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The Anesthesiologist's Bookshelf

Clinical Application of Blood Gases. By B. A. SHAPIRO. Chicago, Year Book Medical Publishers, 1973. Pp. 210, $9.95.

According to the preface of this small monograph " . . . the clinical conclusion surrounding blood gases no longer exists if the physiology in Part I is understood and if the concepts and terminology in Parts II and III are accepted." Alas, that is a big order.

The growth of intensive care has witnessed an explosive expansion in the quality of monitoring techniques. Fifteen years ago an arterial puncture for blood-gas measurements was considered assault and battery. Today, a major surgical procedure without the benefit of an inlying radial-artery cannula is viewed as less-than-optimal care. The change in attitude has been caused by reorientation in laboratory services toward rapid analysis and quick return of results. Abnormal values are recognized early and appropriate therapy can be instituted forthwith. For the service to be successful, quality control must be carefully maintained; the technician must be aware of the potential consequences to the patient if an error of calibration is reported as a valid answer. This has added a new element of responsibility to the laboratory supervisor. It is not enough to teach how instruments are used; the meaning of the measurements must be clarified if potential disasters are to be prevented. To this end, a basic explanatory text is sorely needed. I do believe that the author had this purpose in mind. Unfortunately, the desire to simplify has resulted in a less-than-successful book, and it is difficult to give this text a passing grade, as there is little justification for a presentation replete with deficiencies. Reduced hemoglobin is expressed as "Hb_{CO_2}," 2,3-DPG is called an enzyme, CO_2-combining power is considered to be a measurement of standard bicarbonate, alveoli at the bases of the lung are said to be smaller secondary to capillary congestion, the Laplace relationship is misstated, the presence of carboxyhemoglobin is equated with the effects of anemia, and the need for temperature correction of blood gases is ignored.

Definition of clinical entities and evaluation of therapy are very much a matter of personal taste. I do not agree that the "single most direct and useful blood gas measurement is the arterial P_{CO_2}," unless one focuses exclusively on chronic pulmonary disease, and I submit that availability of the P_{O_2} electrode has revolutionized our approach to respiratory failure. The rationale for defining *acute ventilatory failure* as a condition characterized by a high Pa_{CO_2} and acidemia, while defining *acute ventilatory insufficiency* as the presence of alveolar hyperventilation with alkalemia (*e.g.*, hyperventilation secondary to neurosis or psychosis is considered a form of acute ventilatory insufficiency), escapes me.

The physiologic consequences of hypoxemia secondary to acute pulmonary disease are not easily definable. To conclude that oxygen therapy alone can "effectively support many disease states by preventing increased myocardial work" is wishful thinking, since hypoxemia is a notoriously poor stimulus for increased cardiac output in the sick patient. In fact, misunderstanding on this point has led many to the erroneous conclusion that provision of an FI_{O_2} sufficient to return Pa_{O_2} to normal is curative. Acute respiratory failure, characterized by inappropriate oxygenation, is invariably a combination of inadequate lung and heart function. Measurement of arterial blood gases tells us something about the lungs and very little about the heart until metabolic acidosis ensues. Except for the patient with heart disease, during acute illness cardiac output rarely if ever rises to the level we experience climbing a flight of stairs. Were it to do so chronically, even in the trained individual, then the consequences of a lowered P_{O_2} would be tolerable without the benefit of a ventilator or an increased FI_{O_2}. Our problem is compounded further by the evaluation of oxygen consumption during acute illness, when V_{O_2} is most likely to be elevated.

It is unfortunate that what began as a commendable idea resulted in an unrecommendable product. The basic ingredients for success are there and perhaps deserving of another effort in the future.

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The Biochemical Basis of Neuropharmacology. Second edition. EDITED BY J. R. COOPER, F. E. BLOOM, AND R. H. ROTH. New York, Oxford University Press, 1974. Pages: 272 (paperback). Price: $4.94.

The authors of this already well-known textbook have managed to produce a refreshingly readable and concise work which broadly summarizes the complex and often confusing field of neuropharmacology and neurotransmitters. The chapters are organized as a series of vignettes of each class of neurotransmitter, acetylcholine, catecholamines, serotonin, the amino acids, based on the probability that, with the exception of local anesthetics, all of the neuroactive drugs function at the synapse, usually by an interaction with the neurotransmitter. The new edition is enlarged in the areas of catecholamines, cyclic AMP, prostaglandins and the cholinergic receptor. One of its stronger points is the presentation of experimental techniques along with a critical evaluation of their reliability and sensitivity. The bibliography is limited to recent reviews and general articles, but references to the original sources would enable the reader to attempt a personal digestion of the massive information and appreciate to the full what an excellent job the authors have done.

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