Lactate, blood pressure and infection: tied by faith, untied by man?

"Diseases are conceptually all alike, although each syndrome is cumbersome in its own way". This paraphrase of the beginning sentence of Tolstoi’s *Anna Kareninna* emphasizes the complexity of dealing with a kaleidoscopic mix of signs and symptoms and their interplay. Syndromes are a mainstay of critical care medicine; no other medical specialty deals more regularly with syndromes than intensive care unit (ICU) physicians. Critical illness itself could be considered a syndrome, regardless of its etiology.

Shock is one of the most fascinating syndromes described. Shock was first described in almost philosophical reports starting in the early nineteenth century, then passed through the "decades of measurement" when it was first studied on physiological grounds, and finally arrived at an age when simple, practical concepts are frequently employed to facilitate patient grouping and prognostication (Figure 1). This current approach to medicine should theoretically hasten the development of new therapies, but it can also result in categorization that ignores the initial philosophical and physiological concepts of a syndrome. The same process has occurred with many other syndromes (sepsis, acute respiratory distress syndrome, etc.).

Sepsis and shock are interrelated syndromes. As early as 1868, Edwin Morris wrote that "Thus it would seem that shock and putrid infection, meet hounds of death, hunted this unfortunate man, as it were, in couples: shock held him down while pyaemia fastened her poisoned fangs". In this context, the work presented by Ranzani et al. in this issue of *Revista Brasileira de Terapia Intensiva* is a breath of fresh air that may help us remember both the philosophical and physiological components of the interplay between shock and sepsis, as well as why we should always revisit the criteria used to diagnose a given syndrome. After categorizing a large sample of patients into four groups according to lactate levels and the presence of hypotension, the authors concluded that dysoxic shock (defined as both hypotension and hyperlactatemia) presented with higher mortality. In addition, patients with cryptic shock (i.e., normal blood pressure and high lactate levels) and vasoplegic shock presented with an intermediate mortality between patients with severe sepsis and dysoxic shock.

Several important conclusions from this study highlight the relevance of this work. Most importantly, these findings stress that one should not expect a clear association between hypotension and hyperlactatemia. Even cardiac
output, which is the driving force determining blood pressure, may be unrelated to lactate levels. Moreover, both hypotension and hyperlactatemia reflect an unhinging of the body’s machinery, according to Gross. The role of lactate is much more complex, but it is now clear that anaerobiosis is not the only mechanism involved, as microcirculation disorders and malfunctioning of the biochemical apparatus of the cell have also been shown to be important. Therefore, because these phenomena have a unique physiological background, it is expected that their associations with outcome will be independent, and Ranzani’s work corroborates this concept.

Finally, the authors present extremely relevant findings regarding the epidemiology and prognosis of severe sepsis in our country. It is important to emphasize that the mortality rates due to severe sepsis and cryptic shock (16.8% and 35.2%, respectively) were similar to reports from developed countries. This finding suggests that when widely accepted practices are applied, positive results can be obtained irrespective of the hemisphere where the patient is treated.

REFERENCES


Figure 1 - Concepts in shock.