when arterial blood pH is lowered by the addition of carbon dioxide to the oxygenator respiratory mixture. Recalculation of their data is not possible since $P_{CO_2}$ values were not included.

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References
1. Stadie, W. C., and Martin, K. A.: The thermodynamic relations of the oxygen- and base-

Effect of Cyclopropane and Halothane on the Blood Volume

To the Editor.—I read with interest and concern the article entitled: “The Effect of Cyclopropane and Halothane on the Blood Volume in Man” by Ernest Grable and co-workers, which appeared in Anesthesiology 23: 828, 1962. There are several points I wish to bring out which may be of interest to readers of the above mentioned article.


(2) It is most advisable for anyone who wishes to measure blood volume and in particular when interpreting plasma volume, to read and understand what is being measured. One actually tries to interpret the protein distribution space as plasma volume—a noteworthy misconception.


(3) I wish to congratulate the authors on having mastered the use of iodine$^{125}$ albumin. Over a period of six months we tried to use this radioactive isotope of iodine, generously supplied by E. R. Squibb & Sons, and after many in vitro trials and repeated consultations with colleagues versed in this subject, we have come to the conclusion that I$^{125}$, a very weak gamma emitter (35.4 kev.), is absorbed by chromium$^{51}$, red cells absorb part of the energy—even protein concentration in the solution alters the accuracy in measuring I$^{125}$ concentrations and, therefore, a word of caution is in order on the use of this tracer.

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To the Editor.—The authors appreciate Dr. Albert’s interest in our study. We are further indebted to him for pointing out a typographical error that crept into our manuscript. The sentence referred to should read “Albert... found an increase in blood volume during hypotension caused by the induction of thiopental anesthesia.”

The question of the validity of a plasma tag method for measuring blood volume is too complex to be reviewed in a letter to the editor. During the past two years, we have made over 4,000 blood volume measurements with $^{131}$-albumin and the Volemetron. The reproducibility of the data and the accuracy with which in vivo additions or subtractions of known amounts of blood, plasma, or red cells are recorded are unquestionable.

Our measurements of plasma volume with $^{125}$-albumin were made on samples of cell-free plasma (not whole blood), and in no instance was any trace of Cr$^{51}$ (which would, of course, cause spuriously high counting rates) detected in these samples. The most important factor in obviating error from this source when Cr$^{51}$-RBC and $^{125}$-albumin are used simultaneously is careful, repeated saline “rinsing” of the Cr$^{51}$-RBC tracer doses to remove all Cr$^{51}$ not bound within the red cells. Since $^{125}$ is a low energy gamma emitter (0.027–0.034 Mev.), the instrument used for its assay must have an exceptionally stable high voltage supply and discriminator level. Sample tube dimensions and counting geometry must be calibrated and rigidly standardized. Our experiences with the Volemetron have shown that when these requirements are met $^{125}$-albumin yields the same value for plasma volume as does $^{131}$-albumin, and simultaneous determinations of red cell mass and plasma volume are accurate and relatively simple procedures.

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Correction

To the Editor.—We would like to point out a small typographical error appearing in the last paragraph of our Work in Progress Abstract in the January-February 1963 issue of Anesthesiology (Atrial Activity During Halothane Anesthesia in Man, page 133).

The particular section should have read: “... that it is most likely secondary to sympathetic activity superimposed on the myocardial effect of the anesthetic, ...” instead of parasympathetic as in the printed version.

Admittedly, our criteria for defining the type of autonomic stimulus are highly circumstantial, and one can easily argue for each of these factors as the responsible agent. However, the bulk of the experimental and clinical evidence points to sympathetic innervation as the most likely source and would be less objectionable as a theory from the manner in which we have stated our case.

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