A COMPARITIVE STUDY OF INTRATHECAL FENTANYL WITH HYPERBARIC BUPIVACAINE VERSUS HYPERBARIC BUPIVACAINE WITH NORMAL SALINE FOR ELECTIVECAESAREAN DELIVERY AND IN EARLYPOST-OPERATIVE PERIOD

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CERTIFICATE

This is to certify that the Dissertation **"A COMPARITIVE STUDY OF INTRATHECAL FENTANYL WITH HYPERBARIC BUPIVACAINE VERSUS HYPERBARIC BUPIVACAINE WITH NORMAL SALINE FOR ELECTIVE CAESAREAN DELIVERY AND IN EARLYPOST-OPERATIVE PERIOD"** presented herein by **Dr.M. RAVI KUMAR** , is an original work done in the Department of Anaesthesiology, Government Stanley Medical College and Hospital, Chennai for the award of Degree of M.D.

(Branch X) Anesthesiology under my guidance and supervision during the academic period of 2004-2006.

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DECLARATION

I Dr.M.RAVI KUMAR solemnly declare that the disseration **"A COMPARITIVE STUDY OF INTRATHECAL FENTANYL WITH HYPERBARIC BUPIVACAINE VERSUS HYPERBARIC BUPIVACAINE WITH NORMAL SALINE FOR ELECTIVE CAESAREAN SECTION AND IN EARLY POST OPERATIVE PERIOD "** is a bonafide work done by me in the Department of Anesthesiology, Stanley Medical College and hospital, Chennai, under the able guidance of **Prof.R.MEENAKSHI,** MD., DA., Professor and HOD, Department of Anesthesiology, Govt. Stanley Medical College and Hospital, Chennai – 1.

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INTRODUCTION

Neuromuscular blocking agents are commonly used to facilitate tracheal intubation.

An ideal muscle relaxant should have rapid onset, profound relaxation of all muscles and short duration of action so that the patient's own respiratory function can be restored, should intubation proved to be impossible.

These requirements are best met with Succinylcholine, an ultra short acting depolarizing muscle relaxant. Unfortunately, succinylcholine has many undesirable side effects, some of which may be life threatening.

Rocuronium bromide is a newer steroidal non-depolarizing neuromuscular blocker with a rapid onset and may be a suitable alternative to succinylcholine for rapid control of airway.

There is dose dependent decrease in onset time with Rocuronium and dose of Rocuronium used may influence the rate of onset of satisfactory intubating conditions.

Large doses of Rocuronium (more than 1mg/kg)produces ideal intubating conditions in 30-60 seconds but the duration of action is very much prolonged.

AIM OF THE STUDY

The aim of my study is to evaluate the intubating conditions in 60 seconds using two different doses of Rocuronium with suxamethonium for intubation in children.

It is a study comparing two doses of Rocuronium (0.6mg/kg (2xED95) and 0.9mg/kg (3xED95) with suxamethonium 1.5mg/kg for intubation in children in 60 seconds and observed:-

1) Intubating conditions
2) Hemodynamic changes
3) Adverse effects

PHYSIOLOGY OF NEUROMUSCULAR JUNCTION

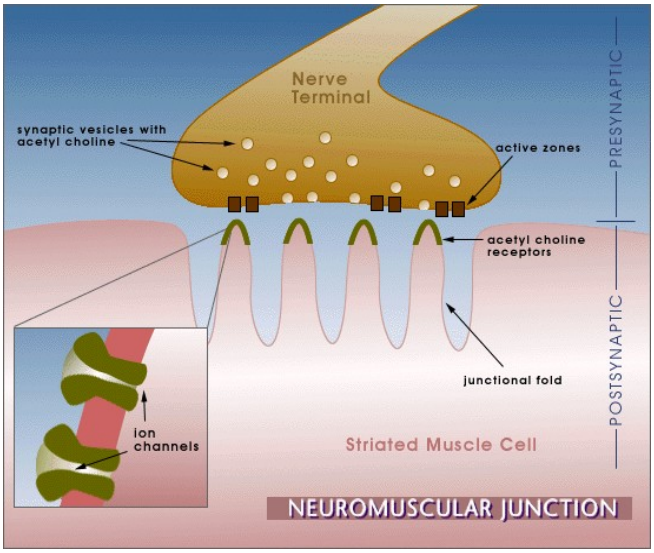
Neuromuscular Junction (NMJ) is a synapse at which an electrical impulse travelling down a nerve is converted into muscle action potential and contraction by chemical transmitters. A motor neuron, along with all the muscle fibres supplied by it forms a motor unit, which follows all or none law of contraction.

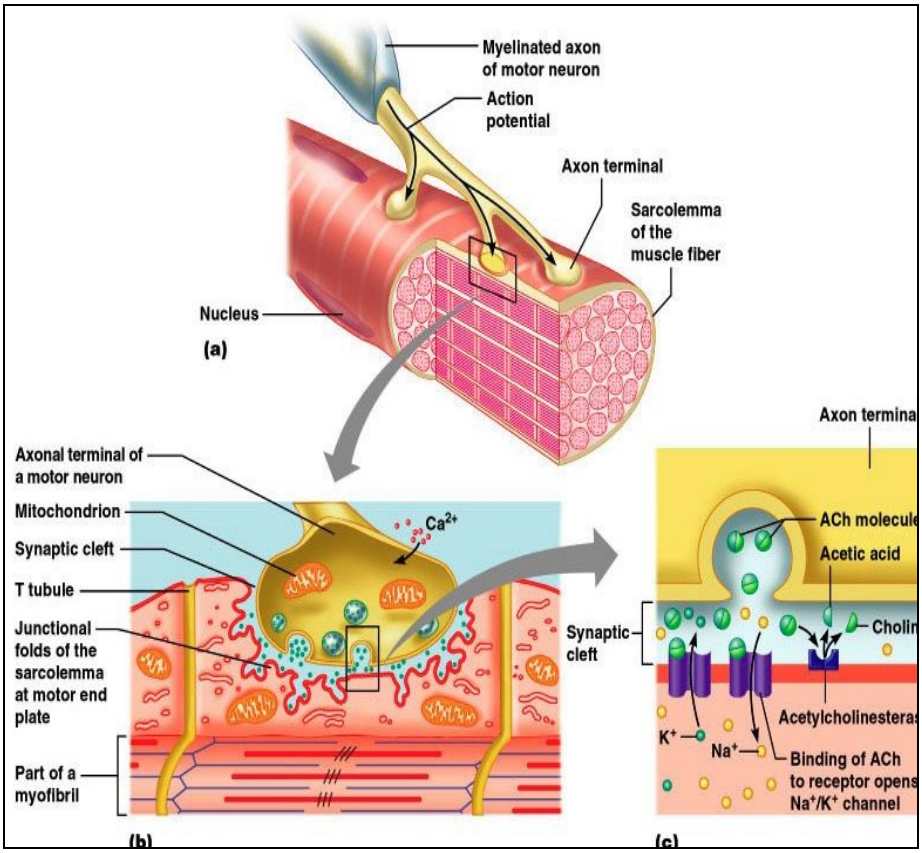
PARTS OF NEUROMUSCULAR JUNCTION

To understand the physiological events occurring during neuromuscular transmission it is essential to understand the anatomy of NMJ, which can be divided into,

1) Presynaptic nerve terminal
2) Synaptic cleft
3) Post Synaptic membrane-Acetylcholine receptors
4) Contractile apparatus

NEURO MUSCULAR JUNCTION





PRESYNAPTIC NERVE TERMINAL:

Presynaptic nerve terminal contains all the apparatus necessary for the synthesis of acetylcholine, which exists in two forms: 20% in the soluble form in the presynaptic axoplasm and 80% in vesicles, which can be further, divided into a readily available pool and a reserve pool. The vesicles are 40-50nm in diameter each containing 1000-10000 molecules of Ach. The walls of the vesicles contain synapsins that help in anchoring the vesicle to the cytoskeletal framework of the

axoplasm.

At the membrane facing the synaptic cleft, there is an electron dense patch, the active zone, around which the readily available pool of Ach vesicles are arranged. Electron microscopy shows small pores between the vesicles at the active zone. These are the calcium channels. The terminal also contains sodium and potassium channels.

The nerve endings on fast muscles are longer and more complicated than those on slow muscles. The reason for this is unclear. These differences in the nerve endings on the muscle surfaces may play a role in the differences in the response to muscle relaxants of fast and slow muscles.

SYNAPTIC CLEFT:

It is 20nm wide space between the nerve terminal and the muscle end plate. The nerve and muscles are held in tight alignment by protein filaments, which span the cleft between nerve and end plate. The muscle surface is heavily corrugated with deep invaginations of the junctional cleft, the primary clefts and the secondary clefts, between the folds in muscle membrane. The shoulders of the folds are densely populated with acetylcholine receptors about 5 million of them in each junction. These receptors are spares in the depths between the folds. Instead these deep areas contain sodium channels. These sodium channels have two component gates, voltage and time dependent gates.

POST SYNAPTIC MEMBRANE – Acetyl Choline RECEPTORS:

The Ach receptors at the NMJ are nicotinic and can be divided into pre synaptic and postsynaptic. The latter are further divided into junctional and extrajunctional.

The Ach receptor is a pentameric transmembrane spaning protein. The 5 protein subunits are arranged in the form of a rosette with a central ion channel. The molecular weight of the receptor is

250000-270000. The mature junctional receptor contains 2 α, β, δ and ε . The immature extra junctional receptor contains 2 α, β, δ and γ which proliferate in abnormal conditions. The extracellular surface of the alpha subunits contains high affinity Ach binding sites.

CONTRACTILE APPARATUS:

The contractile apparatus of the muscle is formed by the myofilament comprising the thin actin filaments and thick myosin filaments, along with tropomyosin, troponin I, T and C. Tropomyosin is attached to the myosin binding site of actin.

The myofilaments combine to form myofibrils. The muscle plasma membrane, the sarcolemma invaginates to form T-tubules which lies in close association with sarcoplasmic reticulum which is a collection of sacs and tubules acting as a reservoir for calcium.

MECHANISM OF NEUROMUSCULAR TRANSMISSION

MECHANISM OF ACETYL CHOLINE RELEASE:

An action potential traveling down the nerve causes the sodium channels in the presynaptic nerve terminal to open, leading to sodium influx. The change in voltage produced by such an influx activates the calcium channels, which open up leading to calcium entry. Calcium mediated activation of calcium-calmodulin dependent protein kinases lead to phosphorylation of synapsins in the vesicle wall, causing the vesicles to break away from the cytoskeletal framework. The vesicles then attach to the active zones with release of Ach molecules. Each nerve impulse causes the release of around 100-400 quanta of Ach. Activation of around 20-25% receptors is essential for impulse transmission.

BINDING OF ACETYL CHOLINE TO RECEPTOR:

The Ach molecule released into the synaptic cleft binds to the alpha subunit. Binding of Ach to both the alpha subunits activates the receptor, leading to configurational changes in the receptor structure and opening up of ion channels. This leads to depolarization of the muscle end plate which when of a sufficient magnitude causes a wave of depolarization to spread across the muscle sarcolemma by means of activation of the voltage dependent gates of the sodium channels in the perijunctional zones. This depolarization wave moving down the T tubule causes release of calcium from sarcoplasmic reticulum. Calcium so released binds to troponin C causing tropomyosin to move and expose the myosin binding sites of actin leading to the formation of cross linkage of actin and myosin heads. They slide over each other leading to shortening of the myofilaments and muscle contraction.

DISSOCIATION OF ACETYL CHOLINE FROM RECEPTOR:

The Ach molecule remains attached to its receptor for a very short period of less than 1 millisecond, after which it dissociates from the receptor and is hydrolysed by the enzyme

acetylcholinesterase. It hydrolyses Ach into acetate and choline, the choline being taken up by the presynaptic nerve terminal and used for further Ach synthesis.

MECHANISM OF NON-DEPOLARISING BLOCKAGE

Non-depolarizing muscle relaxants are drugs having an affinity for the alpha subunits of the acetylcholine receptors mainly at the postjunctional nicotinic receptors and also at the prejunctional sites of nerve ending. Binding of these relaxants to the alpha subunits of Ach receptors can not open the ion channel and it also prevents further binding of Ach molecule. So an action potential is not developed and there is no contraction of muscle fibre. This competitive blockade of Ach receptor is termed as non-depolarising blockade.

MARGIN OF SAFETY:

Atleast 75% of receptors must be occupied before neuromuscular transmission is impaired and if more than 90% of the receptors are occupied, transmission fails.

CHARACTERISTIC FEATURES:

1) Slow onset to maximal effect and slow recovery compared to succinylcholine.

2) The central muscles like the diaphragm, larynx, masseter, orbicularis oculi tend to be affected earlier and recover from the block sooner than those of the peripheral muscles (adductor pollicis) probably as a result of preferential perfusion.

3) Presence of fade and post tetatnic potentiation.

4) Despite flaccid paralysis, the muscles are still able to respond to direct stimulation.

5) The muscle block is reversed pharmacologically by anticholinesterase drugs.

6) Low potency drugs like rocuronium and rapacuronium have rapid onset of action while potent relaxants like doxacurium and pancuronium have relatively slower onset of action and a longer duration of action.

MECHANISM OF DEPOLARISING NEUROMUSCULAR BLOCKADE

Depolarising agents like suxamethonium cause initial depolarization of the endplate due to their acetylcholine like actions, which is transduced into a muscle contraction, but repeated action on the receptor leads to a persistent depolarizing voltage.

This influences the sodium channels in the vicinity of the endplate, the perijunctional area, where the transmembrane voltage causes the voltage gates to remain open and the time gates to remain closed, thereby preventing further ion entry through the channel. Further spread of the depolarization is arrested, and it remains confined to the endplate only. With the progress of depolarization arrested, the channels in the remainder of the muscle are freed of the depolarizing influence and return to their resting state. Thus 3 discrete zones can be delineated namely.

1) Endplate – persistently depolarized.

2) Perijunctional area – Sodium channels still under the depolarizing influence are frozen in closed state.

3) Rest of the muscle – Relaxed, as sodium channels return to resting state.

PHARMACOLOGY

ROCURONIUM BROMIDE

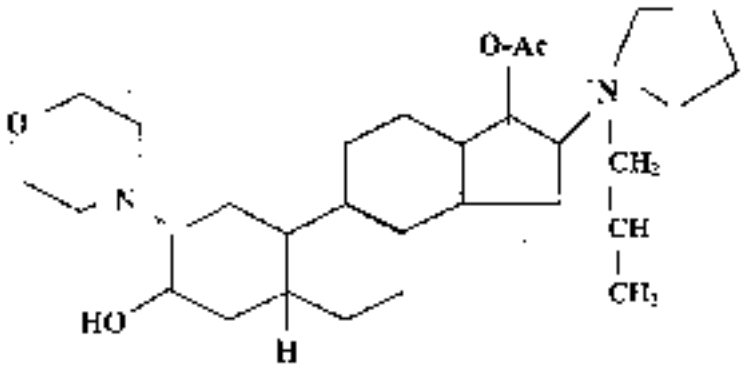
Originally synthesized and studied in the Organon Teknika Laboratories as ORG 9426. Introduced into clinical practice in 1994.

CHEMISTRY:

Rocuronium is a steroidal muscle relaxant of intermediate duration. It is the 2 – morpholino, 3 – hydroxy, 16-N-allyl-pyrrolidino derivative of vecuronium.

1- (2 β, 3α, 5α, 16β, 17β) - 17 - (acetyloxy) - 3 – hydroxy- 2-(4 morpholinyl) - androstan – 16 – yl) – 1 – (2 - Propenyl) pyrrolidium bromide.

MOLECULAR STRUCTURE:



PRESENTATION:

As a clear colourless solution containing 10 mg/ml of Rocuronium bromide. It is available in 5ml & 10ml Vials.

ROUTES OF ADMINISTRATION:

Intravenous. Intramuscular.

Doses:-

ED95	:	0.3 mg / kg
Intubation at 60 – 90 sec	:	0.6 – 0.9 mg / kg
Relaxation (N2O / O2)	:	03 – 04 mg / kg
Relaxation (Vapour)	:	0.2 – 0.3 mg / kg
Maintenance	:	0.1 – 0.15 mg / kg
Infusion	:	8 – 12 microg / kg / min.

ONSET OF ACTION:

89 + / - 36.9 Seconds

DURATION OF ACTION:

2 x ED95 – 27.3 +/- 8.2 min.

3 x ED95 – 53.0 +/- 15.0 min

RECOVERY INDEX:

8 – 17 Minutes

If given by intramuscular injection into the deltoid (1 mg/kg in infants and 1.8mg/kg in children), it permits intubation in approximately 3 minutes.

PHARMACOKINETICS:

Distribution:-

The drug is 30% bound to plasma proteins.

Metabolism:-

Rocuronium is eliminated primarily by the liver with a small fraction (~ 10%) eliminated in the urine. It is taken up into the liver by a carrier mediated active transport system. The putative metabolites 17-desacetyl rocuronium and 16N desallylrocuronium are pharmacologicaly inactive.

TABLE – I

Pharmacokinetics of rocuronium bromide

Age Group	Volume of distribution at steady state (VDss ml/kg)	Clearance ml/Kg/ min	Elimination half; life (min)
Normal Adult	207	2.89 +/- 0.25	70.9 +/- 4.7
Elderly	399 +/- 122	3.67+/-1	97+/-69.1
Children	224	2.67	46 – 55

PHARMACODYNAMICS:

Neuromuscular Junction:

It produces non-depolarising muscle paralysis by competitive antagonism of acetylcholine at nicotinic receptors in the post synaptic membrane of the neuromuscular junction. It also has some prejunctional action.

Non depolarizing blockade is characterized by:-

a) Depression of twitch height

b) Fade with train of four or tetanic stimulation

c) TOF ratio < 0.7

d) Post tetanic facilitation and

e) Antagonism of block by anticholinesterases.

It is 7-8 times less potent than vecuronium but has a faster onset. Paraylsis occurs first in the well perfused fast muscles and last in the diaphragm. Onset of block is faster but less intense at the adductor muscles of the larynx than at the adductor pollicis muscle while the diaphragm is affected later but recovers earlier than the adductor pollicis muscle.

The action of rocuronium can be increased in the presence of hypokalemia, hypermagnesemia, hypocalcemia, hypoproteinemia, dehydration, acidosis, hypercapnia and cachexia. Under hypothermic conditions the neuromuscular blocking effect of rocuronium, is increased and the duration prolonged.

Cardiovascular System:

The autonomic safety ratio for vagal block (3.0 – 5.0) is about 10 times less than that of Vecuronium. Reports of slight to moderate increases in heart rate may be due either to rocuronium producing pain on injection or to its weak vagolytic effect. The heart rate increase may be controlled by the prior administration of fentanyl. Rocuronium in doses upto 0.6 mg/kg has cardiovascular effects that are negligible. At higher doses (0.9 – 1.2 mg/kg) increases in heart rate of 10 to 25% has been observed.

Histamine Release:

Rocuronium is not associated with clinical signs of histamine release in doses upto $5XED_{95}$.

Anaphylaxis / Anaphylactoid Reactions:

In terms of allergy rocuronium appears to have a good safety profile.

Central Nervous Sytem:

No effect as it does not cross the blood-brain barrier. No effect on Intracranial pressure.

Inra Ocular Pressure:

Minimal change in intraocular pressure. Appears to be safe for use in rapid-sequence induction of anesthesia for perforating eye injuries.

Placental Transfer:

Rocuronium does not cross the placenta in significant amounts.

Pseudocholinesterase Inhibition:

This may result in the prolongation of action of drugs that depend on cholinesterase for their metabolism like succinylcholine and mivacurium. The anticholinesterase activity of rocuronium is less than that of vecuronium.

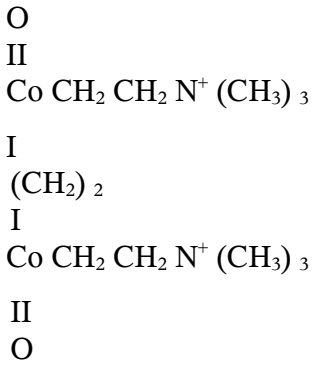
SUCCINYL CHOLINE

It is a Depolarising muscle ideal relaxant. It is the Relaxant for intubation since it is of quick onset and short duration of action.

Chemistry:

It is composed of two molecules of Acetyl choline linked back to back through Acetate – Methyl groups.

Molecular Structure



Presentation

As a solution, it is available in 10ml vials containing 50mg/ml and in vials containing 100mg powder form.

Routes of administration

Intravenous

Intramuscular

Dose

ED_{95}	-	0.51 – 0.63 mg/kg
Intubation dose	-	1 – 1.5mg / kg
Onset of action	-	30 to 60 sec.
Duration of action	-	4 to 10 min.

Pharmacokinetics

Brief duration of action of succinyl choline is due to its rapid hydrolysis by plasma choline esterase which is synthesized in liver. It is metabolised to succinyl mono choline which is subsequently hydrolysed into succinic acid and choline.

Plasma cholinesterase influences duration of action of succinyl choline. Plasma cholinesterase activity is decreased thereby prolonging succinyl choline effect in

1) Liver disease
2) Organophosphorus compounds poisoning
3) Neostigmine, metoclopramide co-administration

In healthy individuals, some times, action of succinyl choline is prolonged due to the presence of Atypical cholinesterase.

Dibucaine No:

It is the percentage inhibition of enzyme activity (Plasma Cholinesterase) by Dibucaine. (local anaesthetic).

Dibucaine inhibits the activity of normal cholinesterase enzyme by 80% compared with only 20% inhibition of activity with Atypical enzyme.

Pharmaco dynamics

Succinyl choline attaches to each of the alpha subunits of the nicotinic cholinergic receptor and mimics the action of Acetyl Choline thus depolarizing the post junctional membrane. Here, the hydrolysis is slow resulting in sustained depolarization (opening) of the receptor ion channels.

Neuromuscular blockade develops because a depolarized post junctional membrane cannot respond to subsequent release of Acetyl Choline (Depolarizing Blockade). It is otherwise called as **Phase I Blockade.**

Phase II Blockade:

- Single large dose of succinyl choline (more than 2mg /kg IV)
- Repeated small doses of succinyl choline
- Prolonged continous infusion

may result in post junctional membranes that do not respond normally to Acetyl choline even when the post junctional membranes have become repolarized (desensitization blockade). Mechanism for this blockade is unknown

Adverse Effects

Cardiac Dysrhythmias

Sinus Bradycardia, Junctional Rhythm, and sinus arrest can occur following administration of succinyl choline.

Hyperkalemia

It is common in patients with

1) Muscular dystrophy
2) Third degree Burns
3) Denervation
4) UMN Lesions.

Myalgia

Post operative myalgia is common following administration of Succinyl choline.

Myoglobinuria

Increased Intra Gastric Pressure

Risk of aspiration is more

Increased Intra Ocular Pressure-

Increased intracranial pressure

Sustained skeletal muscle contraction – masseter spasm following co administration of halothane is not uncommon.

REVIEW OF LITERATURE

Rocuronium Bromide is a non deploarising muscle Relaxant with fast onset.

Dose of Rocuronium used may influence the onset and duration of action. Literature was reviewed to compare intubating conditions with Rocuronium 0.6mg/kg and 0.9mg/kg and with suxamethonium 1.5mg/kg

1) CALUDIA A.Y. CHENG, CINDY ST AUN compared two doses of Rocuronium (0.6mg/kg and 0.9mg/kg) with suxamethonium 1.5mg/kg and concluded that Rocuronium 0.9mg/kg provides similar intubating conditions to suxamethonium.

2) Madhav S.Barve, Roopa Sharma compared intubating conditions and time course of action of Rocuronium & succinyl choline in paediatric patients and concluded that Rocuronium may be a suitable alternative to succinyl choline for intubation in paediatric age group.

3) Mazurek A J ; Hann S. compared Rocuronium versus succinylcholine for Rapid Sequence induction and concluded larger doses of rocuronium may be an alternative to suxamethonium.

4) MC Donald PF, Sanisbary DA, Evaluated onset time and Intubating conditions of rocuronium bromide in children and concluded that intubating conditions are achieved faster with Rocuronium compared to other non depolarizing relaxants.

5) Cooper R, Mirakhur RK, Clarke compared intubating conditions after administration of Rocuronium and suxamethonium and concluded that Rocuronium 0.6mg/kg produces clinically acceptable intubating conditions at 60 seconds after induction with thiopentone 5mg/kg

6) J.D. Crul & Colleagues observed clinically acceptable intubating conditions at 45 seconds with Rocuronium 0.6mg/kg

7) Stoddart compared intubating conditions of Rocuronium 0.6mg/kg with suxamethonium 1 mg/kg in for tonsillectomy patients and onset time was 92 seconds and 42 seconds respectively.

8) J. Viby - Mogenson observed the average clinical duration of intubating dose is shorter in children than adults which may be due to large volume of distribution of control compartment in children.

9) Watanabe K, Chen K., et al (1991) described the pre and postsynaptic effects of org 9426 (Rocuronium) during the onset and recovery from neuromuscular blockade. They found that the relaxant to have moderate potency with rapid onset time, intermediate duration of action and rapid recovery.

10) Meistelman C., et al (1994)[4] found that onset time, intensity of blockade and duration of action were less at the larynx than at the adductor pollicis with 0.5mg/kg of rocuronium. The onset times were 1.4+/- 0.1 and 2.4+/-0.2 minutes at the laryngeal muscles and adductor pollicis, respectively. Maximum blockade was 77+/-5% and 98+/- 1% respectively (P=0.01) and time to 90% recovery was 22 minutes.

11) Jean Paul Cantineau, et al (1994)[5] found that the diaphragm is more resistant than the adductor pollicis to rocuronium 0.6 mg/kg. The onset time for muscle relaxation after 0.6 mg/kg rocuronium was shorter for the adductor pollicis muscle than for the diaphragm

(80+/- 20 Vs120 +/-62 sec). Times for 10% , 25%, 75% and 90% recovery of twitch height were 34+/- 10, 40+/-10, 56+/- 20 and 64+/-21 minutes, respectively for the adductor pollicis and significantly shorter for the diaphragm, 17+/-10, 23+/-9, 33+/-13, and 35+/-10 minutes respectively. The intubating dose of 0.6mg/kg is close to ED95 of 0.5mg/kg for the diaphragm.

12) Peter M.C. Wright., et al (1994)[6] studied the onset and duration of rocuronium and succinylcholine at the adductor pollicis and laryngeal adductor muscles in anaesthetized adults. They found that the onset of effect with succinylcholine was significantly more rapid at the laryngeal adductors (34+/-12 sec) than at the adductor pollicis (56+/-15 sec.) Onset times were similar at the two muscle groups with rocuronium 0.8 and 1.2mg (96+/-29 and 74+/- 36 sec. with 0.8 mg/kg and 54+/-30 and 65+/- sec with 1.2mg/kg at the laryngeal adductors and the adductor pollicis, respectively.) Rocuronium 0.4mg/kg had a more rapid effect at the laryngeal adductors than the adductor pollicis (92+/-29 sec. and 155+/- sec. respectively). They concluded that the laryngeal adductors are more resistant to the action of rocuronium than is the adductor pollicis. Onset of effect of rocuronium in doses greater than 0.8mg/kg is similar to that of succinylcholine at the adductor pollicis but is significantly delayed compared with that of succinylcholine at the laryngeal adductors.

13) De Mey J.C., et al (1994)[7] evaluated onset and intubating conditions of rocuronium bromide 0.5, 0.75 or 0.9mg/kg in adult patients anaesthetized with propofol 2mg/kg and alfentanil 0.5 to 1 μg/kg. They concluded that a dose of rocuronium equal to or larger than 0.6 mg/kg provides acceptable intubations at 60 seconds after administration.

14) Previs TH, Zahn P., et al (1994) Studied the ED95 dose of rocuronium bromide and the

tracheal intubating conditions and time course of actions. They concluded the ED_{95} dose of rocuronium bromide was 0.3mg/kg and duration of action was 20 minutes.

15) Mirakhur R.K. et al (1994)[8] compared onset and intubating conditions of rocuronium bromide 0.6mg/kg and suxamethonium 1mg/kg in adult patients anaesthetized with thiopentone, N_2O in oxygen and small doses of fentanyl. Intubating conditions after rocuronium 0.6mg/kg were found to be clinically acceptable.(good or excellent)in 95% of patients at 60 seconds and in all patients at 90 seconds and in all patients at both times after suxamethonium. There was no significant difference in acceptable intubating conditions between suxamethonium and rocuronium.

16) Scheiber G., et al (1996)[9] Compared 0.6mg/kg rocuronium, 0.1 mg/kg vecuronium and 0.5mg/kg atracurium in children during etomidate-fentanyl-nitrous oxide anaesthesia and found better intubating conditions at 60 seconds after rocuronium than 120 seconds after vecuronium or at 80 seconds after atracurium.

17) K.Barclay., et al (1997)[17] assessed whether low doses of rocuronium improved conditions for tracheal intubation during induction of anaesthesia with propofol 2.5mg/kg and alfentanil 10ug/ kg. They have studied three groups. One group received saline, second group rocuronium 0.1 mg/kg and the third group received 0.3mg/kg rocuronium and they assessed the intubating conditions as judged by jaw opening and laryngoscopy, position of the vocal cords and degree of straining after tracheal intubation. They concluded that injection of rocuronium 0.3mg/kg with propofol and alfentanil provided a high proportion of optimal intubating conditions.

17) Smith I, Saad Rs., et al (1998)[14] Compared the onset of action and the intubating conditions

after rocuronium 0.6mg/kg and vecuronium 0.1mg/kg, when the timing of tracheal intubation was determined by clinical judgement alone like ease of ventilation, jaw and upper airway tone. They concluded that time to laryngoscopy and completion of intubation was markedly shorter in the rocuronium group. Roucronium group also had significantly better intubating conditions.

18) Kirkegaard – Nielsen H., et al., (1999)[10] studied rapid tracheal intubation with rocuronium using a probability based approach. 80 adult patients anaesthetized with fentanyl 2 μg/kg and propofol 2mg/kg randomly received rocuronium 0.0, 0.4, 0.8, or 1.2 mg/kg (n=20/dose). Laryngoscopy was initiated at 40 seconds aiming for intubation at 60 seconds. Doses giving 90 and 95% probability of successful intubation were calculated and found to be 0.83 and 1.04mg/kg respectively. Estimated times until first tactile train of four response after ED_{50} and ED_{95} doses were 32 and 46 minutes respectively. They concluded that after induction with fentanyl and propofol, rocuronium 1.04 mg/kg gives 95% probability of successful intubation at 60 seconds.

19) Andrews JI., et al (1999)[15] Compared rocuronium and succinylcholine for rapid sequence induction of anaesthesia along with propofol and anesthesia was induced using propofol 2.5mg/kg followed immediately by either rocuronium 0.6mg/kg or 1mg/kg or succinylcholine 1mg/kg. Intubating conditions were assessed at one minute and intubation was performed. They concluded rocuronium 1mg/kg given along with propofol in a rapid sequence induction of anaesthesia is clinically equivalent to succinylcholine 1mg/kg.

20) Lam AM., et al (2000) [18] compared the onset and offset time and intubating condition after 1min obtained with rocuronium bromide 0.6mg/kg and succinylcholine 1mg/kg after

induction with propofol and fentanyl. They concluded that rocuronium 0.6mg/kg, when used with propofol and fentanyl for induction, provides intubating conditions similar to succinycholine 1mg/kg at 1 minute.

MATERIALS AND METHODS

This study was conducted at Government Stanley Medical College And Hospital, Chennai.

After institutional approval and informed consent, 60 patients were enrolled in the study.

INCLUSION CRITERIA:-

* Children between 2 – 10 years
* ASA Physical status I & II
* For Elective surgeries posted under G.A.

EXCLUSION CRITERIA:-

* Infants
* Known / suspected difficult Intubation
* H/o any neuromuscular disorder
* Renal / Hepatic disorder
* Known allergy to drugs.
* Surgical procedures of very short duration.
* Patients receiving any medication known to interact with Neuromuscular blocking agent.

PRE OPERATIVE EVALUATION:-

In all the patients, Age, I.P.No, Body Weight, Baseline vital parameters were recorded. History regarding previous anaesthesia, surgery, any significant medical illness, medications and allergy were recorded.

Complete physical examination and Airway assessment was done.

Following laboratory Investigations were done:-

- Haemoglobin %
- Packed cell volume
- Urine – Albumin and sugar
- Chest X-Ray (in selective patients)

PREMEDICATION:

All children received syrup Triclofos 50mg/kg orally one hour before procedure.

INDUCTION OF ANESTHESIA:

After shifting the children to operation theatre, Intravenous line was secured using 22G Cannula in a vein in the dorsum of hand and Isolyte-P infusion was started .

Following monitors were connected to the patients:

1) Non Invasive Blood Pressure
2) Electro Cardiogram Monitor
3) Pulse Oximeter.
4) Precordial stethoscope

These patients were systematically randomized into three groups of twenty each.

Group – I (R6) Thiopentone 5mg/kg + Rocuronium 0.6mg/kg

Group –II (R9) Thiopentone 5mg/kg + Rocuronium 0.9mg/kg

Group-III (S) Thiopentone 5mg/kg + Suxamethonium 1.5mg/kg

Each patient was given Inj. Pentazocine 0.5mg/kg intravenously. After pre-oxygenating these children for three minutes, anaesthesia was induced with Inj. Thiopentone 5mg/kg 2.5% solution given over 15 seconds and followed by:-

Inj. Rocuronium 0.6mg/kg ($2xED_{95}$)

Or

Inj. Rocuronium 0.9mg/kg ($3xED_{95}$)

Or

Inj. Suxamethonium 1.5mg/kg

depending on the group given in less than 5 seconds. Intubation was performed in all children by an experienced anesthetist.(who did not know which relaxant was used for Intubation).

After Intubation and observation of the Intubating conditions and hemodynamic profiles, anesthesia was maintained with 40% oxygen and 60% Nitrous oxide using Jackson-Rees Circuit or closed circuit system with controlled ventilation.

In both rocuronium groups, if additional dose is required Inj. Rocuronium 0.15mg/kg was used. In suxamethonium group Inj. Atracurium 0.5mg/kg and then 0.2mg/kg doses was repeated. And at the end of surgery, Reversal of Neuromuscular blockade was achieved with Inj. Neostigmine 50μg /kg and Inj. Atropine 20μg/kg.

No Regional blockade was given to any of these children before surgery.

The following observations were recorded:-

1) Intubating Conditions:

Intubating conditions were scored by a scoring system used by Mirakhar R.K., Cooper A.R. and Clarke R.S.J (Table II and Table III)

TABLE –II

Scoring of Intubating Conditions

Score	*Jaw Relaxation*	*Vocal Cords*	*Response to Intubation*
0	Impossible to open	Closed (Adducted)	Severe Coughing
1	Open with difficulty	Closing	Mild Coughing
2	Moderate opening	Moving Movement	Slight Diaphragm Movement
3	Easy Opening	Open (Relaxed)	No Movement

TABLE –III

GRADING

Intubating Conditions	Score
Excellent	8-9
Good	6-7
Poor	3-5
Bad	0-2

The following parameters were recorded

- Heart rate
- Systolic blood pressure
- Diastolic blood pressure
- Mean arterial pressure

before induction, during Intubation and 1 min, 3 minutes and 5 minutes after intubation and clinical duration of the Intubating dose was recorded.

EVALUATION OF SIDE EFFECTS:

Patients were monitored for side effects such as signs of histamine release – wheal, flush, bronchospasm, and bradycardia, pain on injection, etc.

STATISTICAL ANALYSIS:

The data was computed and all values were expressed as mean ±SD. The data was analysed using one way anova test, unpaired t test, chi-square test, mann-whitney u test, and kruskal wallis test as appropriate.

OBSERVATION AND RESULTS

The study was conducted on 60 patients randomly allotted into 3 groups as given below:-

TABLE –IV
DRUG DOSAGE AND SCHEDULE

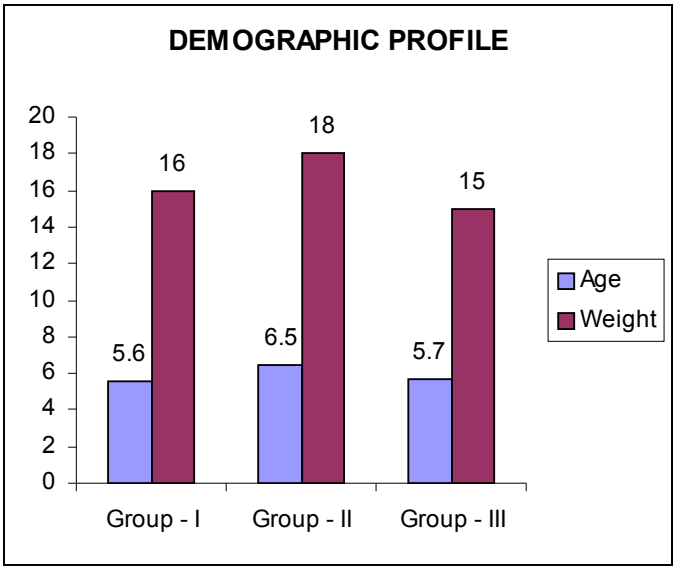
Group	*Drug and Dose*	*Sample Size*	*Abbreviation*
I	Thiopentone 5mg/kg+ Rocuronium 0.6mg/kg	20	R6
II	Thiopentone 5mg/kg+ Rocuronium 0.9mg/kg	20	R9
III	Thiopentone 5mg/kg+ Suxamethonium 1.5mg/kg	20	S

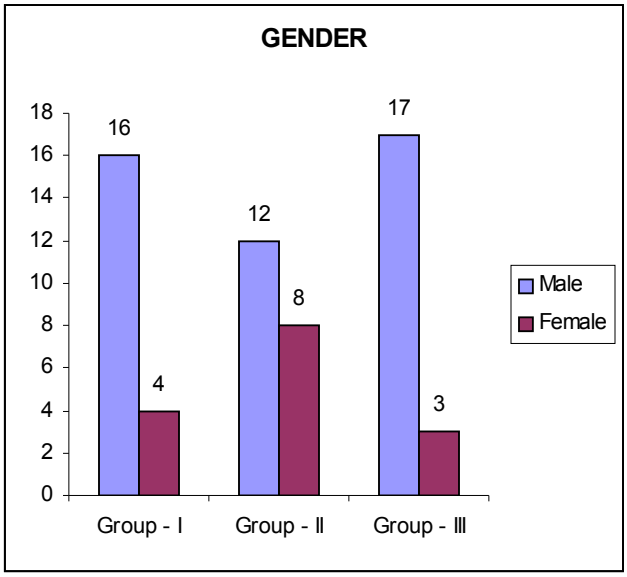
TABLE –V
DEMOGRAPHIC PROFILE

	Group –I	***Group – II***	***Group –III***	***P***
Gender M/F	16/4	12/8	17/3	0.1
Age (Years) Mean ±SD	5.6± 1.96	6.5± 2.19	5.6± 2.17	0.26
Weight(Kg) Mean ±SD	16±3.9	18.35±4.76	15.55±3.9	0.7

P>0.05 – Not significant

Statistical Analysis was done using chisquare test for gender and for weight and age one way Anova test was used.





TRACHEAL INTUBATING CONDITIONS

The scores for Jaw Relaxation, vocal cord position and response to intubation and the total scores were compared between three groups.

TABLE –VI

JAW RELAXATION

Score	Group I (R6)	Group II (R9)	Group III (S)
0 – Impossible to open	-	-	-
1 – Open with difficulty	-	-	-
2 – Moderate opening	15	7	2
3 – Easy opening	5	13	18
Mean ± SD	2.16±0.44	2.65±0.49	2.89±0.32

Statistical Analysis was done using mann whitney u test showing

Group I vs Group II P=0.002 - Significant

Group II vs Group III P= 0.07 – Not Significant

Group I vs Group III P=<0.0001 - Significant

TABLE –VII

VOCAL CORD POSITION

Score	Group I (R6)	Group II (R9)	Group III (S)
0 – Closed	-	-	-
1 – Closing	-	-	-
2 – Moving Movement	13	3	1
3 – Open (Relaxed)	7	17	19
Mean ± SD	2.35±0.49	2.85±0.37	2.95±0.20

Statistical Analysis was done using mann whitney u test showing

Group I vs Group II P=0.0008 - Significant

Group II vs Group III P= 0.30 - Not Significant

Group I vs Group III P=<0.0001 - Significant

TABLE –VIII

RESPONSE TO INTUBATION

Score	Group I (R6)	Group II (R9)	Group III (S)

0-Severe Coughing / Bucking	-	-	-
1 – Mild Coughing	1	-	-
2 – Slight diaphragm movement	16	6	4
3 – No movement	3	14	16

Mean ± SD 2.1±0.42 2.70±0.47 2.8±0.41

Statistical Analysis was done using mann whitney u test showing

Group I vs Group II P=0.002 - Significant

Group II vs Group III P= 0.07 - Not significant

Group I vs Group III P=<0.0001 - Significant

TABLE –IX

TOTAL SCORE

Intubation Score	Group I (R6)	Group II (R9)	Group III (S)
8-9 Excellent	4	18	19
6-7 Good	15	2	1
3-5 Poor	1	-	-
3 – Bad	-	-	-

Statistical Analysis was done using mann whitney u test showing

Group I vs Group II P=<0.0001 - Significant

Group II vs Group III P= 0.01 - Significant

Group I vs Group III P=<0.0001 - Significant

HAEMODYNAMIC PROFILE

The heart rate, systolic pressure, diastolic blood pressure, mean arterial pressure were compared between three groups.

Heart Rate :

Group I	(R6)	-	Table X
Group II	(R9)	-	Table XI
Group III	(S)	-	Table XII

Systolic Blood Pressure:

Group I	(R6)	-	Table XIII
Group II	(R9)	-	Table XIV
Group III	(S)	-	Table XV

Diastolic Blood Pressure:

Group I	(R6)	-	Table XVI
Group II	(R9)	-	Table XVII
Group III	(S)	-	Table XVIII

Mean Arterial Pressure

Group I	(R6)	-	Table XIX
Group II	(R9)	-	Table XX
Group III	(S)	-	Table XXI

Clinical duration of the intubation dose:

Group I	(R6)	-	Table XXII
Group II	(R9)	-	Table XXIII
Group III	(S)	-	Table XXIV

RESULTS

1) Statistical Analysis showed that there was no significant difference between distribution of age, sex, and weight among study groups.

2) All children were succ essfully intubated in 60 seconds without need for second attempt after administration of neuromuscular relaxant.

3) (a) Statistical analysis showed that mean score for Jaw Relaxation was significantly higher in Group II (R9) and Group III (2.65±0.49 in Group II, 2.89± 0.3 in Group III , and 2.16± 0.4 in Group I) .

(b) Mean score for vocal cord position was significantly higher in Group II & Group III when compared to Group I (2.35± 0.49, 2.85± 0.37 2.95± 0.22 in group I, II & III respectively.

(c) Mean score for response to intubation was significantly higher in Group II and Group III when compared to Group I. (2.1± 0.44, 2.7± 0.47, 2.8±0.41 in Group I, II & III respectively)

(d) Mean total intubating score in Group II (R9) and Group III (S) were identical which is significantly higher than Group I. (6.7± 0.86, 8.2± 0.61, 8.7± 0.57 in Group I, II & III respectively).

4) Acceptable Intubating Conditions (Excellent and Good Scores) are observed in almost all the patients except in one patient in Group I (100% in Group II & Group III and 95% in Group I).

But, Excellent Intubating Conditions were produced in most of the patients in Group II and Group III which was significantly higher than in Group I.

5) Statistical anaylsis showed that Mean heart rate, Systolic Blood Pressure, Diastolic Blood Pressure, Mean Arterial Pressure during Intubation in Group I (R6) was higher than in Group II and Group III. (123.6±9.88, 121.2±7.93, 83±5.3, 93.5±5.94 in Group I and in Group II 120.9 ±11.114, 120.1±9.7, 79±7.85, 92.4±7.65) and in Group III 106.8±15, 112±8.56, 75.6±6.28, 88.95±6.4 respectively)

6) Mean heart rate at the end of 5 minutes was higher in Group II (R9) compared to Group I (R6) and Group III (S). (112.7±11.5, 116.64±11.5, 99.75±7.38) in Group I, II and III respectively)

7) Clinical duration of the intubating dose was significantly lower in Group III and among Group I & II, the duration was significantly longer in Group II. (20.35±2.08 minutes in Group I 29.35 ±2.64 minutes in Group II and in Group III 4.6±0.7 minutes).

DISCUSSION

Succinylcholine is an ideal muscle relaxant of choice for Intubation as it is an ultra short acting muscle relaxant producing excellent intubating conditions in seconds and effect wears off in 4-5 minutes usually. But it has got potential adverse effects such as Asystole, Bradycardia, malignant hyperthermia, Raised Intraocular pressure, Raised Intra gastric pressure, Post operative muscle pain. Hyperkalemia etc. and reports of sudden cardiac arrests in patients with undiagnosed muscular dystrophy. Therefore, need exists for a non depolarizing muscle relaxant with a fast onset of action.

Rocuronium is a new steroidal non deploarising muscle relaxant with rapid onset of action and Rapid Onset is believed to be primarily due to its low potency and duration of action is dose dependent. It is short acting with a dose of 0.3mg/kg ($1xED_{95}$) and Intermediate duration of action with 0.6-0.9mg/ kg when more than 1mg/kg is used for Intubation, onset is very quick (less than 45 seconds) but the duration of action is prolonged (more than 1 hour).

In this study we aimed to evaluate the intubating conditions in 60 seconds using smaller doses of Rocuronium (0.6mg/kg ($2xED_{95}$) & 0.9mg/kg ($3xED_{95}$) and compared them with the intubating conditions after 1.5mg/kg of suxamethonium.

There have been several adult studies comparing various doses of Rocuronium with Suxamethonium mimicking rapid sequence induction of Anaesthesia.

We wanted to confirm that Rocuronium is a suitable alternative to suxamethonium even in pediatric age group because the onset time and duration of action are some what different in children

compared with adults.

In the study conducted by R.Cooper 95% patients had developed acceptable intubating conditions at 60 seconds with Rocuronium 0.6mg/kg when the patients were induced with Thiopentone 5mg/kg and Fentanyl 1-3 μ g/kg.

Similarly J.F crul and colleagues observed clinically acceptable intubating conditions at 45 seconds with Rocuronium 0.6mg/kg in patients induced with propofol and alfentanyl 20 μ g/kg.

In the present study. Intubating conditions were excellent with Suxamethonium group and Rocuronium 0.9mg/kg group. When comparing with suxamethonium, Rocuronium 0.6mg/kg was inadequate.

We did not use Atropine with induction as we wanted to observe the Intubation response and vagolytic properties of Rocuronium and Atropine 0.1mg/ml was diluted and kept ready and one patient in Suxamethonium group required 0.2mg bolus once.

The average clinical duration of Rocuronium in pediatric patients is shorter than in adults (26± 7 min observed by J. Viby – Moganson). It may be due to larger volume of distribution of central compartment (V_1) in pediatric population than in adults.

In our study it was 20.35 +/- 2.08 minutes for Group I and 29.35 +/- 2.64 minutes in Group II, and 4.60+/- 0.7 minutes in Group III.

Shorter duration of action for both groups in our study may be due to avoidance of Inhalational

agents and potent opioids No Regional Anaesthesia like Caudal Block was given to any patients in the beginning of surgery.

Agoston S et al (1995) studied the onset time and Intubating conditions of Rocuronium bromide and declared that since there is no parellel correlation between adductor pollicis and laryngeal muscle Relaxation, Neuromuscular transmission monitoring is probably obsolete in regard to intubating conditions.

Cantineau JP, et al (1994)[5] studied the neuromuscular effect of rocuronium on the diaphragm and adductor pollicis muscles in anaesthetized patients and showed that the onset time for diaphragm is slower than for adductor pollicis.

Wright PMC, Caldwell JE, Miller RD., et al (1994)[6] studied onset and duration of rocuronium and succinylcholine at the adductor pollicis and laryngeal adductor muscles in anaesthetized patients and showed that onset of action of rocuronium is slower at the diaphragm than at the adductor pollicis, but faster at the laryngeal muscles than at the adductor pollicis.

We did not monitor the development of neuromuscular block as it has been clearly shown that there is poor correlation between onset time measured at the adductor pollicis and the quality of intubating conditions. So, for more than 40 years authors have abandoned instrumental means to evaluate laryngoscopy and intubating conditions and are using scales which assess clinical criteria only to assess the quality of tracheal intubation. The scale used in the study was used originally by Cooper et al. in their study and is recommended for studies with neuromuscular blockers[8].

HAEMODYNAMIC CHANGES

Thiopentone causes fall in Blood Pressure and Reflex tachycardia.

Pancuronium has vagolytic property and causes hypertension and tachycardia. Vecuronium has no such property and hence provides stable haemodynamics. Rocuronium has got vagolytic properties with larger doses.

In our study mean heart rate, mean systolic, mean diastolic, and mean Arterial pressure at intubation were higher in Rocuronium 0.6mg/kg group when compared to other groups, though statistically insignificant.

Mean Increase in heart rate after 5 minutes was more in Rocuronium 0.9mg/kg Group when compared to other groups.

ADVERSE EFFECTS

The adverse effects looked for were evidences of Histamine Release like Flushing, Wheal, Bronchospasm and Hypotension and pain on injection.

Cooper R., et al (1992) found that there were no significant changes in heart rate or Arterial Pressure after Rocuronium 0.6mg/kg and there was no evidence of Histame Release.

In our study, No signs of histamine release was noted in any of the patients.

One patient of suxamethonium group developed bradycardia which was corrected with bolus dose of Atropine.

SUMMARY

1) Both doses of Rocuronium 0.6mg/kg and 0.9mg/kg produced acceptable intubating conditions (good or excellent) in one minute.

2) But, Excellent intubating conditions identical to suxamethonium was provided with rocuronium 0.9mg/kg group.

3) Duration of action was significantly more in Rocuronium 0.9mg/kg group when compared to suxamethonium.

4) No signs of Histamine release was observed in any of the patients.

5) Increase in Heart rate and Blood pressure at intubation was more in Rocuronium 0.6mg/kg group compared to other groups.

CONCLUSION

Rocuronium 0.9mg/kg ($3xED_{95}$) can be used as an alternative ideal agent to suxamethonium for intubation in children. But while choosing Rocuronium as an alternative to suxamethonium factors such as intubation difficulty and surgical duration should be considered against the potential side effects of suxamethonium.

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A study comparing two doses of Rocuronium with Suxamethonium for Intubation in children in 60 seconds.

PROFORMA

1. Name Age/Sex: P NO. Weight

Diagnosis: Surgery Performed:

Preop Assessment :

Investigations: O/E:

Premedication – Syrup Triclofos 50mg per kg orally one hour before procedure

Shifting to O.T.
IV line Securing
Analgesia used – Inj. Pentazocine 0.5mg / kg IV
Induction – Inj. Thiopentone 5mg/kg
Muscle relaxant used

Group I	-	Inj. Rocuronium 0.6mg/kg
Group II	-	Inj. Rocuronium 0.9mg/kg
Group III	-	Inj. Suxamethonium 1.5mg/kg

Intubation at 60 sec.

Intubation score - **Cooper Scoring System**

Score	*Jaw Relaxation*	*Vocal Cards*	*Response to Intubation*
0	Impossible to Open	Closed (Adducted)	Severe Coughing or Bucking
1	Open with Difficulty	Closing	Mild Coughing
2	Moderate Opening	Moving Movement	Slight diaphragm Movement
3	Easy Opening	Open (Relaxed)	No Movement

Intubation Score

8-9 - Excellent

6-7 - Good

3-5 - Fair

<3 - Poor

No. of Attempts:

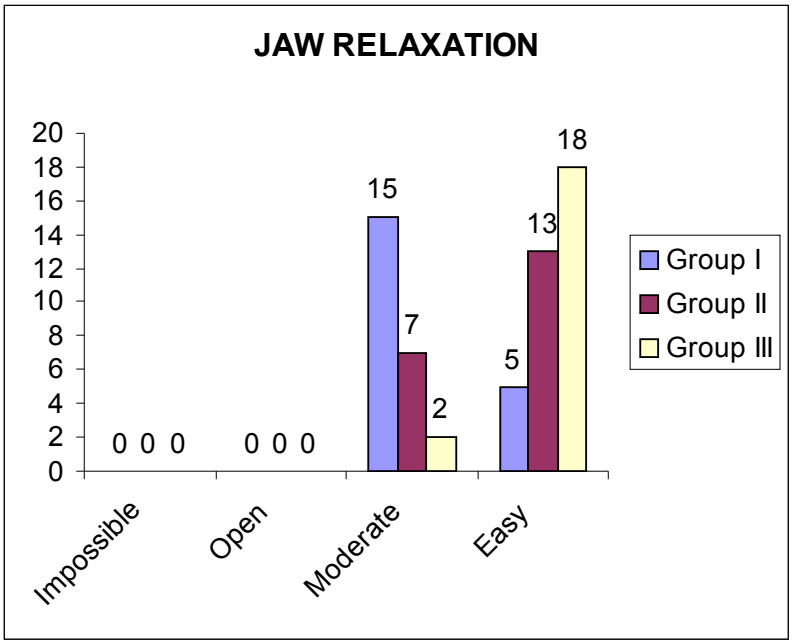
Hemodynamic Response to Intubation

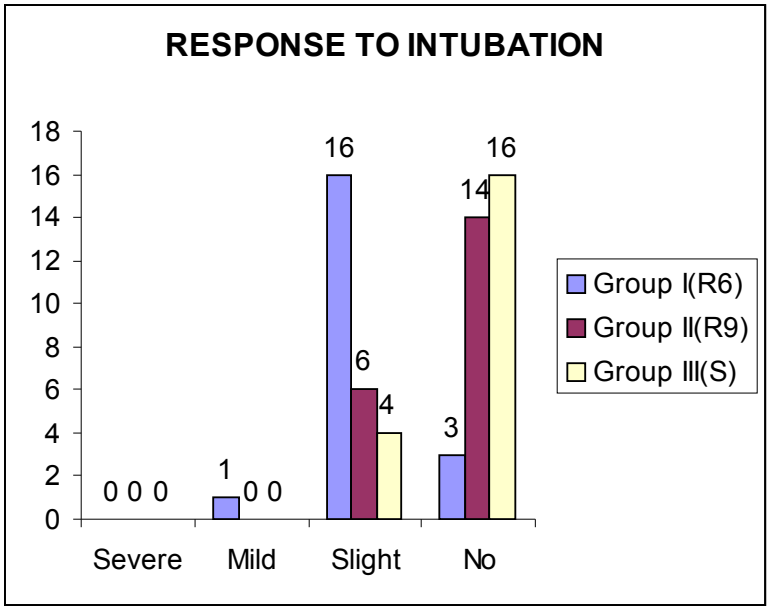
Time	Baseline	Induction	Intubation	1 min.	3 min.	5 min.
HR						
Spo_2						
BP						

Clinical duration of intubating dose :

Adverse Effects noted :

Comments :


JAW RELAXATION
20
18
16
14
12
10
8
6
4
2
0
0 0 0
0 0 0
15
7
2
5
13
18
Group I
Group II
Group III
Impossible
Open
Moderate
Easy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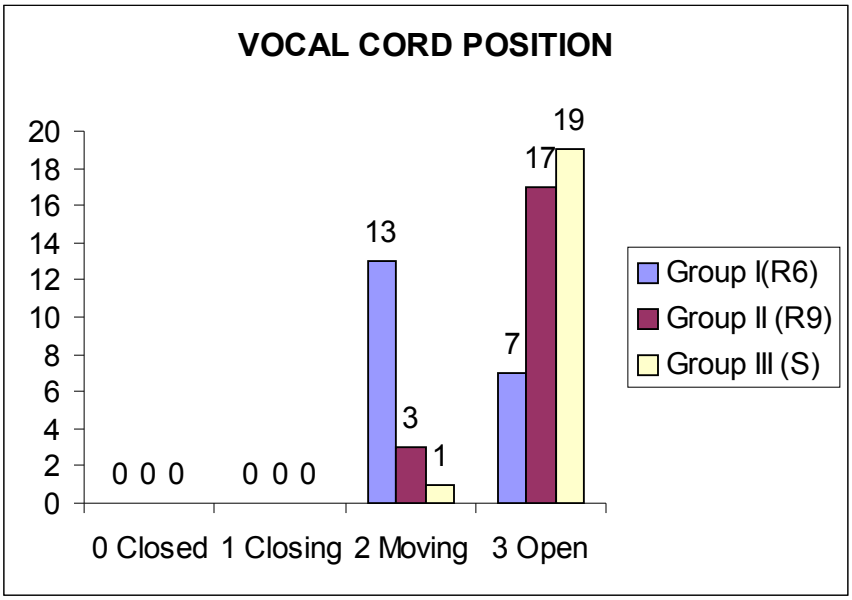
18
16
14
12
10
8
6
4
2
0
0 0 0
1
0 0
16
6
4
3
14
16
Group I(R6)
Group II(R9)
Group III(S)
Severe
Mild
Slight
No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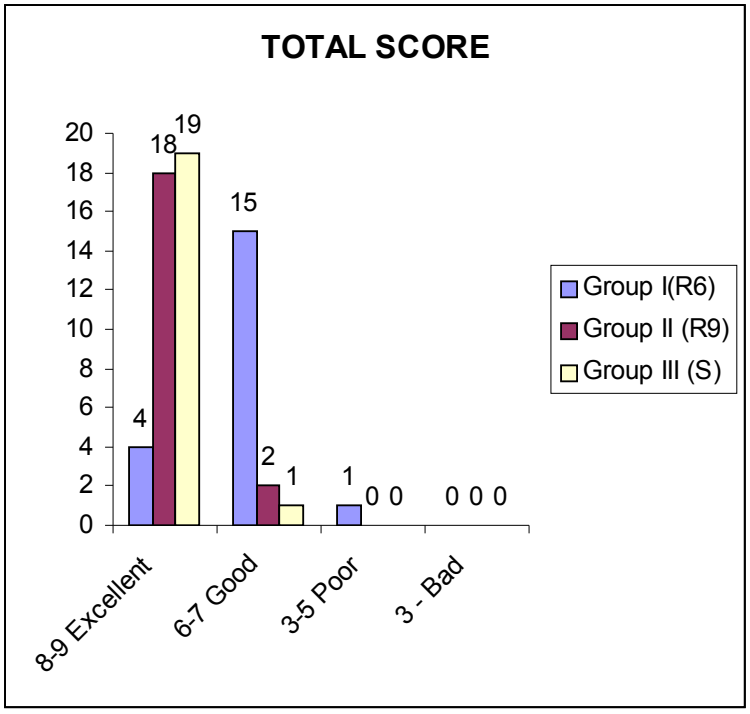
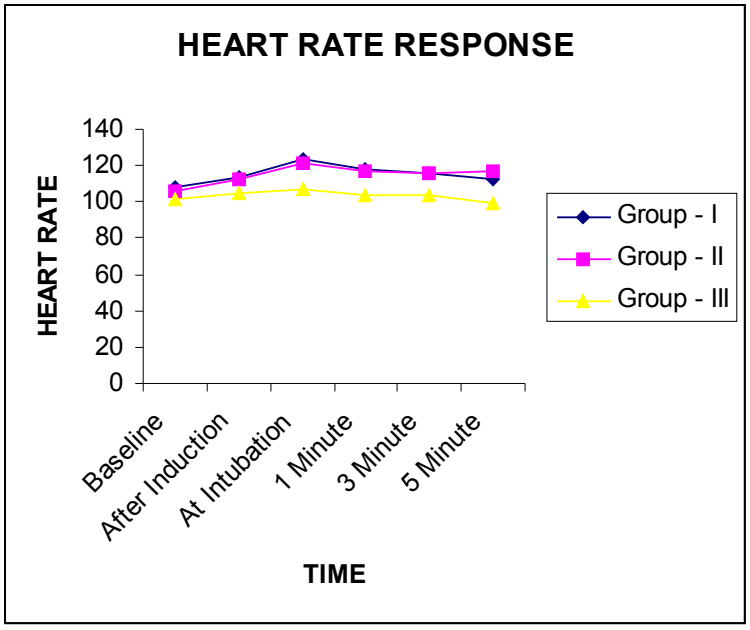
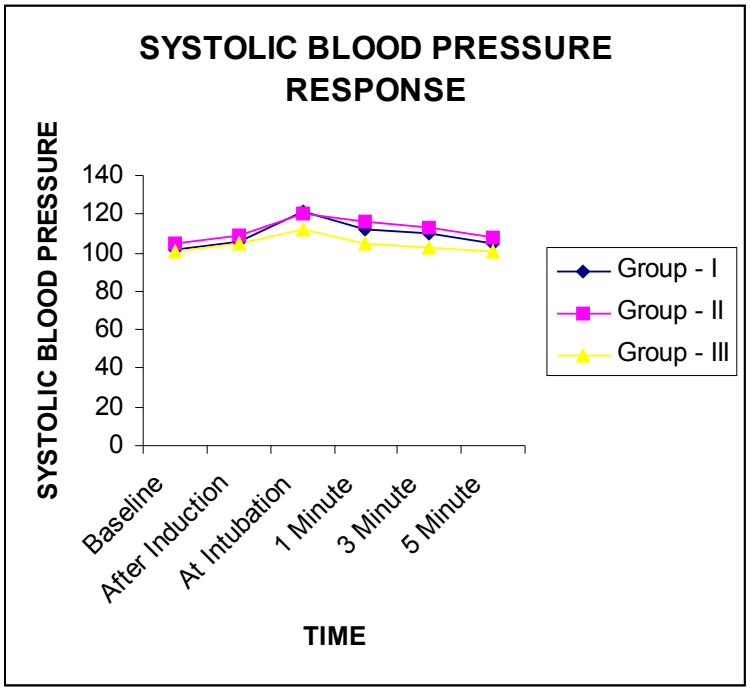
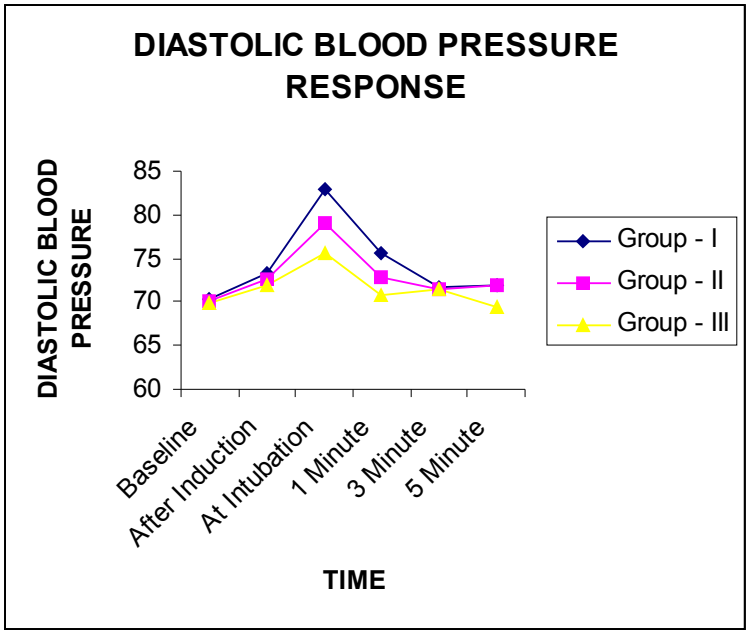
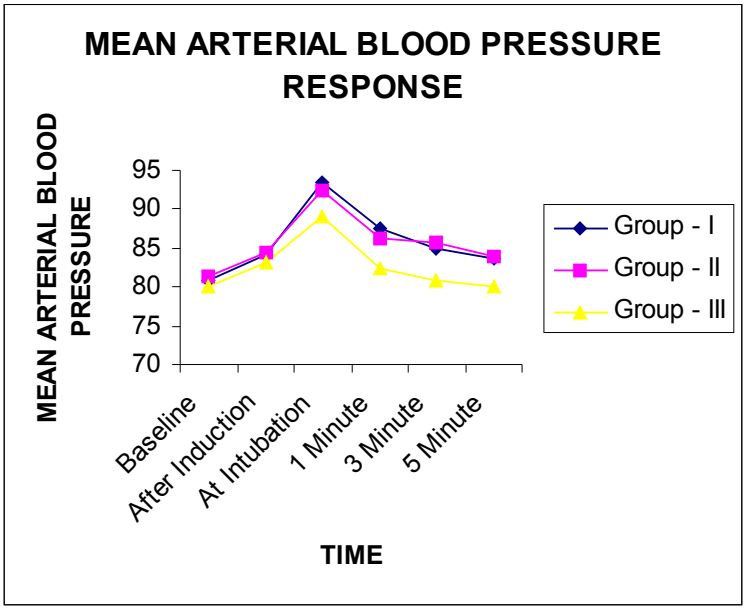
18
16
14
12
10
8
6
4
2
0
0 0 0
0 0 0
13
3
1
7
17
19
0 Closed
1 Closing
2 Moving
3 Open
Group I(R6)
Group II (R9)
Group III (S)


TOTAL SCORE
20
18
16
14
12
10
8
6
4
2
0
4
18
19
15
2
1
1
0 0
0 0 0
8-9 Excellent
6-7 Good
3-5 Poor
3 - Bad
Group I(R6)
Group II (R9)
Group III (S)


HEART RATE RESPONSE
HEART RATE
140
120
100
80
60
40
20
0
Baseline
After Induction
At Intubation
1 Minute
3 Minute
5 Minute
TIME
Group - I
Group - II
Group - III


SYSTOLIC BLOOD PRESSURE RESPONSE
SYSTOLIC BLOOD PRESSURE
140
120
100
80
60
40
20
0
Baseline
After Induction
At Intubation
1 Minute
3 Minute
5 Minute
TIME
Group - I
Group - II
Group - III


DIASTOLIC BLOOD PRESSURE RESPONSE
DIASTOLIC BLOOD PRESSURE
85
80
75
70
65
60
Baseline
After Induction
At Intubation
1 Minute
3 Minute
5 Minute
TIME
Group - I
Group - II
Group - III


MEAN ARTERIAL BLOOD PRESSURE RESPONSE
MEAN ARTERIAL BLOOD PRESSURE
95
90
85
80
75
70
Baseline
After Induction
At Intubation
1 Minute
3 Minute
5 Minute
TIME
Group - I
Group - II
Group - III