dioxide content relative to man. They should be regarded as the standard values to be maintained in cats undergoing artificial ventilation in neurophysiological experiments.

1. *Handbook of Biological Data,* Ed., W. S. Spector, W. B. Saunders Co., Philadelphia, 1956, p271.
2. Yannet, H., *J. Biol. Chem.,* 1940, v136, 265.
3. Ngai, S. H., *Am. J. Physiol.,* 1957, v190, 356.
4. Plum, F., McNealy, D. E., *ibid.,* 1962, v202, 221.
5. Fink, B. R., unpublished observations.
6. Florkin, M., *Ann. de physiol.,* 1934, v10, 500.
7. Bonvallet, M., Hugelin, A., Dell, P., *J. physiol.* (Par.), 1955, v47, 651.

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Plasma Potassium Changes During Rebreathing in the Dog.* (28033)

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A gradual rise in the plasma potassium concentration during breathing of high CO_2 mixtures in the dog has been reported by several investigators(1,2,3). On the other hand, when cats are made to breathe 35% carbon dioxide there is an immediate but transient rise in potassium concentration in the plasma followed by a fall to normal or near normal levels until the animal is allowed to breathe air again, at which time a second elevation takes place(4). This second elevation on reversing the severe hypercarbic acidosis is also regularly observed in the dog(2). Serial sampling of arterial blood at one minute intervals during the first 10 minutes of 30% CO_2 breathing in the dog has revealed no evidence of a transient elevation at this time. However, a transient elevation of plasma potassium concentration has been reported in dogs rebreathing from a bag and therefore, subjected to a gradual elevation of CO_2 over a period of 90 minutes(5). The purpose of this paper was to reexamine this phenomenon.

Methods. Experiments were performed on 10 mongrel dogs anesthetized with intravenous thiopental sodium. After the initial induction, additional anesthetic was administered as required to maintain surgical anesthesia. Arterial blood samples were drawn from a catheter passed through the femoral artery into the abdominal aorta. Rebreathing was carried out by connecting the dog to a 35 liter bag by a cuffed endotracheal tube. Blood pH was determined with a glass electrode at 38° and correction was made, using the factor –.014 pH/°C, when the body temperature of the dog deviated from this value by as much as 0.5°C. Plasma potassium concentration was determined on an internal standard flame photometer. CO_2 and O_2 concentrations in the rebreathing bag were estimated with the Scholander rapid gas analyzer. Oxygen concentration in the bag was maintained well above 20 volumes per cent at all times. Arterial blood samples were drawn before, at regular intervals during the rebreathing, and at 5 minutes following the return to room air breathing. Electrocardiograms were recorded before, during, and following the experimental rebreathing period.

Results. The results are presented in Fig. 1. No transient rise in potassium concentration was evident in these 10 experiments. A gradual rise in plasma potassium concentration took place during the first 30 minutes of the rebreathing period. This increase was statistically significant at this time (P–.01). Between 30 and 60 minutes potassium concentration continued to rise with little or no change between 60 and 90 minutes.

CO_2 tension increased during the 90 minutes and blood pH fell correspondingly. On returning the animals to room air breathing, blood pH rose rapidly to almost normal val-

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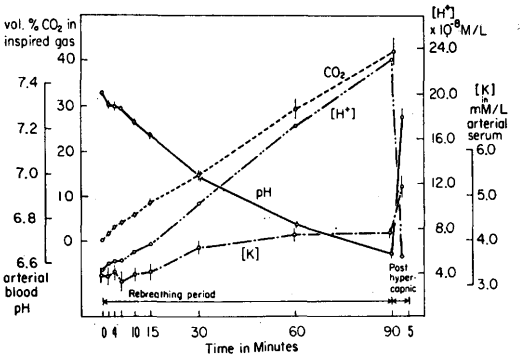

FIG. 1. Changes in plasma potassium concentration and blood pH during and following 90 min of increasing CO_2 concentration produced by rebreathing. Points are mean values of 10 experiments; vertical bars represent standard error on either side of mean.

ues within 5 minutes. During this time, plasma potassium rose abruptly to the highest values observed during these experiments. This increase, which averaged 1.04 meq/l, was highly significant.

Discussion. The transient rise which occurs in plasma potassium concentration in cats breathing high concentrations of carbon dioxide has been attributed to a stimulation of the sympatho-adrenal system resulting in a release of potassium from the liver in response to elevated circulating adrenalin(6). It has been demonstrated that this transient rise can be eliminated by sympathetic blocking agents or by removal of the liver. It seemed unlikely that this explanation would account for a transient rise in dogs that were exposed to a gradual increase in CO_2 tension from rebreathing in a bag since less stimulation of the sympatho-adrenal system would be expected under these circumstances than with sudden exposure to 30% CO_2(5). However, since it has been reported that this transient rise occurred, this phenomenon was investigated.

In our experiments no evidence of a transient rise in plasma potassium was found in any case. No satisfactory explanation is available to account for the difference in results in the experiments of Adrian and Gordon and those reported here. The physicochemical basis of the exchange of potassium between intracellular and extracellular fluids has also been investigated(7).

Summary. Plasma potassium was determined on dogs before and during 90 minutes of rebreathing, and immediately following return to air breathing. Potassium concentration rose gradually with increasing CO_2 tensions during the first 60 minutes of rebreathing and remained essentially constant for the last 30 minutes. On changing from the high CO_2 concentration to air breathing, a further rapid rise in plasma potassium concentration appeared. No evidence of a transient early rise in potassium concentration was observed.

1. Young, W. G., Sealy, W. C., Harris, J. H., *Surg.*, 1954, v36, 636.

2. Brown, E. B., Jr., PROC. SOC. EXP. BIOL. AND MED., 1954, v90, 319.

3. Simmons, D. H., Avedon, M., *Am. J. Physiol.*, 1959, v197, 319.

4. MacKay, J. L., *ibid.*, 1947, v151, 469.

5. Andrews, P. W., Adrian, F. E., Gordon, A. S., *Fed. Proc.*, 1957, v16, 3.

6. Fenn, W. O., Asano, T., *Am. J. Physiol.*, 1956, v185, 567.

7. Brown, E. B., Jr., Goott, B., Lade, R., *The Physiologist*, 1962, v5, 114.

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Effects of Octacosanol on Chick Comb Growth. (28034)

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Previous studies from this laboratory(1) indicate that wheat germ oil contains an unidentified factor or factors which when assayed by the method of Dorfman and Dorfman(2) resulted in a significant increment in chick comb growth. Subsequent studies*

* Ezra Levin, unpublished findings.