

whenever tenderness appeared. It was felt that this complication could be avoided by using a bag with an easily grippable surface and changing hands at suitable intervals.

Unguis Dominantis translated means "nail of the tyrant, despot, one who dominates." Originally, Dr. Dance planned to call the condition Controller's Nail, or *Unguis Controllensis*. However, it seems that French put together two words *contre* meaning counter and *role* meaning register and derived a word that basically means the register

kept at the counter or desk in a hotel. He believes both *contre* and *role* are originally of Latin origin, but apparently their usage together is strictly French. He consulted Latin scholars (Speech and Latin Departments, St. Joseph's College, Rensselaer, Indiana), and they informed him after much disputation that it would be inconsistent to use the words controller and unguis together. He then found the word *dominator* which seemed to approximate his meaning fairly closely. The Latin scholars checked the form and case endings of the phrase *Unguis Dominantis* and assured him this form was correct and appropriate.

CORRESPONDENCE

Metabolic Acidosis and Hyperventilation

To the Editor.—The title of the article "Metabolic Acidosis of Hyperventilation Produced by Controlled Respiration" by Drs. Papadopoulos and Keats [ANESTHESIOLOGY 20: 156, 1959], seems misleading. The observations of slight variable degrees of metabolic acidosis during anaesthesia with or without hyperventilation, hypoventilation, hyperthermia or hypo-

thermia are well known to those who have carried out blood studies on patients during anaesthesia. However, I cannot see how the

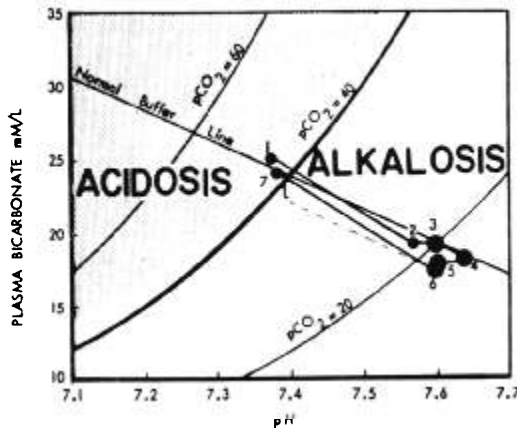


FIG. 1. Represents mean values and standard error of the means of the plasma bicarbonate, pH and pCO₂ from a variable number of observations: before anaesthesia (1), after 10 min. (2), after 1 hr. (3), after 2 hrs. (4), after 3 hrs. (5), after 4 hrs. (6), and post anaesthesia (7). These values are taken from the analysed arterial blood data reported by Drs. Papadopoulos and Keats, ANESTHESIOLOGY 20: 156, 1959, (tables 1 & 2). Observe that after 2 hrs. of hyperventilation there is no change in the plasma bicarbonate (corrected to pH 7.4), and that even after 4 hours of hyperventilation the change (difference of the means) is hardly more than would be expected in the normal daily variation.

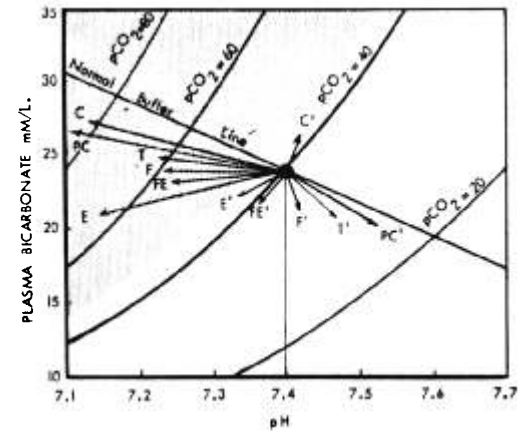


FIG. 2. Represents the usual mean values of plasma bicarbonate, pH and pCO₂ before premedication, and after a stable surgical plane of anaesthesia (patient in the supine horizontal position) with thiopental-d-tubocurarine-nitrous oxide (PC), trichloroethylene-nitrous oxide (T), Fluothane-nitrous oxide (F), Fluothane-ether azeotrope-nitrous oxide (FE), diethyl ether-nitrous oxide (E) and cyclopropane-oxygen (C). The vectors identified with a superscript (') represent the changes observed when pulmonary ventilation is controlled according to a predetermined tidal volume, pressure amplitude, phase ratio and rate of breathing. The other vectors represent the changes observed when ventilation is not adequately assisted or controlled, or when breathing is spontaneous. A non-breathing system or Mapleson A System (Magill attachment) with high gas flows (8 to 14 liters) were used to procure all these data except for cyclopropane-oxygen, for which the closed circle system was used.

ACID-BASE CHANGES ACCOMPANYING ALTERED VENTILATION

	pH	pCO ₂ mm. Hg	[HCO ₃ ⁻] _p mM./L.	Clinical and Anesthetic Conditions
Respiratory alkalosis	7.60 7.65	25 15	23.8 18.0	Fever Hyperventilation during anesthesia with non-volatile agents
Metabolic alkalosis	7.47	43	30.0	Vomiting, excess alkali ingestion (patient with peptic ulcer)
Metabolic alkalosis + respiratory acidosis	7.35	57	30.0	Pneumonia
Respiratory alkalosis + metabolic acidosis	7.48	20	14.0	Hyperventilation during anesthesia with volatile agents
Normal	7.37	40	22.0	Resting, unmedicated
Metabolic acidosis	7.26	38	16.3	Prolonged starvation, blood transfusions, diabetes, nephritis, Addison's disease, severe diarrhea, rewarming after hypothermia
Respiratory acidosis	7.18	70	25.0	Emphysema, hypoventilation during anesthesia with non-volatile agents
Metabolic acidosis + respiratory acidosis	7.20 7.10	57 65	20.0 20.0	Excessive premedication: morphine, meperidine, barbiturates, phenothiazines, etc. Hypoventilation during deep anesthesia with volatile agents, hypothermia

data in the above mentioned paper has shown this to be due to hyperventilation. In fact, a careful analysis of their data does not show any such significant change at all! Figure 1 shows the plot I made from their tabulated data on a pH-bicarbonate-pCO₂ graph. I have borne in mind that the reader cannot weigh their figures properly, because blood samples from different numbers of patients are averaged at each time interval. Moreover, there are only 4 or 5 patients on whom blood data are shown beyond one hour of anaesthesia.

In any case, the shift in acid-base equilibrium from their figures is almost parallel to the normal buffer line.

A slight trend toward metabolic acidosis does in fact occur during prolonged anaesthesia. This is initiated mostly in elderly patients by prolonged starvation and by premedication.

After analysis of complete arterial blood data on a large number of patients, I have found that almost all of them have fallen below the "normal buffer line" within an hour after premedication. This fall was probably initiated by preceding starvation. In my experience,

only patients with peptic ulcer who have been on prolonged alkali therapy regularly have remained above the "normal buffer line" after premedication. On occasion, I have had a very excitable patient, or one on whom I have had difficulty in obtaining the preoperative arterial blood sample, in whom hyperventilation appears to have shifted the pCO₂ to the right of the 40 mm. of mercury isobar, but most are to the left after premedication. Most patients therefore have a slight metabolic and respiratory acidosis before induction of anaesthesia.

The direction of shift thereafter depends largely on the anaesthetic agents and anaesthetic systems employed, the posture adopted for the operation, on whether the patient is allowed to breathe spontaneously or by artificial ventilation, on the duration of anaesthesia, on whether the patient has cardiopulmonary disease, on how much blood and electrolytes are lost and replaced during the operation, and finally, on the site of operation (intrapleural, intrabdominal, or superficial) (fig. 2).

I believe that most anesthesiologists now agree that moderate hyperventilation throughout the period of anaesthesia prevents a shift toward severe respiratory and metabolic acidosis in our patients while they are undergoing severe stress and a period of protracted starvation. This deleterious trend is certainly more difficult to prevent when a large volume of spilled blood must be replaced, because the anaesthetized, starving patient cannot metabolize the acid citrate in the blood for several hours or even for a few days, and cannot maintain a normal blood pH, when large volumes of bank blood with a pH lower than 6.8 must be injected.

The table shows the usual changes in acid-base balance that are seen with several symptoms and during a variety of anaesthetic agents and techniques.

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To the Editor.—We have read Dr. Dobkin's letter and he seems to be saying first, that the title of our paper is misleading because no metabolic acidosis occurred in our patients. He follows this by suggesting that the metabolic acidosis (which did not occur) is due to starvation and not hyperventilation.

We think Dr. Dobkin is concerned that we have not plotted our data on the pH-bicarbonate- $p\text{CO}_2$ graph. (His plot is incorrect, incidentally, since the postanesthesia and before hyperventilation sample labeled as [7] should be [2].) The result of this plot is an estimate of the changes in fixed acids identical to that which we have already presented in our paper as "corrected BHCO_3 " or "plasma BHCO_3 at pH 7.40." The exact significance of changes in corrected BHCO_3 , as well as $(\text{BB} +)\text{b}$, is a matter of debate at the present time. This uncertainty as to the interpretation of such indirect estimates of changes in fixed acids has led such careful workers as Dr. John Bunker of Boston to abandon both these indices and to measure the acids directly, *e.g.*, citrate, lactate, pyruvate, and total keto acids. For precisely this reason we did not rely only on changes in corrected bicarbonate or $(\text{BB} +)\text{b}$ but measured lactic acid as well.

Had facilities been available, we would also have measured changes in other acids and not reported the indirect estimates at all. That Dr. Dobkin ignored the very significant changes in lactic acid is not quite cricket.

Dr. Dobkin does make a valid point in questioning whether these changes are due to starvation. We did not consider this possibility since, (1) the evidence for metabolic acidosis resulting from hyperventilation is now considerable (see our bibliography), (2) all patients had an infusion of at least 50 grams of glucose during four hours of hyperventilation, and (3) an infusion of glucose during anesthesia produces in itself a slight increase in blood lactic and pyruvic acids (Henneman and others; *J. Appl. Physiol.* 12: 164, 1958). We did not eliminate this possibility in our study; Dr. Dobkin offers no evidence to support his alternate hypothesis.

The remainder of his comments and illustrations do not seem to be pertinent to anything in our paper, since anesthetic technique, posture, respiration, etc., were fairly uniform in these patients. None of our patients received a transfusion. His unsupported statement concerning the inability of anaesthetized patients to metabolize acid citrate "for several hours or even a few days" should raise a few eyebrows.

We cannot help note that his figure 2 shows that except for C', all anesthetic combinations, with or without hyperventilation, lead to a vector below the normal buffer line. This is just what we have said in our paper. "Both respiratory acidosis and respiratory alkalosis lead to metabolic acidosis."

Finally, Dr. Dobkin seems to have missed the whole point of our presentation, which was to show that the degree of metabolic acidosis which developed during severe prolonged hyperventilation was slight and was not a valid objection to the routine use of hyperventilation during clinical anesthesia. Our conclusions remain the same: the degree of metabolic acidosis is of no clinical significance and the probable cause of the acidosis is something other than tissue hypoxia from vasoconstriction.

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