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A BRAIN STEM SYSTEM AND THE INHIBITION OF EXTRACELLULAR DEPLETION-INDUCED DRINKING

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Both bilateral lesions of the ventrolateral lateral parabrachial nucleus (VLLPBN) and ablation of the area postrema-medial nucleus of the tractus solitarius (AP-mNTS) in rat produce enhanced water intake to isoproterenol (ISOP). The observation that mechanical stimulation of right atrial receptors by balloon inflation attenuates ISOP--induced drinking suggests that afferents from atrial receptors act to inhibit water intake. This study was conducted to determine whether the exaggerated drinking response of VLLPBN-lesioned rats of ISOP can be explained by the interruption of inhibitory signals arising from the right atrial receptors.

Male Sprague-Dawley rats received either sham lesions or bilateral electrolytic lesions of the VLLPBN. Following recovery from surgery, balloons were implanted into the right atrial/superior vena cava junction in both groups. ISOP-induced drinking tests were performed beginning one week later. In the first test all animals received ISOP (30 μg/kg) and in one half of each surgical group the atrial balloons were inflated and all animals were then allowed to drink. Forty-eight hrs after the first test the ISOP treatment was delivered again to all subjects and the balloons were inflated in the remaining half of the animals. Balloon inflation significantly attenuated ISOP--induced water intake in the sham lesioned animals 60 min after ISOP. In contrast, inflation had no effect on drinking in the VLLPBN lesioned rats. The results suggest that the VLLPBN receives afferent inhibitory input from atrial stretch receptors and support a hypothesis of a brain stem inhibitory thirst mechanism that involves the AP-mNTS, the VLLPBN, and interconnecting neural circuitry.