systematically low value for \( P_{O_2} \) measured by a spectrophotometric method that necessitates hemolysis of the blood, such as the cooximeter.

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REFERENCES

1. Theye RA: Calculation of blood \( O_2 \) content from optically determined \( Hb \) and \( HbO_2 \). ANESTHESIOLOGY 33:653, 1970

To the Editor.—Dr Nahas is correct in pointing out that many previous reports have indicated that spectrophotometric techniques yield slightly higher (0.5–1.5 per cent) values for \( HbO_2 \) than those obtained by the Van Slyke method. These reports confirm the suggestion that uncertainties are involved in calculation of blood \( O_2 \) content from optically determined values for \( Hb \) and \( HbO_2 \) and reinforce the point that any indirect approach to blood \( O_2 \) content must be validated by comparison with the direct method of Van Slyke. Dr. Nahas is also correct in suggesting that if in-vitro methods of handling blood result in significant reductions in levels of 2,3-DPG the affinity of hemoglobin for \( O_2 \) may be increased. This point, however, is not considered particularly relevant to the current discussion, since in my studies all in-vitro handling of blood was carried out without access to a gas phase, thereby precluding opportunity for significant change in \( HbO_2 \) resulting from increased affinity of hemoglobin for \( O_2 \). Furthermore, a sizable number of determinations were carried out at initial \( P_{O_2} \) values exceeding 300 mm Hg which, presumably, results in complete satisfaction of the capacity of \( Hb \) to combine with \( O_2 \).

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Cutaneous Blood Flow with Ether

To the Editor.—In attempting to provide a unitary explanation of the effects of ether, Dr. Gregory and his co-workers (ANESTHESIOLOGY 34: 19–24, 1971) noted that the cardiovascular changes with time are compatible with beta-adrenergic activation, presumably through release of catecholamines or sensitization of the beta receptors.

One of the most striking changes observed was the large increase in skin blood flow. This can be explained only by a decrease of existing alpha-adrenergic activity. Even release from anxiety or loss of temperature control would be mediated through alpha-adrenergic mechanisms. It is difficult to reconcile the concept, as proposed, that ether activates the beta-adrenergic system on one hand and decreases alpha-adrenergic activity on the other.

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To the Editor.—As Dr. Wiklund points out, control of skin blood flow is primarily alpha- and not beta-adrenergic. If, as he proposes, however, the effects were due to a decrease in alpha-adrenergic activity only, then total peripheral resistance and blood pressure should