Nicardipine versus lidocaine for attenuating the cardiovascular response to endotracheal intubation

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Abstract

Purpose. The aim of this study was to compare the efficacy of nicardipine and lidocaine in attenuation of cardiovascular responses to endotracheal intubation.

Methods. In a randomized, double-blind, controlled trial, 60 unpremedicated (ASA I) patients undergoing elective surgery were given either 30μg·kg⁻¹ nicardipine or 1.5mg·kg⁻¹ lidocaine intravenously 2 min before intubation. Laryngoscopy and tracheal intubation were performed 1 min after induction of anesthesia with 5mg·kg⁻¹ thiopentone, followed by administration of 1.5mg·kg⁻¹ succinylcholine intravenously. Blood pressure and heart rate were monitored at baseline and every minute until 4 min after intubation. Repeated-measures ANOVA, Student’s t test, the chi-square test, and 95% confidence intervals were used as appropriate. P < 0.05 was considered statistically significant.

Results. Baseline hemodynamic variables were not different between the groups. After administration of either agents, diastolic blood pressure and mean blood pressure were significantly lower in the nicardipine group. The heart rate in the nicardipine group was significantly higher. The mean between-group differences in diastolic blood pressure, mean blood pressure, heart rate, and rate-pressure product at baseline and 1 min after starting laryngoscopy were statistically significant.

Conclusion. Nicardipine can be used as an alternative to lidocaine in attenuation of cardiovascular response to tracheal intubation in patients without ischemic heart disease.

Key words: Nicardipine, Lidocaine, Cardiovascular response, Intubation

Introduction

Laryngoscopy and tracheal intubation after a standardized induction dose of thiopentone often provokes a reflex increase in both sympathetic and sympathetic-adrenal activity, which may result in hypertension, tachycardia, and arrhythmias [1–6]. These responses, although transient, may be harmful in some patients, particularly those suffering from myocardial or cerebrovascular disease. Many pharmacological techniques, including the use of opioids, local anesthetics, adrenergic blocking agents, and vasodilating agents such as nitroglycerine and sodium nitroprusside, have been devised to reduce the extent of the hemodynamic events [1–3,7–10]. Lidocaine hydrochloride, an aminoethylamide local anesthetic and class IB antidysrhythmic agent, is an acceptable agent for attenuation of the cardiovascular response to intubation and will also diminish cough reflexes, dysrhythmias, and rises in intracranial and intraocular pressure [10]. Kyokong et al. showed that 1.5mg·kg⁻¹ lidocaine administered intravenously 2 min before intubation could attenuate the cardiovascular response to laryngoscopy and intubation [11]. Nicardipine is a new dihydropyridine derivative that acts as a calcium channel blocker. Nicardipine produces an immediate, short-acting, reliable reduction in blood pressure without adverse effects such as hypotension, disturbances of atrioventricular conduction, and myocardial depression [12,13]. Moreover, it produced consistent augmentation of coronary blood flow, oxygen delivery, and aerobic metabolism in ischemic myocardium [14]. Omote showed that 20 or 30μg·kg⁻¹ nicardipine was effective in preventing the circulatory responses to laryngoscopy and tracheal intubation in normotensive and hypertensive patients [15]. However there was no separate study comparing the effects of intravenous lidocaine and nicardipine. This study aimed to compare the efficacy of single rapid administration of lidocaine and nicardipine for controlling these hemody-
namic responses to tracheal intubation under the same anesthetic technique.

Methods

The study was approved by the Ethical Committee of the Faculty of Medicine, Chulalongkorn University. We studied 60 normotensive patients (ASA I) undergoing elective surgery, all of whom had given informed consent. The patients were randomly allocated into two groups (n = 30 for each group) to receive nicardipine 30 μg·kg⁻¹ or lidocaine 1.5 mg·kg⁻¹ intravenously. Patients who were suspected to have difficult tracheal intubation or who had hypertension (systolic blood pressure > 160 mmHg and/or diastolic blood pressure > 95 mmHg as defined by the World Health Organization), renal, hepatic, or gastrointestinal disease were excluded.

No premedication was given. Upon arrival in the operating theater, the patients were monitored by noninvasive blood pressure monitoring (MDE ESCORT, Medical Data Electronics, Arleta, CA, USA) and standard lead II ECG. The baseline blood pressure and heart rate (T₀) were recorded after a resting period of 5 min, and preoxygenation was subsequently given. One minute after baseline blood pressure was recorded (T₁), either nicardipine or lidocaine was administered intravenously in a double-blind fashion. After another minute, anesthesia was induced by intravenous thiopentone 5 mg·kg⁻¹ followed by succinylcholine 1.5 mg·kg⁻¹. Direct laryngoscopy and tracheal intubation was initiated 1 min after administration of thiopentone. Blood pressure (systolic blood pressure, diastolic blood pressure, and mean blood pressure) and heart rate were recorded every 1 min by other independent anesthesiologist until 4 min after intubation (T₈). The study protocol is shown in Fig. 1.

Each intubation was performed by an MD anesthesiologist and was accomplished within 20 s. No patients received topical lidocaine before placement of the tracheal tube. After tracheal intubation, ventilation was controlled with 66% nitrous oxide in oxygen and 0.5% halothane. The study was then completed 4 min after intubation (T₈). The study protocol is shown in Fig. 1.

Results

The demographic characteristics of the patients were comparable in both groups (Table 1). The blood pressure (systolic blood pressure, mean blood pressure, and diastolic blood pressure), heart rate, and rate-pressure product are given as means (SD) (Table 2). The baseline values of systolic blood pressure, mean blood pressure, diastolic blood pressure, heart rate, and rate-pressure product were comparable in both groups.

After the administration of either drugs diastolic blood pressure, mean blood pressure, and heart rate were significantly different between the nicardipine and lidocaine groups (P < 0.01, P = 0.03, and P < 0.001, respectively). Systolic blood pressure and rate-pressure product between groups were not significantly different.

The maximal changes in blood pressure and heart rate occurred 1 min after laryngoscopy was started. The mean differences in diastolic blood pressure, mean blood pressure, heart rate, and rate-pressure product at baseline (T₀) and 1 min after laryngoscopy was started (T₁) were significantly different between groups; [P = 0.01, 95% CI (−17.311, −1.667); P = 0.01, 95% CI (−17.306, −2.494); P < 0.001, 95% CI (13.494, 33.039); and P = 0.01, 95% CI (440.5, 4437.6), respectively, as shown in Fig. 2.

There was one case of occasional premature ventricular contraction (PVC) during laryngoscopy and intubation in the lidocaine group, which spontaneously disappeared within few minutes. Other adverse effects,