CARDIOVASCULAR REACTIONS TO LARYNGOSCOPY AND TRACHEAL INTUBATION FOLLOWING SMALL AND LARGE INTRAVENOUS DOSES OF LIDOCAINE

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IN A PREVIOUS PAPER[1] we described a technique for aerosol anaesthesia of the upper airway prior to induction of anaesthesia and documented the effectiveness of this procedure in preventing cardiovascular reactions to laryngoscopy and tracheal intubation. We expressed the view that the effect of the lidocaine aerosol, especially as it relates to the suppression of arrhythmias, might be due in part to systemic absorption of the local anaesthetic.

Since our first paper controversial views have been expressed on the effect intravenous lidocaine might have on the hypertensive response to laryngoscopy and tracheal intubation.[2]

The purpose of this study was to elucidate these problems.

METHODS AND MATERIALS

Thirty male patients scheduled for elective operations were used in the study. Ages varied between 28 and 85 years. Patients were examined on the day preceding the operation and their informed consent was obtained. A standard premedication of meperidine 1 mg/kg body weight with atropine 0.4 mg was given intramuscularly about one hour before operation. Patients were divided into three comparable groups A, B and C (Tables I and II).

Group A patients served as control and received normal saline intravenously.

Group B patients received 1 per cent lidocaine 0.75 mg/kg.

Group C patients received 2 per cent lidocaine 1.50 mg/kg.

The amount of solution per kilogram was the same in all groups. The contents of the syringes were unknown to the investigator.

Patients were laid supine on the operating table. An intravenous infusion of 5 per cent dextrose in water was set up in a hand vein. A second venopuncture was done in the cubital vein of the other arm for blood sampling. A General Electric surgical monitor was used to display and record the electrocardiogram. Arterial blood pressure was measured using a Roche arteriosonde 1216 automatic blood pressure monitor.

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TABLE I

DETAILS OF THE THIRTY MALE PATIENTS INCLUDED IN THE STUDY

Group	Age	Wt/kg	Operation	Complicating diseases
A-1	78	47	Hemicolectomy	Coronary insufficiency, hypertension
2	49	56	Closure of colostomy	Chronic hepatitis
3	65	69	Urethroplasty	Chronic obstructive lung disease
4	85	78	Hemicolectomy	—
5	69	73	Symes amputation	Hypertension, PVCs, chronic renal failure, diabetes mellitus
6	81	53	Pyelolithotomy	Coronary insufficiency, chron.obstr. lung disease, chron.hepatitis, diabetes mellitus
7	74	70	Sigmoid resection	Coronary insufficiency, anemia
8	59	65	Mediastinoscopy	—
9	60	44	Cholecystectomy	—
10	61	73	Excision iliac aneurysm	Coronary insufficiency, remote myocardial infarct, bifascicular block
B-1	84	66	Meniscectomy	Hypertension, 1st degree A.V. block
2	78	75	Total knee prosthesis	Restrictive lung disease
3	79	51	Hemicolectomy	Coronary insufficiency, PACs
4	79	54	Prostatectomy	Coronary insufficiency, PACs and PVCs, chron.obstr. lung disease, diabetes mellitus
5	28	79	Tympanoplasty	—
6	75	64	Hip pinning	Pulmonary emphysema
7	75	59	Sigmoid resection	Coronary insufficiency, remote myocardial infarct
8	66	66	Palmar fasciectomy	Hypertension, incomplete right bundle branch block
9	51	45	Removal pin (hip)	Coronary insufficiency, remote myocardial infarct
10	75	75	Anterior resection	—
C-1	69	65	Mastectomy	Hypertension, diabetes mellitus
2	72	70	Cholecystectomy	Coronary insufficiency, chronic obstructive lung disease
3	41	55	Gastrectomy	—
4	73	51	Gastrectomy	Chronic obstructive lung disease
5	83	62	Amputation leg	Coronary insufficiency, chronic obstructive lung disease, diabetes mellitus
6	79	55	Amputation leg	Coronary insufficiency, PVCs, diabetes mellitus
7	72	58	Total hip prosthesis	Coronary insufficiency
8	46	78	Meniscectomy	—
9	84	70	Parotidectomy	Hypertension, atrial bigeminy
10	52	74	Cholecystectomy	—

TABLE II

COMPARISON BETWEEN GROUPS OF PATIENTS

	Group A	Group B	Group C
Average age (years)	68.1	69.0	67.1
Average weight (kg)	62.8	63.4	63.8
Complicating diseases	Per cent	Per cent	Per cent
Cardiovascular	50	60	60
Arrhythmias	10	20	20
Respiratory	20	30	30
Renal	10	nil	nil
Hepatic	10	nil	nil
Diabetes mellitus	20	10	30
No complicating diseases	30	20	30

Prior to induction of anaesthesia the electrocardiogram was recorded for one minute and the arteriosonde set to measure blood pressures every minute.

Induction of Anaesthesia

Thiopentone sodium 4 mg/kg was injected intravenously and the patient's lungs were ventilated as required. The appropriate volume of the test solution was now injected intravenously from a coded syringe followed by succinylcholine 1 mg/kg. After fasciculations and ventilation for thirty seconds, the cords were visualized and orotracheal intubation was performed within two to three minutes from the time of injection of the test solution. A bland agent (Lubafax) was used to lubricate the tracheal tubes. Anaesthesia was maintained with nitrous oxide–oxygen 7:3 litres. The lungs were manually ventilated. The electrocardiogram was continually recorded from the time succinylcholine was injected, to one minute after intubation and for two 20-second periods after three and five minutes. The electrocardiogram was also continuously displayed to detect the occurrence of any arrhythmia. A venous blood sample for determination of the lidocaine level was drawn one to two minutes after tracheal intubation. The samples were analyzed by gas chromatography in the ASTRA laboratories, using the method described by Edhorn.[3]

Changes in blood pressure and pulse rate within each group were analyzed statistically using Student's t-test for paired data.

RESULTS

Effects on Blood Pressure and Pulse Rate

Details of mean changes in blood pressure and pulse rate are shown in Tables III and IV. Mean lidocaine venous blood levels are shown in Table V.

Group A

Patients in group A showed significant rises in both systolic and diastolic blood pressure and in pulse rate.

Group B

Patients in group B showed significant rises in diastolic blood pressure only and in pulse rate.

TABLE III

MEAN AND PERCENTAGE CHANGES IN BLOOD PRESSURE

Blood pressure	Group A		Group B		Group C	
	Pre-operative	1-minute post-intubation	Pre-operative	1-minute post-intubation	Pre-operative	1-minute post-intubation
Mean	143.20/79.30	185.00/110.00	146.90/79.90	164.50/100.00	143.30/93.80	173.70/115.60
S.D.	±31.25/±14.96	±35.35/±16.83	±27.16/±10.17	±39.26/±25.16	±32.31/±27.74	±33.80/±26.18
S.E.	±9.82/±4.73	±11.18/±5.32	±8.59/±3.21	±12.41/±7.95	±10.21/±8.77	±10.68/±8.28
Mean percentage rise syst/diast	30.3($P < 0.02$*)/38.7($P < 0.001$*)		11.9($P > 0.2$)/25.2($P < 0.05$*)		21.5($P > 0.05$)/23.2($P > 0.05$)	

*Denotes significant difference to the 0.05 level or better.

TABLE IV

MEAN AND PERCENTAGE CHANGES IN PULSE RATE

Pulse rate	Group A		Group B		Group C	
	Pre-operative	1-minute post-intubation	Pre-operative	1-minute post-intubation	Pre-operative	1-minute post-intubation
Mean	86.80	100.10	80.80	101.90	99.10	107.60
S.D.	±13.88	±10.97	±13.18	±14.02	±24.63	±23.46
S.E.	±4.39	±3.47	±4.17	±4.43	±7.79	±7.42
Mean percentage rise	15.3 ($P < 0.05$*)		26.1 ($P < 0.01$*)		8.5 ($P > 0.4$)	

*Denotes significant difference to the 0.05 level or better.

TABLE V

MEAN LIDOCAINE VENOUS BLOOD LEVELS

	Group B 2–3 minutes following 0.75 mg/kg I.V. bolus	Group C 2–3 minutes following 1.5 mg/kg I.V. bolus
Mean lidocaine blood levels	0.985 μg/ml	1.860 μg/ml

Group C

Changes in blood pressure and pulse rate in group C patients were statistically not significant.

The following electrocardiographic changes were observed at one-minute post-intubation.

Group A

Patient No. 1 showed marked ST depression. Patient No. 4 developed atrial bigeminy. In patient No. 5 infrequent ventricular ectopic beats became frequent and multifocal. Patient No. 6 developed runs of supraventricular ectopic beats.

Group B

Patients No. 1 and No. 2 converted to nodal rhythm. Patient No. 8 developed frequent unifocal ventricular ectopic beats. Patients No. 3 and No. 4 showed no change in their pre-existing arrhythmias.

Group C

No new arrhythmias were noted. In patient No. 6 a pre-existing arrhythmia remained unchanged and in patient No. 9 atrial bigeminy reverted to sinus rhythm soon after the injection of lidocaine.

DISCUSSION

When we started this study our main objective was to examine the assumption made in our previous paper that systemic absorption of lidocaine probably accounted for the absence of arrhythmias after laryngoscopy and tracheal intubation in the group pretreated by a lidocaine aerosol. In view of the well-established anti-arrhythmic effect of lidocaine we were not surprised by the absence of arrhythmias after the 1.5 mg/kg dose but would have expected some protection against ventricular arrhythmias even after the smaller dose. Hayes[4] states that after an intravenous bolus even a blood level of only 0.7 to 1.1 μg/ml is effective since in well perfused organs like the heart the concentration of lidocaine is much higher. It seems, however, that after laryngoscopy and tracheal intubation without topical anaesthesia a blood level above 1.2 μg/ml is required to prevent arrhythmias. In group B one patient had nodal arrhythmias with a plasma level of 1.0 μg/ml, another patient had premature ventricular contractions with 1.2 μg/ml. In group C eight patients had levels of 1.6 μg/ml or more. A suitable sample was not obtained in one patient and another patient had a level of only 0.8 μg/ml.

The correspondence[2] that followed the paper of Denlinger et al.[5] and the results obtained by these authors after intravenous administration of lidocaine 1.5 mg/kg[6] suggested that intravenous lidocaine might not only prevent post-intubation arrhythmias but to a large extent also the hypertension usually observed after tracheal

intubation. Our results did indeed show a borderline statistical protection and the larger dose seemed to prevent tachycardia as well. An explanation of these observations is perhaps possible on the basis of our present knowledge regarding the effect of lidocaine on synaptic transmission[7] and/or on the heart muscle itself.[7–13]

R.H. de Jong[7] states that experimental results suggest that small doses of procaine have a restraining effect on polysynaptic inter-neuronal systems. Other local anaesthetics show similar results. Lidocaine 10 mg/kg inhibits polysynaptic and monosynaptic spinal reflexes in strychninized cats and the linguomandibular reflex is suppressed profoundly by 5 mg/kg of lidocaine. He also states that the experimentally observed depressant effects of local anaesthetics on synaptic transmission can be demonstrated clinically as a suppression of reflexes. He mentions, for example, that intravenous lidocaine is a potent suppressant of the cough reflex in lightly anaesthetized patients.[14]

In this context we should like to mention that Bromage and Robson[15] have observed that systemic absorption of lidocaine obtunds laryngeal reflexes.

The direct cardiac depressant action of lidocaine and its vasodilating effect on the peripheral circulation were studied by many authors.[7–11] McWhirter, *et al.*[12,13] reported two different actions of intravenous lidocaine on the heart: A direct depressant action and an indirect dose dependent stimulant effect. This is caused by inhibition of inhibitory mechanisms at the level of the limbic or higher cerebral centres resulting in an indirect stimulant effect which depends on the integrity of the autonomic nervous system. It can be inhibited by ganglioplegics or vagotomy[7,12,13] and also by central nervous system depressants such as general anaesthetics.[7,12] Therefore, following induction of anaesthesia one would expect the depressant effect of intravenous lidocaine to predominate.

In group B patients sympathetic stimulation caused by laryngoscopy and tracheal intubation resulted in a statistically significant rise in diastolic blood pressure and pulse rate but not the systolic blood pressure (Tables III and IV). This could perhaps be explained by the fact that this small dose of lidocaine (0.75 mg/kg) was just enough to cause myocardial depression but at the same time failed to provoke any indirect cardiac stimulation or peripheral vasodilatation. On the other hand, group C patients (1.5 mg/kg) showed borderline statistical protection against hypertension and tachycardia following tracheal intubation. Here myocardial depression was probably partially reversed by the indirect stimulant effect of lidocaine and apparently not coupled with an increased peripheral resistance. However, the protection against hypertension and tachycardia following tracheal intubation was not as convincing after intravenous lidocaine as it was after inhalation of lidocaine aerosol.[1]

Preinduction aerosol topical analgesia of the upper airways would still be our method of choice to minimize post-intubation cardiovascular reactions in patients with poor myocardial reserve or severe hypertension.[1] The greatly reduced sympathetic stimulation following this technique, with the resulting better protection against hypertension and tachycardia, in spite of a lower lidocaine blood level, are all desirable in patients with latent myocardial insufficiency.

If time or circumstances do not permit topical aerosol anaesthesia, intravenous lidocaine 1.5 mg/kg appears to be a good alternative.

SUMMARY

The efficacy of intravenously administered lidocaine 0.75 mg/kg and 1.5 mg/kg to protect against cardiovascular reactions associated with laryngoscopy and tracheal intubation was studied in two comparable groups of ten patients and compared with a similar control group of ten patients given only saline. Following laryngoscopy and tracheal intubation, the 1.5 mg/kg dose afforded complete protection against cardiac arrhythmias of all types. The smaller dose was ineffectual in this respect. While the larger dose caused borderline protection against hypertension and tachycardia, the smaller dose prevented only the rise in systolic blood pressure. Possible mechanisms to account for these observations are discussed. These include a direct myocardial depressant effect, a central stimulant effect, a peripheral vasodilating effect and finally an effect on synaptic transmission.

RÉSUMÉ

Les effets d'une dose i.v. de 0.75 mg/kg de Lidocaïne et ceux d'une dose de 1.5 mg/kg ont été étudiés chez deux groupes comparables de dix patients chacun, et ont été comparés à ceux observés dans un troisième groupe de dix patients ayant reçu une solution saline.

Lorsqu'elle est administrée avant la laryngoscopie et l'intubation trachéale, la Lidocaïne à la dose de 1.5 mg/kg protège complètement contre tous les types d'arythmies. Une dose de 0.75 mg/kg est inefficace.

La Lidocaïne à la dose de 1.5 mg/kg assure de plus une protection contre l'hypertension et la tachycardie, alors qu'à 0.75 mg/kg elle prévient seulement l'élévation de la tension systolique. Les causes possibles de cette action peuvent être une dépression directe du myocarde, ou une vasodilatation périphérique, ou un effet stimulant central, ou, finalement, un effet sur la transmission synaptique.

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