physiology of fluid and electrolyte disorders of the neonate and infant and the rationale for therapy. It sharply illustrates how a delay between writing and distribution, and medical progress in the interim, can result in publication of well-meaning but unfortunate misinformation. As an example, under techniques of induced hypotensive anesthesia, hypovolemia is recommended as an adjunct. This can only be condemned in today's practice. A perusal of the extensive bibliography reveals that there are very few references as late as 1973.

On the positive side, there is an excellent discussion of pyloric stenosis. The best chapter in the book is concerned with fluid balance in infants receiving respiratory therapy. However, on the negative, there are several incomplete sentences (on pages 54 and 69). Symbols and abbreviations are used without explanation of what they stand for. There is no longer any justification for using stones, pounds, or any system of measurement other than the metric system. In Chapter 1 the neonate is defined as a child in the first 24 hours of life, and eight pages later, as a child as much as 28 days old. There are variable values given for the percentage of body weight of interstitial fluid volume. In the chapter on blood replacement, a 20 per cent blood loss is said to be "allowable if the clinical conditions so indicate," but no details of the clinical conditions are given. Another difficulty is that many of the sentences are awkward and often excessively long, and facts are often difficult to separate from opinion.

There is no question that there is a need for a book on fluids for the newborn and infant during anesthesia and operation. This book may be the first step in that direction. However, the lack of relatively recent references, the awkward and confusing literary style, and the many errors and inconsistencies in the text do not recommend it.

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# Literature Briefs

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*Literature Briefs were supplied by Drs. A. R. Boutros and P. J. Cohen. Briefs appearing elsewhere in this issue are part of this column.*

## Neuromuscular Physiology

**MYASTHENIA GRAVIS** Previous data developed by the authors indicate that myasthenia gravis results from a defect in the acetylcholine receptor. This may be caused by binding of an antibody to the receptor. In order to test this hypothesis, mice were injected daily (for 14 days) with an ammonium sulfate-precipitated aminoglobulin fraction of sera from patients with myasthenia gravis. Pooled blood from normal patients was used for control experiments. The average number of acetylcholine receptors per neuromuscular junction was reduced by 42 per cent in the experimental animals. These mice also showed reduced amplitude of miniature endplate potentials. Some of the animals showed decremental responses with repetitive nerve stimulation, a finding reversed by neostigmine. The authors believe that "this represents the first evidence of a circulating factor in the serum of patients with myasthenia gravis which on passive transfer reproduces features of the disease in experimental animals." (*Toyka KV, and others: Myasthenia gravis: Passive transfer from man to mouse. Science 190:397–399, 1975.*)

## Hepatic Function

**HALOTHANE AND FETAL LIVER** Eight pregnant rats were exposed to 10 ppm halothane 8 hours a day and 5 days per week throughout pregnancy. Control animals were housed in adjoining chambers. Twenty-four hours after delivery, electron micrographs were made of the livers of four randomly picked infants from each litter. The histology of the control liver was normal. On the other hand, degenerative changes (myelin-figure formation, focal cytoplasmic degradation), fatty changes and cellular necrosis were observed in the halothane-exposed animals. Since the concentration of halothane was similar to that found in ambient air of an operating room, the authors suggest that this may represent an occupational hazard to operating room personnel. (*Chang LW, and others: Ultrastructural evidence of the hepatotoxic effect of halothane in rats following in-utero exposure. Canad Anaesth Soc J 22:330–338, 1975.*) ABSTRACTER'S COMMENT: Until other agents are studied, there is no reason to claim that fetal hepatotoxicity is specific for halothane.