3. CURRENT CLINICAL PROBLEMS IN INTERPRETING ELECTROLYTES, ACID–BASE & METABOLITES

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Interpretation of acid-base disturbances and the effect of these on electrolytes are one of the mainstays of clinical practice within a critical care unit. The traditional model for interpretation was devised following work by Sigg-aard-Anderson using nomograms and equations developed by Henderson and Hasselbach, names that should be recognized by the majority of medical students. With a reasonable grasp of chemistry and mathematics this model allowed most physicians to assess the acid-base status of a patient and understand the basics of the interplay of pCO2, bicarbonate and hydrogen ions.

Some of the conclusions drawn using this method of interpretation have permeated medicine and critical care for many years and continue to do so. For example most physicians strongly associate acidosis (1), lactate (2) and lactic acid with a poor prognosis and many of our current treatment strategies are based on these assumptions (3). More recently this dogma has been questioned. Acidosis itself does not appear to be bad for you during exercise so why is it such an issue in critical illness? Despite much consternation there is no evidence to link hyperchloramic acidosis with any pathological consequences (4) and lac- tate may not be harmful but rather have beneficial effects on glycolysis and the Kreb's cycle especially when oxygen delivery is restored (5).

There has recently been renewed interest in the model used to examine acid-base changes with many clinicians adopting Stewart's acid-base theorem (6). This helps explain some of the conundrums of the traditional interpretation of acid–base problems such as hyperchloraemic acidosis but it is much more complex, making it's use at the bedside difficult, and some of the theory does not tally with clinical practice especially in relation to lactate pathophysiology. Some recent authors have suggested that maybe a combination of the two techniques is the optimal way to interpret acid–base physiology (7).

Maybe the problem is not in our assessment of the interplay between the various dependent and independent factors at work in acid–base chemistry, rather it is our limited ability to identify causation. When you are critically unwell you may have a complex combination of both local ischaemia and global hypoperfusion, both of which are interrelated. Lactate levels will rise due to increasing anaerobic metabolism and reduced efficiency of lactate shuttles. Fluid resuscitation is an important part of treatment in these situations but is complicated by many common resuscitation fluids containing exogenous lactate as a buffer. Simply removing this lactate and replacing it with a buffer we know less about (such as acetate) may not make this clearer.

The continuing controversy's in acid–base pathophysiology will be related to the identification of the acids driving the increase in hydrogen ion production and the mechanisms that produce them in critical illness. Hopefully by focusing on these processes instead of the treatment of acidaemia per se, mortality and morbidity will reduce.

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