Blood Acid-Base Status in Acutely Water-Depleted Rats

I. M. LIBERMAN, H. BRAZZUNA, A. CAFANO, and F. GONZÁLEZ

Laboratory of Water and Salt Metabolism, Department of Pathology and
Physiopathology, School of Medicine, Universidad de la República, Hospital de
Clínicas, Piso 15, Montevideo, Uruguay

Received September 29, 1970

Summary. The relevant blood acid-base data were determined in 10 control (C)
and in 10 water-depleted (D) rats. Water-depletion (4.9% of body weight) was
achieved following a 3-h intraperitoneal injection of a hyperosmotic solution. The
results revealed that water-depleted rats failed to develop "contraction" alka-
losis. Actual pH, plasma actual bicarbonate, blood base excess and blood buffer base
were found to be significantly lower in D than in C animals. Blood Pco₂ remained
unchanged. Hematocrit was 25% higher in the D group. Plasma and blood volume
were 33 and 20%, respectively, lower in D than in C rats. The "in vitro" blood
buffer capacity was increased in the D animals; the regression equations from
ApH/Alog Pco₂ were: Y = 8.457 - 0.667 X in the control and 8.231 - 0.571 X in the
water-depleted animals. It is suggested that the observed hydrogen ion accumulation
in the D group could be explained by the tissular hypoxia and deterioration of
kidney function.

Key-Words: Dehydration -- Acid-Base Equilibrium -- Arterial pH -- Plasma
Bicarbonate -- CO₂ Dissociation Curve.

Schlüsselwörter: Dehydratisierung -- Säurebasengleichgewicht -- Arterieller
pH -- Plasmabicarbonat -- CO₂-Bindungskurve.

The acid-base status of blood during short-term, acute hypohy-
dration in man and animals where the water is lost in a few hours has
not been the subject, as far as we know, of investigations. Some authors
recorded that total CO₂, alkali reserve and total base decreased during
acute water depletion in man (Murray, 1966) and dog (Eichelberger
and Hastings, 1937; Libermann, Fernández, Balea, and Puppo, 1966;
Hamburger, Halpern, and Mathé, 1950). Arterial pH was found dimin-
ished in dogs following one-hour osmotic diuresis (Eichelberger and
Hastings, 1937) while in thermal exposures with a duration between
20 min and 12 hours an increase in blood pH was reported in man
(Murray, 1966; Senay and Christensen, 1967) explained by the resulting
hyperpnea and hypocapnia. These experiments give data only on a few

* A preliminary report of this work was presented at the IX. Meeting of the Latin
American Association of Physiological Sciences (A.L.A.C.F.), Belo Horizonte,
Brasil, June 1969.
relevant blood acid-base parameters since they were not directed towards
a search of the action of water depletion on blood acid-base status. On the other hand Cannon, Heinamann, Albert, Laragh and Winters, (1965) demonstrated that edematous patients submitted to an acute water loss develop “contraction” alkalosis, related in part to the rapid and large loss of relatively HCO₃⁻-free fluid from the extracellular fluid space. This investigation, undertaken to evaluate the blood buffer capacity and acid-base parameters in rats following acute water depletion for 3 hours, failed to show this “contraction” alkalosis; on the contrary the water-depleted rats develop a significant metabolic acidosis.

Methods

Twenty adult male albino rats weighing 134—266 g were studied. A randomized experimental block design was used (Lellouch and Lazar, 1966), two animals to each block: a control rat (C) weighing 134—245 g (mean 183 g), and a water-depleted rat (D) weighing 171—248 g (mean 210 g). 15 days before the experiment, the animals were housed in individual cages and were given standard rat chow (Forrêmez Gramon) and tap water “ad libitum”. At the beginning of the experiment all animals were deprived of food and water. A hyperosmotic (550 mOsm/l) solution of glucose and electrolytes¹ was instilled into the peritoneal cavity of the water-depleted rats in doses of 10 ml/100 g body weight, while control rats were only punctured with no injection of liquid into the peritoneum. Previous studies in dogs and rats demonstrated that there are no significant differences in blood acid-base parameters between intact animals and those after a 3—4 hour intraperitoneal injection of an isoosmotic-isotonic solution (Libermann, unpublished data).

Following a 3-hour-experimental period the animals were anesthetized with sodium pentobarbital (35 mg/kg body weight). After laparotomy, fluid was collected by aspiration and measured to the nearest ml in the D rats. Arterial blood was anaerobically sampled with 5 ml syringes, their dead space filled with heparin solution (0.5 mg/ml). Actual blood pH was measured immediately; hematocrit (Ht) and equilibrated pH were measured within 5 min of blood sampling.

The acid-base status of arterial blood samples was determined in duplicate by the equilibration method (Astrup Micro Equipment, AME 1, Radiometer, Copenhagen, Denmark) at 38 °C, and from a nomogram constructed from data obtained in humans (Siggaard-Andersen, 1966). Microhematocrits were measured in an MB International Centrifuge.

The relationship between plasma and blood volume in both groups of animals was obtained from the mean hematocrit difference (Elkinton, Danowski, and Winkler, 1946). The use of changes in hematocrit to estimate changes in plasma and blood volume was based on the assumption that there are no changes either in the red cell mass or in the mean corpuscular volume of the red cells. The first assumption is probably valid in view of the short duration of the experiment. The second assumption is not completely valid, because the increased osmotic pressure of the plasma during the experiment (Libermann et al., 1966) would probably result in a reduction of the volume of the red blood cells. Thus the change in hematocrit would tend to underestimate the reduction in plasma and blood volume.

¹ The composition of the solution was: NaCl 5.77 g/l, CaCl₂ 0.30 g/l, MgCl₂ 0.15 g/l, KCl 0.30 g/l, Na₂CO₃H 3.00 g/l, Na₂SO₄ 0.07 g/l, Na₂PO₄H 0.14 g/l, Glucose 50.00 g/l.