

Book report

Stewart's Textbook of Acid-Base, 2nd edition

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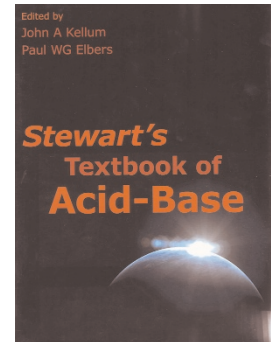
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Stewart's Textbook of Acid-Base. 2nd edition. Edited by Kellum JA, Elbers PWG. Lulu Enterprises, UK Ltd; 2009. ISBN-10: 1409254704; ISBN-13: 978-1409254706. 504 pages.



This multi-author textbook provides a comprehensive understanding of the Stewart approach to acid-base. The book consists of 30 chapters, grouped under three sections. Section 1 is a reproduction of Peter Stewart's original work (9 chapters), section 2 is a detailed primer on quantitative acid-base chemistry (8 chapters), and the third section deals with the clinical applications of the Stewart approach (13 chapters).

Retaining Stewart's original masterly work represents one of the highlights of the book. In the new section, the coverage is broad, and certain chapters such as those on intracellular [H], buffers, and the use of the Stewart model at the bedside are exceedingly well written. The chapters on the Stewart approach during pregnancy and a review of comparative animal physiology are useful additions.

However, criticisms can be made. Important errors of fact have slipped through the proofreading process, along with some controversial assertions. To illustrate:

Table 13.1: The normal plasma ionized calcium concentration is reported as 1 mEq/L, which is equivalent to 0.5 mmol/L. The normal plasma ionized calcium concentration ranges from 1.1 to 1.3 mmol/L. Similarly, the generic intracellular fluid (ICF) calcium concentration is reported as 35 mEq/L, which is equivalent to 17.5 mmol/L. This is exceedingly high. The intracellular [Ca] is of the order of nanomoles per litre.

Table 21.1: The strong ion difference (SID) of gelofusine is reported as 30, whereas the correct value is 34 (manufacturer's data).

Table 28.2: The SID of 5% albumin is reported as 0. The SID of 4% albumin is 12. The SID of 5% albumin is unlikely to be 0, which would result in a highly acidic solution.

Section 27.4: It is suggested that elevated lactate from catecholamines results from increased Krebs cycle activity. It is actually due to accelerated glycolysis. In this section, it is also suggested that reliance on base excess as a resuscitation target in patients receiving catecholamines may lead to an inappropriate diagnosis of hypoperfusion, as base deficits can result purely from catecholamine-induced hyperlactatemia. Similar errors can result from the use of strong ion gap (SIG) (when lactate is not included in the calculation). Finally, it is asserted that standard base excess (SBE) is unstable as partial pressure of carbon dioxide (PCO₂) changes. SBE, unlike actual base excess (ABE), is extremely stable over wide ranges of PCO₂.

Errors and controversial statements such as these might pose a problem to 'Stewart naïve' doctors, the targeted readership. Moreover, the book lacks an index. Whilst this was a deliberate editorial choice, it is my belief that for a quick search for a topic or an equation, an index would have been useful.

An appendix at the end summarizing all of the equations also would have been useful. For example, there are two different versions of the corrected anion gap (AG) equation (presumably because of usage of different units) on pages 324 and 340 and two different equations for SIG on pages 324 and 344.

These criticisms aside, the book on the whole makes excellent reading and is a good value for the money (€69.95). To the many who perceive the Stewart approach as a complex mathematical minefield (particularly those in whose minds the traditional acid-base approaches are deeply entrenched and sacrosanct), I would recommend this book as essential reading. In every chapter, the subject matter is

PCO₂ = partial pressure of carbon dioxide; SBE = standard base excess; SID = strong ion difference; SIG = strong ion gap.

dealt with systematically from the basics to the final conclusion without telescoping any steps at any stage. This book is a valuable addition to every medical library.

Competing interests

The author declares that they have no competing interests.