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A Fresh Look at an Old Question

NORMAL VALUES of arterial *p*H and P_{CO_2} are well-defined for humans at the body temperature of 37°C. When a patient is hypothermic, however, optimal values have not been identified despite considerable interest by anesthesiologists and others. In this issue of ANESTHESIOLOGY, Ream *et al.*[1] present a concept for the management of acid-base balance at any body temperature.

The P_{CO_2} and *p*H of a sample of blood are temperature dependent. When a blood specimen of known *p*H and P_{CO_2} is cooled anaerobically to a lower temperature, the P_{CO_2} decreases and the *p*H increases in a predictable manner. This information has been formalized by Severinghaus[2] and others and is used by most clinical laboratories to correct the *p*H and P_{CO_2} values measured with electrodes maintained at 37°C, to the patient's body temperature. It is at this point that confusion arises. What should the *p*H and P_{CO_2} be when the values presented are either "temperature corrected" to actual body temperature or "uncorrected" at the measuring electrode's temperature?

A survey of 13 anesthesiologists at 12 major institutions reveals that there are three schools of thought. Two of the 12 centers, including Dr. Ream's own institution, use "uncorrected" *p*H and P_{CO_2}. If the values measured at 37°C in the electrode are in the normal range (*i.e.*, *p*H = 7.40 ± 0.04 and P_{CO_2} = 40 ± 4.0 mmHg), this group is satisfied that the patient's acid–base status is acceptable, and any acid–base adjustments are based on "uncorrected" values irrespective of the patient's actual temperature. The second group, consisting of six of the 12 centers, are those that use "temperature corrected" blood values at the hypothermic temperature and attempt to maintain *p*H at 7.4 and P_{CO_2} at 40 mmHg at that reduced body temperature. The condition that they aim for would correspond to hypercapnia and acidosis at the electrode temperature. The policy of the third group postpones the decision, since correction of acid-base status is begun only when the patient has been rewarmed to normothermic levels.

It is surprising that a consensus has not yet been reached for such a long-standing question of clinical management. Work done in the early 1950s on hypothermia without cardiopulmonary bypass led Virtue,[3] in a monograph on hypothermia, to state that "a low carbon dioxide content and a high pH of the blood are desirable but further documentation of this factor is necessary." This statement was based on the information that spontaneous ventricular fibrillation was less of a problem when the *p*H was kept high. The argument presented by Ream *et al.* in this issue supports this position preferring a high *p*H and low P_{CO_2} when body temperature is lowered. As Rahn *et al.*[4] have proposed, this situation maintains constant ionic charges on active protein molecules, thus possibly optimizing enzyme function.

Several theoretic objections to the universal acceptance of this proposal exist. The first deals with changes in cerebral blood flow. As temperature decreases, the cerebral blood flow decreases. If the P_{CO_2} also is lowered, an even greater fall in cerebral flow occurs, and cerebral ischemia might result. Hägerdal *et al.*,[5] however, showed that the cerebral metabolic rate decreased with hypothermia and there was no evidence of tissue hypoxia even with decreases in cerebral blood flow secondary to hypocarbia. They still recommended, however, that hyperventilation not be used in hypothermic patients with cerebrovascular disease. This concern with adequate perfusion of the brain is valid, but the evidence suggests that the response of cerebral blood flow to P_{CO_2} is shifted appropriately with temperature. Cerebral ischemia is therefore unlikely when the uncorrected *p*H and P_{CO_2} values are normal.

Another argument against using uncorrected values as a guide concerns changes in the oxyhemoglobin dissociation curve with temperature and *p*H. As the tem-

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perature decreases, the curve shifts to the left. The same shift occurs with an increase in the *p*H. The two are additive, so that a combination of low body temperature and high *p*H might seem to be deleterious to tissue oxygen supply. This also seems a valid concern, but evidence of problems with tissue oxygenation have not been advanced and it appears equally likely that offsetting changes in hemoglobin affinity compensate for the apparent shifts in the dissociation curve.

Optimal acid–base management during hypothermia still is unsettled. By drawing attention to the investigations of Rahn and Reeves, Ream *et al.* have opened a new way to investigate the problem. From a biochemical viewpoint, there is a strong argument that the appropriate *p*H and P_{CO_2} are those that give normal values when the blood sample has been anaerobically rewarmed to 37°C. Whether this also is relevant from a physiologic standpoint is not clear, but studies of regional blood flow, oxygen utilization, or neuromuscular function can now be examined when compared with the biochemical setpoint for a given experimental temperature. On this basis, during hypothermia, a low P_{CO_2} and high *p*H appear to be reasonable, but further investigation is necessary to show that this biochemical fact is indeed correct physiologically and clinically.

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