Adequate ventilation in hypothermic patients

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Background

In vitro diagnostics of e.g. coagulation, oxygen and acid-base status are commonly performed at a temperature of 37 °C in daily clinical routine, irrespective of the patient's actual body temperature. If this body temperature is decreased in a situation of hypothermia, considerable problems occur in the acid-base balance, because the partial pressures of gases increase massively due to their reduced solubility associated with the warming up to 37 °C.

Unfortunately, the current discussion about optimal ventilation and management of the acid-base balance of a hypothermic patient, is still focused on two options:

1. Keeping uncorrected values in the normal range in hypothermic patients (a procedure known as alpha-stat regulation);
2. The measuring device performs an internal correction – using established known algorithms – of the values taken at 37 °C to the patient's actual body temperature, which has previously been set in the device (a procedure known as pH-stat regulation).

The purpose of the following example is to demonstrate this issue for a patient with a body temperature of 25 °C, considering only the $paCO_2$, which is relevant for ventilation: The uncorrected $paCO_2$ value measured and displayed by the device at 37 °C is approx. 70 mmHg, which seems hardly plausible to the anaesthesiologist (alpha-stat); the $paCO_2$ value measured at 37 °C and corrected to 25 °C is 40 mmHg, a value the doctor is confident with and can be regarded as "correct" ventilation (pH-stat).

Question

Which endexpiratory and thus arterial $paCO_2$ is to be targeted in hypothermic patients and which pH value can then be expected in arterial blood in a status of normocapnia if the patient has a base excess of 0 mmol/l?

Experiments

The acid-base balance of a blood sample was simulated in vitro as follows: Equilibration of fresh blood in the tonometer at 37 °C and 25 °C to pCO_2 40 mmHg and pO_2 100 mmHg (gas mixtures of the gas mixing device adjusted to pH_2O values of 47.1 (37 °C) and 23.8 mmHg (25 °C), respectively). Measuring of the blood pH was made with a pH electrode after calibration with phosphate buffer using temperature-corrected target values [6.841 / 7.383 (37 °C) and 6.865 / 7.410 (25 °C), respectively]. Additionally, the pH and pCO_2 were measured using a BGA device (blood gas analyzer OMNI 9, Roche) with calculation of the base excess (BE, mmol/l). We analysed normal blood (expected BE ± 0 mmol/l) and blood with a quantitative addition of HCl in order to achieve a ΔBE of -10 mmol/l.

Results

Equilibration of a blood sample with a gas mixture of e.g. 40 mmHg pCO_2 can be defined in the alveolus as $petCO_2$ using a capnometer and then confirmed in the blood by blood gas analysis, maybe with minor deviations. This process is simulated in the tonometer with the same positive result as

long as the temperature remains constant for equilibration and measurement.

As expected, the values for pCO_2 (40) and pO_2 (100) were confirmed by the BGA device for both temperatures. The pH values determined for this blood sample with a presumed BE of 0 mmol/l using the two devices are close to the normal value of 7.40 at both 37 °C and 25 °C and thus resulted in a BE of almost 0 mmol/l. As expected, the addition of 10 mmol/l acid reduced the BE of the blood to approximately 10 mmol/l.

If a blood sample is now equilibrated at 25 °C but analysed at 37 °C in a device, this will yield an unusual result: A pCO_2 of approximately 70 mmHg will be measured instead of the expected 40 mmHg, similarly, a pO_2 of approx. 165 mmHg instead of 100 mmHg, which can be explained by the temperature increase in the closed system of the BGA device. The respiratory acidosis produced with a pCO_2 of 70 mmHg will generate a pH of approx. 7.25, but the BE calculated from pH and pCO_2 is still approx. 0 mmol/l, i.e. no change compared with the baseline value. The same holds true for a blood sample with a BE of -10 mmol/l, i.e. the BE remains constant even if an additional respiratory acidosis is mimicked with a pCO_2 of approx. 70 mmHg due to the temperature rise in the device.

If, however, the measurement is made with the BGA device at 37 °C, whilst a conversion to a body temperature of 25 °C is set, the baseline values will be retained, i.e. a normal pCO_2 and pH, so that the BE will be calculated correctly to be approx. 0 mmol/l. A similar result is found for a blood sample with a BE of approx. -10 mmol/l.

Conclusion in vitro

Under in vitro conditions, it can be demonstrated with a clinically sufficient precision that the BE is independant of the temperature: irrespective of the temperature, a blood sample with pCO_2 40 mmHg and BE 0 mmol/l exhibits a pH of 7.40, and a blood sample with a BE -10 mmol/l exhibits a pH of 7.25. A common BGA device has been shown to be able to measure a sample at 37 °C, while that sample had been equilibrated at 25 °C, and to report a correct result after correction to 25 °C. This holds true for the parameters pCO_2, pO_2 and pH and thus for the BE as well. What happens is that a fictitious respiratory acidosis (pCO_2 ↑, pH ↓) is imposed on the blood in the closed system of the analyzer due to the temperature rise to 37 °C leading to a decreased CO_2 solubility. By definition, this acidosis does not allow for any BE change. The analyzer will then be able to reverse this temperature increase mathematically.

Clinical conclusion

For the ventilation of a hypothermic patient, it is recommended to ventilate the patient with a target value of $paCO_2$ 40 ± 5 mmHg under capnometric monitoring ($petCO_2$) and to correct the values obtained by invasive blood measurement at 37 °C to the patient's body temperature to be entered in the BGA device using internal known validated algorithms (procedure known as pH-stat regulation). This will make sure that a physiological patient pH of 7.40 remains as long as the BE is 0 mmol/l. This procedure will keep you on the right track, because it is not necessary to learn „new normal values" for pH (7.40), pCO_2 (40 mmHg), pO_2 (70 - 90 mmHg) and BE (± 0 mmol/l). The metabolism is diagnosed via a temperature-independent BE; the pH corrected for body temperature is used – if it is used at all – for the diagnostics of acidosis and alkalosis.

References

1. Zander R. Optimierung des Säure-Basen-Status unter Hypothermie. Anaesthesist 2007; 56: 912-916
2. Zander R. www.Physioklin.de