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Effect of arterial puncture on ventilation

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ABSTRACT

Background: Clinicians frequently assume that during arterial puncture for measuring arterial blood gases patients hyperventilate from pain and anxiety. This assumption leads clinicians to falsely interpret a $PaCO_2$ and pH near the upper limit of normal as a chronic respiratory acidosis corrected by an acute respiratory alkalosis.
Objective: Determine if participants hyperventilate during arterial puncture from pain and anxiety.
Methods: We recruited participants from a pulmonary function laboratory referred for arterial blood gas measurement. We excluded those with heart failure and included those with any respiratory condition (COPD, asthma, sleep apnea). We measured end tidal PCO_2 ($P_{ET}CO_2$), respiratory rate, and heart rate 15 min before topical anesthesia, during anesthesia, during arterial puncture, and 15 min later. We assessed generalized anxiety before and measured pain during and after arterial puncture.
Results: 24 participants were recruited (age: 54 ± 12 years; men: 54%). $PaCO_2$ was 41 ± 5 mmHg. One had acute respiratory alkalosis. Respiratory rate increased from (19 ± 6 breaths per minute (bpm)) before to a maximum (21 ± 6 bpm) during arterial puncture ($p = 0.001$). Heart rate was stable throughout. The lowest $P_{ET}CO_2$ during the procedure (35 ± 5) was similar to $P_{ET}CO_2$ before the procedure ($p = 0.1$). The change in $P_{ET}CO_2$ and respiratory rate did not correlate with pain, anxiety, or lung function.
Conclusion: Respiratory rate increased slightly during arterial puncture without any change in $P_{ET}CO_2$. Hence, acid–base status must be interpreted without the assumption of procedure induced hyperventilation.

Introduction

Hypoventilation is a manifestation of advanced respiratory diseases. Recognizing it is important to determine prognosis and select appropriate level of respiratory support.[1,2] Because it is difficult in clinical practice to measure ventilation, we measure arterial blood gases to detect chronic respiratory acidosis—the sine qua non of hypoventilation.

Recognizing chronic respiratory acidosis from the measurement of arterial blood gases is easy when the partial pressure of arterial carbon dioxide ($PaCO_2$) is above 55 mmHg. When the $PaCO_2$ is closer to the upper limit of normal, clinicians choose one of three interpretations: normal, a compensation for metabolic alkalosis, or chronic respiratory acidosis with acute respiratory alkalosis that reduces the $PaCO_2$ close to the upper limit of normal.

This issue was recently raised in the attempts to define obesity hypoventilation syndrome (OHS); a serious condition that is becoming more common with the obesity epidemic.[3,4] The measurement of arterial blood gases in obese patients is frequently triggered by elevated serum bicarbonate. But not all obese patients with elevated serum bicarbonates have hypercapnia. About half of the obese patients with obstructive sleep apnea with elevated serum bicarbonate level are hypercapnic.[5] The non-hypercapnic patients with elevated bicarbonate are classically considered not to have OHS.

However, Hart and colleagues proposed that alkalosis in the absence of hypercapnia in obese patients should be added to the definition of OHS.[6] One explanation given to justify this change is that the pain and anxiety from arterial puncture causes hyperventilation and reduces carbon dioxide levels. Others also rely on this justification for labeling participants with normal $PaCO_2$ as having mild OHS.[7] Despite its plausibility, this explanation was tested only once by Morgan and colleagues who found no effect of

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arterial puncture on carbon dioxide in arterialized venous blood.[8] They did not measure the respiratory rate, tidal volume, cardiac output, pain, or anxiety.

In this study we reassess the effect of arterial puncture on ventilation by monitoring the respiratory rate and end tidal carbon dioxide pressure ($P_{ET}CO_2$) during arterial puncture and determining the correlation between the change in these variables and lung function, body weight, pain, and anxiety.

Methods

Setting

This prospective observational cohort study was conducted in a pulmonary function laboratory in a single center from 2014 to 2016. The Institutional Review Board approved the study and the participants provided written consent before enrollment.

Participants

We recruited patients 18 years or older. They were excluded if they did not speak English, were pregnant, had heart failure with reduced ejection fraction (it alters minute ventilation and the relation between end tidal and arterial $PaCO_2$), or used oxygen during the measurement of arterial blood gases.

Procedure

The participants were informed that we would be measuring the exhaled gas during this procedure. After providing informed consent, they sat upright in a chair in the testing room. We fastened a nasal cannula connected to a capnograph (Nonin Respsense, Minnesota, USA) around the participant's head and instructed the participant to breathe normally through the nose with the mouth closed.

One of three technicians with more than ten years of experience performed the arterial puncture. The technician first anesthetized the area with 1% lidocaine and then collected the arterial blood by puncturing the radial artery. After the arterial puncture, spirometry and measurement of lung volumes were performed according to current standards.[9,10]

Measurements

We collected demographic data, medications, and medical history from the medical record. We performed measurements at four times: Fifteen minutes before performing the arterial puncture (time 1), during local anesthesia by lidocaine injection (time 2), during arterial puncture (time 3), and 15 min after arterial puncture (time 4). We measured anxiety using the Generalized Anxiety Disorder Scale (GAD-7). A GAD-7 value > 10 suggests moderate to severe anxiety symptoms.[11] Pain from arterial puncture was measured using a numeric pain scale from 0 (no pain) to 10 (being the worst pain imaginable).[12] The samples were analyzed immediately at a single laboratory accredited by American College of Pathology (ABL800 FLEX analyzer, Radiometer Medical ApS, Brønshøj, Denmark). From the arterial blood samples we recorded the pH, base excess (BE), and the partial pressures of carbon dioxide ($PaCO_2$) and oxygen (PaO_2).

The capnograph uses side stream non-dispersive infrared spectroscopy for quantitative analysis. It provides a capnogram, a numeric value of the end tidal pressure of carbon dioxide ($P_{ET}CO_2$), oxygen saturation by pulse oximetry (SpO_2), respiratory rate (RR), and heart rate (HR). We determined the validity of the $P_{ET}CO_2$ by inspecting the capnogram to ensure that it includes three phases: anatomic dead-space, dead-space alveolar mix, and alveolar plateau. At each step, the recording continued until the investigator identified at least 5 consecutive good quality $P_{ET}CO_2$ waves. The medians of these 5 consecutive values for each variable were used for analysis.

We interpreted the acid-base status by plotting the pH and $PaCO_2$ on a Siggaard-Anderson acid-base chart.[13] The disorders were classified into six major areas: 1) metabolic acidosis, 2) acute respiratory acidosis, 3) chronic respiratory acidosis, 4) metabolic alkalosis, 5) acute respiratory alkalosis, and 6) chronic respiratory alkalosis.

Spirometry and lung volumes

All participants either had spirometry and lung volume measurement performed according to accepted standard on the same day or within 6 months of the index day (Vmax, Care Fusion \ BD, New Jersey, USA).

Statistical analysis

We estimated that a sample of 19 participants was sufficient to detect a change in $P_{ET}CO_2$ of 4 mmHg, a standard deviation of 4 mmHg, and an alpha and beta values of 0.01 and 0.1 respectively. To assess the effect of arterial puncture on ventilation we compared minimum $P_{ET}CO_2$ from time 2, time 3, and time 4 to $P_{ET}CO_2$ at time 1 and maximum RR from time 2, time 3, and time 4 to RR at time 1 using paired *t*-test. We also compared the $P_{ET}CO_2$, respiratory rate, heart rate, and SpO_2 between the four measurements using the Friedman test (the nonparametric equivalent of repeat measurement one-way ANOVA).

We also assessed the correlation of the change in $P_{ET}CO_2$ and the change in RR (as defined above) with the body mass index, FEV_1, FEV_1/FVC, pain, and GAD-7 using the Spearman correlation coefficient.

Results

We recruited 24 participants (Table 1). Most participants were older than 50 years and predominately African American. Eleven participants (46%) were obese (body mass index (BMI) > 30 kg/m^2). Thirteen participants (54%) had smoking exposure (past or current). Five (21%) had obstructive sleep apnea, 12 (50%) had COPD or asthma. Thirteen had a diagnosis of hypertension. Only one participant was on home oxygen therapy.

Spirometry was normal in nine participants (38%), obstructive in seven (29%), restrictive in three (13%), and mixed and nonspecific ventilator defects in five (21%).

The average pain during arterial puncture was 2.4 ± 2.0 (95% CI 1.6 to 3.2). Most participants did not feel any pain after 15 min of the arterial puncture (0.50 ± 1.14, 95% CI 0.02 to 1). Blood was drawn on the first attempt in 22 participants. The majority of participants were not using analgesics (Table 1).

The average GAD-7 scale score was 5.6 ± 5.7 participants (29%) had a score above 10 suggesting moderate to severe anxiety symptoms. Six participants (25%) were on anxiolytic medications.

The pH was 7.42, the $PaCO_2$ was 40.8 ± 4.8 mmHg, and four participants had $PaCO_2 > 45$ mmHg. Based on acid base chart, one participant had acute respiratory alkalosis, three had chronic respiratory acidosis, and one had chronic metabolic alkalosis. The rest were normal. $PaCO_2$ had highest correlation to $P_{ET}CO_2$ at time 2 (0.81). The difference between $PaCO_2$ and $P_{ET}CO_2$ was 5 ± 3 mmHg.