THE EFFECTS OF SUXAMETHONIUM AND PANCURONIUM ON THE HAEMODYNAMIC RESPONSE TO ENDOTRACHEAL INTUBATION

Pages with reference to book, From 51 To 54

Fauzia A. Khan, Rehana S. Kamal (Department of Anaesthesiology, The Aga Khan University Hospital and Medical college, Karachi.)

ABSTRACT

The pharmacological effects of suxamethonium and pancuronium on the cardiovascular system may vary and therefore alter the haemodynamic response to intubation. The arterial blood pressure, the heart rate and the rate pressure product were measured as parameters of haemodynamic change in forty adult ASA. I and II patients undergoing laryngoscopy and endotracheal intubation in a randomised controlled study. The patients were induced with either thiopentone/suxamethonium (Group A) or thiopentone/pancuronium (Group B). There was no significant difference between the groups on comparison of systolic and diastolic blood pressure changes. Pancuronium, however, caused a significantly higher rise in the heart rate after endotracheal intubation compared to suxamethonium. In both groups the maximum rate pressure product occurred one minute after intubation, rising by 56% in the suxamethonium group and 64% in the pancuronium group compared to control values. In conclusion, there were significant and statistically similar increases in systolic and diastolic blood pressures and rate pressure product following intubation in both groups with values significantly above baseline until three minutes post intubation but the increase in heart rate in group A was significantly less than that in group B (JPMA 41: 51, 1991).

INTRODUCTION

In normotensive adults the usual response to laryngoscopy and endotracheal intubation is hypertension and tachycardia which has been well documented in several studies[1-3]. This response was shown to be sympathetically mediated in anaesthetized cats and was due to the stimulation of epipharynx and laryngopharynx[4]. More recent studies have pointed out that in normotensive patients there is a moderate increase in the plasma catecholamine levels following laryngoscopy with little or no contribution from intubation[5]. Mostofthese studies have been done using thiopentone/suxame-thonium induction. It is a generally held view that compared to a rapid sequence induction with thiopentone and suxamethonium, thiopentone and pancuronium results in a greaser degree of haemodynamic stability. The pharmacological properties of a muscle relaxant may seem to be of less significance than the stimulus of laryngotracheal stimulation, but may be contributory to the overall haemodynamic response and this may be of importance in patients who have underlying ischemic heart disease where the increase in myocardial oxygen demand may lead to myocardial ischemia. The objective of our study was therefore to focus on the choice of a muscle relaxant for intubation and to find out whether using thiopentone/pancuronium induction had any advantage or disadvantage over thiopentone/suxamethonium induction as far as the haemodynamic response was concerned. We used the changes in the systolic arterial pressure, the diastolic arterial pressure, heart rate and the rate pressure product as our parameters of haemodynamic change.

METHODS

Forty adult ASA I and II patients of either sex aged between 18-60 years undergoing elective surgery requiring endotracheal intubation were allocated randomly to two groups A and B of twenty each. Patients with a previous history of hypertension were excluded. It was not possible to use a blind technique due to the differences in the onset tilne of the neuromuscular blockade between the two groups. The patients were premedicated with oral diazepam 0.15 mg/kg given two hours before surgery. On arrival in the operating room an 18-gauge I/V cannula was placed on the contralateral arm. EGG (CM5 lead) was displayed continuously and oxygen saturation was monitored throughout the procedure using the finger probe of Ohmeda Biox 3700 Pulse Oximeter. The control readings of systolic, diastolic and mean blood pressure and heart rate were taken after a stabilization period of five minutes. All the patients were preoxygenated for three minutes. Group A patients were then anaesthetized with thiopentone 4 mg/kg over 15 second followed by suxamethonium 1.5 mg/kg over 15 seconds. The patients lungs were ventilated with 33% oxygen and 66% nitrous oxide with a face mask and a Magill's circuit. The Capnograph (Data) sampling catheter was placed under the mask to monitor the end expired carbon dioxide tensions which were kept at 5-5.5%. Intubation was performed exactly 90 seconds after the suxamethonium injection using a standard Mackintosh blade laryngoscope. Male patients were intubated with a 9.0 mm and female patients with an 8.0 mm cuffed Magill's red rubber tube. Group B patients received thiopentone 4mg/kg over 15 seconds followed by pancuronium 0.1 mg/kg over 15 seconds. They were ventilated according to the same protocol as group A patients and were intubated three minutes after the pancuronium injection. All intubatipns were accomplished within 30 seconds by the same anaesthetist. Subsequently, both the groups were ventilated undisturbed for a further 7 minutes maintaining an end tidal carbon-dioxide concentration at 5-5.5%. The group A patients were given pancuronium after the action of suxamethonium had worn off and this time was noted. Monitoring of heart rate and blood pressure was continued and recorded every minute after intubation and the rate pressure product calculated. Statistical analysis was by student's t test and a value of less than 0.05 was considered significant.

RESULTS

There was no significant difference between the groups in respect of age, weight or the distribution of males and females, and the control values of systolic arterial pressure, diastolic arterial pressure, heart rate and the rate pressure product (Table I).

TABLE I. Preoperative Date (Mean ± ISD)

	GROUP A	GROUP B
Number of patients (n)	20	20
Age (years)	39.60±9.36	39.20±10.43
Weight (kg)	60.38±10.78	59.55±12.11
Control Value		
Systolic Arterial Pressure (mm of Hg)	136.7±19.52	132.55±17.62
Control Value		
Diastolic Arterial Pressure (mm of Hg)	81.7±12.22	81.15±9.38
Control Value		
Heart rate (Beats/min)	85.55±16.22	82.95±15.09
Control Value		
Rate Pressure Product	11,572±3,063	11,028±2,623

TABLE II: Changes in the haemodynamic parameters in both groups. Following induction of anaesthesia and tracheal intubation

	Control	After thio-Pentone/ Relaxant Injection	1 Minute Post-Intubation	2 Minutes Post Intubation	3 Minutes Post Intubation
Systolic Blood Pressure					
Group A	136.7± 19.52	130.1± 20.34	166.1± 23.79*	166.7± 22.37*	158.26± 23.06*
Group B	132.5± 17.62	127.5± 21.12	158.7± 22.83*	158.8± 18.02*	150.5± 20.31*
Diastolic Blood Pressure					
Group A	81.7± 12.22	90.1± 13.96	114.7± 14.64*	107.8± 15.04*	97.15± 12.94*
Group B	81.1± 9.38	83.3± 15.36	111.4± 16.07*	103.3± 17.57*	95.5± 17.10*
Heart Rate					
Group A	85.5± 16.26	100.8± 12.76	108.3± 16.44*	100.6± 15.81+	99.15± 15.25+
Group B	82.95± 15.09	99.5± 13.15	115.15± 15.41*	112.3± 16.50*	112.4± 15.78*
Rate Pressure Product					
Group A	11,572± 3,063	13,205± 2,459	18,117± 4,530	17,144± 4,138	15,778± 3,529
Group B	11,028± 2,623	12,632± 2,549	18,227± 3,907	17,706± 2,828	16,802± 2,596

*Statistically significant difference on analysis within the group.
+Statistically significant difference on analysis between the group.

Table II gives the changes in the haemodynamic parameters in the two groups during induction of anaesthesia and upto three minutes following intubation. Systolic Arterial Pressure: The changes in systolic arterial pressure in the two groups are shown in Table II. The systolic arterial pressure in both groups rose significantly above the control values at one minute after intubation and remained significantly elevated until three minutes after intubation. No statistical difference was observed between the groups. Diastolic Arterial Pressure: The changes in the diastolic arterial pressure mirrored the changes in the systolic pressure. The maximum rise occurred at 1 minute post-mtubation. Heart Rate: In both groups the heart rate increased significantly after thiopentone/relaxant injection compared

to the control. The increase was 17.6% in group A and 20.7% in group B. The heart rate in both groups rose further after laryngoscopy and intubation, and remained significantly elevated compared to control until 3 minutes post-intubation (Table II). On comparison between the two groups the different was not statistically significant after thiopentone/relaxant injection one minute after intubation but became significant at 2 minutes and 3 minutes after intubation when the heart rate in group A patients (thiopentone /suxamethonium) remained significantly lower than group B. None of the patients in group A showed bradycardia i.e., heart rate less than 60 beats per minute at any time. Rate Pressure Product: The maximum increase in the rate pressure product compared to the control values was found at one minute after intubation in both groups. In group A (Suxamethonium group) the rate pressure product rose to a mean of 18,117 compared to a control value of 11,572, this rise being statistically significant. In group B (pancuronium group) the rate pressure product rose to 18,277 compared to the control value of 11,028. This rise was again statistically significant. The magnitude of rise from the control was more in the pancuronium group (mean rise of + 7, 199) compared to the suxamethonium group (mean rise of +6, 545). This difference between the two groups was not statistically significant. (Table II). The values of rate pressure product gradually decreased over the next three minutes but remained significantly elevated compared to the control. The absolute values in group A were clinically lower than those of group B. None of patients in group A showed a rise in the rate pressure product compared to the previous readings after being given pancuronium injection.

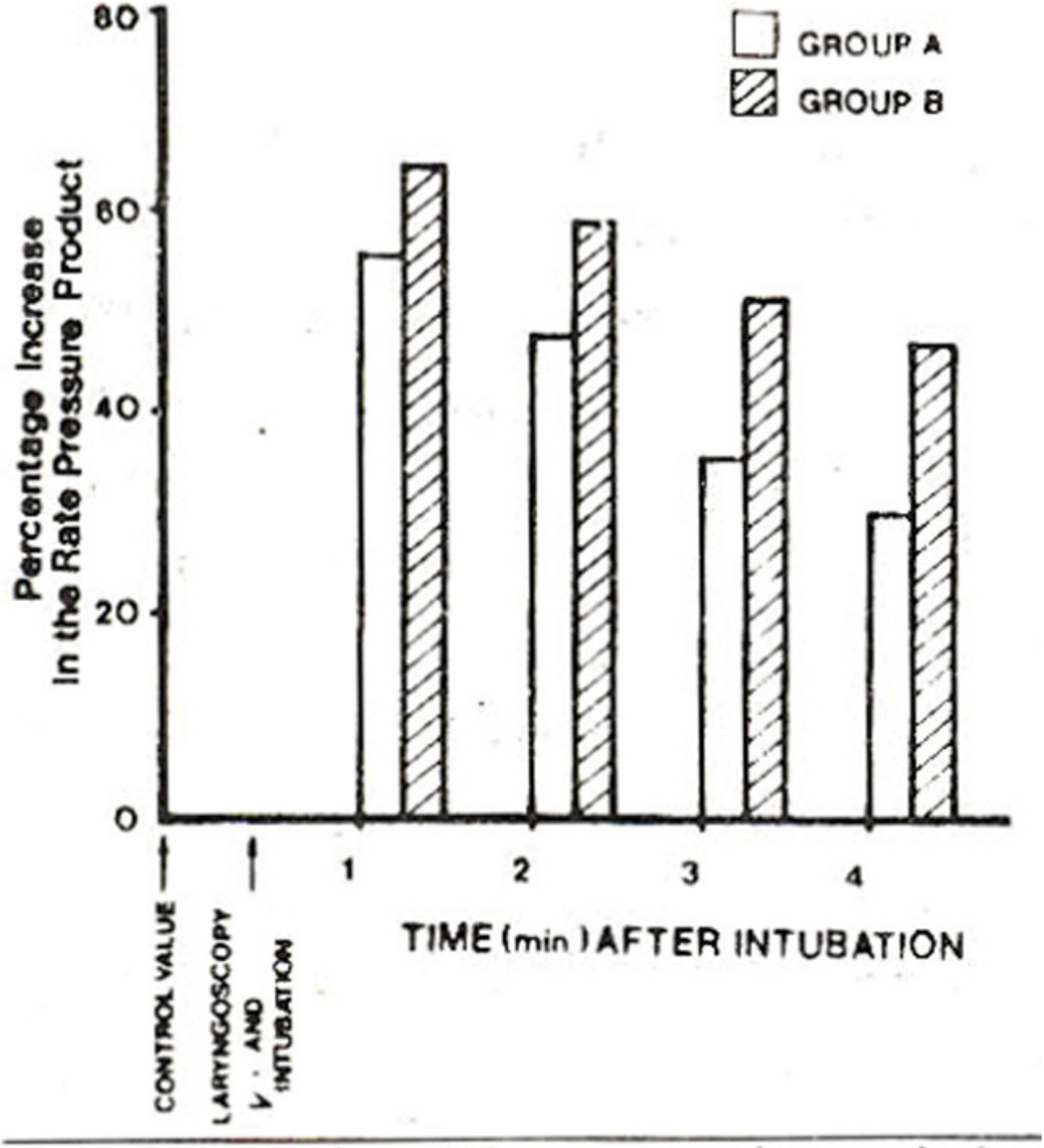

Figure 1. Comparison of the percentage increase in the rate pressure product in the two group compared to the control values. Group A thiopentone/suxamethonium induction ☐ group B thiopentone/pancuronium induction ▨

Figure 1 shows the percentage increase in the rate pressure product compared to the control values in

both the groups at 1,2,3 and 4 minutes after intubation.

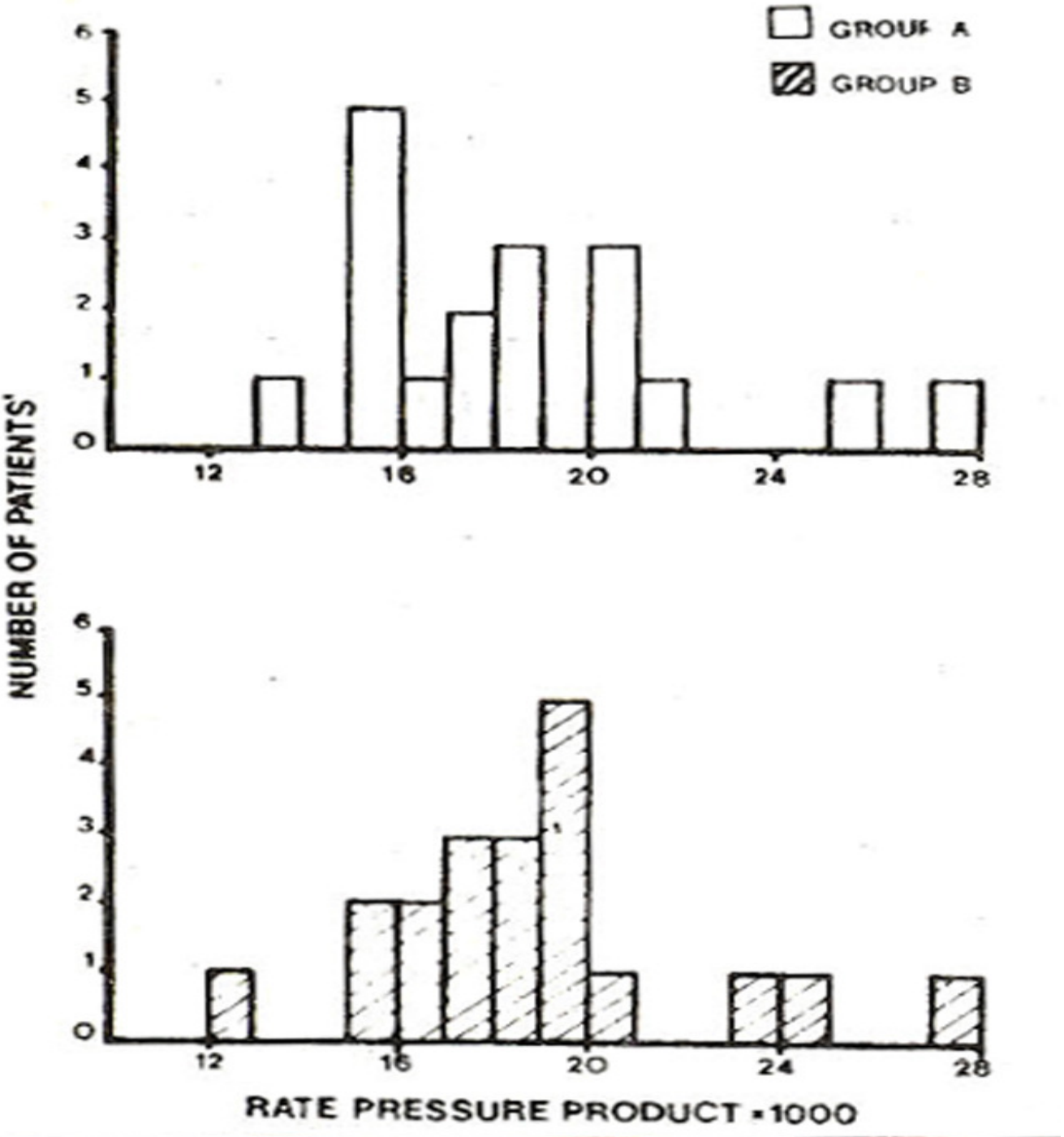

Figure 2. Comparison of the maximum rate pressure product after intubation. Group A thiopentone/suxamethonium induction ☐ group B thiopentone/pancuronium ▨ induction.

Figure 2 compares the maximum rate pressure product reached in different patients in the two groups (Figure 2).

DISCUSSION

The pharmacological properties of induction agents and other drugs used during routine general anaesthesia have been shown to alter the cardiovascular responses to intubation. A bolus dose of thiopentone decreases the arterial pressure, stroke volume and cardiac output. It also causes a decrease in the peripheral venous tone[6]. Its effect on the heart rate is variable, a rise or a no chasnge have both been reorted in different studies[6,7]. It has also been shown that catecholamine response to intubation differs with muscle relaxants[8,11]. Both suxamethonium and pancuronium are widely used to provide muscle relaxation for endotraccheal intubation especially in developing countries where other newer alternatives for muscle relaxation may not be available. The pharmacological effects of the two drugs on the cardiovascular system varies. Suxamethonium may causes bradycardia and a fall in blood pressure due to vagal stimulation[12]. Pancuronium bromide on the other hand causes a moderate rise in the pulse rate and arterial blood pressure of 10-20% and an increasein cardiac output. These changes donot occur in atropinized individuals therefore, indicating that the mechanism is due to the vagolytic effect of the drug[13]. The effect of intubation may also be potentiated by pancuronium due to the enhancement of circulating catecholamines by inhibition of noradrenaline uptake[12]. These pharmacological effects of the durgs though known but their combination of effect with thiopentone bolus and the differences in their effect on the haemodynamic response has not been quantified before in a clinical trial. The blood pressure changes in both groups were similar and did not show any statistical difference on comparison. The only statistical difference observed between the two groups was their effect on the heart rate which remained significantly lower in the thiopentone /suxame-thonium group at 2 and 3 minutes after intubation. None of the patients in either group had bradycardia. This probably reflects that the sympathetic stimulation associated with laryngoscopy masked the vagotonic effect of suxamethonium but pharmacological effect of pancuronium could have been additive. This also accounted for a higher rate pressure product in group B. The rise in the heart rate compared to control that was observed in both groups after thiopentone/relaxant injection, has been reported after thiopentone in other studies[7] and is probably compensatory. The rate pressure product was measured because it is said to reflect the myocardial oxygen consumption[13] and is a guide to the development of myocardial ischemia[14]. The level of rate pressure product where myocardial ischemia occurs varies in different patients but the levels in excess of 22,000 are said to be more commonly associated with angina and myocardial ischemia in patient with underlying ischemic heart disease[15,16]. In our study the rate pressure product rose in both groups following laryngoscopy and intubation with a maximum increase at one minute after intubation. In group A the maximum rise was to 18,117 (control 11,572). This rise was statistically significant and is comparable to figures quoted by Davies[17] with the same technique. It gradually decreased over the next three minutes but did not fall to the baseline. In the pancuronium group (Group B) the maximum rise was again observed at one minute post-intubation, with a rate pressure product of 18,277 (control 11,082) which was statistically significant. This was comparable to the values obtained with thiopentone and pancuronium by Boralessa[18]. The value again decreased over the next three minutes but did not come back to the baseline. Three patients in each group had a rate pressure product above 22,000. None of these patients showed any ST segment changes on the ECG monitor, this was because the patients chosen for study were ASA I and II with no underlying cardiac problems. Further readings of all haemodynamic parameters were taken but were not included in the post intubation response because most of the patients required a nondepolarising muscle relaxant in the suxamethonium group. It has been pointed out by some authors that a rise in the heart rate is more detrimental than the rise in blood pressure. This rise in the heart rate leads to increased myocardial oxygen supply, where as rise in systolic arterial pressure although increases the oxygen demand but also increases the mean and diastolic blood pressure thereby increasing the

perfusion pressure[19]. Keeping this in mind the thiopentone/suxamethonium combination for induction may be a better choice than thiopentone /pancuronium induction as far as the stress on the myocardium is concerned. Another factor which may account for a difference between the results in two groups is the variable interval from the injection of thiopentone to intubation due to the difference in the time of onset of muscle relaxation of the two drugs. Intubation in group A was performed 90 seconds after thiopentone/suxamethomum injections, and in group B 180 seconds after thiopentone/pancuronium injection. This could have resulted in a slightly lighter level of anaesthesia in group B patients and could contribute to the overall haemodynamic response. In conclusion both the techniques caused a significant rise in the systolic and diastolic arterial pressures, heart rate and rate pressure product following intubation. There was no statistical difference between the two groups except in the heart rate which was significantly higher in the pancuronium group and accounted for a higher rate pressure product in that group. Therefore as far as the haemodynamic response is concerned a thiopentone/suxamethonium combination may be preferable to thiopentone/pancuronium.

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