Non-invasive Monitoring of Carbon-dioxide in Newborns and Children

Critically ill newborns and children in an intensive care unit require regular monitoring of their arterial carbon-dioxide tension ($PaCO_2$) to provide information on their ventilatory status. Repeated arterial sampling is not only a waste in terms of manpower and blood volume, it inflicts pain and stress on an already compromised child and also introduces the risk of infection. This can be avoided by utilising non-invasive methods of estimating $PaCO_2$. There are two such methods - transcutaneous monitoring ($tcPCO_2$) and end-tidal monitoring ($PetCO_2$) of carbon-dioxide tension. Transcutaneous monitoring is done by means of a sensor applied to the skin, whereas end-tidal monitoring is done by sampling the end-expiratory gases in intubated patients. The advent of a single skin sensor for monitoring both oxygen tension and carbon-dioxide tension has simplified the measurement of $tcPCO_2$[1,2].

PRINCIPLES OF MEASUREMENT

Transcutaneous Monitoring

The concept that both oxygen and carbon dioxide can be eliminated from the skin surface, is an old one[3]. Studies on transcutaneous monitoring of oxygen have shown that warming the skin to 44° C produces vasodilation and is essential for improving the diffusion of oxygen from the blood vessels and accurately measuring the PO_2[4-6]. Studies using unheated sensors for $tcPCO_2$ monitoring have been promising and one recent study has also shown that skin sensors at either 37, 42 or 44 degrees Celsius are adequate for measuring $tcPCO_2$[7-9]. However, some studies have shown that when heated electrodes are used there is a shorter time lag from the application of the sensor to measurement of $tcPCO_2$, and a marginally better correlation of $tcPCO_2$ and $PaCO_2$[10-13]. The skin sensor consists of a pH-sensitive electrode with an adjacent reference electrode, covered with a membrane permeable to CO_2[14]. Carbon-dioxide from the skin surface diffuses through the membrane, induces pH changes in an electrolyte solution contained between the membrane and the electrode, which are detected by the pH electrode and converted to PCO_2 values.

There are two constant factors that make $tcPCO_2$ values overestimate the $PaCO_2$ in any normal individual. The first is the phenomenon that when blood is warmed the $PaCO_2$ rises because the solubility of CO_2 decreases. The relationship between $PaCO_2$ values at different temperatures is governed by what is known as the anaerobic heating coefficient of blood, which is 4.5% per degree centrigrade[15]. Thus, the $tcPCO_2$ at 44°C has to be divided by a factor of 1.37 to get the value at 37°C. The second phenomenon is that the skin tissue itself produces CO_2 as part of its metabolic processes and this contributes around 4 mm Hg, to the $tcPCO_2$ in healthy infants[16,17].

End-tidal Monitoring

The terminal portion of the expired tidal volume contains alveolar gases, unlike the initial portion which contains gases from the respiratory tract. Since CO_2 diffuses readily across the alveolo-capillary mem-

brane, the PCO_2 measured in this terminal sample reflects the $PaCO_2$. To measure the $PetCO_2$ an adaptor is inserted into the breathing circuit as close to the patient as possible, generally at the ventilator end of the endotracheal tube. The adaptor entrails gases at a pre-set flow rate and sends it to the sampling cell, where the PCO_2 is measured by infrared capnography or mass spectrometry. The measuring instrument generates a capnogram, which is a record of PCO_2 in a time frame. At the beginning of expiration there is a rise in PCO_2, followed by an alveolar plateau. This plateau terminates with the beginning of inspiration. The value in the alveolar plateau just before termination is the $PetCO_2$ and is expected to correlate with the value of $PaCO_2$[18,19].

Even in normal individuals the $PetCO_2$ tends to underestimate the $PaCO_2$. This is because there is always a certain amount of physiological dead space due to ventilation-perfusion mismatch. In the underperfused alveoli CO_2 does not get an opportunity of diffuse out thereby reducing the net $PetCO_2$[20].

Clinical Applications

Several studies have been done to compare $tcPCO_2$, $PetCO_2$ and $PaCO_2$ in newborns and children. Although both non-invasive methods have been found useful, the $tcPCO_2$ monitoring has been found to be consistently superior to $PetCO_2$[21-25]. In a study on 24 newborn infants with pulmonary disease, adjusted $tcPCO_2$ measurements on a population based correction factor led to estimates of $PaCO_2$ within ± 8 mm Hg with 95% confidence interval, whereas this degree of precision with $PetCO_2$ was possible only using calibration for each individual patient[21]. In neonates with respiratory distress syndrome there was a linear correlation between $PaCO_2$ and $tcPCO_2$ ($r = 0.71$, slope = 0.9) while $PetCO_2$ did not correlate as well ($r = 0.52$, slope = 0.42)[22]. $tcPCO_2$ monitoring is ideally suited for sick neonates and children with lung disease such as pneumonia or hyaline membrane disease. $tcPCO_2$ has the advantage over $PetCO_2$ in that it can be used in patients who are not yet ventilated but are likely to develop respiratory failure, but it has the disadvantage that it takes a longer time to calibrate. $PetCO_2$ monitoring is ideally suited for ventilated intraoperative, postoperative or muscle relaxed patients who have normal lungs and require monitoring of CO_2 to adjust ventilation. $TcPCO_2$ or $PetCO_2$ values must be compared atleast once with $PaCO_2$ in a given patient to ensure that they are correlating.

Factors that Falsely Elevate $tcPCO_2$

1. *Respiratory acidosis* : It was widely assumed that the temperature correction of 1.37 and the metabolic factor of around 4 mm Hg could explain the difference between $tcPCO_2$ and $PaCO_2$ at 44°C, and that the $tcPCO_2$ could be theoretically corrected to estimate the $PaCO_2$[26]. However, more recent studies have shown that in regression analyses between $tcPCO_2$ and $PaCO_2$ the slope may be higher than 1.37[27-30]. To resolve this issue one study looked at the difference between the $PaCO_2$ and theoretically corrected $tcPCO_2$ at different levels of $PaCO_2$ and found that this difference was not constant and it increased progressively with rising $PaCO_2$ values[31]. The authors hypothesized that vasoconstriction and opening up of arterio-venous shunts in hypercapnea resulted in accumulation of the tissue CO_2 and thereby higher $tcPCO_2$.

2. *Hypotension* : Animal experiments suggest that with falling cardiac output the $tcPCO_2$ progressively overestimates the $PaCO_2$ possibly because of poorer CO_2 washout from skin tissue[32]. In contrast one study in 24 newborn infants demonstrated that there was a discrepancy between $tcPCO_2$ and $PaCO_2$ values only in extreme hypotension when systolic blood pressure was less than 15 mm Hg[33]. There is no effect of moderate or server hypotension, hypothermia, anemia, polycythemia, edema or tolazoline use on the relationship between $tcPCO_2$ and $PaCO_2$ values.

Factors that Falsely Lower $PetCO_2$

1. *Increased dead space* : Increased anatomical dead space to tidal volume ratio increases the discrepancy between $PetCO_2$ and $PaCO_2$[34]. This discrepancy can be put to use to estimate the dead space in patients with lung disease.

2. *Right to left shunting* : When there is intrapulmonary or intracardiac shunting of blood, a certain fraction of the cardiac output effectively bypasses the lungs Hence the $PetCO_2$ values underestimate the $PaCO_2$ values in infants and children with cyanotic heart diseases[35-37].

3. *Site of sampling the end-tidal gases* : There is a possibility that the CO_2 in the end-tidal sample may get diluted by the fresh gases of the ventilator at the ventilator end of the endotracheal tube. A few studies have looked at the correlation of $PetCO_2$ with $PaCO_2$ when the sampling is done through a sampling catheter at the patient end of the endotracheal tube (distal $PetCO_2$) versus the ventilator end of the endotracheal tube (proximal $PetCO_2$)[38,39]. They have concluded that distal $PetCO_2$, rather than proximal $PetCO_2$, correlates better with $PaCO_2$.

4. *High flow rates for sampling* : Generally flow rates of 150 ml/min are used for sampling the end-tidal gases. If, however, this flow rate is high it may exceed the patient's expiratory flow rate resulting in entrailment of gases from the ventilator circuit along with the patient's expiratory gases. This results in an underestimation of the $PaCO_2$[40].

Factor that Falsely Elevate $PetCO_2$

Studies in adults have shown that a high tidal volume, low respiratory rates or physical activity may elevate the $PetCO_2$ to levels equal to or greater than the $PaCO_2$[41-43].

Undesirable Effects

If $tcPCO_2$ is being monitored with a combined oxygen and carbon-dioxide sensor the skin has to be heated to 44°C. This may result in local burns. Hence the site must be changed every 2 to 4 hours. The problem of burns and of abrasion of upper layers of the stratum corneum by repeatedly removing the adhesive tapes becomes an important issue in premature newborns with delicate skins. There are no significant deleterious effects of $PetCO_2$ monitoring.

Conclusions

Transcutaneous and end-tidal monitoring of PCO_2 have a definite role in the management of neonates and children in the intensive care setting. $TcPCO_2$ slightly overestimates $PaCO_2$. It is best suited for patients with lung disease or right to left shunting and it should be interpreted cautiously in patients with hypercapnea or severe shock. $PetCO_2$ generally underestimates $PaCO_2$. It can be used only in ventilated patients and is best suited for those who are ventilated for conditions other than cardiopulmonary diseases; such as

postoperative patients, patients with neuromuscular diseases, and those who are muscle relaxed.

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