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Original Research Article

Post adenotonsillectomy coughing and laryngospasm in children: preextubation mini-dose suxamethonium, lidocaine, and their combination prove prophylactic

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ABSTRACT

Background: Post adenotonsillectomy coughing and laryngospasm in children entails deleterious sequelae, warranting continued search for effective prophylaxis. The aim was to determine the prophylactic efficacy of intravenous mini-dose (0.1 mg/kg) suxamethonium, 1.5 mg/kg lidocaine 1%, and their combination, against coughing and laryngospasm in children emerging from general anaesthesia after adenotonsillectomy.
Methods: Ethical approval and parental written consent secured, 69 children, aged 1-6 years, of American Society of Anesthesiologists (ASA) class I were randomized into groups A, B, and C, of 23 each. All patients had general anaesthesia induced with propofol 2 mg/kg plus fentanyl 2 µ/kg, maintained with isoflurane 1-1.5% in 100% oxygen, and tracheal intubation facilitated by pancuronium 0.075 mg/kg. Two minutes after reversal dose of neostigmine, groups A, B and C respectively received 1.5 mg/kg lidocaine 1%, suxamethonium 0.1 mg/kg, and 1.5 mg/kg lidocaine 1% plus suxamethonium 0.1 mg/kg, intravenously. Tracheal extubation was done 90 seconds after study drug administration, and coughing and laryngospasm were assessed for 10 minutes using modified Minogue and Likert 4-point scales respectively.
Results: All 69 subjects completed the study. The incidence of mild to moderate coughing was 22 (95.7%) in groups A and B, and 19 (82.6%) in group C, p=1.000. Laryngospasm occurred in none (0.0%) in group C, while in groups A and B its occurrence was 1(4.3%), p=1.000.
Conclusions: Mini-dose suxamethonium or lidocaine 1.5 mg/kg prevented grade 3 coughing and laryngospasm; furthermore, their combination prevented grade 3 coughing and achieved zero incidence of laryngospasm.

Keywords: Adenotonsillectomy, Coughing, Laryngospasm, Lidocaine, Suxamethonium

INTRODUCTION

Paediatric adenotonsillectomy is fraught with different grades of coughing and laryngospasm due to airway irritation during recovery from general anaesthesia.[1] Though occurring as reflex, physiological, airway protective mechanisms, persistent severe coughing can precipitate elevated intracranial, intraocular and intra-abdominal pressures, as well as cause hypotension, arrhythmias, diaphragmatic rupture, rib fractures, pulmonary interstitial emphysema, disruption of surgical wounds and herniations, while prolonged laryngospasm can result in hypoxia, arrhythmias and death.[2-4] From scientific evidence, the risk of laryngospasm is increased by 10-fold in young children, especially infants with hypersensitivity of the airway, and adenotonsillectomy is associated with >20% in its incidence.[5]

The necessity to curb the occurrence of coughing and laryngospasm has led to the use of different pharmacological agents including lidocaine, an amide local anaethetic.[5] Heidari et al documented significantly reduced incidence of stridor, bronchospasm and laryngospasm following tonsillectomy in children upon

the prophylactic administration of 1.5 mg/kg lidocaine.[6] Suxamethonium, a depolarizing neuromuscular blocker, has proven efficacy as rescue drug in the treatment of laryngospasm, and a standard intravenous 1-2 mg/kg or intramuscular 3-4 mg/kg intubating dose was used to break larynxgospasm, until Chung et al showed that the use of intravenous mini-dose (0.1 mg/kg) of suxamethonium effectively relieved laryngospasm without causing fasciculation, apnoea or bradycardia.[7,8] Despite this clinical finding, mini-dose suxamethoniun has continued to be reserved for use as rescue drug and not for prophylaxis; furthermore, its use in the preventive management of laryngospasm has not been reported, thus, necessitating clinical studies to explore its probable prophylactic efficacy.[9] Hence, this study was designed to determine the efficacy of intravenous 0.1 mg/kg suxamethonium, 1.5 mg/kg lidocaine 1%, and their combination, in the prevention of coughing and laryngospasm in children awakening from general anaesthesia after adenotonsillectomy.

METHODS

Following institutional approval from the University of Port Harcourt Teaching Hospital (ethical clearance reference: UPTH/ADM/90/S.II/VOL.XI/1071) for a prospective, randomized, double blind, comparative study, and written informed consent from the parents, 69 children aged 1-6 years, of ASA classification I, scheduled for adenotonsillectomy, were randomized into three groups, A, B and C, of 23 each. All 69 subjects completed the study which was conducted from August, 2021, to June, 2022, in the University of Port Harcourt Teaching Hospital, Port Harcourt, Nigeria.

Sample size determination

Sample size (n) was determined using power analysis formula for interventional study,[10]

$$n=\frac{(Z_\alpha+Z_\beta)^2\ p\ (1-p)}{d^2},$$

where,

n=minimum sample size,

Z_α=one-sided percentage point of the normal distribution corresponding to 100% minus the power; with power of 90% for this study, Zα=1.28,

Z_β=percentage point of the normal distribution corresponding to one-sided significance level; with a significance level of 5% (95% confidence interval), Z_β=1.96.

p1=proportion of outcome in group which received intervention. In a related study, the incidence of laryngospasm in group which received intravenous 1.5 mg/kg lidocaine=5.74% (0.0574).[11]

p2=proportion of outcome in control group. A similar study reported laryngospasm rate of 24.32% (0.2432).[11]

p=average proportion=p1+p2=0.0574+0.2432=0.1503.

d=effect size. For this study, the effect size is 25% (0.25).

Substituting,

With allowance for 10% attrition, adjusted sample size=21+2.1=23 per group. Therefore, for the 3 groups, a total of 69 subjects were recruited.

Randomization and blinding were ensured through recruitment of research assistants and picking of opaque sealed envelopes, with the lead researcher blinded to the subjects' group allocations and study drug preparations. The parents of the subjects picked one out of 69 envelopes from a bag on the morning of surgery under the supervision of a research assistant and a nurse. Each of these envelopes concealed a designation (A, B, or C) in it with an equal number of 23 of each in the bag. The envelope picked was excluded from the rest and the patient allocated to the group so designated. A different registrar anaesthetist (second research assistant), blinded to the outcome of the study, prepared the study agents according to the group and weight-based specifications as well as kept the record using different code for each subject's group and drug against hospital number; the lead researcher administered the study drugs and recorded the parameters. The nurse in the otorhinolaryngology (ORL) surgical ward in conjunction with the second research assistant kept the codes for the purpose of quick access to every child involved in the study, in the event of any adverse effects.

All patients had preoperative evaluation and preparation the day before surgery; the parents stopped solid food 6 hours, breast milk 4 hours but gave clear fluid up to 2 hours prior to surgery. Children aged 1-6 years scheduled for elective adenotonsillectomy, in ASA class I or II and whose parents gave consent comprised the inclusion criteria while parental refusal to participate in the study, respiratory tract infection, ASA >II, obesity, muscular abnormality, anticipated difficult airway, known allergy to study drugs, presence of tracheostomy, family history of malignant hyperthermia, history of bronchial asthma, haemoglobinopathy, epilepsy, >2 unsuccessful tracheal intubation attempts, day-case and emergency surgeries constituted the exclusion criteria.

On the morning of surgery, oral midazolam 0.5 mg/kg mixed with clear glucose-based fluid was administered 30 minutes prior to induction for separation anxiolysis. A multiparameter monitor (Dash 4000®) was attached and a precordial stethoscope fixed to the chest for recording baseline heart rate, breath sound, temperature, non-invasive blood pressure (NIBP) and SpO_2, and for continuous monitoring all through the period of surgery. Laryngoscopy and tracheal intubation were done using

appropriate-size Macintosh blade and cuffed entotracheal tube (ETT), facilitated by intravenous doses of propofol 2 mg/kg, fentanyl 2 mcg/kg and pancuronium 0.075 mg/kg. Sub-maximal ETT cuff inflation was combined with gentle pharyngeal gauze-packing to reinforce airway security.

The following were monitored intraoperatively.

Heart rate and blood pressure (systolic, diastolic, mean arterial) every 2 minutes from the time of commencement of surgery for 10 minutes, then every 5 minutes till the end of surgery;

Peripheral arterial haemoglobin oxygen saturation (SpO_2), to ensure a value >95%;

Continuous peripheral temperature, to maintain normothermia (36.5 - 37.4 ºC);

Intraoperative blood loss (by visual estimation) and intravenous fluid administration.

At the end of surgery, the orohypopharynx was suctioned gently under direct vision using an appropriate-size catheter avoiding the tonsillar beds, throat packs were removed, inhalational agent was discontinued and residual neuromuscular blockade reversed with intravenous 0.05 mg/kg neostigmine preceded by 0.025 mg/kg intravenous atropine. Study drugs were given 2 minutes after reversal dose of neostigmine: group A received 1.5 mg/kg lidocaine 1% in a 5 ml syringe and 0.9% normal saline 1 ml in a 2 ml syringe, group B received 0.1 mg/kg suxamethonium in a 5 ml syringe and 0.9% normal saline 1 ml in a 2 ml syringe, while group C received 1.5 mg/kg lidocaine 1% in a 5 ml syringe and 0.1 mg/kg suxamethonium 1 ml in a 2 ml syringe. Ninety seconds after study drug administration, tracheal extubation was performed in deep plane of anaesthesia at end-inspiration. Children were then observed for the occurrence of coughing and/or laryngospasm for 10 minutes following tracheal extubation, as well as occurrence of any fasciculation or respiratory depression in groups B and C. The SpO_2 and heart rate were monitored continuously; temperature, non-invasive blood pressure, and respiratory rate were assessed and recorded every 2 minutes till observed cardiorespiratory parameters were satisfactory. Thereafter, subjects were transferred to the Recovery room for continued monitoring for a minimum of 45 minutes before shifting to the children ORL ward. Intraoperatively, subjects were given lactated Ringer's according to the 4-2-1 regimen.

Coughing was evaluated using a modified Minogue 4-point scale for this study:[12]

Grade 0 (none): no coughing; grade 1 (mild): 1-2 transient coughs; grade 2 (moderate): 3 coughs each lasting ≤2 seconds, or total duration of coughing <5 seconds; grade 3 (severe): ≥4 coughs each lasting >2 seconds, or total duration of coughing >5 seconds.

Laryngospasm was graded using a Likert 4-point scale:[13]

Grade 0 (none): no signs of compromised airway; Grade 1 (mild)=partial occlusion of vocal cords, inspiratory stridor, decreased tidal volume with SpO_2 >95%; grade 2 (moderate): total occlusion of vocal cords (respiratory silence) evidenced by the use of accessory muscles of inspiration, visible paradoxical chest movements and SpO_2 >85%; grade 3 (severe): features of grade 2 laryngospasm plus SpO_2 <85%, cyanosis and bradycardia.

Data collection and analysis

Data were entered into excel spreadsheet and exported to the Statistical Package for Social Sciences (SPSS) version 20.0 (Armonk, NY: IBM Corp.) for statistical analysis by a Statistician not involved in the study. Statistical significance was set at $p<0.05$.

RESULTS

The patients' demographics, base line mean values in SBP, DBP, MAP, PR, RR, SpO_2, temperature, as well as ASA and mean duration of surgery were comparable across the groups (Table 1).

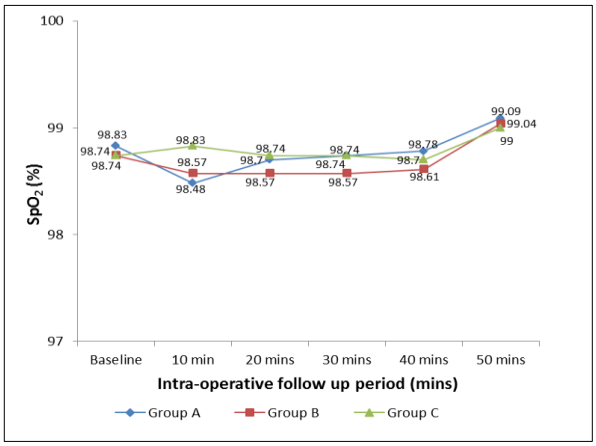

Figure 1: Trends of intra-operative SpO_2 (%) and evaluation period among groups in the study.

Following tracheal extubation, the incidence of coughing was 20 (87.0%), in groups A and B, and 19 (82.6%) in group C, p=1.000. As observed, there was remarkable diminution in the occurrence of laryngospasm in groups A and B, which had same values of 1 (4.3%), while in group C the incidence was 0 (0.0%), p=1.000 (Table 2).

In the assessment of cough severity amongst the children (Table 3), a smaller proportion corresponding to 4 (17.4%) in group A, 3 (13.0%) in group B, and only 1 (4.3%) in group C had grade 2 (moderate) cough, while a

greater proportion experienced grade 1 (mild) cough, with the corresponding values of 16 (69.6%), 17 (74.0%), and 18 (78.3%) for groups A, B, and C. An intergroup analysis of cough severity revealed no statistically significant difference between groups A and B (p=1.000), groups A and C (p=0.407) and groups B and C (p=0.769).

Grade 2 (moderate) laryngospasm was noted in only 1 (4.3%) of the subjects in group A and group B; none (0.0%) of the subjects in group C experienced laryngospasm of any grade (Table 4). On intergroup analysis of laryngospasm severity, there was no significant difference, p=1.000 for groups A versus B, A versus C, and B versus C.

Table 1: Comparison of mean demographic and baseline haemodynamic characteristics across groups in the study.

Variables	Group A (n=23)	Group B (n=23)	Group C (n=23)	P value
Age (years)	4.22±1.48	4.30±1.46	4.13±1.49	0.923
Weight (kg)	16.04±2.96	16.26±2.67	15.74±2.68	0.815
Temp. (°C)	37.19±0.87	37.16±0.11	37.20±0.79	0.268
SpO_2	98.83±0.39	98.74±0.54	98.74±0.45	0.764
SBP (mmHg)	102.48±4.71	101.26±3.63	101.96±3.91	0.604
DBP (mmHg)	55.65±5.07	55.00±5.00	54.78±5.11	0.832
MAP (mmHg)	70.26±1.51	70.17±1.47	69.74±1.25	0.413
HR (b/min.)	113.70±8.34	112.17±6.83	113.17±8.89	0.810
ASA I (%)	23 (100)	23 (100)	23 (100)	
Surgery duration in minutes	41.26±4.42	40.91±4.44	41.30±3.44	0.940

Data are expressed as mean±SD or as number (%); SD=standard deviation.

Table 2: Incidence of laryngospasm and coughing in the study.

Variables	Group A (n=23)	Group B (n=23)	Group C (n=23)	Total (n=23)
	N (%)	N (%)	N (%)	N (%)
Incidence of laryngospasm				
Yes	1 (4.3)	1 (4.3)	0 (0.0)	2 (2.9)
No	22 (95.7)	22 (95.7)	23 (100.0)	67 (97.1)
Fisher's exact test=1.272; p value=1.000				
Incidence of coughing				
Yes	20 (87.0)	20 (87.0)	19 (82.6)	59 (85.5)
No	3 (13.0)	3 (13.0)	4 (17.4)	10 (14.5)
Fisher's exact test=0.338; p value=1.000				

Table 3: Severity of cough across the 3 groups in the study.

Coughing	Group A (n=23)	Group B (n=23)	Group C (n=23)	P value
	N (%)	N (%)	N (%)	
Grade 0	3 (13.0)	3 (13.0)	4 (17.4)	
Grade 1	16 (69.6)	17 (74.0)	18 (78.3)	
Grade 2	4 (17.4)	3 (13.0)	1 (4.3)	1.000^1, 0.407^2, 0.769^3
Total	23 (100.0)	23 (100.0)	23 (100.0)	69 (100.0)

Fisher's exact test: [1]Group A and Group B, [2]Group A and Group C, [3]Group B and Group C.

Table 4: Severity of laryngospasm across the 3 groups in the study.

Laryngospasm	Group A (n=23)	Group B (n=23)	Group C (n=23)	P value
	N (%)	N (%)	N (%)	
Grade 0	22 (95.7)	22 (95.7)	23 (100.0)	
Grade 2	1 (4.3)	1 (4.3)	0 (0.0)	1.000^1, 1.000^2, 1.000^3
Total	23 (100.0)	23 (100.0)	23 (100.0)	

Fisher's exact test: [1]Group A and Group B, [2]Group A and Group C, [3]Group B and Group C.

DISCUSSION

Separately administered, intravenous mini-dose suxamethoniun and intravenous lidocaine demonstrated comparable efficacy in their prophylactic potencies, as evident from the zero incidence of grade 3 coughing and laryngospasm in groups A and B, coupled with the remarkably diminished incidence of grade 2 laryngospasm in the children; to note, the combined administration of the two drugs totally prevented all grades of laryngospasm and grade 3 coughing, as well as better diminished the occurrence of grade 2 coughing in group C, compared to groups A and B. There was no incidence of fasciculation or respiratory depression, post study drug administration in any of the groups.

Preextubation administration of relevant pharmacological agents possessing prophylactic efficacy against post adenotonsillectomy coughing and laryngospasm reduces the incidence of such sequelae.[5] This scientific finding was similarly observed in this study from analysis of the impact of intravenous lidocaine on the severity of measured outcomes. The observation of a 0.0% incidence of grade 3 coughing and laryngospasm in group A lended support to the documentation by Heidari et al that lidocaine was effective against coughing and laryngospasm in children emerging from endotracheal general anaesthesia following adenotonsillectomy, as well as corroborates the empirical report by Sanikop et al regarding recovery profile post paediatric palatoplasty.[6,11]

Following the intravenous administration of 1.5 mg/kg lidocaine 1% prophylactically to children aged 1-6 years in this study, an incidence in laryngospasm as low as 4.3% was observed, which compared with the 0.0% incidence reported earlier by Gefke et al who used a higher dose (2 mg/kg) of lidocaine 2% and an older (>15 years of age) population of children.[14] The evidence that amongst children, the occurrence of laryngospasm was higher in the younger than in the older category had long been documented, with values of 17.4% incidence in laryngospasm in children aged 0-9 years.[15]

Meta-analysis had established the prophylactic efficacy of intravenous lidocaine 1-2 mg/kg against postextubation laryngospam; also, the efficacy of 2 mg/kg intravenous lidocaine for laryngospasm prophylaxis has been documented by Gefke et al.[14,16] For this study, the choice of a dose of 1.5 mg/kg lidocaine was made in consonance with the recommended dose range for its intravenous use documented in the update to 2018 American Heart Association Guidelines.[17] Again, relative to the observation by Sanikop et al of 5.4% incidence in laryngospasm in their 1.5 mg/kg lidocaine group, the reported higher incidence of 16.3% by Manouchehrian et al in the group that received intravenous 1 mg/kg lidocaine 2% indicated likely association between an intravenous lidocaine dose <1.5 mg/kg and a suboptimal prophylactic efficacy against laryngospasm post paediatric adenotonsillectomy.[11,18]

According to earlier scientific reports, the efficacy of intravenous lidocaine is time dependent. Erb et al noted that, compared to the findings of their assessment at 10 minutes, the group of children assessed at 2 minutes after intravenous 2 mg/kg lidocaine recorded a significantly lower incidence of laryngospasm, indicating a short lived drug effect.[19] Thus, judicious timing to perform tracheal extubation soon after onset of action of intravenous lidocaine was warranted for validity of study. Methodologically, therefore, in this study, tracheal extubation was performed at 90 seconds after intravenous lidocaine administration, and assessment of coughing and laryngospasm limited to 10 minutes post ETT removal. An onset of action of within 1 minute after intravenous lidocaine bolus has been documented.[20]

Walker et al have rated suxamethonium as the most reliable pharmacological agent to break laryngospasm.[21] However, due to the occurrence of fasciculations and bradyarrhythmias associated with the recommended clinical doses of 1-2 mg/kg (intravenous) and 3-4 mg/kg (intramuscular), it was conventionally reserved for use as last rescue option in the curative management of laryngospasm.[21] In this study, the low (4.3%) incidence of grade 2 laryngospasm and total absence (0.0%) of grade 3 coughing and laryngospasm noted in group B, following preextubation intravenous administration of mini-dose 0.1 mg/kg suxamethonium, were similar to the observed effects of 1.5 mg/kg lidocaine 1% in group A, and occurred without associated fasciculations and respiratory depression. Drawing an inference from this empirical finding, intravenous mini-dose 0.1 mg/kg suxamethonium possessed prophylactic potency that paralleled the prophylactic efficacy of intravenous 1.5 mg/kg lidocaine 1% against post adenotonsillectomy coughing and laryngospasm in children aged 1-6 years. Again, these observations corroborate the reported unassociation of fasciculation, apnoea and bradycardia with the use of this mini-dose suxamethonium by Chung et al.[8]

A combination of preextubation intravenous mini-dose 0.1 mg/kg suxamethonium and 1.5 mg/kg lidocaine 1% administered to subjects in group C resulting in 100% absence of all grades of laryngospasm and grade 3 coughing, added to a remarkable diminution in grade 2 coughing to 1 (4.3%) without adverse effects, was attributable to the occurrence of positive synergism between the two pharmacological agents. Thus, inferentially, an intrinsic prophylactic potency of mini-dose 0.1 mg/kg suxamethonium against post adenotonsillectomy coughing and laryngospasm achieved favourable synergism, upon its combination with 1.5 mg/kg lidocaine 1% in same paediatric patient.

Younger children, especially infants, had reduced functional residual capacity.[22] Additionally, an autonomic nervous system dysequilibrium characterized by a higher parasympathetic nervous (PNS) tone relative to their sympathetic (SNS) had been reported.[23] Following

computer analysed electrophysiological studies, Harteveld et al observed a cubic trend in PNS activity consisting of an exponential increase from infancy, a plateau in mid-childhood with a decline toward adolescence, and a contrasting linear trend in SNS activity exhibiting a gradual decrease from infancy toward adolescence.[23] Given these peculiar anatomical endowments, the younger category of children pose significant physiological challenge comprising a tendency to rapid desaturation and development of bradycardia during periods of apnoea, precipitating hypoxia and triggering pathophysiological pathways cascading expeditiously to cardiac arrhythmias, hypoxic ischaemic encephalopathy and death, if unrelieved.[4,9] Consequently, post adenotonsillectomy laryngospasm in a child constitutes a nightmare to the paediatric anaesthesiologist.

According to the modified Minogue and Likert 4-point scales used in this study, grade 2 (moderate) cough was identified as 3 coughs, each lasting ≤2 seconds or total duration of coughing <5 seconds, and grade 2 (moderate) laryngospasm as total occlusion of vocal cords (respiratory silence) evidenced by the use of accessory muscles of inspiration, visible paradoxical chest movements and SpO_2 >85%.[12,13] On a critical analysis, while grade 2 cough following tracheal extubation is transient and poses no serious threat to the life of the affected child, grade 2 laryngospasm is invariably a critical phenomenon, demanding prompt effective intervention for the circumvention of an imminent morbimortality. In this study, only one patient in groups A and B experienced grade 2 laryngospasm, which was promptly detected and swiftly aborted with a single intravenous mini-dose of 0.1 mg/kg suxamethonium, without any occurrence of fasciculation, hypoventilation or bradycardia, thus, lending further evidence to the empirical finding of Chung et al.[8]

In any perioperative setting, a patent and functional airway constituted the fulcrum on which the safe conduct of anaesthesia, successful delivery of surgical care to a patient and the immediate postoperative outcome of the clinical interventions are pivoted. Literature documents 15-fold increase in the odds of brain injury or death from airway emergency.[24] To note, that there was high incidence of laryngospasm amongst paediatric populations who received no preextubation prophylaxis had been documented; also, an increasing awareness amongst patients of their rights, and consequent rising trend in the incidence of litigation, had been reported.[25-27] Against this background, a preventive approach, rather than a curative one, is scientifically the rational and preferable choice for the clinical management of perioperative laryngospasm. However, in the event of failed prophylaxis, prompt detection of airflow obstruction and expeditious intervention to achieve swift restoration of airway patency and functionality are desirable goals, considerable as crucial ethical obligations for practising paediatric anaesthesiologists. In this regard, intravenous mini-dose 0.1 mg/kg suxamethonium, by possessing both preventive and curative efficacies against post extubation laryngospasm, coupled with its novel rapidity of onset of 30-40 seconds, and being devoid of causing fasciculation, cardiorespiratory depression and intracavitory hypertension, invariably earns the place of first choice.[28] Therefore, the prophylactic effect of this small dose of suxamethonium against paediatric post adenotonsillectomy laryngospasm is of remarkable perioperative significance when considered against the background of the impending pathophysiolological cascade from hypoxia.

The mechanisms by which intravenous lidocaine and suxamethonium achieved antagonism of coughing and laryngospasm have been linked to their pharmacodynamic properties. Lidocaine reversibly blocks neuronal impulse conduction, and, consequently, tissue response to stimulus, by binding to mainly sodium ion gated channels; its analgesic, anti-hyperalgesic, anti-inflammatory and sympathetic obtunding effects are elaborately documented.[29] Suxamethonium, similar in structure to 2 acetylcholine molecules, binds to post synaptic acetylcholine receptors in voluntary muscles, evokes sustained membrane depolarization and precipitates muscle paralysis antagonizing spasm.[30]

Limitations

A dose-response curve for each drug was not determined and the doses selected were based on previous studies with the assumption that such doses were optimal. Hence, the doses selected for comparison might not be equipotent. Intracavitory pressures were not measured to determine the occurrence of subtle hypertension following the administration of mini-dose suxamethonium as an indicator of latent fasciculations.

CONCLUSION

Intravenous preextubation mini-dose 0.1 mg/kg suxamethonium demonstrated a prophylactic efficacy that equated, and was synergistic, with 1.5 mg/kg lidocaine 1%, against post tracheal extubation coughing and laryngospasm in children following adenotonsillectomy, without causing fasciculations, hypoxaemia and bradycardia.

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