quoted an inhibitory dilution of 500,000 for a human strain of *M. tuberculosis* (inoculum 0·01 mgm.).

The volatile principle was evaluated in 25-gm. Parkes albino mice. These were infected intravenously with the *Ravenel Rv* strain of *M. tuberculosis* (0·1 mgm.). Treatment was started on the day of infection and continued for 14 days, the drug being administered in the diet. The measured daily drug intake was approximately 340 mgm./kgm. mouse weight up to the seventh day. Median survival times of mice, with 19/20 limits of probability, were 14 (12·8–15·3) days for the treated mice compared with 16·3 (15–17·7) days for the untreated controls. This decrease in median survival time is just significant and may indicate toxicity. The pulmonary lesions, however, in the treated mice were indistinguishable from those in the controls.

It may be concluded from these results that the volatile principle of *Adhatoda vasica*, as prepared by us, has no antituberculous properties in mice on oral administration.

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Effect of Isolated Cerebral Hypoxia and Hypercapnia on Sodium Excretion

The healthy organism, as is well known, reacts to a number of influences (such as work, etc.) by decreased sodium excretion. In such cases a relative hypoxia occurs: in pathological sodium retention (cardiac decompensation, famine œdema, anæmia, etc.) an absolute hypoxia (arterial, stagnating or anæmic) can be demonstrated.

It is to be supposed that in all these states a causal relation must exist between the hypoxia and the decreased sodium excretion. We have therefore investigated, in a series of experiments on dogs, the effect of hypoxia on sodium excretion. In these experiments the rate of glomerular filtration was estimated by endogenous creatinine clearance, the sodium was determined by flame photometry, the oxygen content according to Issekutz, and the carbon dioxide according to Van Slyke.

(1) In fifteen experiments we dealt with the effect exercised on sodium excretion by hypoxia of the entire body. The dogs were under chloralose anæsthesia. The hypoxia was induced by inhalation of a nitrogen mixture containing 10 per cent oxygen. In fourteen cases the sodium excretion decreased significantly, to an average of 16 per cent. The sodium excretion occurred in some cases with unchanged sodium filtration, proving the effect to be of tubular origin.

(2) We have also investigated the question whether the fall in sodium excretion could be brought about by isolated cerebral hypoxia. For this purpose we made a preparation in which only neural connexion was maintained between the head and the body.

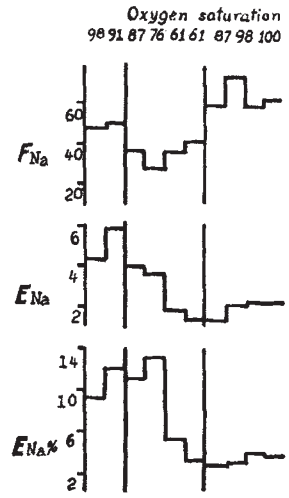

Fig. 1. *F*(Na), filtered sodium (m.equiv.); *E*(Na), excreted sodium (m.equiv.); *E*(Na%), excreted sodium as a percentage of filtered sodium

Circulation in the isolated head was maintained by a Dale–Shuster pump. In all cases the vertebral arteries and veins were ligated and in some instances the spinal veins also. In some cases the sinus caroticus was denervated. In fourteen of sixteen experiments, there was a definite decrease in sodium excretion as an effect of the cerebral hypoxia (Fig. 1). It also occurred in animals in which the spinal veins were ligated, as well as in those with denervated sinus caroticus. Hypercapnia brought about the same results.

The same effect was obtained when stagnating hypoxia was induced in the brain by reducing the perfusion pressure of the Dale–Shuster pump. In these cases, too, the sodium excretion fell to 21 per cent of the original.

Finally, during some of these experiments, we considered the effect exercised on sodium excretion by hypoxia of the isolated body. In parabiosis, the head of the acceptor animal was perfused through the carotids with blood (100 per cent oxygen saturation) from the donor dog. Hypoxia of the body of the acceptor animal was induced by inhalation of an oxygen–nitrogen gas mixture with reduced oxygen content. Sodium excretion decreased here, too, as in isolated head hypoxia.

From these experiments the conclusion is drawn that isolated cerebral hypoxia and hypercapnia lead to a decreased sodium excretion. We believe this volume-regulating phenomenon to be of importance. Our results, which show that the effect also occurs in body hypoxia, indicate that there are receptors sensitive to hypoxia not only in the brain but also throughout the whole organism. It is still a question, however, which of these receptors are the most sensitive.

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