

EVALUATION OF THE EFFECT OF SECOND DOSE OF SUCCINYLMCHOLINE ON THE PULSE RATE IN NON-ATROPINISED PATIENTS UNDER GENERAL ANESTHESIA

Abrar A Abdul-Salam^{*}, Jasim M Salman[@] & Salam N Asfar[#]

^{*}MB, ChB, FICMS Anesthesiology, Al-Sadir Teaching Hospital, Basrah. [@]MB, ChB, DA, FICMS, Assistant Professor of Anesthesiology, Basrah College of Medicine. [#]MB, ChB, MSc, Professor of Anesthesiology, University of Basrah, College of Medicine, Basrah, IRAQ.

Abstract

From the time when succinylcholine(scoline) introduced to clinical anesthesia, intravenous route of administration of the drug has been broadly practiced. Severe bradycardia and arrhythmias may from time to time produced by single or frequent doses. The mechanism of bradycardia caused by succinylcholine administration has not been completely explained, it has been found that succinylcholine produce a unpredictable effects on the sinoatrial node. In our hospital , a second dose of succinylcholine is usually used for short surgical procedures that required good relaxation like anorectal surgery and orthopedic manipulation under anesthesia.

This study aimed to discuss the conditions under which bradycardia appear during general anesthesia with oxygen, propofol, scoline, halothane, along with the use of second dose of scoline and assess the need for atropine to prevent this effect.

Fifty patients aged between 20-50 years of American Society of Anesthesiologists (ASA) class I were included in this study, anesthesia was induced with propofol, scoline, along with oxygen halothane mixture, then 2nd dose of scoline was given 10 minutes later. Frequent pulse rate were recorded.

There was considerable decrease in pulse rate of the patients after the 2nd dose of succinylcholine, 36% of the patients developed bradycardia (pulse rate below 60), and the mean reduction in pulse rate was (23±13 beats/min). However there is no need for the use of atropine since all of the patients return to normal rates (60-100 beats/min) within 1 minute spontaneously.

Conclusion: It has been found that the use of scoline in one dose produce decrease in pulse rate but not reach to the bradycardia level, while the uses of another dose will result in significant reduction in pulse rate that can reach to the level of bradycardia. All patients return to normal level of pulse rate within 1 minute, therefore, there is no need to use atropine neither preoperatively nor after developing of bradycardia since it's a self limiting phenomenon.

Introduction

Suxamethonium chloride (scoline) (succinylcholine) is still the preferable neuromuscular blocking agent. It is a depolarizing neuromuscular blocking drug utilized to paralyze the patients temporarily as an adjunct to general anesthesia, to facilitate endotracheal intubation, and to provide skeletal muscle relaxation during surgery¹.

The popularity of succinylcholine is due to its rapid onset (30-60 seconds) and short duration of action. The extremely

short duration of action of succinylcholine (5-10 minutes) is due to its rapid hydrolysis by pseudo-cholinesterase in the liver and plasma²⁻⁴.

In therapeutic use (The usual intubating dose is 1-1.5 mg/kg IV), bradycardia, hypotension, arrhythmias and cardiac arrest may develop. Sever hyperkalemia and ventricular dysarrhythmias may develop in patients in catabolic states or underlying neuromuscular conditions, including extensive burns, traumatic muscle injury, paraplegia, hemiplegia,

muscular dystrophy, multiple sclerosis, prolonged pharmacologic neuromuscular blockade, upper motor neuron injury or extensive denervation of skeletal muscle⁵. Because of the resemblance of succinylcholine to ACH it is not only stimulate nicotinic receptors, it stimulate all the cholinergic receptors so the entire parasympathetic nervous system and parts of sympathetic nervous system (sympathetic ganglions, adrenal medulla, and sweat glands) are stimulated also. Low dose of scoline can produce negative inotropic and chronotropic but higher doses usually increases heart rate and contractility and elevate circulatory catecholamine level⁶. Different types of arrhythmias have been reported such as nodal bradycardia and ventricular ectopy⁷.

Bradycardia occurs in adults after the second dose of scoline administration approximately 3-8 minutes after the first dose⁸. The mechanism of bradycardia has not been fully elucidated. Succinylcholine and succinylmonocholine effects on sinoatrial node were studied in dogs, it has been concluded that the positive chronotropic effect of succinylcholine may be mediated through beta-adrenergic receptor in the sinoatrial node and that the negative chronotropic effect of succinylmonocholine may be from excitation of cholinergic receptors in the sinus node⁹. A metabolite, succinylmonocholine, appears to sensitize muscarinic cholinergic receptors in sinoatrial node to the second dose of scoline resulting in bradycardia⁶. There are many reports of cardiac arrhythmias following the administration of succinylcholine to normal children including sinus bradycardia¹⁰ sinus tachycardia^{10,11} ventricular ectopics and ventricular bigeminy¹¹ but a causal relationship between the arrhythmias and succinylcholine has not been clear. Proposed arrhythmogenic means of succinylcholine-induced arrhythmias including acute hyperkalaemia and

inflection of parasympathetic outflow through actions on pre- and post-synaptic nicotinic and muscarinic receptors¹². Succinylcholine at doses causing neuromuscular relaxation rarely causes effects attributable to ganglionic blockade. However, cardiovascular effects are sometimes observed that are probably due to the successive stimulation of vagal ganglia (manifested by bradycardia) and of sympathetic ganglia (resulting in hypertension and tachycardia¹³).

In order to overcome this effect, intravenous atropine (0.02 mg/kg in children, 0.4 mg in adult) is given prophylactically prior to the 1st dose in children and before the 2nd dose in adults still recommended in anesthetic practice¹⁴.

This study aim is to discuss the situations under which bradycardia appear during general anesthesia with oxygen, propofol, scoline, halothane, along with the use of 2nd dose of scoline and assess the need for atropine to prevent this effect.

Patients and Methods

Fifty patients were presented for elective anorectal (mostly fissure in ano) and orthopedic surgery under general anesthesia, their age ranged between 20 and 50 years.

All patients were belonging to American Society of Anesthesiologists (ASA) class 1, and were free from cardiovascular diseases.

Exclusion was made for those patients who are less than 18 and more than 50 years old, ASA classes II and above, diabetic patients, patients who have a history of chronic drugs use that had effect on the heart rate like B-adrenergic blocker, a-blocker, Ca-channel blocker, ACE inhibitors, severe hepatic dysfunction that can affects drug metabolism, and any cardiac condition congenital or acquired that can affect heart rate.

All patients were monitored with a standard monitoring devices that are available in our operating room which includes pulse-oximeter and non-invasive blood pressure monitoring.

A baseline pulse rate was recorded before the induction of anesthesia.

Anesthesia was started with pre-oxygenation for five breaths followed by induction dose of propofol (2.5 mg/kg) then a paralyzing dose of succinylcholine 1 mg/kg was administrated. Endotracheal intubation was done 45 seconds after induction. Anesthesia was maintained by halothane-oxygen mixture. Pulse rate was recorded within 1 minute, 10 minutes after the 1st dose. A 2nd dose of scoline 30 mg was injected 10 min after the 1st dose and also the pulse rate was recorded within 1 min., 2 min., 5 min., and 10 minutes after the 2nd dose.

Data was analyzed by using SPSS program (version 20), considering a p-value of <0.05 as statistically significant.

Results

The characteristics of the total fifty patients participating in this study are as follows: age of the patients included in this study ranged from 20-50, mostly were in between 20-30 years (48%). The mean age was 34±8 years as shown in table I.

Table I: Age of the patients.

Age (years)	No.	percentage
20-30	24	48%
30-40	13	26%
40-50	13	26%
Total	50	100%

Most of the patients were males 32 patients(64%) and 18 females(36%) as demonstrated in table II.

Table II: Gender of patients participating in the study.

Gender	No.	percentage
Male	32	64%
Female	18	36%
Total	50	100%

In regard to weight and height, the body mass index of the patients, table III shows that 32 patients (64%) were in the normal BMI. Mean±SD was 25±4.

Table III: Body mass index of patients included in the study.

BMI	No.	percentage
18-25	32	64%
25-30	7	14%
30 or more	11	22%
Total	50	100%

Table IV, demonstrates that most of the need for the second dose of scoline was for anal surgery in 42 cases (84%) while in 8 cases (16%) was for manipulation of the shoulder joint under general anesthesia.

Table IV: Types of operations

Operation	No.	Percentage
Anal surgery	42	84%
Manipulation Under GA	8	16%
Total	50	100%

The mean change of pulse rate among the patients is shown in table V. The mean initial baseline pulse rate was 96±10 beat/min. while following the first dose of scoline during induction was 90±10 beat/min. and after the second dose it was significantly dropped to 65±13 beat/min.

Table V: Pulse rate changes in patients included in this study.

Pulse Rate	Initial		Following induction		After 2nd	
	No.	%	No.	%	No.	%
Mean±SD	96±10		90±10		65±13	
Bradycardia <60	0	0	0	0	18	36
Normal 60-100	34	68	45	90	32	64
Tachycardia >100	16	32	5	10	0	0
Total	50	100	50	100	50	100

*P=0.001(P<0.05) statistically significant

The mean rate of reduction of the pulse rate following the second dose of scoline was significant (P=0.03) in 5 patients (10%), also there was a major drop of pulse rate 20-40 beats/min. in 21 patients(42%) as shown in table VI.

Table VI: Rate of reduction in the pulse rate after the second dose.

Rate of reduction Beats/min.	no. of cases	Percent.
<20	24	48%
20-40	21	42%
>40	5	10%*
Total	50	100%

* P=0.03(P<0.05) statistically significant

Discussion

All the fifty patients included in this study were subjected to short surgical procedure under general anesthesia.

Forty two patients underwent anal surgery (84%) and eight patients had orthopedic surgery (16%). Since these procedures are short to the use of long acting muscle relaxants, the use of scoline in frequent doses seems to be sufficient to cover such procedures. In this study we use two doses of scoline, the first one at the induction and the others after 10 minutes in order to provide optimal surgical relaxation.

The data reported in this study indicates that the incidence of succinylcholine-

induced bradycardia in adults and middle age group (20-50 years) belonging to ASA I is unaffected by gender, BMI, or type of surgery.

It has been concluded that after the 1st dose of scoline, there is a reduction in mean pulse rate from 96±10 to 90±10 however it is considered statistically insignificant (p value more than 0.05) but it has been found that 22% of the patients (11 patients) are converted from initially tachycardia to normal range of pulse rate (60-100 beats/min).

After the second dose, the mean pulse rate is reduced from 90±10 (beats/min) to 65±13 (beats/min) which is statistically significant.

Eighteen patients (36%) developed bradycardia (pulse rate less than 60 beats/min), while the remaining 64% become within the normal level.

Ten percent of the cases develop more than 40 beats reduction in pulse rate which is statistically significant, and should be taken seriously.

Fortunately, all the patients who developed bradycardia recover spontaneously within 1 min, although they didnot return to the initial base line pulse rate but they reach to the normal level.

Baraka suggested that propofol-suxamethonium sequence may be trailed by strict bradycardia in patients who had not taken atropine. Bradycardia may be disallowed by premedication with atropine. In disparity to thiopentone, propofol seems to lacks central vagolytic activity and may have a central vagotonic effect which can amplify the muscarinic effects of suxamethonium¹⁵.

Williams results agree with our results, but he used thiopental sodium, methoxyflurane instead of propofol and halothane, he concluded that bradycardia became more severe when scoline was given immediately after the 1st dose and less severe when it gave 8 minutes later and the concomitant use of thiopental decrease this effect¹⁶.

Another study was done to review the effects of self-taming on the cardiovascular effects of scoline, especially after repeated succinylcholine administration. The conclusion of this study is that the use of self-taming in healthy young patients did not provide protection against subsequent succinylcholine administration. If this technique is used, cardiac monitoring is necessary and care should be observed if succeeding doses of scoline are to be administered¹⁷.

Conclusion

According to this study, the use of scoline in a healthy young and middle age patients of both gender produce the following effects:-

1-The 1st dose results in decrease in pulse rate but no bradycardia.

2-The 2nd dose produces significant reduction in pulse rate that reach to the level of bradycardia.

3-Fortunatly this bradycardia is a self limiting phenomenon and all the patients return to normal level of pulse rate within 1 minute spontaneously, therefore there is no need to use atropine neither preoperatively nor after developing of bradycardia.

It is good that all patients should have monitoring with pulse oximeter and ECG monitor before the start of general anesthesia and throughout the procedures, also a vagolytic (anti-cholinergic) agents such as atropine should be available in the theater for immediate use.

References:

- 1-Appiah-Ankam J, Hunter JM; Pharmacology of neuromuscular blocking drugs, Br J Anaesth, Critical Care and Pain 2004;4;2-7
- 2-Bertram and Katzung; Basic & clinical pharmacology, Tenth Edition, McGraw-Hill companies, 2007; ch 27: p424.
- 3-Lee C. Structure, conformation and action of neuromuscular blocking drugs, Br J Anesthesia, 2001; 87:755-769.
- 4-Morgan GE, Mikhail MS, Murray MJ; Clinical Anesthesiology, 4th edition, McGraw-Hill companies, 2006;ch 9, p223.
- 5-Savarese JJ ; Pharmacology of muscle relaxants and their antagonists. In: Miller RD (editor): Anesthesia, 5th ed. Churchill Livingstone, 2000.
- 6-Farman J. V.:Circulatory Effects of Atropine during Halothane Anesthesia, BJ. Anaesth. (March) 1997; 39:226-235.
- 7-Mathias JA, Evans-Prosser C, Churchill-Davidson HC, The role of non-depolarizing Drugs in the prevention of Suxamethonium bradycardia, Br J Anesthesia 1970, 42; 609-613.
- 8-Takahashi K, Kaya K, Satoh M; A mechanism of Succinylcholine in induced arrhythmias, Masui,1986;14:942-947.
- 9-Isamu Yasuda, Toshio Hirano, Keisuke Amaha, Hiroto Fudenta, Obara S. Chronotropic Effects of Succinylcholine and Succinylmonocholine on the Sinoatrial Node: Anesthesiology 10 1982, Vol.57, 289-292. doi
- 10-Barreto RS. Effects of Intravenously administered succinylcholine upon cardiac rate and rhythm. Anesthesiology 1990; 21: 401-4.
- 11-Perez HR. Cardiac arrhythmia after succinylcholine. Anesth. & Analg. 1980; 49: 33-7.
- 12-Nigrovic V; Succinylcholine, cholinoreceptors and catecholamines proposed mechanism of early adverse hemodynamic reactions, Can Anaesth Soc J, 1984; 31: 382-94.
- 13-Leiman BC, Katz J, Butler BD; Mechanisms of succinylcholine-induced arrhythmias in hypoxia or hypoxic: hypercarbic dogs, Anesth & Analg. 1987; 66: 1292-7.
- 14-William Evelas, Gabob Racz, and Allen B. Dobkin; A study of plasma potassium and electrocardiographic changes after a single dose of succinylcholine, Can. Anaes. Soc. J, July 1969; vol. 16, no. 4.
- 15-Baraka A.; Severe bradycardia following propofol-suxamethonium sequence. Br J Anaesth. 1988 Oct;61(4):482-3.
- 16-Williams, Deutsch, Linda, Bullough & Dripps, Effects of intravenously Administered Scoline on Cardiac Rhythm and Arterial Pressure in Anesthesia, Anesthesiology 22;947 (1969).
- 17-Magee D.A., Sweet P.T. and Holland A.J.C.; Cardiac effects of Self-Taming of Succinylcholine and repeated Succinylcholine administration, Can. Anaesth. Diac, 1987.