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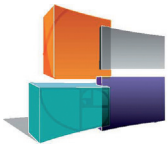

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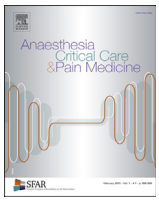

Original Article

Mini-fluid challenge can predict arterial pressure response to volume expansion in spontaneously breathing patients under spinal anaesthesia

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ABSTRACT

Introduction: The objective of this study was to test whether stroke volume (SV) variations in response to a fixed mini-fluid challenge (ΔSV_{100}) measured by impedance cardiography (ICG) could predict an increase in arterial pressure with volume expansion in spontaneously breathing patients under spinal anaesthesia.

Methods: Thirty-four patients, monitored by ICG who required intravenous fluid to expand their circulating volume during surgery under spinal anaesthesia, were studied. Haemodynamic variables and bioimpedance indices (blood pressure, SV, cardiac output [CO]) were measured before and after fluid challenge with 100 mL of crystalloid, and before/after volume expansion. Responders were defined by $\geq$ 15% increase in systolic arterial pressure (SAP) after infusion of 500 mL of crystalloid solution.

Results: SAP increased by $\geq$ 15% in 20 (59%) of the 34 patients. SAP, SV, and CO increased and HR decreased only in responders. SV variations in response to mini-fluid challenge and volume expansion differed between patients who showed arterial responsiveness and those in whom SAP did not increase with volume expansion (11.6% [9.1–19.3] versus 2.5% [1.3–7], $P < 0.001$, and 22.4% [11.7–36.6] versus 0.9 [0–5.5], $P < 0.001$, respectively). ΔSV_{100} predicted an increase of arterial pressure with an area under the receiver operating characteristic (AUC) curve of 0.89 ($CI_{95\%}$: 0.73–0.97, $P < 0.001$). The cut-off was 5%. Baseline SAP and HR were not predictive of arterial responsiveness ($P > 0.05$).

Conclusion: A ΔSV_{100} over 5% accurately predicted arterial pressure response to volume expansion during surgery.

1. Introduction

Spinal anaesthesia (SA) is commonly used for surgery of the lower half of the body. A major clinical haemodynamic effect, associated with SA during surgery, is arterial hypotension (AH), which has been associated with increased mortality and morbidity [1,2]. Intravenous fluid administration is frequently the first-line treatment for AH in the operating theatre, but with inconsistent results. One main explanation could be that AH can be due to the interplay of several pathophysiological mechanisms that may interact differently over time: decreased systemic vascular resistance (SVR), increased venous capacitance, decreased cardiac output (CO), and decreased blood volume [3–5]. These effects can also vary according to the level of SA. According to the primary pathophysiological mechanism involved, AH treatment may include fluid expansion and/or vasopressors. During general anaesthesia, blood pressure and heart rate (HR) have been demonstrated to be unsuitable for assessing cardiovascular status and blood volume [6]. Devices measuring CO and dynamic preload indices have been validated as useful tools for predicting fluid responsiveness and optimizing CO [7,8]. Because dynamic preload indices can predict an increase of CO in response to volume expansion (i.e. fluid responsiveness), they are assimilated with markers of preload dependence (a surrogate for blood volume) [9]. However, dynamic preload indices are only reliable predictors

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in anesthetized and intubated patients under positive mechanical ventilation [9]. Another approach consists of testing preload dependence by a limited fluid challenge. Muller and colleagues demonstrated that an increase in the subaortic velocity time index (a surrogate for stoke volume) during a low-volume infusion could predict a further increase of CO with volume expansion [10]. In this study, the authors used transthoracic echocardiography, which would be difficult to apply in awake patients under spinal anaesthesia in the operating theatre. Impedance cardiography (ICG) is a non-invasive and operator-independent device that can be used to continuously assess CO and stroke volume (SV) [11]. As AH during surgery under SA can be related to various mechanisms (vasoplegia, hypovolemia), a non-invasive haemodynamic approach that evaluates preload status and cardiac output in this clinical setting should be validated in order to provide an appropriate AH treatment. The primary objective of this study was therefore to demonstrate that in patients with AH during surgery, a variation of SV in response to a limited fluid challenge measured by ICG could predict a further increase of arterial pressure during volume expansion. Secondly, we aim to demonstrate that in patients with AH during surgery, pulse pressure changes were correlated to those of SV with fluid expansion.

2. Methods

2.1. Ethics

This study was approved by the Institutional Review Board (IRB)[1] for human subjects. Informed consent was waived, as the IRB considered the protocol to be part of routine clinical practice.

2.2. Patients

A prospective, observational study was conducted over a two-month period (November to December 2013) at two institutions (anaesthesia department of CHU Amiens and anaesthesia department of Clinique Sainte-Isabelle, Abbeville). Patients undergoing orthopaedic surgery under spinal anaesthesia and monitored by NICCOMO, in whom the anaesthetist decided to infuse intravenous fluids to treat arterial hypotension were included. In the absence of a consensual definition during general and spinal anaesthesia, we defined AH as a systolic arterial pressure less than 100 mmHg. This definition was based on studies evaluating AH during SA [12,13]. Non-inclusion criteria were patients with chronic obstructive pulmonary disease, prior cardiac or thoracic surgery, or arrhythmia. Exclusion criteria were patients with arrhythmia and drug administration during the study period.

2.3. Anaesthesia

Each patient was monitored by 3-lead electrocardiogram, pulse oximetry and non-invasive blood pressure monitoring. Non-invasive systolic and diastolic arterial blood pressures were measured every 3 min by a plethysmographic method. All patients underwent spinal anaesthesia with spontaneous ventilation through a facemask with oxygen. Spinal anaesthesia was induced with a single intrathecal dose of 10 to 12.5 mg of bupivacaine (bupivacaine 5 mg/mL) and 2.5 μg of sufentanil. Bupivacaine and sufentanil were injected through a 27-gauge needle between the L3/4 or L4/5 interspaces. The extent of blockade was tested by pinprick every 5 min until the maximum height was reached. No patient received prophylactic ephedrine or fluid loading.

[1] IRB contact information: Comité de Protection des Personnes Nord-Ouest II, CHU, Place V.-Pauchet, 80054 Amiens cedex 1, France.

2.4. Impedance cardiography monitoring

After cleaning the skin with alcohol, two sensors including gel pads were carefully placed on each side of the thorax along the midaxillary line, and two sensors were placed on each side of the neck just above the clavicle. An ear clip was placed on the ear lobe. Bioimpedance values were recorded continuously (beat-by-beat) on the ICG monitor (NICCOMO^{TM}, Imedex, France). An ICG quality indicator, corresponding to the percentage of evaluable heart beats, was determined. Only patients with an ICG signal quality higher than 50% were included in the study. Heart rate was obtained continuously from the three-lead electrocardiogram. The reproducibility of SV measurements was tested before the study; the intraobserver and interobserver variability for SV measurements was 1.8% ($CI_{95\%}$: 0.9–6.6) and 2.1% ($CI_{95\%}$: 0.7–6), respectively. All values were analysed offline using dedicated NICCOMO^{TM} software. All values represented the mean of twenty consecutive beat measurements.

2.5. Study protocol

The study was composed of four steps. A first set of measurements (heart rate [HR], systolic arterial pressure [SAP], diastolic arterial pressure [DAP], SV, CO, systemic vascular resistance [SVR]) was recorded at baseline (T1). A first fluid administration of 100 mL of Ringer lactate was infused over 1 min. A second set of measurements was then immediately recorded (T2). A third set of measurements was recorded 5 minutes after the second set and was called baseline 2 (T3). Volume expansion consisted of the infusion of 500 mL of Ringer lactate over 10 min. A final set of measurements was recorded immediately after the end of volume expansion (T4).

3. Statistics

A minimum of thirty patients would be sufficient to demonstrate that ΔSV_{100} can predict a greater than 15% increase of SAP in response to volume expansion with an AUC greater than 0.85 for a power of 80%, an alpha risk of 0.05 and a beta risk of 0.2. Thirty-six patients were recruited after taking the exclusion criteria into account. The distribution of variables was assessed using the D'Agostino-Pearson test. Data were expressed as medians (25–75), or proportions (percentages), as appropriate. SAP measured before and after volume expansion (VE) was used to define responders and non-responders. A positive response was defined as a greater than 15% increase in SAP in response to volume expansion (between T3 and T4) [14]. A Wilcoxon test was used to make within group comparisons (T1 versus T2, T3 and T4). Differences between responders and non-responders were compared by Mann-Whitney tests. The Spearman method was used to test linear correlations. Receiver operating characteristic curves (ROC) were generated for ΔSV_{100}, baseline HR, and baseline SAP in relation to their capacity to predict an increase in SAP in response to volume expansion. Association between cardiovascular variables at baseline (HR, SAP, MAP, DAP, SV, SVR, ΔSV_{100}) and arterial pressure responsiveness was assessed using a univariate logistic model. Variables with a P value less than 0.10 were included in a multivariate logistic model with a backward selection procedure. Differences with a P value < 0.05 were considered statistically significant. Statistical analysis was performed using IBM® SPSS® Statistics 21 (IBM).

4. Results

Thirty-six patients were included during the study period. Two of these patients were excluded due to poor quality ICG signals. Finally, 34 patients undergoing orthopaedic surgery (hip [n = 24] or