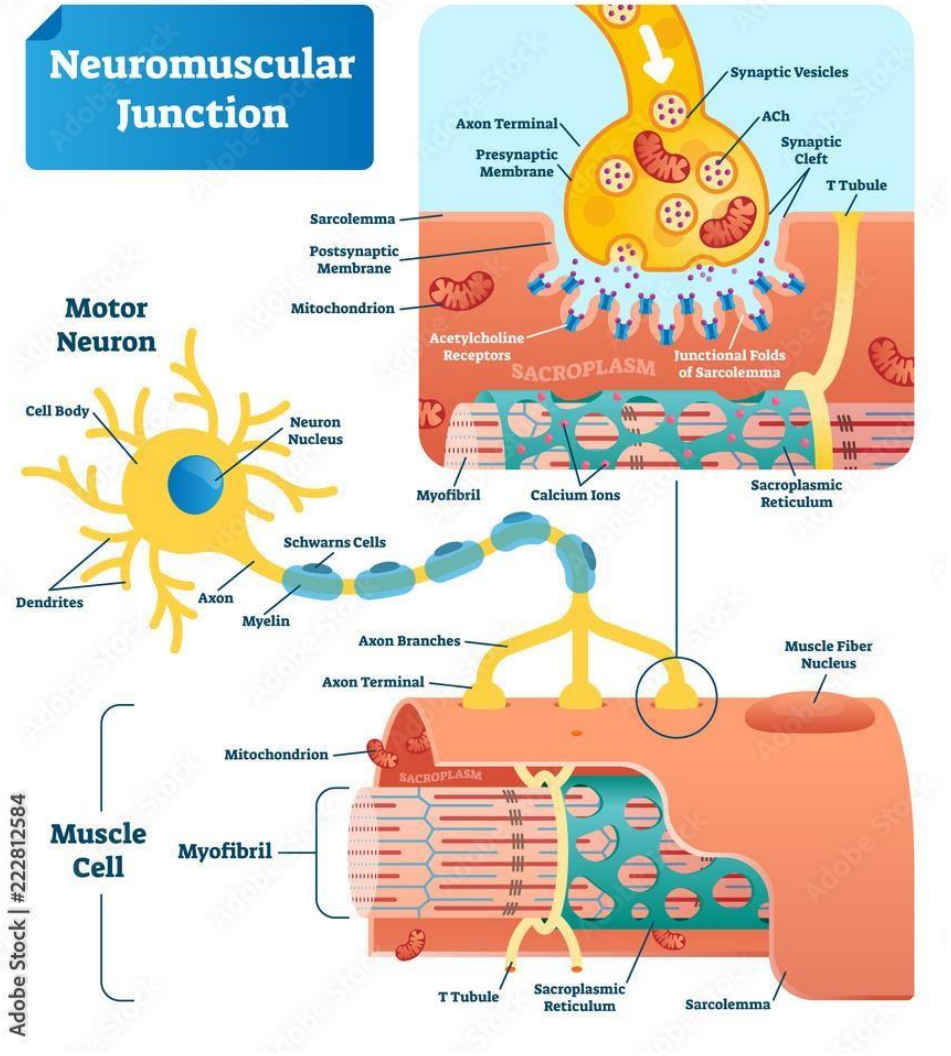


Fig 2: Physiology of Neuromuscular Junction

Neuromuscular junction includes two additives:

A nerve terminal frames the presynaptic portion of the neuromuscular junction, and a muscle terminal primes the postsynaptic structure.

The synaptic cleft is located between the two.

The nerve terminal loses its myelin coating at the neuromuscular junction and is isolated from the fluid around it by one or more Schwann cells. The presynaptic membrane (the nerve terminal's membrane directly across from the Muscle terminal)

is layered to produce active zones as it becomes thicker. Acetylcholine-containing vesicles are grouped up against these active zones. Additionally, voltage-gated calcium channels are arranged along the sidewalls of these active zones. Voltage gated calcium channels activate when an action potential reaches a nerve terminal, causing a significant calcium ion influx. Acetylcholine molecules are released into the synaptic cleft as a result of calcium ions' attractive pressure on the vesicles grouped in zone 1. The acetylcholine molecules are delivered in quanta, which are evenly sized packets. By raising intracellular calcium levels, one can increase the frequency of these quantas. Large sized vesicles are present as the reserve pool in Zone 2. These vesicles are produced when the nerve is repeatedly stimulated, increasing the amount of acetylcholine that is available for impulse transmission.

Acetylcholine receptors are present at the nerve terminal, or the presynpatic Ach receptor. The mobilisation of vesicles from their storage site to active sites is probably something they are participating in.

Synaptic Cleft

Between the presynaptic and postsynaptic membranes is where it is located. It measures 20 to 30 nanometers in length and is also known as a junctional cleft. It is made up of a thin layer of reticular fibres that are filled with ECF. These fibres serve to firmly hold the terminals of muscular nerves together.

When a nerve impulse (action potential) travels via the axon into the nerve terminal, acetylcholine, the transmitter at the neuromuscular junction, is released from the presynaptic nerve ends. Choline acetyltransferase is used to create it from choline and acetate, and it is then stored in the terminal's vesicles. Each of these 45 nm-long vesicles contains between 5000 and 10,000 acetylcholine molecules.

Acetylcholine is released in quanta, or packets, and each quantum corresponds to a single vesicle's contents. 200 to 400 quanta, or 1 to 4 million acetylcholine molecules, are released into the synaptic cleft during an action potential. The binding of vesicles, their fusion, and the release of acetylcholine all depend on calcium.

If the calcium concentration is lower or if magnesium is antagonistic to calcium, the release process may be inhibited.

Postsynaptic modifications[7,8]

Nicotinic cholinergic receptors make up the muscular endplates. These receptors are distributed across the cellular membrane and are made of five glycoprotein subunits arranged in the shape of a rosette. The two identical α nicotine subunits and three additional β, δ and ε subunits, make up the nicotinic subtype at the neuromuscular junction. Acetylcholine binding sites are located on the outside of the 2 subunits. A hole forms in the centre of the rosette when two acetylcholine molecules attach simultaneously to each binding site, allowing cations like sodium and potassium to migrate along concentration gradients.

Because of the negative voltage inside the cell, the fundamental alteration is the movement of sodium ions into the cellular part. This results in the endplate's interior portion being less negative, which causes depolarization. The perijunctional region and the folds of the synaptic cleft contain an excessive density of sodium channels, which open when the membrane depolarization reaches a critical point and further depolarize the cell by allowing sodium access. Through the activation of the sodium channels, this depolarization generated an action potential that travelled the whole length of the muscle fibre.

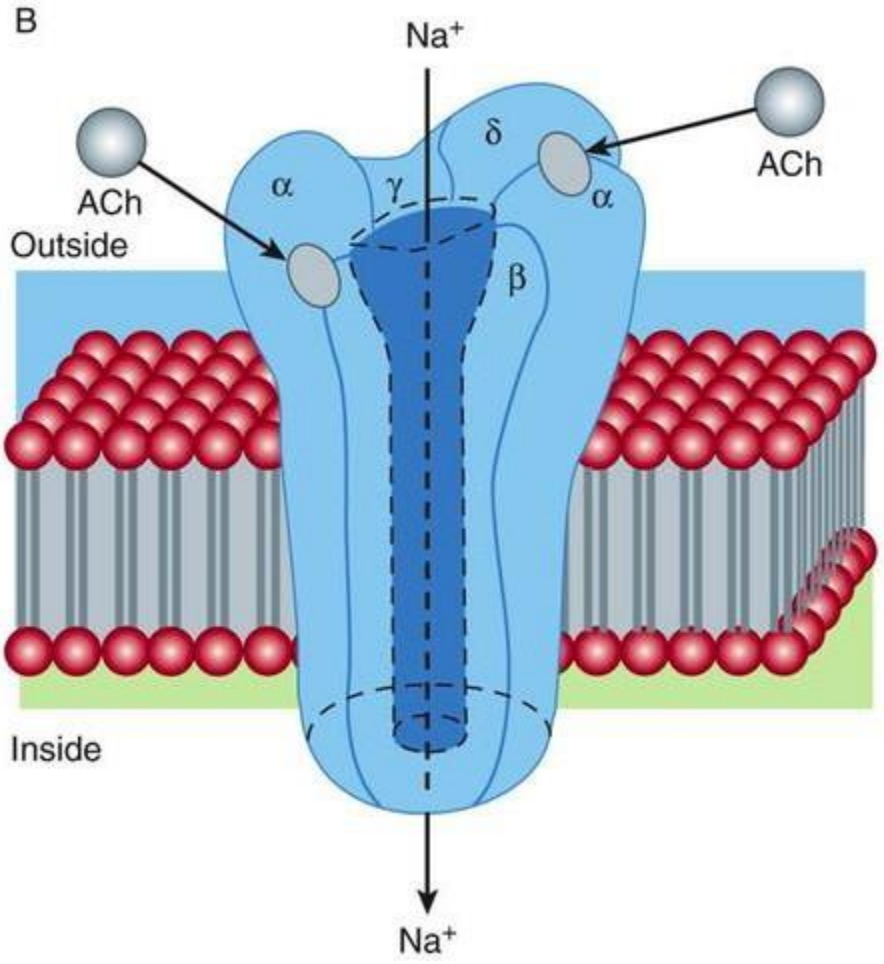


Fig 3: Nicotinic Acetylcholine receptor

PHARMACOLOGY OF NEUROMUSCULAR BLOCKING DRUGS[8]

The primary pharmacological action of neuromuscular blocking medications is to obstruct nerve impulse transmission at the neuromuscular junction, resulting in muscle relaxation and the cessation of patient movements during surgery. These medications can be divided into depolarizing neuromuscular blocking drugs (succinyl choline) and non-depolarizing neuromuscular blocking pharmaceuticals based on the clear differences in their mechanisms of action.

Non depolarizing neuromuscular blocking drugs may be categorised on the basis in their action into long, intermediate and short acting tablets. The non depolarizing

neuromuscular blocking drugs can also be categorized based on chemical class (steroidal, benzylisoquinolinium or different compounds).

Mechanism of action of non-depolarizing neuromuscular blockading drugs:

Non depolarizing neuromuscular blocking medicines work by interacting with nicotinic acetylcholine receptors at the neuromuscular junction without activating those ion receptor channels to block neuromuscular junction activity. Without causing a change in the structure of those receptors, they compete with acetylcholine for binding to the subunit of the post junctional nicotinic acetylcholine receptors. However, the movements on the post junctional sites are highly essential. Non depolarizing neuromuscular blocking medicines also operate on the pre junctional nicotinic acetylcholine receptors.

Non-depolarizing neuromuscular blockade characteristics[9-11]

Characteristic skeletal muscle responses to electrical stimulation from a peripheral nerve stimulator in the presence of non-depolarizing neuromuscular inhibition include:

a) Reduce the twitch reaction to a single stimulus

b) Unsustained response under continuous stimulation (fade)

c) After tetanic potentiation

d) Other non-depolarizing neuromuscular blocking medications are potentiated

e) Conflict brought on by anticholinesterase medications

Drugs taken before, during, or after surgery may enhance the effects of non-depolarizing neuromuscular blocking drugs. These include:

1. Antibiotics with aminoglycosides

2. Aerosolized anaesthetics

3. Diuretics

4. Regional anaesthetics

5. Magnesium

6. Lithium

Non-drug factors that affect how much neuromuscular blockade is reduced by non-depolarizing neuromuscular blocking medications include the following:

1. Hypothermia

2. Acid-base modification

3. Adrenocortical malfunction

4. Thermal damage

5. Allergic responses

Non-depolarizing intermediate-performing neuromuscular blocking medications:

The medications Atracurium, Rocuronium, Vecuronium, and Cistracurium are categorised as non-depolarizing, intermediate acting neuromuscular blockers. As opposed to long-acting, non-depolarizing neuromuscular blocking medications, these medications have an effective clearance mechanism that reduces the likelihood of a significant cumulative effect. These medicines have the following advantages over long-acting, non-depolarizing neuromuscular blocking medications:

i. most neuromuscular blockades start out similarly (with the exception of Rocuronium which has a fast onset just like Suxamethonium)

ii. roughly one-third of the action's duration

iii. 30% to 50% quicker healing

iv. little to no cumulative effects

Atracurium[12,13]

It is a non-depolarizing, intermediate acting bisquaternary benzylisoquinolinium neuromuscular blocker. The neuromuscular junction's pre and post synaptic cholinergic receptors are the site of action. Its start of effect is 3 to 5 minutes after administration, and its action lasts 20 to 35 minutes. Its ED95 is 0.2 mg/kg. (17) . 0.5 mg/kg is the intubating dose, and 0.1 mg/kg to 0.2 mg/kg is the maintenance dose. (18) It produces the release of histamine, which may cause hypotension, tachycardia, and cutaneous flushing.

At normal body temperature and pH, atracurium spontaneously degrades without the aid of enzymes in a process known as Hofmann elimination. Hydrolysis by universal plasma esterases is a second pathway of metabolism that is active concurrently. These pathways are unaffected by renal and hepatic function. As a result, both healthy individuals and those with impaired renal or hepatic function experience the same length of atracurium-triggered neuromuscular blockade. The best metabolite of atracurium's two metabolic routes is laudanosine. About 70% of laudanosine's excretion in the bile and the remaining percentage in the urine is dependent on the liver for clearance.

PHARMACOLOGY OF ATRACURIUM[13]

It is an intermediate acting, non-depolarizing, neuromuscular blocking bisquarternary benzyl isoquinolinium medication. The substance is a racemic blend of ten stereoisomers.

Excretion & Metabolism:

Due to atracurium's considerable metabolization, less than 10% of it is eliminated intact via the biliary and renal routes, which suggests that its pharmacokinetics are independent of renal and hepatic pathways. The metabolism is controlled by two different methods.

Hydrolysis of an ester

Non-specific plasma esterases catalyse this action.

Hofmann's Exclusion:

Spontaneous non-enzymatic chemical breakdown employing a base-catalyzed process at physiological pH and temperature that results in the metabolites acrylate and laudanosine.

Dosage:

The recommended intubating dose is 2 ED 95, which has an ED 95 of 0.2 mg/kg, a time to first effect of 3 to 5 minutes, and a time to last effect of 20 to 35 minutes. Succinylcholine is administered following succinylcholine with an initial dose of 0.25 mg/kg, followed by increasing doses of 0.1 mg/kg every 10–20 minutes.

A 5–10 g/kg/min infusion can successfully take the place of intermittent bolus. When compared to adults, atracurium may appear shorter in children and infants.

Storage:

A solution containing 10 mg/mL of atracurium is available. It should be kept between 2 and 8 degrees Celsius because opening it to room temperature causes it to lose 5% to 10% of its efficacy per month. It must be used within two weeks of being stored at room temperature.

Adverse effects and clinical factors:[14]

At doses above 0.5 mg/kg, atracurium causes a dose-structured histamine release that becomes significant.

Tychycardia and hypotension

The only time cardiovascular side effects occur is when doses are higher than 0.5 mg/kg. A transient reduction in systemic vascular resistance is another effect of atracurium. These effects are lessened by injecting at a slow rate over five to ten minutes.

Bronchospasm:

Atracurium is typically not administered to people with bronchial asthma. Patients with a history of asthma are characterised as having severe bronchospasm.

Toxicity of Laudanosine:

The breakdown of the tertiary amine laudanosine, produced via Hofmann elimination, is linked to the central nervous system (CNS) and can cause an increase in the minimum alveolar concentration (MAC) or possibly the onset of seizures.

Laudanosine toxicity often only occurs when a patient has received a significant cumulative dose or has liver failure. The liver converts laudanosine into bile and urine for excretion.

Temperature and pH Sensitivity:

Hypothermia and acidity typically lengthen the time that Atracurium is in motion.

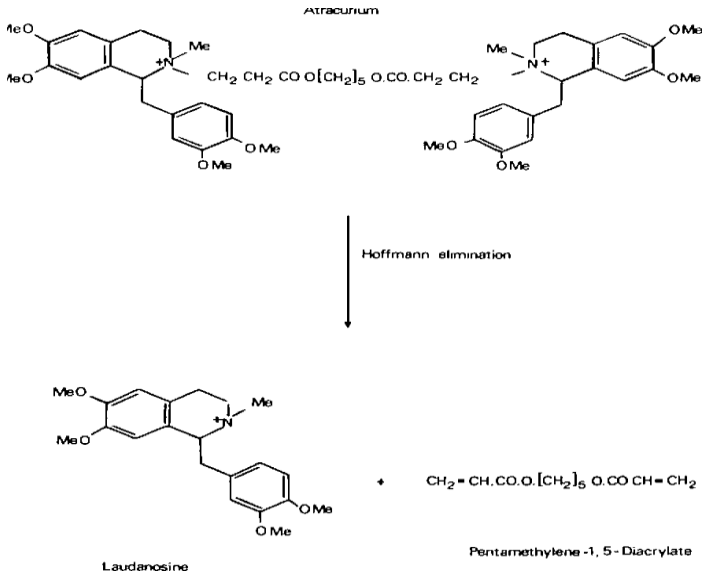
Incompatibility:

Atracurium will precipitate as a free acid if it is added straight into a line with an alkaline solution, such as thiopentone sodium**.**

Immune Reactions:

Rare anaphylactoid reactions caused by atracurium were hypothesised. The most plausible routes are acrylate-mediated immune activation and direct immunogenicity. There have been reports of IgE-mediated antibody reactions to medicines that replace ammonium, particularly treatments that limit neuromuscular activity.

CHEMICAL STRUCTURE



RAPID SEQUENCE INDUCTION (RSI)[15]

Rapid sequence induction (RSI) has been utilised for a long time for patients who are prone to aspirating stomach contents into their lungs during anaesthesia induction. It comprises intubation without face mask ventilation after a cricoid pressure-induced loss of consciousness. It is important to intubate the trachea as fast and safely as possible. This approach is employed daily during urgent surgery.

History

The dangers of pulmonary aspiration of stomach contents while unconscious have long been recognised. In a 1950 investigation of anesthesia-related mortality, the great Britain Association of Anaesthetists found 43 cases of regurgitation and aspiration-related deaths.

By 1956, there had been an additional 110 deaths from aspiration of gastrointestinal contents. Thiopental has traditionally been used in "classical" RSIs induction drug and scholine as a neuromuscular blocker. Succinylcholine was introduced in 1951, while thiopental was first used for military anaesthesia during World War II. It took another 40 years for etomidate to be introduced, and another 20 years for propofol to be available. Before the 1940s, tracheal intubation was not used on a regular basis. Thiopental and succinylcholine are still helpful, reliable substances despite their age.

Sellick first described his "simple method" for minimising reflux of stomach or oesophageal contents in 1961, prior to intubation with a cuffed endotracheal tube. Since then, the cricoid cartilage has been compressed against the bodies of the cervical vertebrae to impede upper end of the oesophagus. 1 To protect the airway, anaesthesia was previously induced while the patient was upright.

With the availability of new medications, tools, and information over the previous ten years, RSI has changed as a practise. The practise is diverse, according to a recent survey of all trainees and representative anaesthetists. Numerous anaesthetists employ various alternatives to succinylcholine as well as various induction drugs. These include opioids and non-depolarizing neuromuscular blockers. This study did in fact confirm that many anaesthetists do not adhere to recommended practises.

In nations other than the UK, rapid sequence induction is used less frequently. There are no extensive studies that demonstrate the effectiveness of RSI or that it helps to lower demise rate. This article encourages the development of a well-thought-out strategy while examining the numerous RSI components.

Consequences of aspiration

The quantity of fluid entering the bronchial tree affects how severe the symptoms of aspiration are. In reaction to the aspiration of solid and liquid particles, Mendelson first identified two symptoms in 1946. 3 He compared the reaction to aspirating contents to respiratory attacks., which leaves patients not doing well but stabilizes over the course of 24 to 36 hours.

Larger amounts of stomach acid cause stomach contents to enter the bronchial system and cause infection, than smaller volumes of pH-neutral liquids. Acidic contents causes bronchiolar spasm, as well as surrounding bronchus cause congestive and exudative reactions, which disrupt normal intrapulmonary circulation. The other pattern is characterized by solids blocking a bronchus or bronchiole, resulting in atelectasis and distal collapse.

Bluish discolouration, increase heart rate, breathlessness, mediastinal shift, consolidation, and further morbidity accompany the massive lobar collapse that

follows. Gas transport is hampered by chemical pneumonitis, as well as by collapse, consolidation, and edema brought on by blockage and inflammatory reactions.

For all emergency and non-compulsory patients, Warner and colleagues reported an overall death rate after aspiration of 1 in 71829. According to a 1993 French study, 1 in 33 000 anesthetics resulted in death and 1 in 7400 episodes of aspiration.

Rapid sequence induction

Indications

All patients who are under emergency anesthesia incur the danger of aspirating their stomach contents if their larynx isn't working properly. Although they may not always be appropriate or easily accessible prior to emergency surgery, where there are time limitations on patient stabilisation , fasting, and prokinetic medicines lessen this risk. A nasogastric tube can be used to remove some of the stomach contents.

The following are some factors linked to elevated aspiration risk:

1. Pathology of the abdomen, especially blockage or ileus

2. A delayed emptying of the stomach (e.g., Ache, trauma, opioids, alcohol, vagotomy).

3. A hiatus hernia, a weak lower oesophageal sphincter, and gastroesophageal reflux disease

4. A change in consciousness that impairs laryngeal reflexes

5. Neuromuscular or neurological disease

6.Pregnancy

7. Troublesome airways and

8. Disturbances in metabolism.

Throughout the whole perioperative phase, but especially during the induction and post-anaesthesia recovery, these patients are at risk for aspiration.

According to Warner and colleagues, 1 out of every 8000 anesthetics given to ASA grade I and II patients resulted in aspiration; the incidence was higher in ASA grade III and IV patients (1/343). To reduce this risk, rapid collection induction is performed.

Requisite actions[15,16]

To perform RSI, one must avoid aspiration, swiftly achieve intubation, and be prepared for the possibility that one would not be able to stop regurgitation or achieve intubation. It's critical to keep in mind that 50% of difficult intubations take place without any prior warning symptoms.

Preoxygenation

Beginning with preoxygenation of the affected person, RSI has recently advanced. Preoxygenation is caused by oxygen being used to replace nitrogen in the patient's lung's functional residual capacity (FRC). As a result, the affected person's reserve of oxygen during apnoea will be improved (vide infra).

The alveolar air flow (VA) divided by the FRC (l=FRC/VA) is the time constant of a wash-in exponential function that charges the oxygen present and the FRC during preoxygenation. Given here the alveolar air flow (minute ventilation less dead space ventilation) is around 50 ml/kg/min and that the normal FRC is roughly 40 ml/kg, it might take 0.8 minutes. The exponential approach can be completed 98% of the way in four minutes (or around 3 minutes for preoxygenation while the patient inspires normally). As a result, the recommended preoxygenation duration is typically three

minutes, but it can be altered based on the patient's alveolar breathing or changes in functional residual capacity.

Encourage the patient for four maximal (vital capacity) breaths as an alternative tactic. This method makes preoxygenation more quickly completed. This procedure necessitates the use of a reservoir bag that is sizable enough to accommodate the affected person's necessary capacity. It has been discovered that this approach is less effective, particularly in elderly people.

Technique

As RSI can cause loss of awareness and neuromuscular block without guaranteeing the capacity to mechanically ventilate the patient's lungs, the anesthetist should be ready for any eventuality before removing RSI. This necessitates thorough instruction in safe intubation, a method to remove vomit or other secretions, and a practice intubation procedure. It's essential to have complete monitoring and a helper who knows how to apply cricoid pressure.

It is necessary to inspect the equipment, which should have functional suction, capnography, and the correct endotracheal tubes and laryngoscopes. The cart must be able to fall over effortlessly, head first. long-bore intravenous A cannula is connected to circulating fluid in order to ensure that medications are delivered swiftly to the brain. For successful intubation, the patient must be in the right position.

To maximize the amount of oxygen the patient can receive from their functional residual potential at some time during induction, pre-oxygenation using oxygen at 100% is crucial. As soon as the expired oxygen fraction is >85%, oxygen is supplied for 3 to 5 minutes.

A predetermined dose of an induction drug is given, then immediately a neuromuscular blocking agent. Prior to losing consciousness, cricoid pressure (20–40 nor 2- 4 kg) is applied. The trachea is intubated after the jaw has relaxed and succinylcholine-associated fasciculations have stopped. Ventilation, capnography, and/or chest auscultation should be used to confirm the endotracheal tube's placement. after tracheal placement and verification of cricoid pressure release.

Selection of an induction agent

Intravenous induction, which allows loss of awareness in seconds time, shortens the period between the loss of consciousness and intubation. The best induction medication should have a speedy onset, quick anesthesia recovery, and little to no side effects on the digestive and circulatory systems.

Thiopental provides rapid loss of consciousness at an expected dose (three to seven mg kg1) with a well-defined cease factor. It works much more quickly than etomidate or propofol. However, it takes a while to take effect, and its adverse effects could be fatal. Thiopental has an abnormally high anaphylaxis rate (1 in 20 000).

Propofol has the capacity to reduce laryngeal reflexes, which facilitates intubation. Compared to thiopental, it starts acting later and causes higher cardiovascular depression.

The main advantage of etomidate is cardiovascular stability, which makes it helpful for inducing anesthesia in patients who have cardiovascular diseases. However, due to its slow onset and tendency to suppress the adrenal glands, its use is restricted. These hazards more than the benefits of its cardiovascular stability in the vast majority of people. When the circulatory system collapses, ketamine should be considered for emergency anesthesia.

In conclusion, it is much more practical to match the induction agent choice to the patient who will be under anaesthesia. Etomidate should only be administered to patients who have a predominance of cardiovascular co-morbidity and only when there is a high risk of aspiration.

Selection of a neuromuscular blocking substance[10]

For neuromuscular block, succinylcholine has traditionally been considered the favored substance. A neuromuscular blocking medication for RSI should have the following qualities: a quick onset time to reduce the risk of aspiration and hypoxia, quick recovery to make it easier to resume breathing in the event that intubation is unsuccessful, and minimal systemic and hemodynamic side effects. Although succinylcholine has traditionally been preferred, it does not meet these criteria. Even though it starts and ends quickly, it has a lot of side effects, some of which might be harmful to one's way of life.

Bradycardia, malignant hyperpyrexia, hyperkalaemia, and muscle pain can all be brought on by succinylcholine. Anaphylaxis and histamine release both happen far too frequently. Increased intraocular, intracranial, and intragastric stress can come from a weak lower oesophageal sphincter, which can also cause passive regurgitation. Succinylcholine apnoea, which develops in those who are genetically predisposed, can hinder the repair of neuromuscular features in 0.001-0.03% of the population. The duration of the neuromuscular block might range from 20 minutes to several hours, depending on the genotype of the pseudocholinesterase.

Cricoid force

The discovery of cricoid pressure dates back to 19611. The oesophageal lumen is entirely stopped by extending the neck and pressing the cricoid cartilage on the fifth

cervical vertebra's body. Head and neck extension increases the anterior curvature of the cervical backbone while stretching the oesophagusand preventing its lateral displacement. A helper places their thumb and finger on either side of the cricoid cartilage and exerts pa ressure of 20 N (2 kg weight). This continues until the patient is intubated and the cuff is inflated.

Cricoid pressure has a mixed record of success in reducing regurgitation, according to studies. In cricoid pressure analysis of 26 high-risk inductions, three instances of immediately after regurgitation of the stomach or oesophageal contents showed that the approach was effective in preventing regurgitation.

The unique procedure has been improved, and the timing and amount of pressure applied have been quantified. It has been determined that for the method to be successful, an assistant must become proficient in applying cricoid stress and must regularly use the skill.

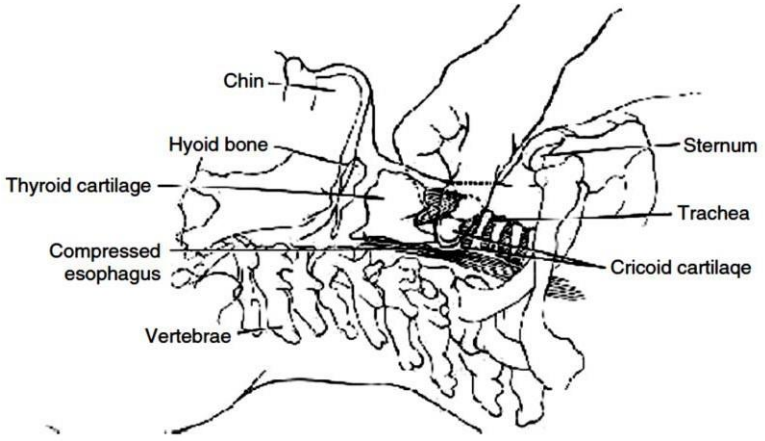


Fig 5: Cricoid Pressure

Tracheal intubation

The traditional approach is direct laryngoscopy, which is recommended. Both the MacIntosh laryngoscope and Murphy's kind of PVC tracheal tube are regularly used.

When the patient is in the sniffing position (neck flexion and extension at the atlanto-occipital joint), intubation is simpler. However, when this is not recommended, as in the event of suspected or verified cervical spinal injury, intubation is more difficult. This kind of intubation must be done by a trained laryngoscopist. Intubation is made easier with the use of a stylet and, on occasion, the best external laryngeal manipulation (OELM). This expert advices consistently employing a style in an emergency because it may help you save time. If the patient has an anterior larynx, OELM might be useful. The assistant should be told by the laryngoscopies to press on the thyroid until a clear view is gained.

Verification of tube role

Once the patient has been intubated, it is essential to make sure the tube is (1) in the trachea and not the oesophagus, and (2) not going straight down one of the most important bronchi.

Direct visibility of the tube among cords71 or the use of fiberoptic bronchoscopes are the best 2 approaches that may be failsafe. The tube is in the trachea according to numerous established strategies, although most of them are false. 70 (Table 1) The latter is frequently impractical, but the former may not always be a possibility. To determine the amount of carbon dioxide in exhaled gas, there are two almost foolproof methods: employing an oesophageal detector device or a capnometer of the spectrophotometric or less sophisticated calorimetric kind. The mechanisms required for at least one, if not both procedures, should be accessible in areas where tracheal intubation is done, according to this author.

Auscultation over each axilla is the tried-and-true technique for considering or disqualifying endobronchial intubation. The disadvantage of using this strategy in an

emergency is that the patient might also be suffering from serious pathology in one lung area, which would account for the difference in breath sounds. As a result, it is usually okay to position the tube directly in front of your eyes and stop it when the upper cuff boundary is 1-2 cm below your vocal chord. An x-ray of the chest is also beneficial, but it takes time.

Table 1: Methods of verification of tracheal tube position

Non-failsafe methods	Failsafe methods
Tactile orotracheal tube placement test	Direct visualization of tube between cords
Chest wall movement and auscultation	Use of fibreoptic laryngoscopes and bronchoscopes
Endobronchial intubation	
Epigastric auscultation	
Reservoir bag compliance and refilling	
Reservoir bag movements with spontaneous breathing	**Almost failsafe methods**
Cuff manoeuvres and neck palpation	
Sound of expelled gases during sternal compression	Direct visualization of CO_2 in exhaled gas
Tube condensation of water vapour	Oesophageal detector device
Electronic metal detectors	
Video stethoscope	
Use of nasogastric tubes, gastric aspirates, introducers and other devices	
Transtracheal illumination	
Pulse oximetry and detection of cyanosis	
Chest radiography	

Complications

Before attempting the method, it is crucial to understand the potential complications of conducting RSI. The main risk comes from intubating the patient without first determining whether it is possible to ventilate them. Failure to ventilate a paralysed patient may result from this. For this example, each anaesthetist wants to have a plan in place. Similar scenarios include a failed intubation and the requirement to wake the patient. In order to best protect the airway, this action should be carried out while maintaining cricoid pressure. The most popular RSI medications have relatively high anaphylaxis occurrence rates; the anaesthetist should be able to manage anaphylaxis

and care for a vulnerable patient. Although it won't result in a harmful operation, the risk of attention during the procedure may cause people a great deal of distress.

When cricoid pressure is used, it can have negative effects on the body, including oesophageal occlusion failure, laryngeal distortion that affects laryngoscopy vision, and, less frequently, oesophageal rupture during active vomiting. If there is vigorous vomiting rather than passive regurgitation, the cricoid pressure needs to be relieved**.**

Terminating anaesthesia

Detail-oriented behaviour is necessary during the duration of anaesthesia in patients who have required RSI. When transitioning from a profound state of anaesthesia to full consciousness or vice versa, the risk of aspiration is highest. The patient must be fully awake and demonstrating the ability to take intentional action or respond to orders before being extubated. This proves that the patient can defend their own airway once the cuffed tube is withdrawn. In the event of regurgitation, the patient's airway is equally safeguarded either seated or in the left lateral position.

Review of Literature

KF Koh et al (1994)[15] study 80 people joined the study and were randomly assigned to 4 groups, each with 20 participants, by KF Koh et al. (1994), in order to compare the intubating conditions using succinylcholine and atracurium based on the "timing principle." All of the subjects in groups 1, 2, and 3 received 0.5, 0.75, or 1 mg.Kg-1 of atracurium, whereas group four, which served as the control group, received 1.5 mg.Kg-1 of succinylcholine. Fentanyl 1 mg/kg was given to the study group, followed by atracurium 3 minutes later and thiopentone 4-6 mg/kg when ptosis started. After three minutes, the control group administered 4-6mg.Kg-1 of thiopentone and succinylcholine. They also received 0.025mg.Kg-1 of atracurium and 1 kg-1 of fentanyl. One minute after giving thiopentone, the tracheal intubation was completed. The intubating circumstances were assessed by a laryngoscopist who was unaware of the induction sequence. On the day after surgery, interviews with every participant were conducted. The intubation rankings of Group 1 participants were lower than those of the other 3 groups, and Groups 2, 3, and 4 did not differ from Group 1 in any way. The method employed and excessive hemodynarnic alterations had no correlation. With the exception of one patient, everyone else was able to cough comfortably following atracurium injection and prior to thiopentone-induced anaesthesia. This led to the conclusion that atracurium might be employed instead of succinylcholine at the moment of rapid sequence induction while still adhering to the timing principle.

A prospective randomised controlled study was conducted by **Duggappa AK et al.[17] (2018)** to determine the ideal priming dose of atracurium that can hasten the onset of action. Ninety participants were divided into Group A, Group B, and Group C at random. Along with the priming dosage, an average dose of 0.5 mg/kg body weight of

atracurium was administered to each participant. A different priming dose was given to every group. Group A received 0.05 mg/kg of body weight, Group B received 0.025 mg/kg of body weight, and Group C received saline as the priming dose. Regarding demographic information, all patient characteristics were comparable. In groups A, B, and C, the mean times for the train-of-four (TOF) count to reach zero were 147, 193, and 218 seconds, respectively. Two patients in groups A and B who received the priming drug atracurium at a dose of 0.05 mg/kg body weight experienced ptosis three minutes after priming. The individuals showed no further negative effects. According to the results of this investigation, atracurium priming decreased the time needed for TOF count to reach zero by 71 seconds when used at 0.05 mg/kg body weight and by about 25 seconds when used at 0.025 mg/kg body weight. Adverse effects were rare and clinically insignificant.

The timing and intubation score with the use of triple the (ED95) of rocuronium or cis-atracurium in morbidly obese patients were evaluated in a study by **EM Taher et al.[18] (2020)**. In this study, sixty morbidly obese adult patients were randomised to either the ROC group or the CIS group based on the muscle relaxant used to induce anaesthesia. The ROC group received 0.9 mg/kg of rocuronium, whereas the CIS group received 0.15 mg/kg of cis-atracurium. Recorded data included the onset and duration of relaxation, intubation score, intubation time, and frequency of negative effects. Additionally, any variations in the hemodynamic parameters were kept track of. It was found that the use of triple (ED95) rocuronium during rapid sequence anaesthesia induction significantly sped up intubation time from 104.55.17.91 seconds to 89.14.20.43 seconds and decreased the onset of relaxation from 96.00.13.29 seconds to 84.00.18.50 seconds. It also increased the duration of relaxation from 58.76.9.27 seconds to 69.86.8.38 seconds. In both groups, the intubation score was

comparable. Conclusion: Compared to cis-atracurium, the administration of treble the (ED95) rocuronium during fast sequence anaesthesia induction among morbidly obese patients dramatically decreased the time required for intubation and the beginning of relaxation, with minimal impact on the intubation score.

In order to ascertain the dosage and effectiveness of atracurium without priming for the rapid sequence induction of anaesthesia**, P Chalermkitpanit et al.[6] (2020)** carried out a randomised controlled study. In this study, three groups were randomly assigned to 115 surgical patients who were under general anaesthesia. All of the patients received the same doses of atracurium (0.6 mg/kg, 0.75 mg/kg, or 1 mg/kg) without a priming dose after receiving the IV anaesthetics propofol and fentanyl at rates of 2–3 mg/kg and 1 g/kg, respectively. Within one minute of the study drugs being administered, tracheal intubation was carried out. As the main endpoints, the intubating conditions, vocal cord movement, and diaphragm movement were assessed. The success rates of intubation with atracurium doses of 1 mg/kg, 0.75 mg/kg, and 0.6 mg/kg were seen to be 51.4%, 43.6%, and 26.3% without coughing or bucking. 84.6% of patients receiving 0.75 mg/kg of atracurium and 86.5% of patients receiving 1 mg/kg of atracurium had excellent or good intubating conditions. When comparing the effects of the 1 mg/kg dose of atracurium to the 0.6 mg/kg dose, there were discernible variations in the paralysis of the vocal cords and diaphragm. According to the findings of this study, under certain circumstances, a high dose of atracurium administered without priming can be used for a rapid sequence induction of anaesthesia as a substitute for a neuromuscular blocking agent.

In a study published in **2020, Surbhi and S Nanda[19]** evaluated the effectiveness of Cisatracurium Besylate at a loading dose of 4 ED95 and examined its effects on histamine release, intubating conditions, duration of action, and onset. 30 patients

between the ages of 18 and 60, of either gender, who were undergoing elective surgery under general anaesthesia with endotracheal intubation participated in this study. Using a consistent anaesthetic technique, all of the patients were put to sleep. Cisatracurium injection at a dose of 0.2 mg/kg made it easier to intubate the endotrachea. Only clinical observations were used to assess the level of muscle relaxation. This study found that a 4 ED95 dose gave almost all study participants an excellent to good laryngoscopic view within 3 minutes. The intubating conditions were excellent or good for each patient. Cisatracurium had an intermediate duration of effect, and the resulting muscular relaxation may be easily undone with neostigmine. Histamine release was not clinically demonstrated. Based on these results, the study came to the conclusion that a loading dose of 2 mg/kg (4 ED95) of cisatracurium was an efficient neuromuscular blocking agent that did not release histamine.

In a study published in **2020, Sahu et al[20].** compared the effectiveness of intravenous (IV) injections of atracurium (0.5 mg/kg) and cisatracurium (0.2 mg/kg) for intubating patients undergoing endoscopic retrograde cholangiopancreatography (ERCP). Group A received a 0.5 mg/kg intravenous injection of atracurium besylate, while Group B received a 0.2 mg/kg intravenous injection of cisatracurium besylate. One hundred adult patients between the ages of 18 and 60 who were scheduled for ERCP procedures under general anaesthesia were chosen. Time to reach the maximum blockade, length of action, time needed to intubate, hemodynamic parameters during and after 1, 2, 3, 5, and 15 minutes of intubation, and the emergence of negative effects. Compared to 19 patients in the atracurium group, 36 patients in the cisatracurium group had good intubating conditions according to the Cooper et al score. Group B was found to have a better overall intubating condition, with a significantly longer time to the maximum blockade than the cisatracurium group. When

cis atracurium was used instead of atracurium, the mean intubation time was shorter (13511.1 seconds vs. 1449.48 seconds). Hemodynamic parameters during intubation and at 1, 2, 3, 5, and 15 minutes were comparable in both groups, but Group B experienced prolonged neuromuscular blockade. Both groups did not experience any negative effects. Based on these findings, it was determined that, as compared to 0.5 mg/kg atracurium, 0.2 mg/kg cisatracurium provides good intubating conditions with early onset of action, extended duration of action, no major hemodynamic alterations, and no side effects.

The effectiveness of cis atracurium and atracurium in terms of intubation and maintenance doses, hemodynamic response, and cost effectiveness in kidney transplant patients were compared in a study by **Jirasiritham S et al.[21] (2004).** 46 patients with end-stage renal disease were enrolled in this study. 23 people from each group received tracheal intubation and continued use of a muscle relaxant while receiving their medication. While cis atracurium served as the study (S) group, atracurium served as the control (C). When compared to the atracurium group, the S group's mean dosage for intubation and maintenance was lower. Hemodynamic status did not differ statistically between the two groups. This study found that even though the price of cisatracurium was higher, the cost-minimization analysis revealed that it was actually lower per case.

In patients scheduled for open heart surgery, Cis atracurium and Atracurium's hemodynamic effects were evaluated by **Ghorbanlo M et al[22] in 2016.** All adult patients with cardiac disease whose ejection fraction was 35% or below were included in this randomised prospective double-blind research trial. All of the chosen patients were divided into two groups of 30 each at random. All of the patients were given

midazolam, etomidate, and either 0.2 mg/kg of cis atracurium or 0.5 mg/kg of atracurium within one minute of induction during the induction stage. At various times during the anaesthesia and surgical processes, the hemodynamic indices were measured and evaluated. The authors found no statistically significant differences in the groups' hemodynamic parameters, which they attributed to the groups' similar systolic and diastolic pressure ratios. Based on the findings, it was determined that using cis atracurium as a muscle relaxant is better and more advantageous.

S. Thukral et al[23]. (2018) conducted a study to compare the effects of atracurium and cis atracurium on neostigmine reversal, action onset, action duration, and 25% recovery from last supplemental dose. Sixty patients were equally and randomly assigned to Groups A and B, with Group A receiving atracurium 0.5 mg/kg and Group B receiving cis atracurium 0.2 mg/kg as a loading dose. In addition to the haemodynamic data, the Train of Four (TOF) response was used to capture the onset time, block duration, and recovery time. It was shown that the Cis atracurium group's mean duration of action was substantially longer than that of the Atracurium group. Comparing Cis atracurium besylate to Atracurium, the mean 25% recovery is higher. Cis atracurium has a quicker onset than atracurium, good intraoperative hemodynamic parameters, and a superior recovery profile, according to this study's findings.

R Ranjan et al.24. (2021) evaluated the neuromuscular blockade and recovery qualities of atracurium with cis atracurium in a prospective, randomised experiment. In total, 100 patients were included in the trial, split into two groups of 50 patients each. Group A received atracurium (0.5 mg/kg), whereas Group B received cis atracurium (0.15 mg/kg). When comparing the start and duration of action, recovery time, hemodynamic circumstances, and indicators of histamine release, the atracurium group's mean recovery time was 25% lower than the cis atracurium group's. The mean

recovery time from the reversal was substantially longer in the cis atracurium group compared to the atracurium group. According to this study, cis atracurium (0.15 mg/kg) exhibited a quicker onset and a longer half-life of effect than atracurium (0.5 mg/kg). At this dose, cis atracurium provided improved intubating circumstances, fast neuromuscular blocking with a longer duration of action, stable hemodynamic conditions without histamine release, and protracted muscle paralysis in compared to atracurium.

In a study published in 2021, **J. C. Makwana et al[25].** compared the effectiveness of atracurium and cisatracurium in terms of the timing and length of their effects as well as their effects on hemodynamic stability. 40 patients from Group 1 received atracurium 0.5 mg/kg for induction. Cisatracurium 0.1 mg per kg was administered to 40 patients in group 2 as an induction dose. In comparison to cisatracurium, atracurium's onset of action was quicker. Using the Cooper's scoring system, it was determined that after 3 minutes of the administration of cisatracurium, vocal cord movement was still evident while it was absent in the case of atracurium. The authors came to the conclusion that while cisatracurium's duration of action was greater than atracurium's, the former's onset of action was faster in the atracurium group. Similar to this, cisatracurium demonstrated superior hemodynamic stability than atracurium.

MATERIALS AND METHODS

STUDY DESIGN: Randomized control study

STUDY PERIOD: January 2021 to June 2022

STUDY PLACE: B.L.D.E(Deemed to be University) B. M. Patil Medical College, Hospital and Research Centre, Vijayapur, Karnataka.

SAMPLE SIZE: 115 patients

CRITERIA FOR INCLUSION

1. Individuals with ASA grades I and II.
2. Individuals between the ages of 20 and 65..
3. Patients willing to give informed and written consent.
4. Patients of both the sexes
5. Patients undergoing various surgical procedures

EXCLUSION CRITERIA

Patients with any one of the following were excluded:

1. Individuals who are severely obese
2. Pregnant patients
3. Patients who may be challenging to intubate
4. ASA grade III and IV individuals
5. Patients not willing to give written informed consent

After receiving written informed consent and approval from the Institutional Review Board, 112 patients who were scheduled to undergo elective surgery under general

anaesthesia with endotracheal intubation and required intraoperative neuromuscular blockade lasting were included in the trial.

The 112 patients who were a part of the trial were randomly split into 2 groups of 56 each. 3 patients not considered due to difficult intubation.

Group A: 56 patients receiving 0.75mg/kg [3ED95]

Group B: 56 patients receiving 1 mg/kg [4ED95].

During the preoperative visit, a thorough history of the patient was taken before the procedure started. Vital signs had been obtained and a clinical examination had been done.

The Modified Mallampati Score, thyromental distance, mento hyoid distance, and mouth establishing were used to evaluate the airway. All patients received the recommendation to fast for eight hours overnight following pre-anesthesia examination.

The patient was brought to the operating room on the day of the procedure, and before anaesthesia was administered, patients were informed about the Train-of-4 (TOF) measurements that would be carried out postoperatively and any potential pain or discomfort they might experience. 10 ml/kg of ringer lactate solution was given after setting up an IV line.

Following the pre-anesthesia check-up, the TOF monitor was connected along with the electrocardiogram, non-invasive blood pressure, pulse oximetry, surface temperature probe, capnography, and acceleromyograph (TOF GUARD, organon Technika Laboratories).

Glycopyrrolate 0.2 mg, ondansetron 4 mg, midazolam 0.03 mg/kg, paracetamol 1 gm, and diclofenac sodium 75 mg were all given intravenously to all patients. After preoxygenating with 100% O2 for 3 minutes, they were induced with fentanyl 1 mg/kg, followed by propofol 2-3 mg/kg, or until loss of vocal contact. Later, an acceleromyograph was used to record the contraction of the ipsilateral adductor pollicis muscle after the wrist had been stimulated with supramaximal stimuli of 0.2 ms length delivered in a TOF mode at 2Hz once per 15s. The T1 of TOF and TOF ratio, which were calculated at the end of control stimulation, will serve as the baseline value.

Atracurium was given in varying doses—0.75 mg/kg (3ED95) for group A patients and 1 mg/kg (4ED95) for group B patients. The patients were allowed to remain apneic for one second, with face cover ventilation allowed in the event of desaturation. Within one second of the administration of the investigational medicines, intubation was carried out by trained anaesthesiologists using a video laryngoscope.

The intubating conditions were deemed to be the primary endpoint by the two anesthesiologists who were blinded to the atracurium components. The intubating conditions were divided into four categories: excellent (simple section of the cylinder without hacking or kicking), excellent (entry of the cylinder with only a little hacking or kicking), reasonable (section of the cylinder with moderate hacking or kicking), and poor (condition in which the cylinder was kicked or hackled heavily) (unrealistic to intubate).

The vocal cord and stomach development during intubation were also graded into 4 categories: excellent (completely deadened), excellent (incompletely incapacitated with slight development), reasonable (half-deadened with moderate development),

and poor (insufficiently deadened with severe development) (unparalyzed stomach with vocal lines shut).

Utilizing a TOF Watch®SX (Organon Ltd., Dublin, Ireland) with the transducer against the thumb to measure the level of the neuromuscular bar, the fringe nerve was stimulated. The number of muscular jerks, often known as a Train of Four (TOF) estimation, is spoken to the muscles.

PACU TOF Measurement

At the PACU, all patients were given 4l/min oxygen via mask and underwent noninvasive electrocardiographic, pulse oximetric, and monitoring of the heart rate. The examination of RNMB utilising TOF stimulation (four pulses of 0.2 ms duration for 2s at a frequency of 2Hz; current intensity, 50 mA), which was performed four to five times with an interval of 12s between stimulations, revealed the greatest of the two consecutive repeatable TOF ratios. TOF measurements were carried out repeatedly every 5 minutes until the TOF ratio reached 0.9, or 90% or greater. The first TOF reading and the TOF ratio reaching 0.9 or higher were documented. It was noted and not repeated if the patient had already achieved a TOF ratio of 0.9 or higher. The patient will be taken out of the study if the TOF ratio is less than 0.9 30 minutes after extubation and they have hypoxia. The TOF ratio was measured every five minutes for statistical analysis until the TOF was greater than or equal to 0.9, which could take up to thirty minutes following extubation. The time it took for TOF to increase to more than or equal 0.9 was also noted.

There will be no restriction on the use of intraoperative pain medications until the procedure is complete. General sedation will be maintained with 60% N2O, 40% O2, and isoflurane.

The circulatory strain, pulse, and heartbeat oximeter were recorded each moment for 10 minutes, trailed by observing like clockwork after the enlistment of sedation.

The antagonistic impacts of atracurium because of histamine discharges, for example, cutaneous flushing, rash, bronchospasm, and hypotension, were On the off chance that patients created supported high aviation route pressure, wheezing, desaturation, or unsuitable hypotension, at that point, the correct administration was performed.

FEATURES ASSESSED:

Preinduction, preintubation, and post-intubation measurements of non-invasive blood pressure, systolic arterial pressure (SAP), diastolic arterial pressure (DAP), mean arterial pressure (MAP), pulse rate, and SPO2 will be taken at 1, 3, and 5 minutes. Any arrhythmias or issues that arise during intubation, such as local wounds, bleeding, regurgitation, laryngospasm, or a decrease in SPO2, will be documented.

Statistical analysis:

The data was entered into an excel worksheet, and SPSS 20 was used for the analysis. The use of descriptive statistics was done. Data from categorical measurements were provided in number (%) and results from continuous measurements were presented as meanSD. In order to determine the association between two category variables, the chi square test will be employed. A P value of 0.05 or lower was regarded as statistically significant.

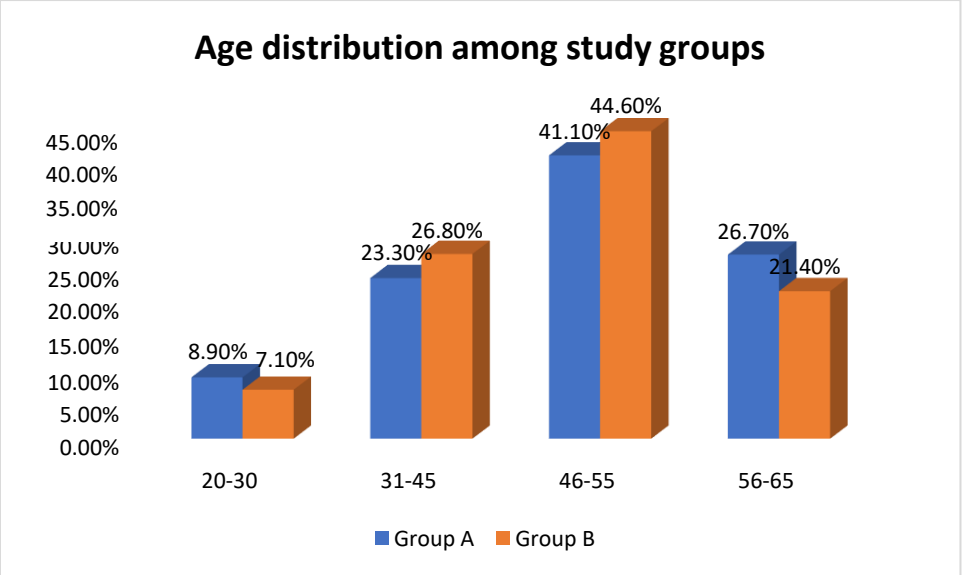
Picture showing TOF being measured

RESULTS

Table 2: Shows how old each study group is.

Age Group (in years)	Group A	Group B	Total
20-30	5 (8.9%)	4 (7.1%)	9
31-45	13 (23.3%)	15 (26.8%)	28
46-55	23 (41.1%)	25 (44.6%)	47
56-65	15 (26.7%)	12 (21.4%)	27
Total	56	56	112

Figure 6: shows how old each study group is.

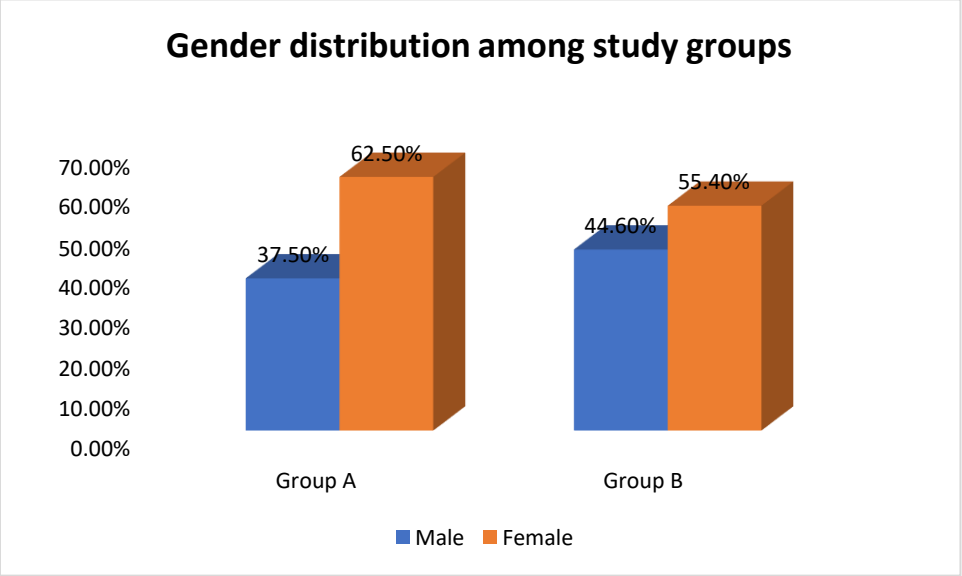


The majority of the study participants in groups A and B (41.1% and 44.6%, respectively) were between the ages of 46 and 55.

Table 3: Study group sex distribution

Sex	Group A	Group B	Total
Male	21 (37.5%)	25 (44.6%)	46 (41.1%)
Female	35 (62.5%)	31 (55.4%)	66 (58.9%)
Total	56	56	112

Figure 7: Sex Distribution among study groups

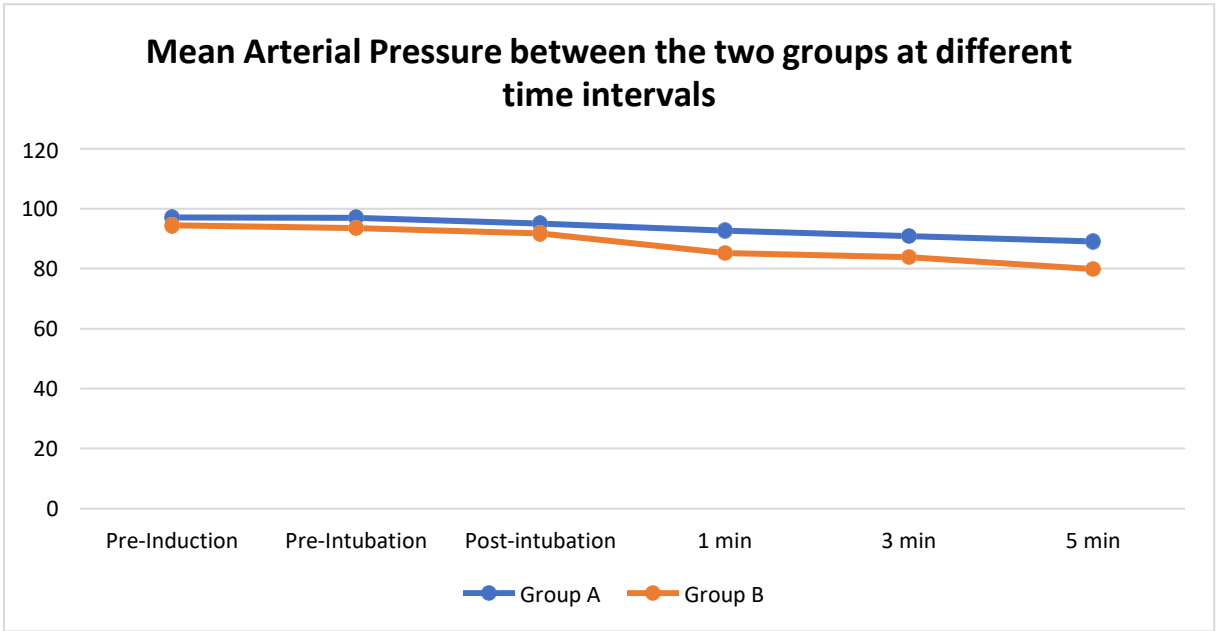


In groups A and B, there are more female patients (62.5% and 55.4%, respectively).

Table 4: compares the two groups' mean arterial pressure at various time points.

Time	Group A	Group B	P-value
Pre-Induction 1 min	84.10 ± 3.6	83.47 ± 3.3	0.025
Pre-Induction 3 min	86.31±6.95	86. 28±6.74	0.024
Pre-Induction 5 min	86.46±8.91	83.24±9.53	<0.05
Pre-Intubation 1 min	85.93 ± 3.6	83.57 ± 3.91	<0.001
Pre-Intubation3 min	85.50±7.62	86.41±8.62	<0.001
Pre-Intubation 5min	83.61±9.54	83.36±7.57	<0.001
Post-intubation 1 min	82.73 ± 3.3	80.20± 3.26	<0.001
Post-intubation 3 min	80.89± 2.8	70.8 ± 2.90	<0.001
Post-intubation 5 min	80.10 ± 3.6	70.90 ± 3.3	0.031

Figure 8: compares the two groups' mean arterial pressure at various time points

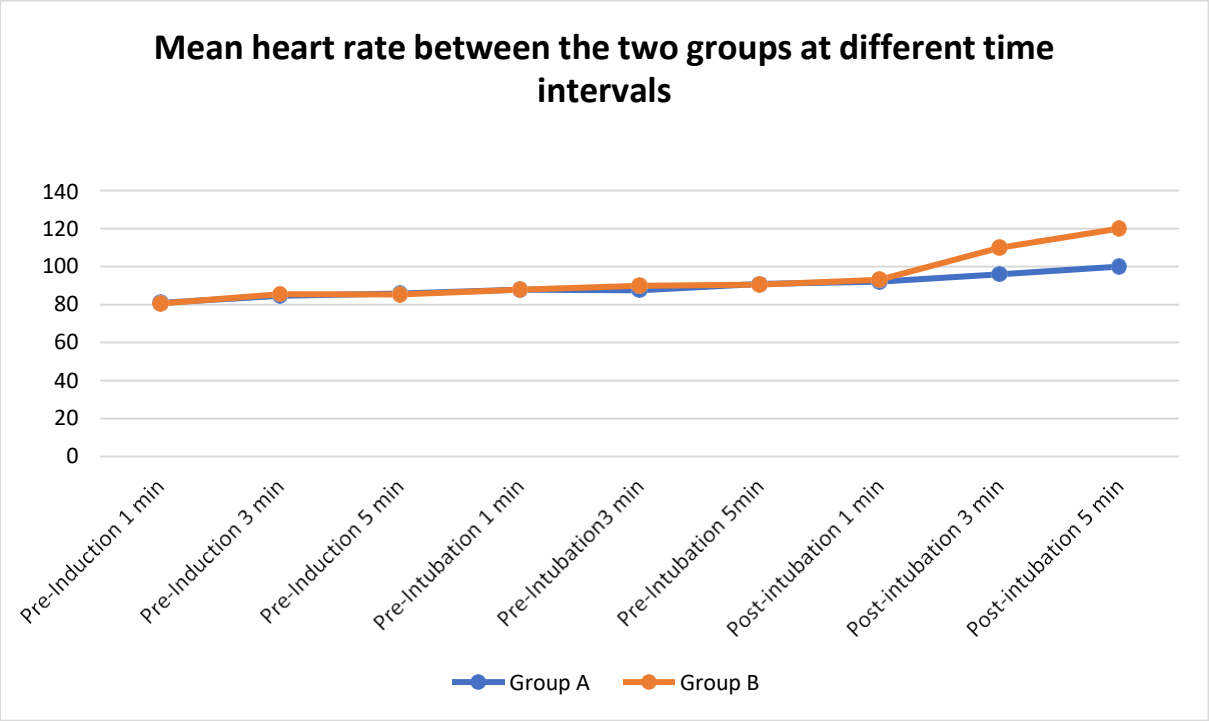


Mean arterial pressure readings were taken for patients in both groups before induction, throughout induction, during intubation, and 1, 3, and 5 minutes after intubation. These results showed that arterial pressure was similar in both groups at all time points during pre-induction and pre-intubation. After intubation, however, the group that received 1 mg/kg of atracurium had mean arterial pressure readings at 1, 3, and 5 that were considerably lower than the group that received 0.75 mg/kg of atracurium.

Table 5: compares the two groups' average heart rates at various time points.

Time interval	Group A	Group B	p-value
Pre-Induction 1 min	80.9 ± 17.5	80.6 ± 14.9	<0.001
Pre-Induction 3 min	84.6 ± 16.1	85.5 ± 18.3	<0.001
Pre-Induction 5 min	85.9 ± 17.2	85.3 ± 18.5	<0.001
Pre-Intubation 1 min	87.8 ± 17.4	87.8 ± 17.7	<0.001
Pre-Intubation3 min	87.6 ± 17.0	89.8 ± 17.1	<0.001
Pre-Intubation 5min	90.9 ± 17.5	90.6 ± 14.9	<0.001
Post-intubation 1 min	92.84±7.29	93.63± 7.32	<0.01
Post-intubation 3 min	96.72±9.01	110.50±8.51	<0.01
Post-intubation 5 min	100.23±768	120.21±7.55*	<0.01

Figure 9: compares the two groups' average heart rates at various time points.

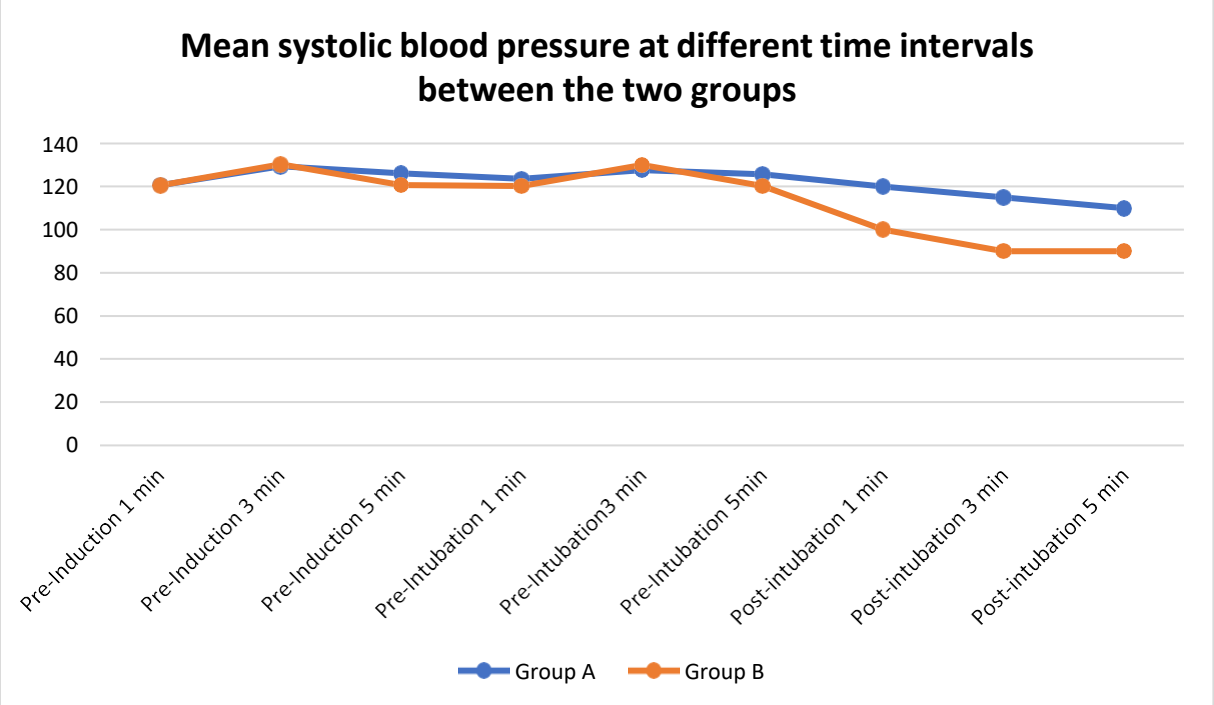


Prior to induction, during induction, during intubation, and 1, 3, and 5 minutes following intubation, mean heart rates for patients in both groups were noted. These results showed that arterial pressure was similar in both groups at all time points during pre-induction and pre-intubation. Nevertheless, at 1, 3, and 5 hours after intubation, the mean heart rate was considerably higher in the group receiving 1 mg/kg of atracurium than it was in the 0.75 mg/kg atracurium group.

Table 6: Mean SAP for various time periods for the two groups

Time	Group A	Group B	p-value
Pre-Induction 1 min	120.60 ± 12.98	120.56 ± 11.88	0.635
Pre-Induction 3 min	129.42 ± 14.10	130.30 ± 13.36	0.023
Pre-Induction 5 min	126.13± 18.54	120.61± 17.74	0.035
Pre-Intubation 1 min	123.65± 22.64	120.24± 22.02	<0.05
Pre-Intubation3 min	127.57 ± 17.16	130.06 ± 16.48	<0.001
Pre-Intubation 5min	125.72 ± 12.98	120.16± 11.88	<0.001
Post-intubation 1 min	120.10±9.53*	100.97±6.77	<0.05
Post-intubation 3 min	115.68±9.71	90.24±8.11	<0.05
Post-intubation 5 min	110.76±9.17	90.86±7.35*	<0.05

Figure 10: Mean SAP pressure between groups at various time intervals

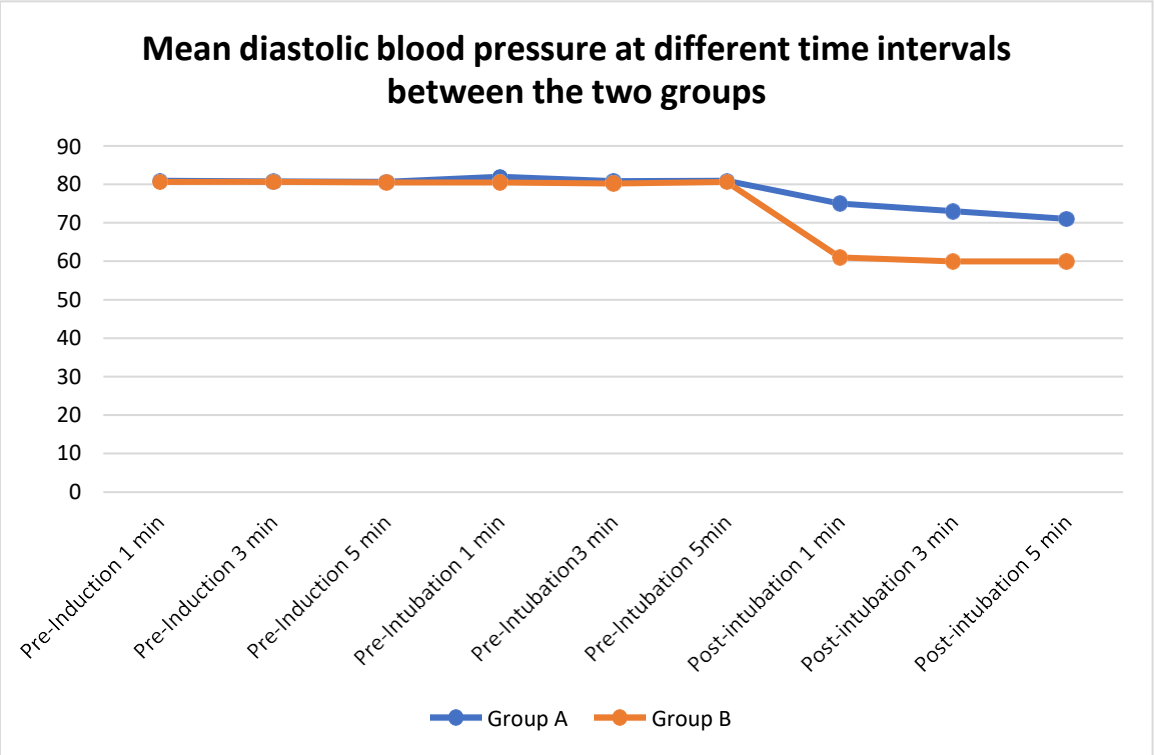


For patients in both groups, the mean SBP was recorded prior to induction, during induction, during intubation, and 1, 3, and 5 minutes after intubation. These findings demonstrated that during pre-induction and pre-intubation, arterial pressure was comparable in both groups at all time points. However, after intubation, the group that received 1 mg/kg of atracurium had a significantly lower mean SBP at 1, 3, and 5 than the group that received 0.75 mg/kg of atracurium.

Table 7: Compares the two groups' average DAP over various time periods.

Time	Group A	Group B	p-value
Pre-Induction 1 min	80.93± 7.6	80.63 ± 10.30	<0.001
Pre-Induction 3 min	80.73±9.67	80 .67±11.27	<0.001
Pre-Induction 5 min	80.63±8.39	80.47±17.26	<0.001
Pre-Intubation 1 min	81.97±8.42	80.50 ±15.64	<0.001
Pre-Intubation3 min	80.80±7.18	80.23 ±13.91	<0.001
Pre-Intubation 5min	80.93±11.2.6	80.63 ± 10.30	<0.001
Post-intubation 1 min	75.37±8.10	61.32±7.15	0.023
Post-intubation 3 min	73.60±5.59*	60.46±8.02	0.036
Post-intubation 5 min	71.47±6.12	60.20±7.07	0.021

Figure 11: Mean DAP at different time intervals comparing the two groups



For patients in both groups, mean DAP were recorded prior to induction, during induction, during intubation, and 1, 3, and 5 minutes after intubation. These findings demonstrated that during pre-induction and pre-intubation, arterial pressure was comparable in both groups at all time points. But after intubation, the 1 mg/kg atracurium group showed a significantly lower mean diastolic arterial pressure at 1, 3, and 5 compared to the 0.75 mg/kg atracurium group.

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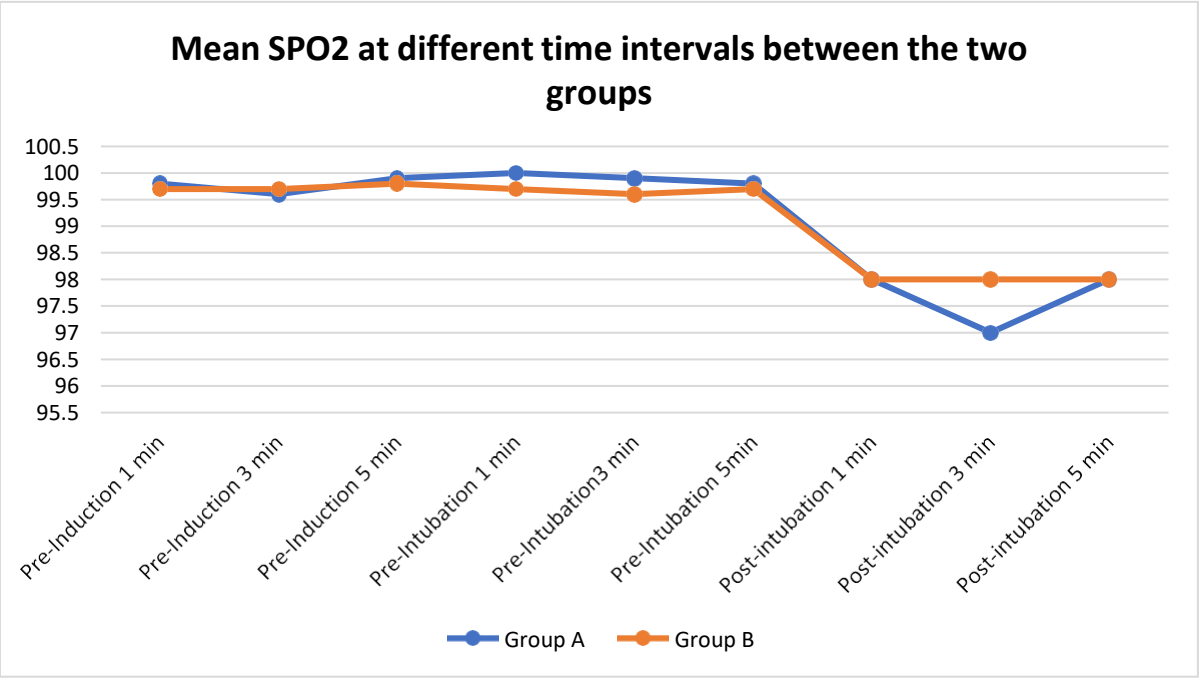
Table 8: Mean SPO2 at different time intervals between the two groups

Time	Group A	Group B	p-value
Pre-Induction 1 min	99.8 ± 1.04	99.7 ± 0.77	<0.001
Pre-Induction 3 min	99.6 ± 0.65	99.7 ± 0.25	<0.001
Pre-Induction 5 min	99.9 ± 0.65	99.8 ± 0.37	<0.001
Pre-Intubation 1 min	100.0 ± 0.61	99.7 ± 0.25	<0.001
Pre-Intubation3 min	99.9 ± 0.76	99.6 ± 0.25	<0.001
Pre-Intubation 5min	99.8 ± 1.04	99.7 ± 0.77	<0.001
Post-intubation 1 min	98.83±6.12*	98.97±7.83	<0.05
Post-intubation 3 min	97.76±8.10	98.65±7.85	<0.05
Post-intubation 5 min	98.34±7.82	98.83±6.12	<0.05

Figure 7: Compares the two groups' mean SPO2 values at various time points.

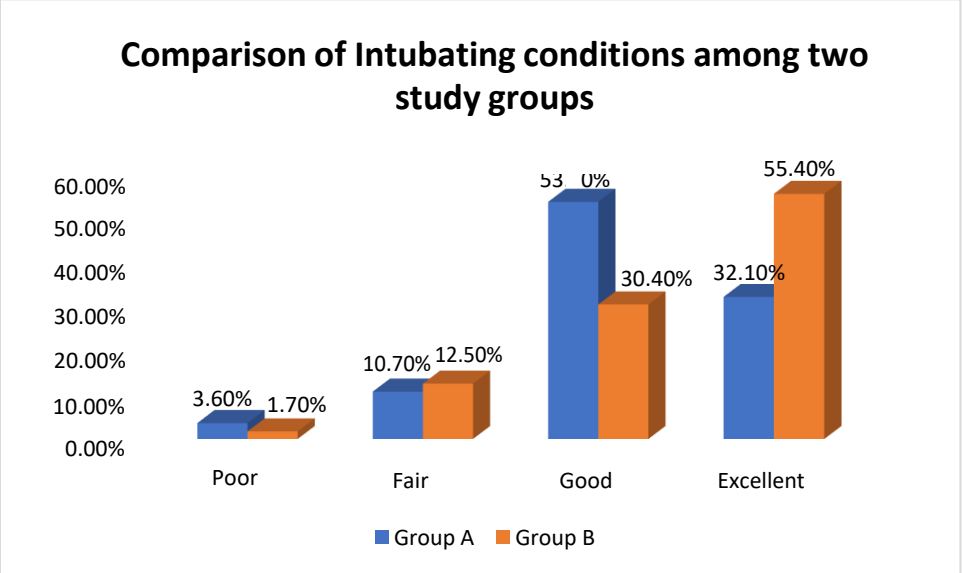


Comparing mean SPO2 levels revealed that neither group's mean SPO2 levels changed over the course of the time intervals.

Table 9: Comparison of Intubating conditions among two study groups

Intubating conditions	Group A	Group B
Poor	2 (3.6%)	1 (1.7%)
Fair	6 (10.7%)	7 (12.5%)
Good	30 (53.6%)	17 (30.4%)
Excellent	18 (32.1%)	31 (55.4%)
Total	56	56

Figure 13: Comparison of Intubating circumstances among two study groups



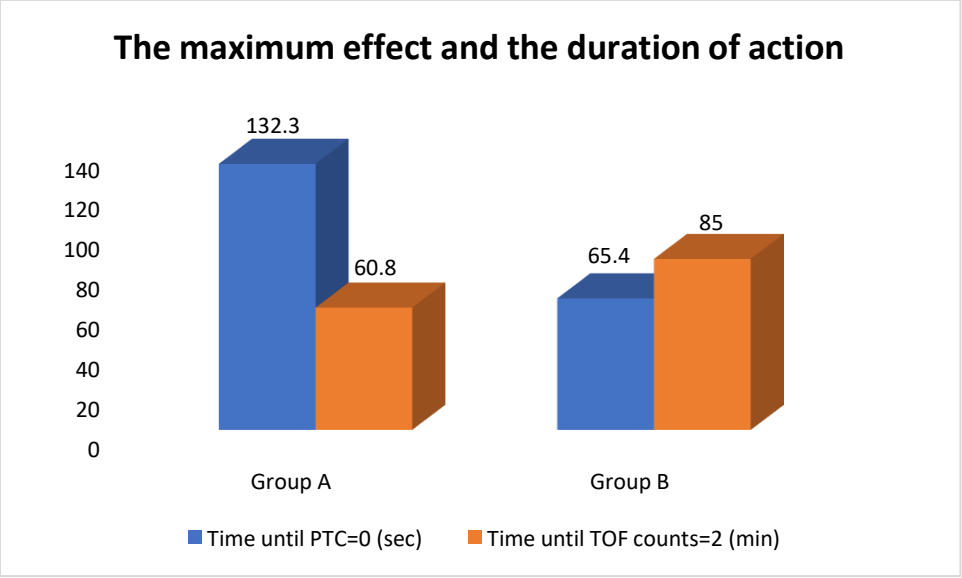
Intubating circumstances were excellent in 55.4% of the patients in 1mg/kg atracurium group, compared to 32.1% in 0.75mg/kg group. In the 0.75mg group, more patients

(53.6%) had favourable intubation circumstances. Only one patient received a higher dose, and their intubating conditions were poor.

Table 10: According to the peripheral nerve stimulator, atracurium's maximum effect and length of action

	Group A	Group B	p-value
Time until PTC=0 (sec	132.3±14.65	65.4±10.6	0.013
Time until TOF counts=2 (min)	60.8±9.70	85.0±11.40	0.001

Figure 13: According to the peripheral nerve stimulator, atracurium's maximum effect and duration of action



The time of onset and spontaneous recovery were 65.4±10.6 and 85.0±11.40 min for 1mg/kg atracurium compared to 132.3±14.65 and 60.8±9.70 min in 0.75mg/kg.

DISCUSSION

Tracheal intubation is necessary as part of the rapid sequence induction (RSI) of anaesthesia, which is often performed in emergency conditions, to lessen the risk of aspiration and hypoxia. Succinylcholine and high-dose rocuronium have been established as standard neuromuscular blockading medicines due to their fast and reliable beginning of an action (NMBAs). Due to the possibility of prolonged excretion and lingering neuromuscular blockade, rocuronium must be avoided in patients with renal impairment. In individuals with renal failure, atracurium is safe to use and has no effect on ICP or IOP. But succinylcholine can occasionally be harmful for people who are hyperkalemia, have accelerated intracranial pressure (ICP).

A greater dose of atracurium—roughly three to four times its ED95—would enable quick tracheal intubation in comparison to a high dose of rocuronium, according to a previous study that indicated that atracurium's effectiveness became comparable to that of the latter. The potency of NMBAs is inversely associated with the rate at which they start to work. The timing principle method, which involved giving a bolus of high-dose atracurium before the induction, was used to successfully validate fast tracheal intubation in an earlier study7. Nevertheless, during the induction of anaesthesia, patients may feel uncomfortable, have trouble breathing and coughing, develop diplopia, or suffer changes in awareness. The primary method, which calls for administering 0.04-0.06 mg/kg atracurium two to three minutes before to a single bolus dosage, made it possible to intubate patients quickly—in less than a minute. [8,9]

Patients at high risk for gastric aspiration or patients who are uncooperative should not use the timing principle or the priming method. Rapid sequence induction does not

have any supporting clinical statistics when excessive doses of atracurium are used without priming.

In our study, we compared the effectiveness of two different atracurium doses for the quick induction of anaesthesia.

Intubating condition

The current study showed intubating situations were excellent in 55.4% of the patients in 1mg/kg atracurium institution, compared to 32.1% in the 0.75mg/kg group. More frequent of patients in 0.75mg atracurium group had accurate intubation conditions (53.6%). Only one affected person in 1mg/kg atracurium group showed terrible intubating conditions.

These results agree with those of El Kasaby et al.26 who found that cisatracurium had excellent intubating properties at dosages greater than 2 ED 90–95. This result agrees with a study by Bluestein et al. in which the intubating dose of cisatracurium was suggested to be 0.15 mg/kg.

On the opposite, Mohamed A A et al[27] found great differences in intubating conditions among groups that acquired 4 times ED95 atracurium with priming, which have been advanced significantly faster whilst as compared with the group who received three instances of ED95 atracurium and not using priming.

According to P. Chalermkitpanit et al.6, only a smaller percentage (26.3%) of patients in the control group who received 0.6 mg/kg atracurium had appropriate intubating conditions. In addition, 94.9% and 89.7% of patients, respectively, developed clinical paralysis of the vocal cord and diaphragm after administering 1 mg/kg of atracurium. These results support the use of an overdose of atracurium for a quick tracheal intubation.

Onset time

In the prevailing study, the time of onset had been 65.4±10.6 sec for 1mg/kg atracurium compared to 132.3±14.65 sec in 0.75mg/kg

Results of studies by Mellingh off et al[28]. 3, Bluestein et al.[29], who also indicated the time of onset , are similar to those of our study. These trials, which had statistical significance identical to our study, revealed that the point of beginning of action of three ED 95 doses of Cis atracurium and Atracurium was quicker than that of two ED 95doses.

This result is consistent with the findings of Ginsberg B et al.30 and AA. Mohamed et al.27, who found that raising the dose of atracurium from 3ED95 (0.75mg/kg) to 4ED95 (1mg/kg) significantly sped up the onset times in groups III and IV (1 mg/kg) as compared to groups I and II (0.75mg/kg).

When comparing three groups of cis atracurium at different dosages (2 ED95, 4 ED95, and 6 ED95 doses) with atracurium (2 ED95 doses), El-Kasaby et al.26 reported similar outcomes. They claimed that cis atracurium at larger dosages (4 and 6 ED95) causes motion to start much more quickly.

Bluestein et al.29 also compared atracurium (2 ED95) with three different dosages of cis atracurium (2 ED95, 3 ED95, and 4 ED95), and they found that the effects on the initiation of motion were similar.

TOF

Recovery of neuromuscular function results from a decrease in plasma concentrations, and distribution typically accounts for a larger portion of this decrease.

Recovery now depends at least 25%-75% more on drug eradication than on drug distribution.

The longer spontaneous recovery period and much faster atracurium onset were benefits of the larger dose. The existence of TOF = 2 as the timeframe for less extreme neuromuscular blockade allowed the present study to observe the spontaneous recovery period.

In the current study, the time of onset and spontaneous recovery have been 65.4±10.6 and 85.0±11.40min for 1mg/kg atracurium as compared to 132.3±14.65 and 60.8±9.70 min in 0.75mg/kg.

In the observe by way of P Chalermkitpanit et al[6], the spontaneous recovery time for 1 mg/kg atracurium was 80.86 ± 14.26 minutes, even as Lennon RL et al[31] reported that the length of duration of action as 57minutes.. Hui L et al[32] and Xue FS et al[33] confirmed that onset time and recovery time of atracurium had been affected by age and gender. Women have shorter onset times and longer recovery times than men.

The recommended time for 25% recovery from reversal in the R Ranjan et al. study24 was 32.4 1.90 min in the atracurium group, compared to 49.46 1.86 min in the cis atracurium group (P 0.001). Carroll et al.34 reported that atracurium 0.5 mg/kg (47-48 min) and cis atracurium 0.15 mg/kg (51-59 min) took longer to reach 25% recovery after drug delivery, but the difference eventually became inconsequential. Atracurium had a mean 25% recovery time of 32.11 3.2 min while cis atracurium had a mean 25% recovery time of 51.61 2.5 min, according to Shyamlal et al23 (P 0.05). Additionally, they observed that atracurium and cis atracurium turned had healing times of 2.1 0.3 and 2.5 0.2, respectively. When Bergeron et al.35 evaluated the three different

cisatracurium dosages of 0.05 mg/kg, 0.15 mg/kg, and 0.3 mg/kg, they found that while the recovery time increased, the onset was not significantly different in adults.

Hemodynamic Effects

Concern has been raised about histamine release caused by bolus of high dosage atracurium lowering blood pressure. The 1 mg/kg dose of atracurium demonstrated a statistically significant reduction in mean arterial pressure, SBP, and DBP following intubation in the current trial when compared to the lesser doses.

At the time of intubation, the heart rate in the 1 mg/kg group significantly increased in comparison to the 0.75 mg/kg group. The adjustment of reduced systemic vascular resistance brought on by histamine release may have caused tachycardia. A prior study that administered an excessive dosage of atracurium noted the similar hemodynamic pattern of mild hypotension and tachycardia. Similar conclusions were made by Koh KF et al. (2015), Mohamed AA et al. (2017), and Naguib M et al. (2016).

The maximal MABP and HR adjustments of patients receiving cis atracurium were minor and similar to those seen in patients receiving two times the ED95 of atracurium, according to Lien et al.36 and Basta et al.13. Similar hemodynamic outcomes were reported for atracurium and cisatracurium by Yazdanian et al37, but they determined that atracurium had a better cost-benefit ratio. Because of cis atracurium's stereospecificity, there was no histamine release signal even at higher dosages (8 ED95).

Summary

• Majority of study subjects in each group A and group B had been from 46-55 age group (41.1% 44.6% respectively).

Patients in groups A and B have a higher proportion of female patients (62.5% versus 50.54%).

• For patients in both groups, mean arterial pressures have been recorded prior to induction, during induction, during intubation, and 1, 3, and 5 minutes following intubation. It was confirmed that, compared to the lower doses, the mean arterial pressure significantly decreased at 1, 3, and 5 minutes after the onset of anaesthesia.

• In comparison to the lower doses, the mean heart rate significantly increased at 1, 3, and 5 minutes following anaesthesia induction.

The SBP was significantly lower at 1, 3, and 5 minutes following the induction of anaesthesia as compared to the lower dose, according to a comparison of mean SBP values.

• The DBP at 1, 3, and 5 minutes after anaesthesia induction significantly decreased as compared to the lower dose, according to a comparison of mean DBP values.•

Comparison of Mean SPO2 confirmed that there was no exchange in mean SPO2 ranges in both the groups.

• Intubating conditions had been exceptional in 55.4% of the patients in 1mg/kg atracurium group, compared to 32.1% in 0.75mg/kg group. More number of patients in 0.75mg group had excellent intubation conditions (53.6%). Only one affected person in higher dose group showed bad intubating conditions.

- The time of onset and spontaneous recovery have been 65.4±10.6 and85.0±11.40 min for 1mg/kg atracurium in comparison to 132.3±14.65 and 60.8 ±9.70 min in 0.75mg/kg.

Conclusion

In the current investigation, we found that intubation had a high dose-dependent success rate. Atracurium provided excellent intubating circumstances with a shorter onset time at 1 mg/kg. Conclusion: In a few situations, a very strong dose of atracurium administered without priming may be employed as an alternate neuromuscular blocker for sequential induction of anaesthesia.

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IEC/No.09/2021
Date-22/1/2021

B.L.D.E. (DEEMED TO BE UNIVERSITY)

(Declared vide notification No. F.9-37/2007-U.3 (A) Dated. 29-2-2008 of the MHRD, Government of India under Section 3 of the UGC Act, 1956)

The Constituent College

SHRI. B. M. PATIL MEDICAL COLLEGE, HOSPITAL AND RESEARCH CENTRE

INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Institutional ethical committee of this college met on 11-01-2021 at 11 am to scrutinize the synopsis of Postgraduate students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has been accorded Ethical Clearance

Title: Determination of dose and efficacy of atracurium for rapid sequence induction of anaesthesia: A randomized prospective study.

Name of PG student: Dr Manchala Priyanka Department of Anaesthesiology

Name of Guide/Co-investigator: Dr Vijaykumar T K Professor of Anaesthesiology

DR .S.V.PATIL
CHAIRMAN,IEC

Institutional Ethical Committee
B L D E (Deemed to be University)
Shri B.M. Patil Medical College,
VIJAYAPUR-586103 (Karnataka)

Following documents were placed before Ethical Committee for Scrutinization:

1. Copy of Synopsis / Research project
2. Copy of informed consent form
3. Any other relevant documents.

INFORMED CONSENT FORM

B.L.D.E(Deemed to be University)

SHRI B.M PATIL MEDICAL

COLLEGE HOSPITAL AND RESEARCH

CENTRE, VIJAYPURA-586103, KARNATAKA

TITLE OF THE PROJECT: "Determination of dose and efficacy of atracurium in rapid sequence induction of anesthesia"

PRINCIPAL INVESTIGATOR: **DR. MANCHALA PRIYANKA**

Department of Anesthesiology

BLDE (Deemed to be University)

Shri B.M Patil Medical College and Research Centre,

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PG GUIDE: **DR. VIJAYKUMAR T.K**

Professor

Department of Anesthesiology

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Shri B.M Patil Medical College Research Centre,

Sholapur Road Vijaypura-586103

I have been informed that this study is "Determination of dose and efficacy of atracurium in rapid sequence induction of anesthesia". I have been explained about this study in the language which I understand. I have been explained about the reason for doing this study and selecting

me/my ward as a subject for this study. I have been told that my participation in the above study is voluntary, and I am aware that I can opt-out of the study at any time without having to give any reasons for doing so. I am also informed that my refusal to participate in this study will not affect my treatment by any means.

I agree to participate in the above study and cooperate fully. I agree to follow the doctor's instructions about my treatment to the best of my knowledge.

CONFIDENTIALITY:

I understand that medical information produced by this study will become a part of this Hospital records and will be subjected to the confidentiality and privacy regulation of this hospital. Information of a sensitive, personal nature will not be a part of the medical records but will be stored in the investigator's research file and identified only by a code number. The code key connecting the name to numbers will be kept in a separate secure location.

If the data are used for publication in the medical literature or teaching purposes, no names will be used, and other identifiers such as photographs and audio or videotapes will be used only with my special written permission. I understand that I may see the photograph and videotapes and hear audiotapes before giving this permission.

REQUEST FOR MORE INFORMATION:

I understand that I may ask more questions about the study at any time, and Dr Manchala Priyanka available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study, which might influence my continued participation.

If during this study, or later, I wish to discuss my participation or concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me and that a copy of this consent form will be given to me for my careful reading.

REFUSAL OR WITHDRAWAL OF PARTICIPATION:

I understand that my participation is voluntary, and I may refuse to participate or may withdraw consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital.

I also understand that Dr. Manchala Priyanka will terminate my participation in this study at any time after she has explained the reasons for doing so and has helped arrange for my continued care by my own physician or therapist.

INJURY STATEMENT:

I understand that in the unlikely event of injury to me/my ward, resulting directly to my participation in this study, if such injury were reported promptly, then medical treatment would be available to me, but no further compensation will be provided.

I understand that by my agreement to participate in this study, I am not waiving any of my legal rights.

I have been explained the purpose of this research, the procedures required, and the possible risks and benefits, in my own language.

I have been explained all the above in detail, and I understand the same. Therefore I agree to give my consent to participate as a subject in this research project.

Patient's Signature: Witness Signature :

Name :

Date :

DR. VIJAYKUMAR.T.K **DR.MANCHALA PRIYANKA**

(Guide) (Investigator)

STUDY SUBJECT CONSENT STATEMENT:

I confirm that **DR. MANCHALA PRIYANKA** has explained to me the purpose of this research, the study procedure that I will undergo, and the possible discomforts and benefits that I may experience in my own language.

I have been explained all the above in detail in my own language, and I understand the same. Therefore, I agree to give my consent to participate as a subject in this research project.

Participant: Date:

Witness to the above signature Date:

SCHEME OF CASE TAKING:

PROFORMA

STUDY “Determination of dose and efficacy of atracurium for rapid sequence induction of anesthesia"

Name of the patient :

I.P. No. :

Age : Sex:

M	F

Weight: Date of Admission:

Diagnosis:

Consent was taken for study:

Y	N

Group allocated :

A	B

Pre- anesthetic evaluation :

Chief complaints :

Past History :

a) Presence of any comorbid condition - Diabetes/ Hypertension/ Ischemic heart disease/ Cerebrovascular accident / Asthma/ Epilepsy/ Bleeding disorder/ Drug allergy/ any other .

b) Drug Therapy

c) H/o previous anesthetic exposure :

Family History :

General Physical Examination:

- General condition :
- Pallor / Icterus / Cyanosis / Clubbing / Lymphadenopathy / Pedal edema.
- Temperature:
- Pulse rate:
- Respiratory rate:
- Blood Pressure :

Airway Assessment:

- Thyromental Distance:

- Mento hyoid distance:
- Mouth Opening:
- TMJ movement:
- Teeth:
- Dentures:

Mallampatti grade :

Systemic Examination :

- Cardiovascular system
- Respiratory system
- Central nervous system

Others

Table 1:Baseline patient characteristics			
		Atracurium dose 0.75mg/kg(n=39)	Atracurium dose 1 mg/kg(n=37)
Age(year)			
Gender			
Female			
Weight(Kg)			
Height(cm)			

BMI(Kg/m^2)			
ASA physical status			
I			
II			
Operations			
Breast Surgery			
Thyroid surgery			
Laparoscopic Surgery			
Exploratory Laparotomy			
Colostomy			
Others			

The value presented as mean ±SD and Frequency (%)

Table 2 :Intubating Conditions		
Intubating Conditions	Atracurium dose 0.75mg/kg(n=39)	Atracurium dose 1 mg/kg(n=37)
Poor		
Not Possible to intubate		
Fair		
Passage of tube with moderate coughing/bucking		
Good		
Passage of tube with slight coughing/bucking		
Excellent		

Passage of tube without coughing/bucking		

Value presented as frequency (%).*1mg/kg vs 0.6mg/kg: P=0.01.*1mg/kg vs 0.6mg/kg: P=0.03.

Table 3: The maximum effect and the duration of action of atracurium according to the peripheral nerve stimulator		
	Atracurium dose 0.75mg/kg(n=39)	Atracurium dose 1 mg/kg(n=37)
Time until PTC=0 (sec)		
Time until TOF counts= 2 (min)		

Value presented as mean ±SD. PTC =Post-tetanic count, TOF:Train-of-force.*1mg/kg vs 0.75mg/kg and 0.6mg/kg: P<0.01. †0.75 mg/kg vs 0.6 mg/kg: P<0.05

Investigations:

- Complete blood picture
- Blood group and type:
- Random Blood sugar :
- Urine routine:
- ECG:
- Chest X-ray PA view :
- Peripheral nerve stimulation
- Any other :

ASA Grade :

Diagnosis:

<u>Premedication</u>:

Induction:

Drug	Dose	Time

Variables	Heart rate	SAP	DAP	MAP	ECG changes	SP02	Meantime of intubation
Pre-induction1 min							
Pre-induction3min							
Pre induction 5 min							
Pre-intubation1 min							
Pre-intubation 3min							
Pre-intubation 5min							

Post-intubation 1min							
Post-intubation 3min							
Post-intubation 5min							

Signature of the Anesthesiologist:

Name:

Designation:

DETERMINATION OF DOSE AND EFFICACY OF ATRACURIUM FOR RAPID SEQUENCE INDUCTION OF ANAESTHESIA: A RANDOMIZED PROSPECTIVE STUDY

by Dr Priyanka Manchala

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