

CORRESPONDENCE**Effects of Apnea on Blood Acid-Base Parameters**

To the Editor.—By means of artificial re-breathing, Eger and Severinghaus (ANESTHESIOLOGY 22: 419, 1961) recently evaluated the rate of rise of alveolar P_{CO_2} in five apneic, anesthetized patients, before and after hyperventilation. These workers observed a rapid elevation in alveolar P_{CO_2} of about 13 mm. of mercury during the first minute of apnea, followed by a linear increase at an approximate rate of 3 mm. of mercury/minute. These findings prompted our investigation of blood acid-base parameters in a patient during a five-minute period without ventilation.

Anesthesia was induced in a clinically healthy subject prior to elective surgery by intravenous administration of thiopental. Capillary blood specimens were collected immediately before and at one-minute intervals after complete suppression of spontaneous respiration by a dose of succinylcholine. pH measurements made in an Astrup Micro-analyzer were used to obtain values for acid-base parameters by nomographic interpolation (table). Cessation of respiration for one minute was associated with a rise in blood P_{CO_2} of 10.9 mm. of mercury which is close to alveolar P_{CO_2} values observed by Eger and Severinghaus for the same apneic period. However, throughout the entire apneic period the increase in blood P_{CO_2} /minute averaged 8 mm. of mercury, which was about double the mean rate of rise in alveolar P_{CO_2} noted by these workers.

Astrup analyses indicate that accumulation of CO_2 was accompanied by a fall in blood pH as well as a rise in actual bicarbonate levels. The decline of pH was 0.04 unit/minute, thereby producing a moderate degree of respiratory acidosis during the apneic period. Concomitant increases in actual bicarbonate may be interpreted as reflecting a compensatory mechanism. It is noteworthy that standard bicarbonate levels showed little fluctuation at the time intervals studied. Therefore, these data support the proposal of Astrup and co-workers (Lancet 1: 1035, 1960) that standard bicarbonate levels are largely independent of ventilation.

Our investigation suggests that on an individual basis it may be difficult to predict the rate of rise of blood P_{CO_2} with any degree of certainty. As pointed out by Eger and Severinghaus, any factor which affects CO_2 storage or metabolism in the body is capable of altering the accumulation rate. Thus CO_2 homeostasis may best be evaluated by repeated blood measurements during surgical anesthesia.

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Acid-Base Parameters by Nomographic Interpolation Obtained from pH Measurements

Minutes of Apnea	pH	P_{CO_2} (mm. Hg)	Actual Bicarbonate (mEq./liter)	Standard Bicarbonate (mEq./liter)	Total CO_2 (mEq./liter)	Base Excess (mEq./liter)	Buffer Base (mEq./liter)
0	7.46	33.1	24.5	25.0	25.5	+2.5	45.2
1	7.36	44.0	23.8	23.6	25.1	+1.0	42.0
2	7.33	53.5	27.5	24.8	29.1	+1.5	51.2
3	7.29	55.0	25.0	24.2	25.7	+1.5	42.8
4	7.26	70.0	29.5	24.5	31.6	+2.0	49.5
5	7.26	73.0	32.0	24.2	34.2	+1.8	53.0