It is, in fact, apparent that Schwartz and his colleagues on the one hand, and Astrup and his associates on the other, agree on the physicochemical events which occur during alterations in carbon dioxide tension. The difference is related to how to describe these events. The Astrup school has adopted a nomenclature including something called base excess (or deficit) which reflects (1) alterations in the distribution of bicarbonate across cell membranes, and which may be entirely independent of any alterations in non-volatile acids or base and, (2) alterations in the concentrations in blood of non-volatile acids or bases. The measurement of base excess, accordingly, does *not* tell which is going on. It certainly does *not* justify the therapeutic implications which have been commonly adopted. It *has* served as a source of widespread confusion, in short, a cruel hoax on the physician and student who have tried to understand acid-base balance in physiologic terms. Schwartz and his associates have presented a readily understood physiologic approach which should form the basis for a much wider understanding of the nature of acid-base disturbances.

Drs. Siggaard-Andersen and Engel correctly state that Shaw and Messer were aware that an elevation in P_{CO_2} is accompanied by a shift in bicarbonate from blood to tissue. Siggaard-Andersen also reported, in 1962, a small difference between *in vivo* and *in vitro* titration curves, to which he attributed little importance. Schwartz claimed no priority for the idea, although he has, of course, provided to date by far the most complete data. The issue is rather the importance of the difference between acid-base as studied in blood and that of intact man, which remained for Schwartz and Relman to put forward.

The report of the conference on acid-base terminology and interpretation from the New York Academy of Medicine does not, in my opinion, clarify the issues, but rather solidifies the conflicting interpretations. Likewise further debate, which I myself have encouraged, is less apt to resolve the issues than are new data. The current report by E. B. Brown, Jr. and R. L. Clancy (J. Appl. Physiol. 20, 885, 1965) confirms the *in vivo* titration curves of Schwartz and his colleagues and supports their interpretations.

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Oxygen Tension Temperature Factor

To the Editor.—Dr. Hedley-Whyte and his co-workers [1] believe that, to measure arterial oxygen tension when hemoglobin is not fully saturated, the oxygen electrode must be kept at the temperature of the patient. Few would deny the desirability of this approach but our experience of maintaining a 24-hour service for measurement of blood P_{O_2} suggests that this is frequently impracticable. We believe, furthermore, that a satisfactory correction factor may now be applied for partially desaturated blood.[2] At full saturation our measurements of the thermal coefficient of P_{O_2} agree precisely with those of Hedley-Whyte and Laver,[3] and in partially desaturated blood, the coefficient rises towards the value calculated for desaturated blood by Bradley *et al.*[4] Our measured value approached the calculated value at a saturation of about 90 per cent, at which level the change in dissolved oxygen is of little importance.

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Percentage Saturation of Hemoglobin	Exponential Factor for Thermal Coefficient	Percentage Change for 1°C
100	0.005	1.2
99	0.013	2.9
97	0.021	4.8
90	0.028	6.3

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To the Editor.—Professor Nunn states that as a result of his recent work, a satisfactory oxygen tension temperature correction factor may now be applied for partially desaturated blood; for fully saturated blood his results confirm our earlier findings. Unfortunately, the practical use of Nunn's factors is limited by the fact that a base excess or deficit will affect the hemoglobin dissociation curve. Nunn's experimental design did not take into account these nonrespiratory acid-base changes. At tensions above full saturation of hemoglobin this problem does not occur and our nomogram will provide accurate correction for the effect of temperature on oxygen tensions.[1] We have discussed this problem more extensively in our recent book,[2] moreover Severinghaus has recently included the influence of base excess or deficit in his "Blood gas temperature correction slide rule." [3] If no nonrespiratory acid-base changes are present, as Professor Nunn states in his letter, his factors are acceptable for clinical use when it is impossible to maintain oxygen electrodes accurately calibrated at the temperature of the patient, and if the oxygen dissociation curve is *accurately known.* Unfortunately, wide variations from patient to patient may occur in the top end of the dissociation curve.

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Correction

To the Editor.—An error appeared in the article "Splanchnic Circulation During Cyclopropane Anesthesia" (ANESTHESIOLOGY 26: 312 (May–June) 1965) on page 316, right-hand column, line 27. The phrase ". . . involved in the response has been shown to . . ." should be ". . . has *not* been shown to . . ."

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AORTIC REGURGITATION Aortic regurgitation due to prolapse of one or more of the aortic leaflets is a less widely appreciated complication of ventricular septal defect and the combination of the two produces a typical clinical picture. The resultant aortic regurgitation imposes an additional hemodynamic burden upon the left ventricle, usually progressive in severity and often leading to left ventricular failure. Operative correction of the combined lesion may become imperative. (*Braunwald, E., and others: Ventricular Septal Defect and Aortic Regurgitation, Amer. J. Med. 39: 552 (Oct.) 1965.*)