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Effects of double administration of nicardipine of the cardiovascular response to tracheal intubation in hypertensive patients

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Abstract: The efficacy of intravenous nicardipine in attenuating the cardiovascular responses to laryngoscopy and tracheal intubation was studied in 20 hypertensive patients. Ten patients received intravenous 1 mg of nicardipine 1 min before induction (N1 group). The other ten received 1 mg of nicardipine 1 min before induction and an additional 1 mg just before laryngoscopy (N2 group). In the N1 group, arterial pressure and heart rate increased significantly after tracheal intubation. In the N2 group, arterial pressure did not increase but heart rate increased more than that in N1 group. There was no significant difference in rate pressure product between the two groups.

Key words: Nicardipine, Tracheal intubation, Hypertension, Arterial pressure, Heart rate

Introduction

Laryngoscopy and tracheal intubation cause significant changes in the hemodynamic state, including hypertension, tachycardia, and arrhythmia [1,2]. Especially in the hypertensive patient, the extent of these cardiovascular changes is much more marked than in the normotensive patient. These changes may be dangerous if the patient has myocardial disease [3].

Small doses of fentanyl, local anesthetics, α- and β-adrenoceptor antagonists (blockers), prostaglandin E_1, and calcium channel antagonists, such as nifedipine, nicardipine, and diltiazem, are effective in controlling the hemodynamic responses to laryngoscopy and intubation in normotensive patients [4–14]. However, there are *few* reports concerning nicardipine given to hypertensive patients during induction of anesthesia.

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The purpose of the present study was to evaluate the efficacy of nicardipine in attenuating the cardiovascular responses to laryngoscopy and tracheal intubation in hypertensive patients, and to compare a single injection with two injections of that drug.

Materials and methods

Twenty patients who had been treated with antihypertensive drugs and whose systolic blood pressure was above 160 mmHg or diastolic blood pressure was above 90 mmHg upon arrival in the operating room were included in the study. All patients consented to participate in this study. They were randomly assigned to a single-administration group (N1 group) and double-administration group (N2 group).

Premedication consisted of atropine, 0.01 $mg \cdot kg^{-1}$, and midazolam, 1–2 mg, intramuscularly 30 min before induction. On arrival of the patient in the operating room, lead II of the electrocardiogram (ECG) was placed. Arterial pressure (AP) was monitored by an automatic oscillometric blood pressure monitor (Dynamap, Critikon, Tampa, Florida). After baseline measurements, all patients received a single rapid intravenous injection of nicardipine 1 mg. One min after the administration of nicardipine, thiamylal 5 $mg \cdot kg^{-1}$ and succinylcholine 1.2 $mg \cdot kg^{-1}$ were given intravenously and laryngoscopy was started 1 min later. In the N2 group, an additional 1-mg dose of nicardipine was administered just before laryngoscopy. Tracheal intubation was completed within 20 s. After tracheal intubation, anesthesia was maintained with either sevoflurane or isoflurane and 66% nitrous oxide in oxygen.

AP and heart rate (HR) were recorded at baseline (T0), 1 min after nicardipine (T1), before laryngoscopy (T2), immediately after intubation (T3), and at 1 min (T4), 5 min (T5), and 10 min (T6) after intubation.

All values were expressed as mean ± SD. Statistical

analysis was performed using analysis of variance with repeated measures and paired *t*-test. Non-paired *t*-test was also used for intergroup comparison. A *P* value of less than 0.05 was considered significant.

Results

There were no significant differences between the groups in age, weight, or height (Table 1). Baseline values in systolic, mean, diastolic AP, and HR were also comparable in both groups.

APs were significantly lower after injection of nicardipine (T1), before intubation (T2), and 1 min (T4), 5 min (T5), and 10 min (T6) after intubation compared with the initial values in both groups. In response to laryngoscopy and intubation (T3), AP in the N1 group returned to the baseline value, but AP in the N2 group was significantly lower. APs were less in the N2 group compared with the N1 group immediately after (T3) and at 10 min (T6) after intubation (Fig. 1).

HR in the N2 group increased before intubation (T2), just after intubation (T3), and 1 min after intubation (T4), but HR in the N1 group increased only just after intubation (T3). HR in the N2 group was significantly higher than that in the N1 group just after intubation (T3) (Fig. 2).

The rate pressure product (RPP) decreased after injection of nicardipine (T1), before intubation (T2), and at 5 min (T5) and 10 min (T6) after intubation in both groups. However, there was no significant difference in RPP between the groups (Fig. 3).

No abnormalities in ECG were observed in any patients. No patients required any pressor drugs for treatment of excess hypotension.

Discussion

The hypertensive response to laryngoscopy and tracheal intubation may be enhanced and may prove dangerous to hypertensive patients [3]. For example, tachycardia and hypertension resulting from laryngoscopy and tracheal intubation may lead to myocardial ischemia and prolonged regional myocardial dysfunction in patients with reduced coronary vascular reserve [15,16]. Therefore, in those patients, prevention of hypertension following tracheal intubation by using some form of pretreatment should be considered.

Table 1. Patient characteristics of the two groups

	Sex (F/M)	Age (years)	Weight (kg)	Height (cm)
N1 group (n = 10)	8/2	56.2 ± 7.6	55.5 ± 5.5	154.7 ± 6.0
N2 group (n = 10)	7/3	62.1 ± 10.7	56.5 ± 7.4	151.1 ± 11.4

Values expressed as mean ± SD.

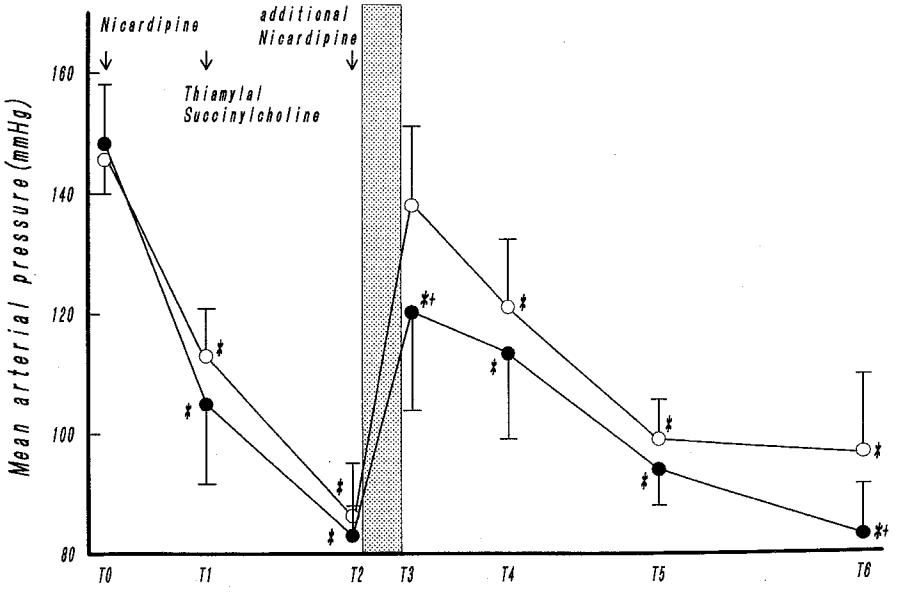

Fig. 1. Mean arterial pressure during induction of anesthesia and tracheal intubation. *T0*, baseline; *T1*, 1 min after nicardipine; *T2*, before laryngoscopy; *T3*, immediately after intubation; *T4*, 1 min; *T5*, 5 min; *T6*, 10 min after intubation. *Open circles*, N1 group; *solid circles*, N2 group. *Arrowhead*, injection of drugs; *dotted area*, duration of laryngoscopy and tracheal intubation. $^{*}P < 0.05$ comparing each stage to baseline value within the same group $^{+}P < 0.05$ comparing with two groups in each stage

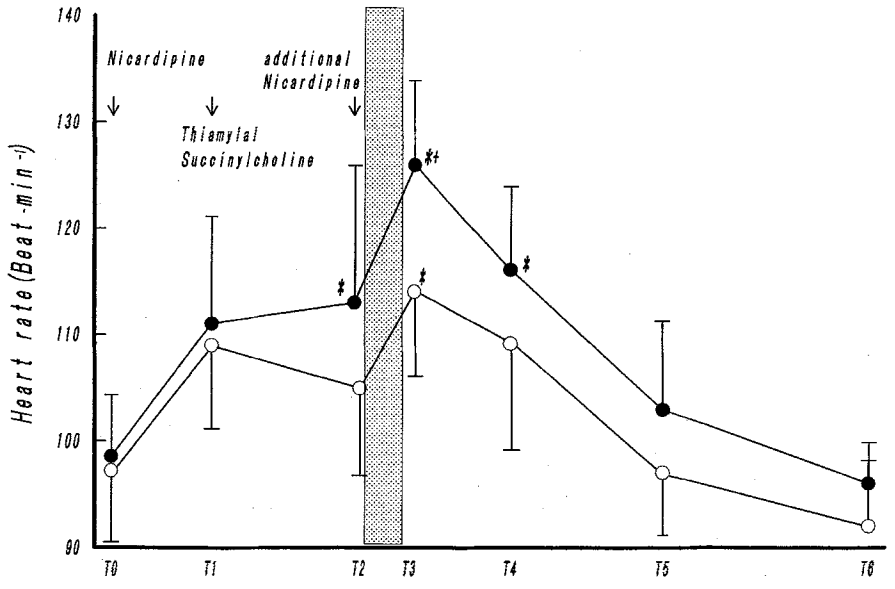

Fig. 2. Heart rate during induction of anesthesia and tracheal intubation. *T0*, baseline; *T1*, 1 min after nicardipine; *T2*, before laryngoscopy; *T3*, immediately after intubation; *T4*, 1 min; *T5* 5 min; *T6*, 10 min after intubation. *Open circles*, N1 group; *solid circles*, N2 group. *Arrowhead*, injection of drugs; *dotted area*, duration of laryngoscopy and tracheal intubation. $*P < 0.05$ comparing each stage to baseline value within the same group. $^{+}P < 0.05$ comparing with two groups in each stage

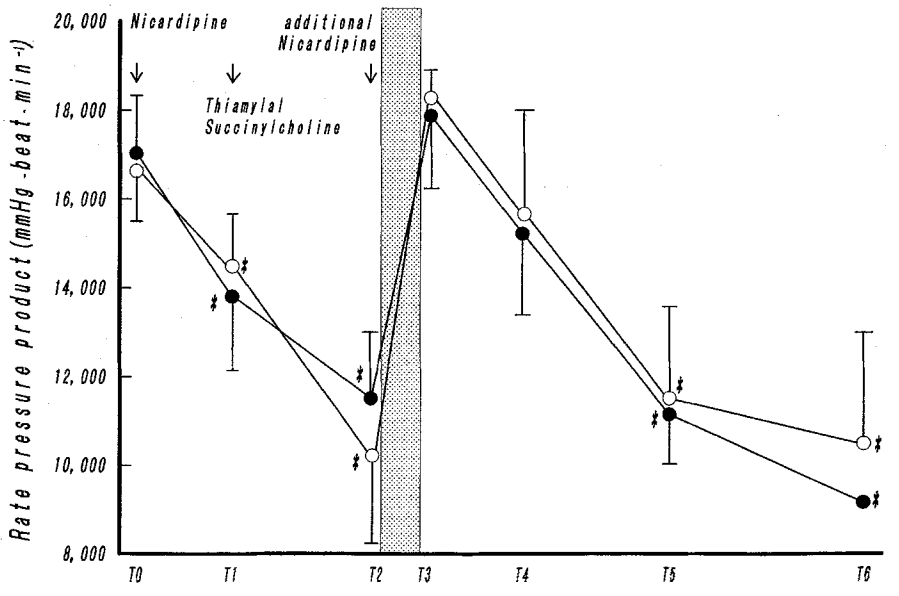

Fig. 3. Rate pressure product during induction of anesthesia and tracheal intubation. *T0*, baseline; *T1*, 1 min after nicardipine; *T2*, before laryngoscopy; *T3*, immediately before intubation; *T4*, 1 min; *T5*, 5 min; *T6*, 10 min after intubation. *Open circles*, N1 group; *solid circles*, N2 group. *Arrowhead*, injection of drugs; *dotted area*, duration of laryngoscopy and tracheal intubation. $*P < 0.05$ comparing each stage to baseline value within the same group

The present data demonstrated that single intravenous nicardipine could not attenuate pressor response since AP increased toward the hypertensive range, but an additional 1-mg dose of nicardipine could suppress the increases of AP which resulted from laryngoscopy and tracheal intubation. The calcium channel antagonist, nicardipine, acts on the cardiovascular system by inhibiting the influx of Ca^{2+}, and has been shown to be effective for stable, variant, and unstable angina and for severe hypertension [17,18].

The onset of hypotension induced by nicardipine is approximately 20–40 s after intravenous administration and the maximal effect is at 2 min after administration. Mean AP (MAP) begins to return toward the baseline values after only 3 min and the complete recovery is after 5–7 min [13,19]. Thus, the administration of nicardipine 1 min prior to laryngoscopy seems to be effective since the peak effect of nicardipine occurs at the moment of intubation. Mikawa et al. [13] have indicated that administration of nicardipine, 15 and 30 $\mu g \cdot kg^{-1}$, 1 min before laryngoscopy attenuated the increases in MAP and RPP following tracheal intubation. In the present study, it was necessary to decrease AP toward the normotensive range by the first administration of nicardipine. Since tracheal intubation was usually started more than 2 min after nicardipine injection, AP suppression may not be sufficient if the peak antihypertensive effect of nicardipine is not timed to coincide with the maximal increase of AP by intubation. Therefore, the additional nicardipine might

be useful for hypertensive patients during tracheal intubation.

However, the additional nicardipine increased HR significantly more than the single administration, although there was no significant difference in RPP between the groups. The increased HR and decreased AP due to double administration of nicardipine may be dangerous for the patient with ischemic heart disease. It is generally believed that patients with ischemic hear disease can tolerate an increase of AP better, and conversely can only poorly tolerate a decrease in AP and tachycardia. Even if the RPP value remains at a safe level during myocardial ischemia, a low RPP associated with low AP becomes dangerous for the patient suffering hypertension and coronary artery disease [20–23].

In conclusion, double administration of nicardipine can prevent pressor responses following laryngoscopy and tracheal intubation in hypertensive patients. However, the usefulness of this technique for patients with ischemic heart diseases should be evaluated with additional studies and clinical experiences in the future.

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