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Original contribution

Pulse oximetry–derived pleth variability index can predict dexmedetomidine-induced changes in blood pressure in spontaneously breathing patients☆,☆☆

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Abstract

Study objective: Hypertension or hypotension in patients receiving continuous infusions of dexmedetomidine (DEX) is often due to changes in vascular resistance caused by α_2 receptor stimulation. We investigated whether baseline perfusion index (PI) and pleth variability index (PVI), derived from pulse oximetry readings, could predict DEX-induced changes in the hemodynamic status in spontaneously breathing patients.

Design: Observational study.

Setting: Operating room.

Patients: Patients (American Society of Anesthesiologists performance status 1 or 2) scheduled to undergo lower extremity or abdominal procedures under regional anesthesia were approached.

Interventions: The PI and PVI were set as baseline upon arrival in theater and were then measured at 2.5-minute intervals. Upon attaining stable hemodynamic status under spontaneous breathing, intravenous administration of DEX was initiated at 6 $\mu g\ kg^{-1}\ h^{-1}$ for 10 minutes, followed by continuous infusion at 0.6 $\mu g\ kg^{-1}\ h^{-1}$.

Measurements: Blood pressure, heart rate, PI, and PVI were measured. Hypertension was defined as an increase in systolic blood pressure (SBP) >15% and hypotension as a decrease in SBP <15% from baseline.

Main results: Baseline PI and PVI correlated with the degree of change in SBP. The maximum percentage increase as well as the maximum percentage of decrease in SBP from baseline correlated with baseline PI ($r = 0.418$ [$P = .005$] and $r = 0.507$ [$P < .001$], respectively) and PVI ($r = -0.658$ [$P < .001$] and

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$r = -0.438$ [$P = .003$], respectively). PVI < 15 identified DEX-induced hypertension (sensitivity 94%, specificity 85%) and PVI > 16 identified DEX-induced hypotension (sensitivity 83%, specificity 64%).
Conclusions: PVI may predict DEX-induced changes in blood pressure in spontaneously breathing patients.
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1. Introduction

Hypertension and hypotension in patients receiving continuous infusions of dexmedetomidine (DEX) are often the consequence of increased or decreased vascular resistance, respectively, due to α_2 receptor stimulation. These conflicting hemodynamic changes may be induced by initial loading doses of DEX at various time points and lead to a critical condition even in nonintubated patients, although an initial loading is needed for rapid onset of anesthesia. If the changes in blood pressure could be predicted, assessment and treatment of hypertension and hypotension caused by DEX would be possible.

A previous study has demonstrated that the condition of patients determines which site (central nervous system or peripheral vessels) is most affected by DEX administration [1]. For example, systolic blood pressure (SBP) can decrease because of the sedative effect of DEX in patients who are in an emergency condition and who are experiencing pain or stress. In contrast, SBP can increase because of a peripheral vasoconstrictive effect in patients who have received sufficient amounts of anesthetics. However, no useful and objective indicators of DEX-induced hemodynamic changes in conscious patients have been found to date.

The perfusion index (PI) and pleth variability index (PVI), derived from pulse oximetry, have been used to evaluate peripheral perfusion dynamics from changes in peripheral vascular tone. The PI is defined as the ratio between constant absorption and pulsatile absorption and reflects the amplitude of the plethysmographic waveform. PVI can automatically detect the maximum and minimal PI value over a sufficient period of time. Evaluation of patients' peripheral vascular status may be helpful to predict DEX-induced hemodynamic changes. Moreover, because the probe for pulse oximetry is attached only to a finger and is therefore noninvasive, it is helpful, particularly in conscious patients where arterial and central venous catheters are typically not used to measure hemodynamic parameters.

This study was aimed to examine whether baseline PI and PVI could be useful to predict DEX-induced hemodynamic changes in spontaneously breathing patients. We aimed to determine optimal cutoff points for PI and PVI to predict DEX-induced hypertension and hypotension.

2. Materials and methods

2.1. Study population

This study was approved by the Institutional Ethics Committee of Asahikawa Medical University, Asahikawa, Japan, and was performed according to World Medical Association Ethical Principles for Medical Research Involving Human Subjects. We investigated patients attending the Asahikawa Medical University Hospital from February 2014 to February 2015. Forty-two patients scheduled to undergo lower extremity or abdominal procedures under regional anesthesia were approached prior to anesthesia; patients provided oral and written informed consent to enter in this study. The exclusion criteria were an American Society of Anesthesiologists (ASA) classification of III or greater, age below 18 years, pregnancy, inability to give informed consent, or the presence of severe arteriosclerosis obliterans, arrhythmia (atrial fibrillation), heart failure (ejection fraction <40%), renal failure (plasma creatinine level >2 mg/dL), and previous use of medications involving α- or β-adrenergic agonists or antagonists.

2.2. Hemodynamic status monitoring

Premedication was not needed. Upon arrival in the operating room, each patient was placed in the supine position and was given an intravenous infusion of bicarbonate or acetate Ringer's solution, at 100 to 200 mL/h. Standard monitoring by means of electrocardiography, automated noninvasive blood pressure (NIBP) monitoring, and pulse oximetry was carried out. The automated NIBP device cuff was attached to the right arm, and SBP and diastolic blood pressure were measured at 2.5-minute intervals. The probe of the pulse oximeter (Masimo Radical-7; Masimo Corp, Tokyo, Japan) was attached to the left fourth finger. Baseline NIBP, heart rate (HR), PI, and PVI were monitored in the supine position. The attending anesthesiologists were blinded to the values of PI and PVI.

When hemodynamic status had stabilized under spontaneous breathing, DEX was administered at 6 $\mu g\ kg^{-1}\ h^{-1}$ for 10 minutes and then modulated to 0.6 $\mu g\ kg^{-1}\ h^{-1}$. At least 20 minutes after the initiation of DEX administration, appropriate regional anesthesia, according to the surgical site (intrathecal anesthesia, lumbar plexus block, femoral nerve block, or sciatic nerve block), was administered by the anesthesiologist.

NIBP, HR, PI, and PVI were recorded for 20 minutes during administration of DEX. Hypertension was defined as an increase in SBP to a value greater than 15% or 20 mm Hg from baseline and hypotension as a decrease in SBP to a value of less than 15% or 20 mm Hg from baseline, as defined in a previous study [2]. When SBP increased more than 30% from baseline, 0.5 mg nicardipine was dispensed intravenously, and when SBP decreased more than 30% from baseline, 5 mg ephedrine was dispensed intravenously as a rescue medication. We dispensed 0.5 mg atropine intravenously if severe

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