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Die klinische Bedeutung von Base Excess und Laktatkonzentration

Clinical Relevance of Base Excess and Lactate Concentration

Since a new generation of modern blood gas and metabolite analyzers is available with an option for a great variety of clinical parameters, the determination of the base excess (BE, mmol/l) in blood, and of the lactate concentration (cLact, mmol/l) in plasma are successfully used as a diagnostic tool in many clinical disciplines such as anesthesiology, surgery, obstetrics, internal and transfusion medicine. Therefore, this subject was presented in a special session on the annual meeting of the Deutsche Interdisziplinäre Vereinigung für Intensiv- und Notfallmedizin (DIVI 2000) on November 2000 in Hamburg, main results of which will be published in the following issue.

A direct proportionality can be expected between the change of the BE in blood (Δ BE), and that of lactate concentration in plasma (Δ cLact), i.e., the generated H^+ -ions, and the lactate-anions are from the same source of lactic acid production. In the contribution by Zander, the validity of such a relationship is clearly demonstrated from the measurement in athletes under exercising conditions for BE up to -30 mmol/l, and the corresponding change of lactate in plasma, but not if related to blood. However, there are also many cases in which this proportionality can not be found. For example, in patients on the ICU, receiving exogenous lactate or bicarbonate from infusion solutions or if the generated H^+ -ions are not from hypoxic lactic acid production but ATP-hydrolysis. Since the BE includes all the H^+ -ions which are released into the extracellular fluid, it is an excellent parameter for diagnosis of the non-respiratory acidosis, and, in combination with the measured lactate concentration, it can be used for therapeutic control of lactate infusions or as hypoxic marker for additional information.

In a recent study of a great number of polytrauma patients, it could be shown that the measured BE at admission into the hospital within the first 24 hours is also a significant prognostic indicator related to hemodynamics, needs of blood transfusions and volume, and mortality. For BE as low as -6 mmol/l, the predicted mortality is already as high as 25%. From the data in the contribution of Rixen *et al.*, taken from the register of trauma patients of the Deutsche Gesellschaft für Unfallchirurgie, it is also evident that the posttraumatic time course of the BE is significantly correlated to the mortality of the patient. So, the BE may be useful as a feed back in the therapy of the trauma-induced oxygen deficit.

In obstetrics, the routine measurement in umbilical blood is that of pH alone, reflecting both the respiratory and non-respiratory side of the newborn acid base status. Using the complete formula for correcting oxygen saturation, this calculated BE is a true non-respiratory parameter, irrespective of whether obtained from measured pH, pCO_2 , cHb and sO_2 in arterial or venous umbilical blood. The successful use of correctly calculated BE is well documented by a great number of obstetric protocols in the contribution of Roemer to show that from about 8000 neonates at the time of birth, the mean umbilical BE is in the range -5.5 to -6.8 mmol/l.

The general hypothesis that any lactic acidosis is caused by tissue hypoxia, is critically discussed by Knichwitz. From literature and own data he shows that hyperlactatemia (cLact >2 mmol/l) can only be correctly interpreted as a marker for hypoxia if the metabolic acidosis is also indicated by the corresponding BE. Furthermore, it must be certified that the function of the liver is sufficient for hepatic clearance of accumulated lactate.

As already mentioned, the proportionality between BE and the plasma lactate concentration disappears and makes diagnosis

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impossible, if solutions containing large amounts of lactate (up to 55 mmol/l) are infused into the patient. This problem is thoroughly investigated by *Raum et al.* in an animal model. Whereas the time course of measured lactate concentration is interpretable in hemorrhagic animals without infusion, it is not in animals receiving lactate containing infusion solutions, irrespective of whether hemorrhaged or not. The doses infused are comparable to those in polytrauma patients, however, distributed over a longer period.

In another contribution of *Zander*, possible interactions of the administered infusion solutions and blood products with the acid base status of the patient are discussed. These are dilution acidosis from decreased extracellular bicarbonate concentration and infusion alkalosis. The latter is caused from metabolism of infused metabolizable anions such as lactate, acetate, malate or gluconate in the intact liver, a process in which HCO_3^- is produced, and H^+ -ions are consumed. This iatrogenic metabolic alkalosis must be compensated by hypoventilation, which leads to arterial hypoxia from diminished oxygen uptake in the lungs, even though the mainly hepatic oxygen consumption is increased. To the clinician, these consequences are often difficult to overlook, since declaration of the infusion solutions is insufficient with respect to possible metabolic effects of the containing anions. Furthermore, in stored packed erythrocytes with added acidic stabilizing solutions (CPD, SAG-M) over a period of up to 42 days, the measured BE is in the range -25 to -55 mmol/l, and the concentration of lactate from glycolysis up to 30 mmol/l.

From all these contributions, it is evident that the base excess and the lactate concentration play a significant role in the different areas of clinical medicine.