Serum Potassium Levels in Hyperventilated Dogs. (27758)

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How much pulmonary ventilation an anesthetized patient should have is a difficult question to answer. Since all general anesthetic agents now employed cause some respiratory depression, most anesthesiologists augment their patient's pulmonary ventilation by positive pressure applied intermittently either by hand or by means of a mechanical ventilator. Many anesthesiologists believe that since euventilation is difficult to achieve, passive hyperventilation should be employed, that is, always to err on the side of a high p O_2 and low p CO_2 in the arterial blood. Some possible adverse physiological effects of a markedly lowered p CO_2 have been suggested by the work of Kety(1), Sugioka(2), and others. These effects relate principally to the vasoconstrictive effect of a low p CO_2 upon certain blood vessels with a resulting ischemia of the supplied area. Other possible adverse effects of hyperventilation, however, relate to electrolyte changes in blood plasma, principally those of potassium and sodium. Brown, *et al.*, noted no consistent change in plasma potassium although urinary potassium excretion increased during hyperventilation(3). Robinson(4) has recently presented evidence that hyperventilation is not deleterious to the patient, even though certain marked chemical changes may occur in the blood. Following a marked, passive hyperventilation, he noted a significant drop in serum potassium and sodium in awake subjects but only a slight drop in serum potassium in anesthetized subjects. Von F. Schaub found a large decrease in serum potassium in actively hyperventilated subjects(5). Sieker showed a relation of blood glucose to the serum potassium under hypoxic hyperventilation(6). In order to clarify these discrepancies the following study was undertaken.

Method. To investigate serum potassium during prolonged passive hyperventilation, 17 mongrel dogs, average weight 13 kg, were studied. After anesthesia was induced and maintained at moderate levels with halothane in room air, the dogs were heparinized and control blood samples were drawn. The dogs were then passively hyperventilated with a sinusoidal pump (intermittent positive pressure) in a non-rebreathing system for 6 hours. Blood samples were drawn at intervals. The pump was stopped and spontaneous respirations resumed for about 20 minutes at which time another control blood sample was drawn. All dogs were allowed to survive at least 2 days, some for several weeks, before being sacrificed.

All blood was drawn through polyvinyl catheters directly into siliconed, heparinized syringes containing 1 cc of mercury. The syringes were gently inverted several times immediately prior to whole blood analysis. pH's and CO_2 contents were done on whole blood and potassiums were determined in serum (blood clotted with protamine and fibrin removed).

Serum potassiums were determined with flame photometry on 13 dogs. Blood gas contents were determined with the method of Van Slyke and Neill, and pH's with glass electrode on 6 dogs. The pH meter was calibrated at least twice a day with standard buffer at pH 7.0. Rosenthal temperature corrections were employed. From the above data arterial p CO_2's were calculated. Alveolar p CO_2's were determined with an infrared analyzer and appropriate calculations, on 6 dogs. The CO_2 analyzer was calibrated before and after each experiment with various standard gas samples ranging from about 2-8% CO_2.

To determine hemolysis, hemoglobin determinations were done on several of the serum samples. With an all plastic system from artery to syringe, heparinization of the dog, and no "flushing" of the catheters, serum hemoglobin levels of less than 2 mg % were obtained. This amount of hemolysis should not contribute significantly to the serum potas-

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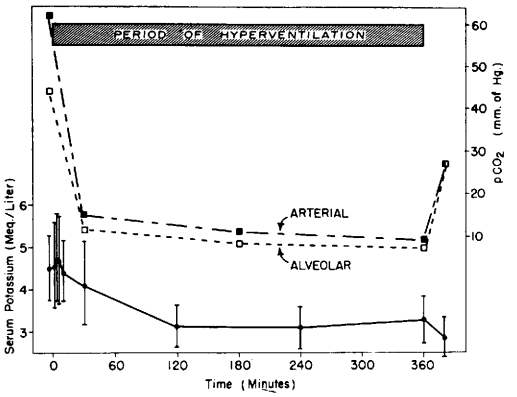

FIG. 1. p CO_2's indicated by ordinate on right. Arterial: solid squares and dashed lines. Alveolar: open squares and dotted lines. Serum potassiums indicated by ordinate on left and by solid dots and lines. Vertical bars represent stand. dev. Three points (4, 30 and 120 min) were each compared with the first control sample (−3 min) by means of the "t" test. 4 min: $p < 0.15$; 30 min: $p < 0.05$; 120 min: $p < 0.01$.

sium levels, and indeed is probably close to physiological levels in the dog. The serum appeared clear and colorless to the eye until serum hemoglobin levels of about 20 mg % or more were reached.

Results and comments. The p CO_2 levels (Fig. 1) show how markedly hyperventilated the dogs were. Although the drop in serum potassium during the first 30 minutes followed the decreased p CO_2 somewhat, the reverse was not true during recovery, at least during the time studied. The arterial and alveolar p CO_2's showed a linear correlation, $r = .926$. The fact that the initial alveolar p CO_2 was lower than the arterial p CO_2 is probably a sampling error in the alveolar gas collection during a period of relatively small tidal volumes due to anesthetic respiratory depression.

Serum potassium showed a slight initial increase (maximal at 4 minutes) (Fig. 1). This is not significant, but suggestive, and has not been reported previously to our knowledge, although it is suggested by the data of Gold *et al.*(7). It then dropped sharply to significantly low levels in 2 hours. This confirms the work of Robinson and Von F. Schaub.

It is interesting that the serum potassium remained at very low levels even after 20 minutes of spontaneous respirations following the hyperventilation period. The relation of glucose to serum potassium levels under these conditions is yet to be determined. The dogs in this study were probably kept slightly hyperglycemic by a slow continuous 5% glucose intravenous drip, but blood glucose was not determined.

It is hoped that this slight water and glucose loading will maintain a more nearly steady physiological state than no loading at all, as has been done previously. Thus any plasma potassium changes seen are not secondary to hypoglycemia.

All dogs recovered and were apparently well at time of sacrifice.

Although severe respiratory alkalosis and serum hypokalemia were seen, there was no apparent respiratory, cardiovascular, nor cerebral damage. Whether severe passive hyperventilation is a deleterious procedure has not been demonstrated.

Summary. Anesthetized dogs which were passively hyperventilated for 6 hours developed severe respiratory alkalosis. Serum potassium concentration increased slightly in the first 4 minutes, then decreased markedly during the next 2 hours. For at least 20 minutes after cessation of hyperventilation the K level did not begin to increase, even though the arterial p CO_2 did so.

The authors wish to thank Dr. F. G. Hall, Dept. of Physiology, for use of laboratory facilities, and Dr. W. P. Wilson, Dept. of Psychiatry, for technical assistance in serum potassium determinations.

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Received May 17, 1962. P.S.E.B.M., 1962, v111,