4.4.2. Lactate

Lactate has, for decades, been the most popular metabolizable anion in a wide variety of infusion fluids, in particular Ringer's lactate (Hartmann's solution).

A number of considerations argue against the use of lactate, especially in patients with pre-existing elevated plasma lactate concentrations (lactic acidosis):

Lactic acidosis is a manifestation of disproportionate tissue lactate formation in relation to potentially impaired hepatic lactate metabolism. It makes no sense to further increase oxygen consumption in a patient with pre-existing tissue hypoxia. In a patient with lactic acidosis, Ringer's lactate will invariably exacerbate pre-existing acidosis by producing dilutional acidosis; unnecessarily increase the risk of rebound alkalosis; and preclude the diagnostic use of lactate as an important marker of hypoxia.

These considerations will be discussed in more detail below, making comparisons with acetate where appropriate.

Lactate Metabolism

At the basal metabolic rate (BMR), the myocardium, muscle, brain, intestinal mucosa, and red blood cells produce approximately 1 mmol of lactate/kg/h, and more than half of it is eliminated by the liver [20, 32, 89].

$$CH_3\text{-}CHOH\text{-}COONa + 3\ O_2 \rightleftharpoons 2\ CO_2 + 2\ H_2O + NaHCO_3$$

At the BMR, gluconeogenesis accounts for approximately 20 % and oxidation for approximately 80 % of lactate metabolism [20]. When lactate is supplied exogenously, up to 70 % of lactate can be used as a substrate for gluconeogenesis [139].

Intrahepatic gluconeogenesis ceases once pH falls below 7.1, or a BE of –15 mmol/L [15, 64]. Incipient hepatic dysfunction (increases in bilirubin and SGOT) quickly results in lactate concentrations as high as 8 mmol/L, which are associated with very high mortality [34]. Compared with acetate, lactate infusion is characterized by a relatively slow onset of alkalization and, therefore, has been called "delayed HCO_3^- infusion" [28]. Peak lactate turnover has been reported to be approximately 450 mmol/h [30]. As glucose levels may increase quite significantly after lactate administration [2, 8, 171], it comes as no surprise that intraoperatively administered Ringer's lactate may cause glucose concentrations to double in diabetics [171].

The D-lactic acidosis issue is not covered here because in Europe only physiological L-lactate is used, whereas racemic lactate (D and L) is traditionally used in the United States [176].

Does Lactate Increase Oxygen Consumption?

Oxygen consumption in laboratory animals increased very rapidly after the administration of lactate [6, 16]. Similarly, healthy volunteers given a bolus of 330 mmol of lactate showed an increase in O_2 consumption by almost 30 %, and this was mainly due to an increase in hepatic (almost 30 %) and muscle oxygen consumption (over 40 %) [2].

Lactate Clearance

The rate of lactate metabolism – above all in the liver – has become a major criterion for evaluating the therapeutic management of critically ill patients [1, 44, 52, 77, 121, 177]: "Changes in lactate concentration can provide an early and objective evaluation of the patient's response to therapy" [177].