

Effects of Intratracheal Lidocaine on Circulatory Responses to Tracheal Intubation

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Hypertension and tachycardia following laryngoscopy and tracheal intubation during various forms of light general anesthesia are well documented.¹⁻⁴ Methods to avoid these potentially harmful responses while preserving the advantages of minimal anesthetic depth in critically ill patients have been sought. Intratracheal administration of lidocaine, 4 per cent, is frequently used in clinical practice; however, there has been no objective evaluation of its effectiveness in blocking the circulatory responses to intubation.⁵ This study in cardiac patients anesthetized with morphine and nitrous oxide provides data establishing the effects of intratracheal lidocaine on circulatory responses to intubation.

METHOD

Twelve consecutive patients scheduled for elective cardiac surgery were randomly divided into two treatment groups: Group I (trachea to be sprayed with saline solution, 3 ml/70 kg) and Group II (trachea to be sprayed with lidocaine, 4 per cent, 3 ml/70 kg). The assignment code was not made known to the investigator until the data from the entire series of 12 patients had been tabulated. The patient groups were comparable in terms of age, sex, weight, physical status, and cardiac lesion. Operative procedures consisted of nine coronary-artery grafts, two valve replacements, and one mitral commissurotomy. One patient (included in the lidocaine group) had a preoperative his-

tory of hypertension. Mean preinduction blood pressures were $131.7 \text{ torr} \pm 9.3 \text{ SE}$, $71.3 \text{ torr} \pm 3.1 \text{ SE}$ in the lidocaine group and $129.2 \text{ torr} \pm 7.4 \text{ SE}$, $68.0 \text{ torr} \pm 5.7 \text{ SE}$ in the saline solution group.

Direct, continuous arterial and central venous pressure monitoring was established prior to the induction of anesthesia. At the time of study, anesthesia was achieved with morphine, iv, (mean dose = $0.996 \text{ mg/kg} \pm 0.120 \text{ SE}$) supplemented by nitrous oxide, 50 per cent, in oxygen. The patients were given *d*-tubocurarine, 3 mg, iv, followed by a continuous infusion of succinylcholine, 0.2 per cent. Complete neuromuscular blockade was maintained throughout the study period. Ventilation was controlled to maintain PaCO_2 between 30 and 40 torr. At 3-minute data collection period (to obtain control values) preceded initial laryngoscopy (MacIntosh #3 blade) and tracheal spray. Continuous written records of blood pressure and electrocardiogram were obtained. During the subsequent 5-minute-period, continuous data recordings were obtained while ventilation was controlled via face mask. Laryngoscopy was then repeated and the trachea was intubated with a sterile, disposable cuffed tube (I.D. 8 or 9 mm). The prestretched cuff was inflated with the minimal amount of air necessary to prevent gas escape during controlled ventilation. A second 5-minute data collection period preceded any change in patient position or surgical stimulation.

Systolic blood pressure, diastolic blood pressure, and heart rate were averaged over 10-second segments of the record, taken at precisely 1-minute intervals. The entire record (paper speed = 25 mm/sec) was scanned for arrhythmias. Statistical analyses were calculated by Student's *t*-test for paired data.

No drug other than the continuous succinylcholine infusion and nitrous oxide was

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§ LTA disposable cannula, Abbott Laboratories.

¶ Electronics for Medicine IR-4 photographic oscilloscope.

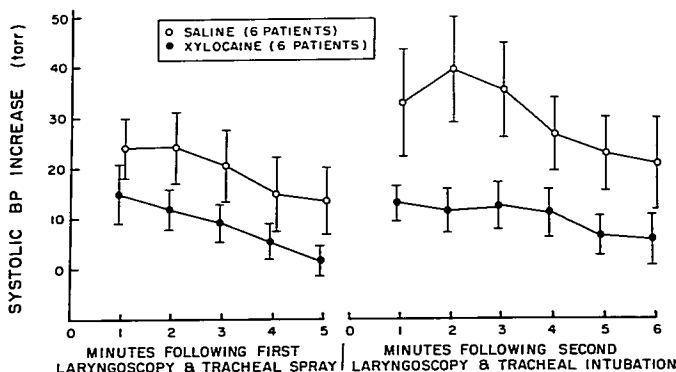


FIG. 1. Systolic blood pressure increase vs. time.

given during the 14 minutes required for the study, with the single exception of one patient in the saline solution group who needed intravenous injection of chlorpromazine for hypertension caused by tracheal intubation.

RESULTS

Control arterial blood pressures were $108.2 \text{ torr} \pm 4.7 \text{ SE}$ / $60.6 \text{ torr} \pm 2.7 \text{ SE}$ in the lidocaine group and $108.6 \text{ torr} \pm 4.9 \text{ SE}$ / $54.4 \text{ torr} \pm 2.5 \text{ SE}$ in the saline solution group.

Significant increases in systolic blood pressure ($P < 0.05$) followed both laryngoscopy plus tracheal spray and laryngoscopy plus tracheal intubation (fig. 1). Patients whose tracheas were sprayed with saline solution had significantly higher mean peak increases in systolic blood pressure (39 torr) following intubation than did the group whose tracheas were sprayed with lidocaine (13 torr). Five of six patients in the saline-solution group had peak increases in systolic blood pressure of at least 30 torr, and one of these patients had an increase of 78 torr. No patients in the lidocaine group had a peak increase in systolic blood pressure that exceeded 20 torr. At each time interval, systolic blood pressure in the saline solution group was higher than systolic blood pressure in the lidocaine

group. Although less profound, there were similar differences between diastolic blood pressures and heart rates in the two groups following tracheal intubation (figs. 2 and 3). Postintubation arrhythmias did not occur in either treatment group.

DISCUSSION

Tracheal intubation in the absence of coughing, hypoxia, or hypercarbia may cause hypertension and tachycardia by reflex sympathetic discharge.² That hypertension during induction of anesthesia in critically ill patients may be harmful is substantiated by reports of cerebral hemorrhage, left ventricular failure, and life-threatening cardiac arrhythmias.^{4,6,7,8} Hypertension may be particularly hazardous in patients with coronary artery disease, such as those studied in the present series. Just as sympathetic stimulation from various forms of stress may provoke angina in the conscious patient, it is possible that reflex sympathetic stimulation in the anesthetized patient may produce myocardial ischemia by increasing myocardial work.⁴ In the present group of six normotensive patients anesthetized with morphine-nitrous oxide who were not treated with intratracheal lidocaine, the mean increase in systolic blood pressure was 39 torr. This magnitude of

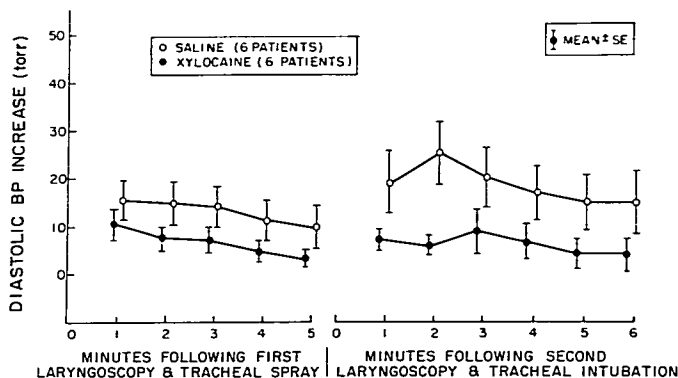


FIG. 2. Diastolic blood pressure increase vs. time.

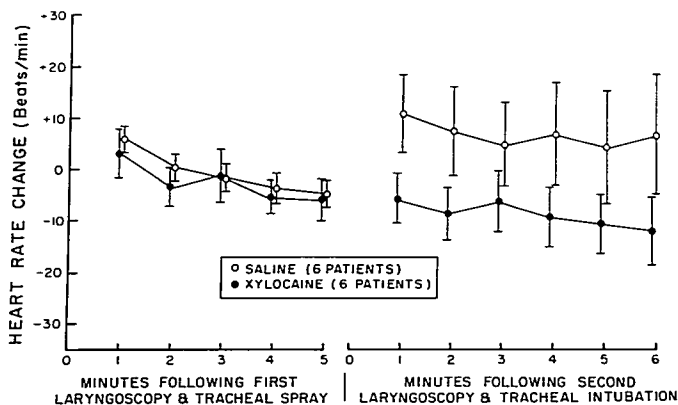


FIG. 3. Heart rate change vs. time.

response is somewhat greater than the 25-torr increase in mean arterial blood pressure previously reported to follow intubation in normotensive patients during thiopental-nitrous oxide anesthesia.⁴

Hypertension and tachycardia following laryngoscopy and tracheal intubation have been reduced by deep anesthesia^{1,2} and

blocked by phentolamine.⁹ Translaryngeal topical anesthesia has been shown to block the hypertensive responses in five of seven patients anesthetized with thiopental and halothane.¹⁰ However, in that study marked increases in blood pressure were noted following the translaryngeal injection itself. Wycoff, in 1960, reported a decrease in the

hypertensive response when a combination of translaryngeal block, superior laryngeal nerve block, and laryngotracheal spray was employed in patients during barbiturate anesthesia.³ King *et al.*¹¹ demonstrated that topical anesthesia (cocaine or tetracaine applied to the base of the tongue, pharynx, epiglottis and trachea with an atomized spray or cotton swab plus tracheal cannula) does not prevent the hypertensive response to tracheal intubation in awake patients. Thus, the response to tracheal intubation is not abolished by a combination of topical and general anesthesia. However, the present study indicates that this hypertensive response in patients anesthetized with morphine and nitrous oxide can be *significantly decreased* by a simple intratracheal spray with lidocaine, 4 per cent.

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