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Original contribution

Effects of nicardipine on the onset time and intubation conditions of rocuronium-induced neuromuscular blockade☆

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Abstract

Study objective: The objective of this study was to identify the effects of nicardipine on neuromuscular blockade of rocuronium, such as the onset time and intubation conditions, using a nicardipine dose that attenuates cardiovascular responses during endotracheal intubation.

Design: Randomized, double-blinded, placebo-controlled clinical comparison was used as the design of this study

Setting: The study was conducted at the operating room of a university hospital.

Patients: Participants of this study comprise 78 American Society of Anesthesiologists physical status 1 and 2 patients, aged 18 to 60 years who were undergoing elective surgery under general anesthesia.

Interventions: The nicardipine group was given an intravenous bolus of 20 μg/kg nicardipine before tracheal intubation: the control group was given an intravenous bolus of a comparable volume of normal saline before tracheal intubation.

Measurements: Using a TOF-Watch SX monitor, the time from the end of the injection of rocuronium to maximum depression of T1 (onset time) was measured. Intubation was performed 1 minute after rocuronium administration, and the status of the intubation conditions was assessed. The mean blood pressure and heart rate were each measured after endotracheal intubation. Rate pressure product values were also calculated.

Main results: Intubation conditions were clinically acceptable in 37 (94.9%) of 39 patients in group N compared with 29 (74.4%) of 39 in group C ($P < .05$). The onset time of rocuronium was significantly faster in group N than in group C ($P < .05$). The mean blood pressure was significantly lower in group N than in group C ($P < .05$). The heart rate was significantly higher in group N than in group C ($P < .05$). Rate pressure product values showed no significant difference between the two groups ($P > .05$).

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Conclusions: Pretreatment with 20 μg/kg nicardipine improves intubation conditions, shortens the onset time of rocuronium, and attenuates cardiovascular responses to tracheal intubation.

1. Introduction

Instrumental manipulation of the pharynx and tracheal intubation may result in tachycardia, hypertension, and increased plasma catecholamine concentrations. These responses, although transient, may be harmful in some patients, particularly those suffering from myocardial or cerebrovascular disease [1,2]. Many pharmacological techniques have been studied to reduce the extent of the hemodynamic events, including the use of high-dose opioids, local anesthetics, adrenergic blocking agents, and vasodilating agents such as nitroglycerin and sodium nitroprusside [3–6]. Nicardipine is a dihydropyridine derivative that acts as a calcium channel blocker. The onset of action of nicardipine is rapid and its duration is fairly short. Nicardipine has been shown to produce dose-dependent decreases in mean arterial pressure associated with reflex tachycardia without major side effects [6–8]. In previous studies, 20 to 30 μg/kg nicardipine has been reported to effectively attenuate transient hypertensive responses to laryngoscopy and tracheal intubation [9–11]. Therefore, nicardipine appears to be a suitable agent for attenuating the circulatory responses to laryngoscopy and tracheal intubation.

Several experimental and clinical studies involving the interactions between calcium-channel blocking agents and neuromuscular blocking agents have been reported. Calcium-channel blockers have been shown to increase the neuromuscular effect of depolarizing or nondepolarizing neuromuscular blockers in isolated preparations or intact animals [12–18]. In addition, potentiation of the neuromuscular blocker effects by calcium-channel blockers has been described in humans [19,20]. However, few investigated the effects of nicardipine on the onset time and intubation conditions of rocuronium-induced neuromuscular blockade using a dose of nicardipine that provides hemodynamic stability during tracheal intubation. We hypothesized that the pretreatment with nicardipine improves intubation conditions, shortens the onset time of rocuronium, and attenuates cardiovascular responses to tracheal intubation during anesthetic induction. For this, we planned a study to identify the effects of nicardipine on the onset time of rocuronium, intubation conditions, and hemodynamic response during endotracheal intubation.

2. Materials and methods

The present study was carried out after obtaining approval from the Institutional Review Board and receiving informed consent from 78 patients (American Society of Anesthesiologists [ASA] physical status 1-2), aged 18 to 60 years, who were undergoing elective surgery under general anesthesia. The patients were allocated into 2 groups of 39 each, where group N (n = 39) was given an intravenous (IV) bolus of 20 μg/kg nicardipine before tracheal intubation and group C was given an IV bolus of a comparable volume of normal saline before tracheal intubation. Patients with a history of cardiovascular instability (such as hypertension, myocardial infarction, or arrhythmias), asthma or obstructive pulmonary disease, obesity (body mass index >30 kg/m^2), neuromuscular disease, kidney disease, liver disease, central nervous system disease, and cerebral infarction, as well as those who were prescribed medication that affect neuromuscular blockade, hypersensitive to the drugs used in the study, parturient, and expected to have or had a history of a difficult airway, were excluded from the study.

All patients underwent 8 hours of nil per os and were premedicated with 2 mg of midazolam and 0.2 mg of glycopyrrolate intramuscularly 30 minutes before surgery. On arrival in the operating room, electrocardiograms, oxygen saturation, noninvasive blood pressure, and endotracheal CO_2 were monitored. All patients were connected to a bispectral index monitor (model A-2000; Aspect Medical Systems, Natick, MA). Induction of anesthesia was performed after sufficient hemodynamic stability was obtained, and 100% oxygen was delivered. Group N was given an IV bolus of 20 μg/kg nicardipine, and group C was given an IV bolus of a comparable volume of normal saline. Normal saline (3 mL) or 20 μg/kg nicardipine (in 3 mL saline) was administered 1 minute before induction in a double-blind fashion. All study drugs (nicardipine, isotonic saline, rocuronium) were prepared by 1 anesthesia nurse, and the attending anesthesiologist was unaware of the group allocations.

Next, 2 mg/kg propofol was given for 1 minute and 15 seconds. After confirming that the patient was unresponsive to a verbal stimulus, and the eyelash responses were absent, calibration of the electrical nerve stimulator was performed, and 0.6 mg/kg rocuronium was administered for 10 seconds. Endotracheal intubation was performed 1 minute after rocuronium administration using a Macintosh laryngoscope and with a polyethylene endotracheal tube. An 8.0- or 7.5-mm-diameter tube was used in male or female patients, respectively. Intubation was performed by same expert anesthesiologist unaware of the injected drugs. Each intubation was accomplished within 20 seconds. Cases in which tracheal intubation was not completed at first attempt were regarded as failed cases and were excluded from the