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Original Article

Does intravenous atropine affect stroke volume variation in man?

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ABSTRACT

Objectives: Currently there are no reports of the effect of increasing heart rate (HR) induced by intravenous atropine on stroke volume variation (SVV). We hypothesized that increasing HR alters the value of SVV. This prospective study aimed to investigate changes in SVV values by increasing HR induced by intravenous atropine in patients with good cardiac function. We also re-evaluated the effect of intravenous atropine alone on hemodynamics including new hemodynamic parameters such as SVV.
Methods: Patients were chosen as participants of this study if, 30 minutes after anesthesia induction, HR was below 65 beats/min. Baseline hemodynamic values were recorded, and then the patients received intravenous atropine (0.01 mg/kg; max 0.5 mg). These values were recorded again after intravenous atropine every minute for 5 minutes.
Results: Ten American Society of Anesthesiologists (ASA) physical status I–II patients aged 37–65 years who were scheduled for elective surgery were included. Intravenous atropine significantly increased HR at the 1–5 minute time points, mean arterial pressure at the 1–4 minute time points, and cardiac output at the 1–3 minute time points compared with baseline values but did not significantly change SVV, stroke volume index, pressure of end-tidal CO_2, and systemic vascular resistance.
Conclusion: Administration of intravenous atropine did not change SVV, and we present this as a novel finding.

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1. Introduction

Standard hemodynamic parameters such as heart rate (HR), arterial pressure (AP), and central venous pressure are poor markers of hypovolemia and cardiac output (CO), and are not reliable in detecting volume responsiveness.[1] Dynamic markers such as SVV (stroke volume variation), PPV (pulse pressure variation), SPV (systolic pressure variation) in the mechanically ventilated, and passive leg raise (PLR) in those breathing spontaneously are superior to static markers such as central venous pressure (CVP) in predicting fluid responsiveness.[2]

SVV can be affected by various factors; we previously reported that the rapid infusion of fluid may significantly influence these different parameters,[3] and other situations such as intravascular volume status,[4] depth of airway pressure and tidal volume,[4–6] and intraabdominal pressure[7–9] can affect SVV. Furthermore, we recently reported that SVV can be affected by induced hypertension and hypotension[10] and by induced hypotensive anesthesia,[10] and also that SVV is affected by landiolol,[11,12] an ultra-short-acting adrenergic β_1 receptor blocking agent.[11]

The addition of a muscarinic anticholinergic drug to anesthetic premedication to decrease secretions and prevent harmful vagal reflexes was mandatory in the era of ether anesthesia.[13] Further, the primary indication for atropine is the treatment of reflex-mediated bradycardia[14] during surgery because atropine increases HR. However, there are no reports, to our knowledge, on the effect of increasing HR induced by intravenous atropine on SVV, and we hypothesized that increasing HR alters the value of SVV, and if so, SVV values might be overestimated, underestimated, or misinterpreted. The aim of this prospective study was to investigate changes in SVV values by increasing HR induced by intravenous atropine in patients with good cardiac function. Furthermore,

Conflicts of interest: The authors declare there are no conflicts of interest.

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because only a few studies have reported the effects of intravenous atropine alone on hemodynamics, we re-evaluated these effects of intravenous atropine alone on hemodynamics including new hemodynamic parameters such as SVV.

2. Methods

2.1. Patients and treatments

Approval for this study was obtained from the Institutional Review Board of the International University of Health and Welfare Hospital, Tochigi, Japan, and written informed consent was obtained from all participants. We registered this study in the "UMIN Clinical Trial Registry" (ID: UMIN000007557). The participants of this study were patients scheduled to undergo elective abdominal surgery. All patients were American Society of Anesthesiologists (ASA) physical status I and II, and none had known diabetes mellitus; hypertension; cardiovascular (including non-sinus rhythm and 2° or 3° A-V block), pulmonary, endocrinologic, neurologic, or autonomic diseases; or diseases that affect intravascular fluid volume or balance, such as gastrointestinal obstructive or inflammatory diseases. All patients underwent preoperative fasting for at least 8 hours, and no premedication was given to any of the patients.

Induction of anesthesia was performed with propofol (initial effect-site concentration = 4 μg/mL) and 1 μg/kg remifentanil intravenously (IV) in total, and rocuronium (0.6 mg/kg) IV. After induction of anesthesia, a 23-gauge catheter was inserted in the left or right radial artery for direct arterial pressure monitoring, and the patients' lungs were mechanically ventilated by means of a semi-closed circle system at a fresh gas flow of 6 L/min (O_2, 2 L/min and air, 4 L/min). Controlled ventilation was set at 10 breaths/min, with a tidal volume of 8 mL/kg and an inspiratory/expiratory ratio of 1:2. Later, the effect-site concentration of propofol was adjusted to achieve a target Bispectal index (BIS) between 40 and 60 and stable circulatory variables (propofol was administered by a plasma target-controlled infusion method).

Systolic arterial pressure (SAP), mean arterial pressure (MAP), diastolic arterial pressure (DAP), HR, pressure of end-tidal CO_2 ($P_{ET}CO_2$), SVV, cardiac output (CO), stroke volume index (SVI), and systemic vascular resistance (SVR) were continuously monitored with a standard monitor (CARESCAPE Monitor B850; GE Healthcare, Helsinki, Finland) and the FloTrac/Vigileo system (software version 03.06; Edwards Lifesciences, Irvine, CA, USA).

If the patient's HR was below 65 beats/min 30 minutes after induction of general anesthesia, the patient was chosen as a participant, and the baseline values of SAP, MAP, DAP, HR, $P_{ET}CO_2$, SVV, CO, SVI, and SVR were recorded. Then, the patients received atropine IV (0.01 mg/kg; maximum 0.5 mg). SVV, CO, SVI, and SVR were recorded 20 seconds after SAP, MAP, DAP, HR, and $P_{ET}CO_2$ were recorded because the Vigileo system samples the pressure waveform at 100 Hertz over 20 seconds, capturing 2000 data points for analysis, and parameter calculations were provided at the end of every 20 second timeframe.[3,15] These values were recorded again after atropine IV every minute for 5 minutes. All of these studies were conducted before the surgery began, and just 100 mL of normal saline was administered to the patients to maintain minimal change in SVV values for general anesthesia induction and during the study.[11]

2.2. Statistical analysis

Sample size was estimated from preliminary data obtained from six patients. An assumption was made that a 0.5 point change in CO between the baseline values and those 2 minutes after the injection of atropine would be clinically relevant. Power analysis suggested that a minimum of eight patients would be needed for $\beta = 0.1$ and $\alpha = 0.05$. To compensate for potential dropouts, we enrolled 10 patients in this study. This analysis was performed using GraphPad StatMate 2.00 (GraphPad Software, Inc., La Jolla, CA, USA).

Values are expressed as the mean ± standard deviation (SD). Comparisons of SAP, MAP, DAP, HR, $P_{ET}CO_2$, SVV, CO, SVI, and SVR were performed with paired *t* tests with Bonferroni's correction to determine whether there were significant differences between the parameters ($p < 0.05$). A p value < 0.05 was required to reject the null hypothesis. All analyses were performed with GraphPad Prism 5.04 (GraphPad Software, Inc.).

3. Results

Patient characteristics are shown in Table 1. Values after intravenous atropine are shown in Table 2. Administration of intravenous atropine significantly increased SAP at 2 minute time points, MAP at 1–4 minute time points, DAP at 1–5 minute time points, HR at 1–5 minute time points, and CO at 1–3 minute time points compared with baseline values (Table 2), but it did not significantly change $P_{ET}CO_2$, SVV, SVI, and SVR (Table 2).

4. Discussion

One of our main results is that intravenous atropine did not change SVV, and this is the first study, to our knowledge, that has measured SVV after intravenous atropine. SVV is defined as:

$$\text{SVV}\ (\%) = 100 \times (\text{SVmax} - \text{SVmin})/[(\text{SVmax} + \text{SVmin})/2], \qquad (1)$$

where SV = stroke volume and maximal and minimal values for SV were determined as SVmax and SVmin, respectively, over a single respiratory cycle of paced breathing.[3,10] Because SV and SVI were unchanged after atropine administration (Table 2), the numerator and denominator in this formula clearly did not change. Therefore, the reason for the invariance in SVV value was the invariance of maximum SV and minimum SV, and this phenomenon can be explained by the fact that the absolute SVV values were unchanged in this study (Table 2).

In the 1960s, Farman[16] investigated the circulatory response to the injection of 0.6 mg atropine during nitrous oxide, oxygen, and halothane anesthesia with spontaneous breathing, and found that SV fell by 17% in man. By contrast, Farman and Kennedy[17] then studied the effect of atropine (0.012 mg/kg) on the circulation of artificially ventilated patients anesthetized with nitrous oxide and tubocurarine and found that there was no significant change in SV. They concluded that the response to atropine depends not only on the effect of the drug on the heart, but also on the control state of the circulation, both of which are heavily influenced by (1) other anesthetic drugs; (2) the arterial $PaCO_2$ (in their study, the patients hyperventilated); and (3) the mechanical effect of artificial ventilation. They also investigated the circulatory responses to intravenous atropine in artificially ventilated patients anesthetized with

Table 1
Demographic data of study group.

Variable	Measured value
Patients (M/F)	10 (6/4)
Age (y)	54.0 ± 14.3 (37–65)
Body weight (kg)	58.9 ± 14.5
Height (cm)	156.7 ± 11.5
Body surface area (m^2)	1.58 ± 0.23

Data are presented as mean ± standard deviation (range).

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