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CHANGES IN RENAL PARAMETER DURING SALINE INFUSION IN DOGS.

by

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An intravenous infusion of isotonic saline solution causes an increase in the urinary excretion of water and sodium which regulates the constancy of the extracellular fluid volume and its osmolality. The mechanism involved in the diuresis and natriuresis are open to question. Most of the reported experiments were designed with a saline infusion via a peripheral vein which created problems of plasma and extracellular fluid volume expansion. These, in turn lead to changes in several other systemic parameters which might trigger extrarenal mechanisms.

The observations of Henry et al (3) that blood volume receptors in the thoracic vascular bed determined volume diuresis by inhibiting the release of antidiuretic hormone strengthened the concept that the kidneys are the effector organs of a reflex arc, rather than primary effectors. The search for neural pathways of a reflex arc responsible for the renal volume response has not been successful (1, 2, 4,). Cardiac denervation (1) has been occasionally reported to attenuate the renal response to iso-oncotic infusions, but never to abolish it. This led investigators to search for humoral component(s) or renal parameters which the vascular expansion might directly exert on the geometry of the kidney resulting in natriuresis.

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The primary aims of this study are to determine whether kidneys can function as primary effectors during the infusion of saline solution; and to determine whether urinary excretion of sodium is under the influence of plasma sodium concentration or plasma osmolality.

The experiments were performed on 13 mongrel dogs which were divided into four groups. Group I, composed of six dogs, were infused with hypertonic and hypotonic sodium chloride, group II (three dogs) were infused with hypertonic and hypotonic choline chloride, group III (two dogs) were infused with hypertonic and hypotonic lithium chloride and group IV (two dogs) were infused with hypertonic and hypotonic rubidium chloride. Either the hypertonic solution (537 mOsm/kg) or the hypotonic solution (27 mOsm/kg) were infused directly into either the left or the right renal artery at the rate of 2.5 ml/min instead of the control isotonic saline solution (280 mOsm/kg). During experimental period the contralateral kidney received isotonic saline solution continuously as during the control period.

It was found that the infusion of hypertonic saline solution caused an elevation of the sodium concentration in that renal artery by 4.59 ± 1.18 (S.D.) uEq/ml. This minute change in the filtered load of sodium caused a significant increase in V , $U_{Na} \cdot V$ and $U_{Cl} \cdot V$. Hypertonic lithium chloride and rubidium chloride of the same osmolality (537 mOsm/kg) also caused a significant increase in V and $U_{Cl} \cdot V$, but $U_{Na} \cdot V$ was not significant increase. When the systemic effect of hypertonic cholinechloride was ruled out, it was found that choline caused a non-significant increase in $U_{Na} \cdot V$. All hypertonic perfusates induced osmotic diuresis because its C_{Osm} were significantly increased. Infusion

of hypotonic saline solution (27 mOsm/kg) caused a decrease in renal arterial plasma sodium concentration by 4.94 ± 2.03 (S.D.)/uEq/ml. This responded in decreases in V , U_{Na} , V and U_{Cl} , V . Hypotonic choline chloride, lithium chloride and rubidium chloride of the same osmolality (27 mOsm/kg) caused slight increase in U_{Na} , V and V . In the experimental kidney there were no significant difference in GFR, ERPF, U_K , V , Hct, mean arterial blood pressure, plasma volume, and plasma protein concentration from comparison to the control value.

These results suggested that kidney can function as primary effector to various perfusates introduced directly to them. Each can function independently of the other. The changes that occur do not depend on just the sodium concentration or the osmolality. The transport mechanisms together with their capacities, will initiate and determine the pattern and the degree of diuresis. This primary response can further be modified by the action of several hormones such as aldosterone and ADH, which are triggered by generalized systemic changes.

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