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STUDY OF THE EFFECTIVENESS OF INTRAVENOUS LIGNOCAINE IN OBTUNDING THE CIRCULATORY RESPONSE TO INTUBATION

F. E. Amadasun
Department of Anaesthesiology, University of Benin Teaching Hospital, Benin City, Nigeria.

ABSTRACT

The effectiveness of intravenous lignocaine in mitigating the acute rise in haemodynamic variables on endotracheal intubation was evaluated.

Eighty (80) patients were sequentially assigned to two groups, L and C. Group L patients had 1.5 $mgkg^{-1}$ i.v. lignocaine, 3 minutes before laryngoscopy, and 4 $mgkg^{-1}$ thiopentone for induction of anaesthesia. Group C (control) group had 4$mgkg^{-1}$ thiopentone for induction. Tracheal intubation was facilitated in both groups with 1.5$mgkg^{-1}$ suxamethonium chloride.

Intravenous lignocaine significantly attenuated the postintubation rise in heart rate and rate-pressure product, without significant effect on systolic arterial pressure (SAP), diastolic arterial pressure (DAP) and mean arterial pressure (MAP).

The study thus showed that intravenous lignocaine modestly attenuates the haemodynamic response to intubation and that not all the haemodynamic variables are affected.

KEYWORDS: ***Intravenous Lignocaine, Circulatory Response, Intubation.***

INTRODUCTION

Intravenous lignocaine is widely used to mitigate the acute rise in blood pressure and heart rate associated with laryngoscopy and intubation during anaesthesia and critical care practice[1-4]. In spite of its popularity with anaesthestists in this respect, not all believe in its efficacy[5-8]. This study was designed to test the hypothesis of its efficacy in our local population, especially as there is a dearth of materials on the subject in the local literature.

MATERIAL AND METHODS

This study spanned six months and was carried out at the University of Benin Teaching Hospital, Benin City, Nigeria. It was prospective, controlled, sequential and involved adult male and female patients for elective surgical procedures. The approval of the Ethics Committee of the hospital and informed patient consent were obtained.

Eighty (80) patients with physical status ASA 1 or 2 between the ages of 18 and 67 years were included in the study. There were 36 males and 44 females in the study. Patients with intercurrent diseases, those taking drugs with cardiovascular effects and those for emergency or day care surgery were excluded from the study. All the patients were seen in the ward the day before surgery for preoperative assessment. There, each patient's heart rate (HR) was determined by palpation of the radial pulse, the systolic and diastolic arterial pressures (SAP, DAP) were measured with sphygmomanometer and strethoscope, using the Riva Rocci method, and the 4th Korotkoff sound was used to determine the DAP. The mean arterial pressure (MAP) and the rate-pressure product (RPP) were derived mathematically. Anaesthetic premedication consisted of 10mg oral diazepam given one hour before induction of anesthesia.

In the operating theatre, the patients were assigned sequentially to one of two groups L and C, i.v. lignocaine and control groups. Each patient was weighed and monitors were attached including non-invasive electronic blood pressure monitor (Siemens Sirecust 610 model) and a pulse oximeter (Ohmeda Biox 355e model). The baseline (preinduction) heart rate, systolic, diastolic and mean arterial pressures were determined by the electronic blood pressure monitor, and the rate-pressure product was calculated and recorded.

Each patient was preoxygenated for five minutes. Group L patients were given 1.5$mgkg^{-1}$ lignocaine by slow intravenous injection, three minutes before laryngoscopy and intubation, i,e. during preoxygenation. Anaesthesia was induced with 4$mgkg^{-1}$ thiopentone by slow i.v. injection. Followed with 1.5$mgkg^{-1}$ suxamethonium chloride. Group C patients (control), were given 4$mgkg^{-1}$ thiopentone by slow i.v. injection followed with 1.5$mgkg^{-1}$ suxamethonium chloride. After adequate relaxation as judged by depressed lower jaw tone, laryngoscopy was undertaken in both groups with a long blade Magill laryngoscope, and tracheal intubation with the appropriate size of polyvinyl chloride (PVC) endotracheal tube.

One minute after intubation, the heart rate, systolic, diastolic and mean arterial pressures were recorded from the electronic oscillometer and the rate pressure product was calculated and recorded. Cases of difficult and/or repeated laryngoscopy and/or intubation that took over 30 seconds were excluded from the study. Intubation time was taken to be the time from oral passage of the laryngoscope to successful placement of the endotracheal tube. The difference in the values of the preinduction and postintubation haemodynamic variables in each group, represented the circulatory response to laryngoscopy and intubation.

*Correspondence: Dr. F. E. Amadasun

The results were subjected to statistical analysis and rational deductions derived. Student's t test was used to test statistical significance, and the null hypothesis was rejected at $P < 0.05$.

RESULTS

All the patients in both groups L and C were comparable in number, age, sex, weight and intubation times (Table 1). Table 2 shows the results of the mean cardiovascular values and the values after intubation for both groups. The percentage change of the haemodynamic variables after intubation is shown in table 3.

Compared to the control group, i.v. lignocaine significantly attenuated the postintubation rise in HR ($P<0.01$) and RPP ($P<0.05$). The postintubation rise in SAP, DAP and MAP were not significantly affected.

Table 1: **Patient Characteristics**

Group	No of Patients	Sex (M/F)	Mean age (yr.) ± S.D.	Mean Wt (kg.) ± S. D	Mean intubation time (sec)
L	40	20/20	38.6 (± 13.3)	62.9 (± 18.1)	27.8
C	40	16/24	41.4(± 7.4)	64.7 (± 6.3)	29.5

Table 2: **Mean Cardiovascular Values (±SD, P>0.05, N.S. = Not Significant)**

Haemodynamic Variable	Group L (i.v. Lingnocaine)	Group C (Control)
HR (bpm)		
A	80.6(± 10.5)	80.6((± 7.9)
B	83.1((± 18.1)	91.2((± 17.0)
C	101.7((±14.1)	122.1((± 13.0)
D	18.6((± 12.2)	30.9((± 19.3)
	P<0.01	
SAP (mmHg)		
A	133.0((±22.4)	144.6((±13.8)
B	132.5((±21.6)	124.6((±12.1)
C	169.1((±24.4)	162.1((±35.1)
D	36.6((±17.0)	37.5(±25.4)
	N.S	
DAP (mmHg)		
A	79.0(±8.8)	75.5(±8.4)
B	85.9(±18.6)	84.6((±12.2)
C	128.0(±22.1)	126.6(±26.6)
D	42.1(±18.1)	42.5(±24.4)
	N.S	
MAP(mmHg)		
A	97.3(±11.9)	88.2(±7.5)
B	102.3(±20.2)	98.6(±13.3)
C	139.6(±22.3)	141.6
D	37.3(±10.8)	42.0(±24.4)
	N.S	
RPP(mmHg beat min^{-1})		
A	10,600.1(±1687.3)	9,240.0(±1479)
B	10,981.6(±3951.4)	11,428.1(±2782.2)
C	17,376.7(±3951.4)	19,868.1(±4624.6)
D	6,395.1(±2748.3)	8,440.0(±4306.6)
	P<0.05)	

A = preoperative values in the ward
B = pre-induction values in the operating theatre
C = post-intubation values
D = C-B post-intubation change in haemodynamic values.

Table 3: **Percentage Increase in Haemodynamic Variables after Intubation**

Haemodynamic variable	Group L	Group C
HR	22.4	33.9
SAP	27.7	30.1
DAP	49.0	49.6
MAP	36.5	42.6
RPP	58.2	75.8

DISCUSSION

The use of intravenous lignocaine to attenuate the pressor response to laryngoscopy and intubation accords well with its pharmacological profile. Systemic lignocaine has circulatory depressant effect[9].

In this study, i.v. lignocaine given three minutes before intubation affected the pressor response modestly (HR, RPP), without significant effect on SAP, DAP and MAP Wilson et al[10], using a similar technique got a 21.7% postintubation rise in HR (vs. 22.4% in this study). The issue of the optimal timing of i.v. lignocaine before intubation was investigated by Tam, Chung and Campbell[2]. Complete attenuation of the pressor response was observed by them when the i.v lignocaine was given three minutes before intubation. Splinter et al[3] in a study of 150 geriatric patients, concluded that i.v. lignocaine given 4 to 4.5 minutes before intubation, attenuated the pressor response. They agreed with Tam et al. that in younger adults, the optimal time to administer i.v. lignocaine is three minutes.

Miller and Warren[5] in a study of 45 Chinese women randomly allocated to a control group or one of three treatment groups to receive i.v. lignocaine, 1, 2, and 3 minutes before laryngoscopy could not demonstrate any significant difference between the groups. The time-specific frame of i.v. lignocaine in obtunding the pressor response may be related to its phamacokinetics. On intravenous injection, onset of action is 45 – 90 seconds, and peak affect is at 1 – 2 minutes[9]. The drug thus have a short life span. Perhaps its optimum antipressor effect on intubation is exerted at about its peak effect. The mechanism of its postintubation antipressor effect is believed to be via the depression of laryngeal reflexes[11]. It also decreases the MAC values of volatile anaesthetic agents by 10-28%[12] and reduces thiopentone requirements by 13.3%[13]. It is therefore, possible that part of its antipressor effect may be the deepening of anaesthesia when the anaesthetic agents are used with lignocaine.

Patients in the study would have benefited from continuous ecg monitoring, but this was not available. Considering the fact that vasoactive substances were being used, the ecg would have been useful in picking abnormal rhythms during the procedure. Fortunately, there was no serious cardiovascular incident during the study.

CONCLUSION

This study has shown the attenuative effect of i.v. Lignocaine on the postintubation rise of some haemodynamic variables (HR, RPP) and a seeming lack of effect on SAP, DAP and MAP. Its use in obtunding the pressor response to intubation will therefore depend on the desired haemodynamic effect. Most probably, where a complete or profound attenuation is desired, more effective agents and/or techniques may be more appropriate.

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