Excerpt

Body fluid homeostasis and arterial pressure are intimately related to the point that their control share many common mechanisms. The diagram shown in Figure 4.1 illustrates an interactive network (antidehydration network) activated by the dehydration of the two major body fluid compartments, extracellular [represented by the production of angiotensin II (ANG II)] and intracellular (represented by hyperosmolarity). The operation of the network involves redundancy and reciprocity and results in effector mechanisms that counteract dehydration. Although highly simplified (many important factors, e.g., aldosterone, are omitted), the network diagram suggests complex control systems orchestrated by the brain. When deranged, the operation of such systems may lead to pathologies, for example, hypertension. The brain circuit that counteracts dehydration has two main entrances or input paths for sensory information arising from the periphery (blood and viscera). They are located at opposite poles of an axis of multiple connections formed between hindbrain and forebrain. The preoptic periventricular tissue surrounding the anteroventral third ventricle (AV3V) forms a key region in the forebrain pole that integrates mechanisms to control the antidehydration network. The AV3V and the lamina terminalis share the organum vasculosum (OVLT) and the ventral median preoptic nucleus (MnPO). Also belonging to the lamina terminalis is the subfornical organ (SFO). The AV3V extends from the OVLT to the periventricular preoptic tissue until the rostral limits of the anterior hypothalamic area (Figure 4.2) (Brody and Johnson 1980; Menani et al. 1988b). The OVLT, along with the SFO, functions as a primary sensory station of the forebrain that monitors humoral factors such as circulating ANG II and osmolarity (Johnson 2007; McKinley et al. 2001; Chapter 2). The other entrance to the antidehydration brain circuit is located in the hindbrain and involves primary visceral sensory inputs in the nucleus of the solitary tract (NTS) and another circumventricular organ devoid of blood–brain barrier, such as the OVLT and SFO, the area postrema (AP). The AV3V functions as a nodal structure that integrates and redistributes signals originated in visceral sensory organs to pattern generators of neuroendocrine, autonomic and somatic effector actions against dehydration and reduction in blood volume. It has an intimate connection with the lamina terminalis and connects with many other areas in the fore and hindbrain. As suggested in Figure 4.3, signals generated in the OVLT and SFO make their way out of the lamina terminalis through projections to forebrain structures such as the paraventricular and supraoptic nuclei of the hypothalamus (PVN and SON, respectively), the lateral hypothalamus and the medial septal area (MSA) (Brody and Johnson 1980). The AV3V
region also direct or indirectly connects with areas of the hindbrain that control blood pressure, including the NTS, AP and the rostral ventrolateral medulla (RVLM) (Johnson 2007; Ricardo and Koh 1978; Saper et al. 1983; Whalen et al. 1999). The NTS and RVLM are the main areas of the medullary circuitry involved in cardiovascular control. The NTS located dorsally in the hindbrain is the site of the first synapse of baroreceptor, chemoreceptor, and cardiopulmonary receptor afferent fibers in the central nervous system, whereas the RVLM located in the ventral surface of the hindbrain is the main premotor sympathetic nuclei, projecting directly to the intermediolateral (IML) column in the spinal cord and responsible for the generation and maintenance of sympathetic vasomotor tone (Guyenet 2006). The reciprocal direct or indirect connections of the AV3V distributed along the forebrain–hindbrain axis form the neuroanatomical basis for the AV3V as an integrative region. An important indirect connection of AV3V with hindbrain is made through the PVN, which mono- or polysynaptically connects with sympathetic neurons in IML (Westerhaus and Loewy 1999) or indirectly affects sympathetic activity through connections with the RVLM (Yang and Coote 1998). Moreover, the AV3V also has connections with salivatory nuclei in the pons (Hübschle et al. 2001). The role of the lamina terminalis for the sensory integration of thirst and sodium appetite has deserved a special review in a relatively recent past (Johnson 2007). In this chapter, we first briefly refresh data on the general role of AV3V on the control of body fluid homeostasis and its role for the secretion of the atrial natriuretic peptide (ANP). Then, we review more recent data emphasizing the involvement of AV3V with salivary secretion, hindbrain control of cardiovascular function, and brain plasticity, in this order. Pilocarpine, a useful cholinergic agonist for therapeutics and experimental investigation about salivation, also affects arterial pressure and fluid balance. Early evidence for a central action of pilocarpine-induced salivation derived from studies with damage to the AV3V, but now we see that the same damage also interferes with the cardiovascular effects of pilocarpine. A role for AV3V on salivation linked to thermoregulation is also discussed. Then we show how AV3V influences the control of arterial blood pressure, first by presenting its role to sustain arterial pressure taking hemorrhage as a model, and second, by discussing compelling evidence for its role in the modulation of hindbrain mechanisms involved with short-term and long-term control of arterial pressure. Finally, we recall early evidence for brain recovery from AV3V damage and its implication for brain plasticity associated with sensitization of sodium intake before leading to conclusions.

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