

Physiol. 20: 885, 1965; Brackett, N. C., Cohen, J. J. and Schwartz, W. B.: J. Clin. Invest. 43: 777, 1964 and New Eng. J. Med. 272: 6. 1965). One is led to wonder why the obvious difference in these slopes remained undetected during the years when several approaches to quantitation of the nonrespiratory component of acid-base disturbances were being presented (Singer, R. B., and Hastings, A. B.: Medicine 27: 223, 1948; Astrup, P., and others: Lancet 1: 1035, 1960).

The paper by Shock and Hastings (J. Biol. Chem. 112: 239, 1935) is commonly referred to as demonstrating that there is no difference between the *in vitro* and *in vivo* slopes. Astrup, Anderson, Jorgenson and Ingle refer to this paper as follows: "The effect of equilibrating whole blood seems to be approximately the same *in vitro* as *in vivo* (Shock and Hastings, 1935)—i.e., as if the respiratory function was standardized." When the data of Shock and Hastings are examined it is found that the *in vivo* buffer curves of normal subjects were compared with an *in vitro* curve drawn from L. J. Henderson's data, (Blood. New Haven, Yale University Press, 1928). Apparently their *in vitro* curve was constructed from the data presented by Henderson on page 127, since the slope of a curve drawn from these data is identical with that in Shock and Hastings' figures. On page 124, Dr. Henderson indicates the source of the blood used for construction of this table. He says, "For the present purpose, we have at our disposal data concerning these curves and other necessary data obtained from studies of the blood of TJF, a patient suffering from pernicious anemia. The measurements were all made on one day, March 23, 1927, at a

time when the feeding of liver during a period of two months had restored the blood to a nearly normal condition." He goes on to say that the amount of hemoglobin was in the low range of normal and the red cells were indeed somewhat different from those of normal men. At any rate, the *in vitro* slope from the blood of this patient plotted as pH against bicarbonate was $-21.6 \text{ mM HCO}_3/\text{liter/pH}$. It can be calculated from the empirical equation given by Van Slyke (Quantitative Clinical Chemistry, Vol. I. Baltimore, Williams and Wilkins, 1931, p. 912) that this slope equivalent to blood containing hemoglobin is approximately 10 g./100 ml. Blood with normal hemoglobin concentration has an *in vitro* slope of -28 to $-29 \text{ mM HCO}_3/\text{liter/pH}$. In other words, Shock and Hastings were comparing the slopes of *in vivo* buffer curves of normal humans with an *in vitro* slope that was only 75 per cent of normal. The fact that no difference in slope appeared was fortuitous.

It is also unfortunate that Davenport (A B of Acid-Base Chemistry. Chicago, University of Chicago Press, 1958) used the same source of data to establish the slope of his "normal buffer curve," and accordingly uses the value -21.6 as the slope of this curve throughout his monograph. On the other hand, perhaps it is fortunate that what is presented as an *in vitro* "normal buffer curve" accidentally has approximately the slope of the *in vivo* curve (Brown, E. B., Jr.: Ann. N. Y. Acad. Sci.: In press).

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Carbon Dioxide Tensions to Determine Adequacy of Ventilation

To the Editor:—A recent article by Slater and his associates (Arterial Oxygen Tension Measurements During Nitrous Oxide-Oxygen Anesthesia, ANESTHESIOLOGY 26: 642, 1965) revealed the difficulty encountered in maintaining adequate PaO_2 levels with high concentrations of nitrous oxide and constant tidal volumes for upper abdominal surgery. They

suggest that atelectasis may result from abdominal packing and retractors and that it might be eliminated by intermittent hyperinflation of the lungs.

In an earlier report (Markello, King, and Cutter: Hyperventilation Studies During Nitrous Oxide-Narcotic Relaxant Anesthesia, ANESTHESIOLOGY 24: 225, 1963, figure 1) we

showed that oxygenation of arterial blood could be well maintained using 80 per cent N₂O and constant tidal volumes of 500 ml. merely by increasing the rate of ventilation. Although arterial oxygen tensions were not measured, the progressive improvement of arterial saturation from preoperative control (91 per cent) to the end of anesthesia (98 per cent) indicated absence of atelectasis. These patients, too, all underwent upper abdominal surgery and were very similar to those reported by Slater. We also noted as did Slater that when Pa_{CO}₂ was maintained in the region of 30 mm. of mercury oxygenation of arterial blood could not be well maintained. This demonstrates the fallacy of using a nomogram or carbon dioxide tensions to determine adequacy of ventilation when 20 per cent oxygen is being used. We suggest therefore that moderate hyperventilation be routinely employed when high concentrations of N₂O are being used.

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To the Editor:—We would like to underscore Dr. Markello's comment on the fallacy of using "carbon dioxide tensions to determine adequacy of ventilation" not only when 20 per cent oxygen is used, but, as we have previously demonstrated, whenever a patient is anesthetized.^{1, 2} Furthermore, the vagaries of hemoglobin dissociation (depending on P_{CO}₂, pH or temperature) do not permit assessment of pulmonary function from a single arterial O₂ saturation reading. The mean values given by Markello, Cutler and King (ANESTHESIOLOGY 24: 225–230, 1963) for their patients were as follows (the corresponding arterial oxygen tensions were obtained from the Blood-Gas Calculator * designed by Dr. John W. Severinghaus, assuming a base excess of zero and a body temperature of 38° C.):

Hyperventilated Group				
	P _{CO} ₂ (mm. Hg)	pH	% O ₂ Sat.	P _O ₂ (mm. Hg)
Control	37	7.41	91	63
2 hours	15	7.63	94	57
End anesth.	15	7.63	98	91.5
15 minutes after ces- sation of hyper- ventilation	37	7.38	80	49.5

Nonhyperventilated Group				
	P _{CO} ₂ (mm. Hg)	pH	% O ₂ Sat.	P _O ₂ (mm. Hg)
Control	35	7.39	93	72.7
2 hours	35	7.37	97	103
End anesth.	34	7.38	97	102
15 minutes after ces- sation of controlled ventilation	41	7.32	87	63

The data demonstrate a rather interesting paradox: a fall in P_{CO}₂ (due to hyperventilation) and a rise in O₂ saturation despite a fall in arterial oxygen tension (marked shift of the dissociation curve to the left with a rise in pH). The authors do not specify the circumstances under which the two hour samples were taken. However, in the study by Slater *et al.* (ANESTHESIOLOGY 26: 642, 1965) samples drawn when the "intra-abdominal packs and retractors had been in position for 20 minutes" (patients being ventilated with 20 per cent oxygen) revealed a mean arterial P_O₂ of 58 mm. of mercury, a value in remarkable agreement with Markello, Cutler and King.

The tendency to use O₂ saturation for the assessment of pulmonary function during controlled ventilation has caused a great deal of confusion. In a sense O₂ saturation is an unfortunate choice since it does not reflect either the total amount of oxygen carried per unit volume of blood (O₂ content) or the partial pressure of oxygen (P_O₂) which is important in determining diffusion rates. Interpretation of O₂ saturation is also limited by variations in the position of the dissociation curve caused by massive transfusion with ACD blood or other systemic disorders.^{3, 4, 5}

With our present knowledge it is too early to imply that controlled ventilation with 20

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per cent oxygen during surgery is either dangerous or safe until we have a better definition of critical levels of oxygenation. We do believe, however, (1) that reliance on arterial O_2 saturation during controlled ventilation can lead to a sense of false security, (2) that ventilation during anesthesia with oxygen concentrations equivalent to ambient air may lead to P_{O_2} levels well below the values found in the awake state, and finally (3) that (as shown so nicely by Markello, Cutler and King) CO_2 removal can be achieved very readily without much change in the level of oxygen tension. These facts place the burden of proof squarely on the anesthetist who claims that his patient is adequately oxygenated.

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POSTHYSTERECTOMY BLEEDING Posthysterectomy bleeding sufficient to require transfusion of 500 ml. or more of blood occurred in 0.8 per cent of a series of 4,421 hysterectomies. Postoperative hemorrhage was more common in patients who had undergone vaginal hysterectomy. When hemorrhage began early (1-12 hours) following the vaginal procedure, it was more likely to be serious and to be from an intra-abdominal source. Abdominal exploration was required in about half of these patients with early postoperative hemorrhage. Late hemorrhage following vaginal hysterectomy was less serious, as was postoperative bleeding following abdominal hysterectomy. The source of bleeding was usually in the vaginal vault and could be controlled by packing or vaginal sutures. (Smith, R. D., and Pratt, J. H.: *Serious Bleeding Following Vaginal or Abdominal Hysterectomy*, *Obstet. Gynec.* 26: 592 (Oct.) 1965.)

INTENSIVE CARE UNIT Cross infection in a 4-bed intensive care unit in a 200-bed community hospital was responsible for a 10 per cent incidence of acquired staphylococcal infections. All patients now have nasal cultures on admission and all staff have weekly cultures. Topical antibiotics are used on all staphylococci carriers and the patient infection rate is now 2 per cent. (Crockett, G. S., and others: *An Intensive Care Unit: Two Years' Experience in a Provincial Hospital*, *Brit. Med. J.* 2: 1173 (Nov. 13) 1965.)