Blood Gases in Clinical Practice. By L. LAPUERTA. Springfield, Ill., Charles C Thomas, 1976. Pages: 117. Price: $12.50.

This book is described as a simple and basic aid to respiratory therapists, residents, and physicians. Absence of the "confusing barrage of theory" frequently seen in textbooks is emphasized. Regrettably, the author has failed completely to attain his stated goal. The number of factual and conceptual errors in the 110 pages of text is too great to catalog individually. One of the most serious errors occurs as a consequence of an attempt to simplify the Henderson–Hasselbalch equation. The resulting statement suggesting that *p*H is approximately equal to the quotient of bicarbonate divided by P_{CO_2} (*sic*) will leave the uninitiated believing that normal *p*H should be about 0.6 (24 mEq/l/40 mm Hg) and wondering why the value 7.4 recurs throughout the text. All health professionals for whom this material is intended must surely have sufficient basic preparation in physical and biological sciences so that it should not be necessary to attempt to simplify concepts to the point where they become meaningless. Much of the text is devoted to discussions of respiratory failure in various circumstances. These are not very helpful. We are given no objective criteria for diagnosis of respiratory failure or for institution and discontinuation of artificial ventilation, but only admonished to use our good clinical judgment. Illustrations are few, and some graphs are difficult to interpret because of failure to label ordinate and/or abscissa. Repeated errors in grammar and syntax are distracting. Many symbols used are not in accord with standard usage.

This book is likely to be extremely confusing for the beginner, and offers nothing for the individual already conversant with the simplest principles of acid–base and blood-gas physiology. It cannot be recommended for use by health professionals at any level.

NORMAN A. BERGMAN, M.D.
Department of Anesthesiology
University of Oregon Medical School
Portland, Oregon 97201

Literature Briefs

Peter J. Cohen, M.D., Editor

Literature briefs were supplied by P. J. Cohen, M.D. Briefs appearing elsewhere in this issue are part of this column.

Theories of Anesthesia

NALOXONE ANTAGONIZES N_2O The analgesic effects of N_2O were tested in mice by the use of an intraperitoneal injection of phenylquinone that normally causes "writhing." After administration of phenylquinone the mice were placed in a clear plastic enclosure and exposed to various mixtures of N_2O and O_2. N_2O produced a dose-related inhibition of writhing, with an ED_{50} of 55 per cent N_2O. Injection of naloxone had no effect on the incidence of writhing. However, pretreatment with naloxone significantly reduced the analgesia produced by N_2O. In treated animals, 55 per cent N_2O produced no analgesia. While control animals exposed to 80 per cent N_2O had 84 ± 6 per cent analgesia, naloxone-treated mice showed only 37 ± 4 per cent analgesia. The authors conclude that either narcotic antagonists have analgesic antagonist properties not previously identified or nitrous oxide "releases or potentiates an endogenous analgesic or opiate within the central nervous system." (*Berkowitz BA, Ngai SH, Finck AD: Nitrous oxide "analgesia": Resemblance to opiate action. Science 194:967–968, 1976.*)

Respiratory Physiology

HYPOXIC RESPIRATORY DRIVE It is well known that hypoxic ventilatory drive (HVD) is markedly diminished in man born and raised at high altitude. How early does this abnormality develop and is it reversible? The authors examined HVD in eight children and young adults (aged 7–16 years) who had cyanotic congenital heart disease (arterial oxygen saturation 55–83 per cent). A similar study was done in another group of 13 subjects (aged 7–17) whose cardiac defects had been repaired. HVD was blunted in the cyanotic group, indicating that this alteration could appear in children after as little as seven years of hypoxia. The diminution of sensitivity varied directly with the magnitude of chronic arterial hypoxia. The ventilatory responses were normal in all 13 subjects who had undergone repair of their cardiac lesions, demonstrating that this abnormality is reversible. These results differ from those of other investigators, who have reported continued blunting of HVD after repair of the cardiac lesion. The authors attribute part of these differences to persistence of arterial hypoxemia in some subjects in the latter study. (*Blesa MI, and others: Normalization of the blunted ventilatory response to acute hypoxia in congenital cyanotic heart disease. N Engl J Med 296: 237–241, 1977.*)