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Sulfate Retention in Dogs Following Bilateral Adrenal Extirpation.

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The results of previous work by the senior writer¹ and his co-workers² on the functional significance of the suprarenal cortex, indicated that the cause of death in double operated cats and dogs is probably acid intoxication, due to retention of acids formed in the course of normal metabolism. The acidosis is apparently not due to the formation of abnormal acids within the organism, but due to defective elimination of non-volatile acids.

Study of the acid-base equilibrium of normal and bilaterally adrenalectomized cats showed quite clearly that the animals with marked symptoms of adrenal insufficiency were suffering from an uncompensated non-volatile acidosis. According to the data available, the acidosis was attributed to an increase in phosphoric and undetermined organic acids. However, it was evident that the marked fall in bicarbonate, observed in our double operated animals, was not occasioned to any considerable extent by the rise in inorganic phosphorus, which although marked, never exceeded two millimols, whereas the carbon-dioxide fell from 6 to 10 millimols. We stated that "increase in organic acid (which in our data included the sulfate ion) is the one change of sufficient magnitude to be responsible for the bicarbonate fall. Further efforts on our part to determine the nature of the organic acid or acids ended in failure, hence it was decided to test for sulfate retention, to see if the sulfate ion could be responsible for the observed changes.

Dogs were used as experimental animals, and only those animals surviving 4 to 6 days or longer, following the second operation, were employed for blood tests.* The results obtained are of interest. The blood of normal unoperated and unilaterally operated dogs have inorganic sulfate values averaging 2 mg. per 100 cc. Double-operated animals, showing no symptoms of adrenal insufficiency, also have normal amounts of blood inorganic sulfate. However, when serious symptoms of adrenal insufficiency intervene, the rise in inorganic sulfate becomes apparent and increases up to the time of death. Values as high as 12 mg. sulfur per 100 cc. of blood are not uncommon. The increase is most marked when the animal is verging on coma. Inorganic phosphate and inorganic sulfate be-

* All operations under careful anesthesia.

have similarly with respect to the stage of adrenal insufficiency during which the greatest increases occur. Increased concentration of inorganic phosphate and sulfate in the blood parallels the increase in severity of the symptoms and the degree of acidosis. Some of the data obtained from 6 of our 25 cases are presented in Table I.

TABLE I.
Sulfate retention in adrenalectomized dogs.

Unilaterally operated	Sulfur mg. per 100 cc.	Bilaterally operated	1st symptoms noticed	Survival period	Sulfur per 100 cc.	Hd	CO ₂ capacity vols. %	CO ₂ content vols. %	P mg.	Sugar, mg. per 100 cc.	Condition anti-mal when bled
Mo.-day											
6/8 at 10 A.M.	0.9 mg.	6/15 at 4 P.M.	6/18 at 1 P.M.	5 days	11.9	7.26	27.	20.6	10.	56	weak
6/9 at 11 A.M.	1.2 "	6/16 at 2 P.M.	6/19 at 9 P.M.	6 "	10.7	7.25	25.	19.2	9.2	50	weak
5/18 at 2 P.M.	1.4 "	5/26 at 9 A.M.	5/29 at 10 A.M.	5½ "	11.1	7.22	22.	19.0	12.1	45	verging on coma
5/21 at 3 P.M.	1.2 "	5/29 at 3 P.M.	6/2 at 11 A.M.	5 "	9.4	7.27	27.	21.5	13.1	57	weak
5/24 at 11 A.M.	1.2 "	5/30 at 9 A.M.	6/3 at 8 A.M.	6½ "	12.2	7.25	26.	21.4	10.2	62	weak
5/20, 10 A.M.*	1.4 "	5/28 at 5 P.M.	5/31 at 2 P.M.	4½ "	9.1	7.27	32.1	28.1	12.5	53	weak

* Venous blood used.

It is evident from our data that retention of the sulfate ion plays a considerable rôle in the acid intoxication of adrenal insufficiency. Whether or not the rise in inorganic phosphorus and sulfate are sufficient to account entirely for the degree of acidosis observed, the writers are not prepared to say. It seems likely that the fall in CO_2 is to a considerable extent due to retention of these two acids. There is also probably a slight increase in organic acid or acids of undetermined nature.

¹ Swingle, W. W., and Eisenman, A., *Am. J. Physiol.*, 1927, **lxxix**.

² Corey, E. L., *Am. J. Physiol.*, 1927, **lxxix**, 633.

³ Zwemer, R. L., *Am. J. Physiol.*, 1927, **lxxix**.