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Pulse oximeter-enhanced accuracy of capnometry in children with cyanotic heart disease

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Abstract *Objectives:* To evaluate the relationship between the arterial end-tidal partial pressure of carbon dioxide (PCO_2) difference (ΔpCO_2) and the degree of desaturation in children with cyanotic heart disease (CHD) and to come to a more reliable estimation of the arterial carbon dioxide partial pressure ($PaCO_2$) from the end-tidal carbon dioxide partial pressure (PET-CO_2). *Design and setting:* In part retrospective, in part prospective observational study at a university children's hospital. *Subjects and interventions:* We retrospectively assessed the relationship between the arterial oxygen saturation as measured by means of pulse oximetry (SpO_2) and the arterial to end-tidal PCO_2 differences (ΔPCO_2) from the records of medical or surgical interventions in 43 patients with CHD. We derived a $PaCO_2$-PET-CO_2 correction formula that was prospectively validated in 34 patients with CHD. *Measurements and results:* In the retrospective part we found a significant correlation between SpO_2 and ΔPCO_2 (r^2=0.84, p<0.001). The regression equation (corrected PET-CO_2=raw PET-CO_2–0.36×SpO_2+39) was used in the prospective part to calculate the corrected PET-CO_2. The r^2s for the correlations between $PaCO_2$ and uncorrected and corrected PET-CO_2 were 0.17 (p<0.05) and 0.94 (p<0.001), respectively. The uncorrected PET-CO_2 bias was 13.0 mmHg, the bias ± 2SDs was –0.1 and 26.2 mmHg. The corrected PET-CO_2 bias was –0.6 mmHg, the bias ± 2SD's was –4.0 and 2.9 mmHg. *Conclusions:* Correcting the PET-CO_2 for the degree of hypoxia using the SpO_2 in artificially ventilated infants and children with CHD results in a clinically applicable estimation of the $PaCO_2$. As both SpO_2 and PET-CO_2 can be monitored continuously and non-invasively, this could facilitate artificial ventilation management in children with CHD.

Keywords Pediatric anesthesia · Respiratory monitoring · Capnometry · Cyanotic heart disease

Introduction

In anesthesia, it is common practice to adjust ventilator settings based on the end-tidal carbon dioxide partial pressure (PET-CO_2) assuming a more or less constant relationship between arterial carbon dioxide partial pressure ($PaCO_2$) and PET-CO_2. In otherwise healthy patients, this may in general yield $PaCO_2$ values within the normal range but physiological disturbances and interventions may affect this relationship in the more seriously ill [1, 2, 3, 4, 5]. This explains, in part, why capnometry has not gained wide acceptance in ICUs [6].

In children with cyanotic heart disease (CHD) capnometry has been described as "unreliable" as significant arterial to end-tidal PCO_2 differences (ΔPCO_2) are commonly found [7, 8, 9, 10, 11]. Moreover, Burrows and

Lazzell found a considerable inter- and intra-individual variability in the ΔPCO_2 in children with mixing and right-to-left shunting type heart disease [7, 8]. As the PET-CO_2 considerably and variably underestimates the true $PaCO_2$, adjusting minute ventilation guided by capnometry could lead to hypoventilation, hypercapnia and respiratory acidosis in these cases and could add to an already present metabolic acidosis. Due to the pathophysiology of CHD, we assumed a relationship between the degree of desaturation and the magnitude of this ΔPCO_2 [9, 10].

The aims of the present study were to evaluate the relationship between the ΔPCO_2 and the arterial oxygen saturation as measured by means of pulse oximetry (SpO_2) and, if a consistent relationship were found, to assess whether the SpO_2 could be applied as a correcting factor to estimate the $PaCO_2$ from the PET-CO_2 in patients with CHD.

Material and methods

The hospital ethics committee approved the study and judged that informed parental consent was not required as only clinically available data were used and the set-up was in part retrospective and in part observational. The study consisted of two elements: a retrospective study to evaluate the relationships between SpO_2 and ΔPCO_2 in order to arrive at a more reliable estimation of the $PaCO_2$ from the PET-CO_2 corrected for the degree of hypoxia (derivation set), and a prospective observational study to test the clinical usability and reliability of the correction formula (validation set). In both the retrospective derivation and the prospective validation studies, all interventions were performed under general anesthesia using midazolam in combination with either sufentanil or morphine.

The trachea was intubated with an endotracheal tube of adequate diameter so that there was no audible air leak below 35 cmH_2O peak inspiratory pressure. The lungs were ventilated with an oxygen/air mixture (FIO_2 of 0.21 in duct-dependent lesions; in all other cases the FIO_2 was 0.5) using a Dräger Cicero (Dräger, Lübeck, Germany) ventilator (OR) or a Siemens Servo 300 (Siemens Elema, Solna, Sweden) ventilator (PICU). The PET-CO_2 was monitored by means of a mainstream capnometer (sensor: Hewlett Packard M1016A, monitor: Merlin, HP, Böblingen, Germany). The capnometer was calibrated before each use. Further monitoring consisted of pulse oximetry (probe: Nellcor Oxisensor II D-20 or N-25, Nellcor Puritan Bennett, Pleasanton, Calif., monitor: Merlin, HP), ECG, invasive arterial and central venous pressures and central and peripheral temperature measurements. Arterial blood gas samples were taken during normothermic "steady states" before and after interventions and determined immediately after sampling in triplicate on a Chiron 855 or 865 blood gas analyzer (Bayer Diagnostics Manufacturing, Sudbury, UK).

All records of children with congenital cyanotic heart disease requiring medical or surgical interventions in the period January 1999–June 2000 were scrutinized. As capnography was infrequently used in the PICU in those days, most retrospective data were derived from anesthetic records. The records of 43 patients were selected for analysis. This selection was made based on completeness of relevant clinical and laboratory data. Interventions included medical therapy (e.g. alprostadil therapy), intervention catheterization (e.g. atrial balloon septostomy) and surgery. All drug therapy interventions and all but one Rashkind procedures were performed in the PICU. SpO_2 data of 95% or more either before or after intervention were disregarded to prevent non-linearity, leaving 50 of a possible 86 data sets for analysis. The correction formula derived from the retrospective data was validated in 32 patients with CHD both in the OR (n=16) and PICU (n=16). All post-intervention measurements were taken in the PICU. Nine of the 16 patients undergoing surgical interventions were preoperatively artificially ventilated and on inotropic support and/or alprostadil therapy in the PICU. In these cases the pre-intervention measurements were also taken in the ICU bringing the total of measurements performed in the PICU to 57. Disregarding SpO_2 data of 95% or more yielded 57 of a possible 64 data sets.

The data were analyzed by means of linear regression, Student's t-test for paired data and Bland-Altman analysis [11]. A p less than 0.01 was considered significant. We also assessed the effects of raw and corrected PET-CO_2 data on medical decision-making with respect to adjusting the ventilator minute volume setting to achieve the desired $PaCO_2$.

Results

The demographic data are shown in Table 1. In all but three cases interventions led to improvements in the clinical condition of the patient with an increase in SpO_2 and a decrease in ΔPCO_2. Three Rashkind procedures failed.

Regression analysis revealed a good correlation between SpO_2 and ΔPCO_2 (r^2=0.84, p<0.001) (Fig. 1). The resulting regression equation, corrected PET-CO_2=raw PET-CO_2–0.36×SpO_2+39, was then used to calculate the $PaCO_2$ from the PET-CO_2 for any SpO_2 and subsequently validated in a prospective study. The SpO_2 values in this study ranged from 23–88% before, to 46–100% after, intervention. The comparisons between actual $PaCO_2$ and corrected PET-CO_2s are shown in Figs. 2a and 2b.

Table 1 Demographic data (*TGA* transposition of the great arteries, *Switch* arterial switch operation, *Rashkind* balloon septostomy, *ToF* tetralogy of Fallot, *Shunt* aortopulmonary shunt, *HLHS* hypoplastic left heart syndrome, *TA* tricuspid atresia, *PA* pulmonary atresia, *BT* Blalock-Taussig shunt, *AVSD* atrioventricular septal defect)

		Derivation group	Validation group
Patients (n)		43	32
Disease	Therapy		
TGA	Switch	12	7
	Alprostadil	7	9
	Rashkind	4	4
ToF	Shunt	3	2
	Correction	3	2
HLHS	Norwood	2	5
	Rashkind	2	3
PA/TA	BT shunt	4	0
AVSD	Banding	4	0
Age, median (range)		11 days (2 days – 11 years 1 month)	56 days (1 day – 11 years 9 months)

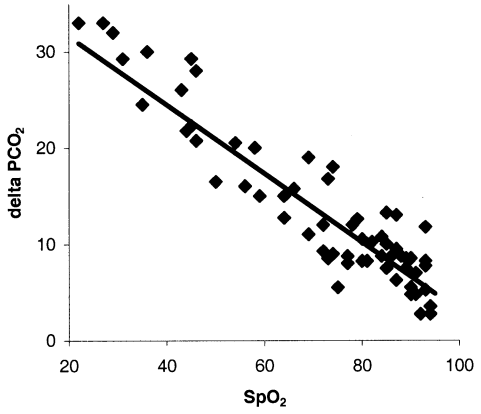

Fig. 1 Correlation between arterial saturation as measured by means of pulse oximetry (SpO_2; %) and arterial to end-tidal differences in carbon dioxide partial pressure (ΔPCO_2; mmHg) (For explanation see text)

The r^2 for the correlation between $PaCO_2$ and raw PET-CO_2 was 0.17 ($p<0.05$), while the r^2 for the correlation between $PaCO_2$ and corrected PET-CO_2 was 0.94 ($p<0.001$) (Fig. 2a). The raw PET-CO_2 bias was 13.0 mmHg, the bias ± 2SDs was –0.1 and 26.2 mmHg, respectively (Fig. 2b). The corrected PET-CO_2 bias was –0.6 mmHg, the bias ± 2SDs was –4.0 and 2.9 mmHg, respectively (Fig. 2b). The difference between actual values and values obtained by means of the ΔPCO_2 correction was statistically not significant. Decision-making with respect to ventilator setting would have been accurately supported in 92% of the cases when using the corrected PET-CO_2 values, whereas in only 5% of the cases would a decision based on the raw PET-CO_2 values have been correct.

Discussion

The PET-CO_2 is normally determined by alveolar ventilation, pulmonary perfusion, CO_2 production (VCO_2) and the ventilation-perfusion (V/Q) relationship [12, 13]. In CHD, alveolar ventilation is normal in general, pulmonary perfusion low, V/Q mismatch considerable and VCO_2 decreased [7, 8, 9]. In CHD, low pulmonary perfusion and/or right-to-left shunting of low oxygen/high carbon dioxide venous blood leads to cyanosis and a large arterial end-tidal PCO_2 difference (ÄpCO$_2$) [10]. This, in turn, leads to other effects, e.g. shift of the oxygen dissociation curve, an increase in hemoglobin concentration and depressed metabolism as it becomes oxygen delivery-dependent, thus leading to a decrease in VCO_2.

We found an inverse relationship between SpO_2 and ΔPCO_2. When validating the correlation equations to correct the PET-CO_2 for the degree of hypoxia in a prospective group, we found an adequate agreement between actual $PaCO_2$ and corrected PET-CO_2, while the

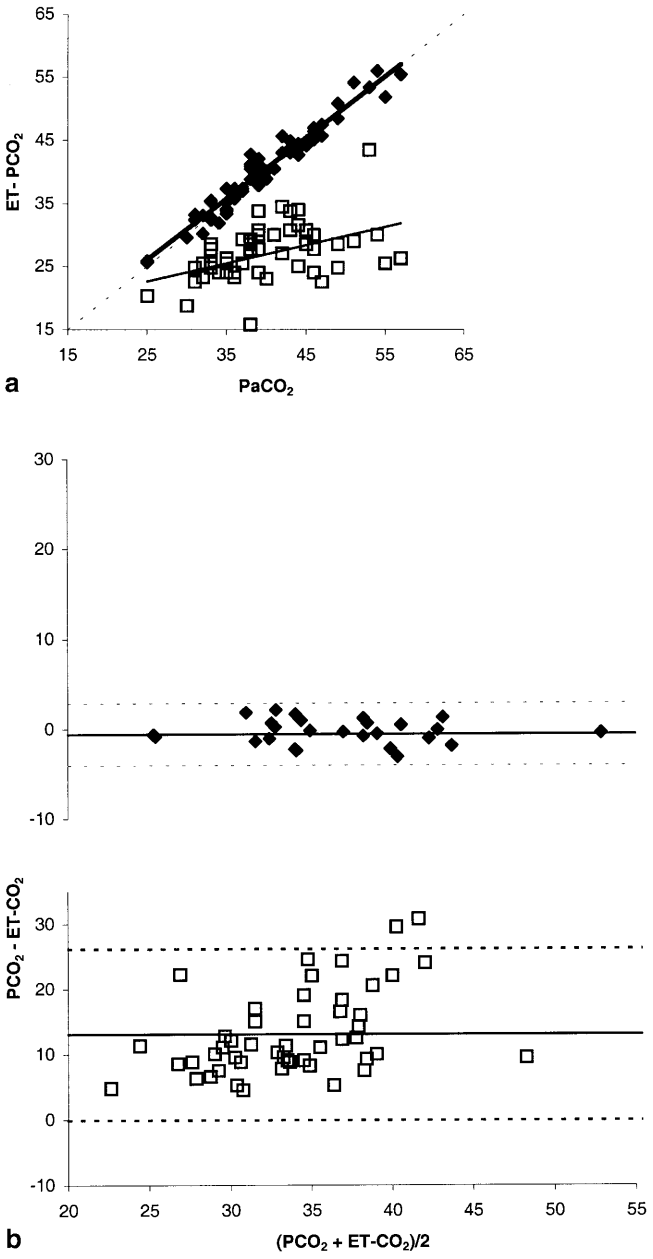

Fig. 2 a. Scattergram. *Open squares* and *thin line*: arterial carbon dioxide partial pressure ($PaCO_2$) versus raw end-tidal carbon dioxide partial pressure (PET-CO_2; mmHg), *closed diamonds* and *bold line*: $PaCO_2$ versus corrected PET-CO_2. The *dotted line* represents the line of identity **b.** Bland-Altman plots. Top section (*open squares*): bias and 2 SDs limits of arterial carbon dioxide partial pressure ($PaCO_2$) versus raw end-tidal carbon dioxide partial pressure (PET-CO_2); bottom section (*closed diamonds*): same for $PaCO_2$ versus corrected PET-CO_2. All units are millimeters of mercury

agreement between actual $PaCO_2$ and raw PET-CO_2 was poor. The corrected PET-CO_2 values proved sufficiently reliable to support artificial ventilation management, whereas most of the decisions based on the raw capno-

metric data would have been wrong. One advantage of PET-CO_2 and SpO_2 monitoring over blood gas analysis is that both capnometry and pulse oximetry are non-invasive and continuous techniques while blood gas analysis is invasive, discontinuous and expensive. Although we are aware that pulse oximetry-enhanced accuracy of capnography represents a gross simplification of the pathophysiology of cyanosis in congenital heart disease and disregards various other contributors, its application yields reliable and continuous information on gas exchange and the effects of surgical or medical interventions and may reduce the need for arterial blood gas analysis. Due to the procedural differences, it is likely that this correction has a broader applicability and a larger impact in the PICU than in the OR.

In conclusion, capnometry is a useful tool in respiratory monitoring but has important physiological limitations when used to estimate $PaCO_2$ in children with CHD. The performance of capnometry in children with CHD can be enhanced when used in combination with SpO_2 monitoring and correcting the PET-CO_2 data for the degree of hypoxia.

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