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Labyrinthine and other supraspinal inhibitory controls over head-and-body ventroflexion

Sergio M. Pellis*, Vivien C. Pellis* and Philip Teitelbaum

Department of Psychology, University of Florida, Gainesville, FL 32611 (U.S.A.)

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The vestibular head righting reflex can be demonstrated by holding an adult rat vertically downward, so that the snout points downward. In this situation, the animal dorsiflexes its head and neck, bringing the head towards its normal orientation in space. Bilateral labyrinthectomy not only blocks this response, but releases an actively maintained ventroflexion of the head and neck. Bilateral electrolytic lesions of the lateral hypothalamus (LH) exaggerate such ventroflexion in labyrinthectomized rats. By themselves, LH lesions had no such effect. Therefore, it is argued that there are vestibular and supraspinal inhibitory mechanisms which, in the intact adult animal, keep this ventroflexion response in check. In addition, when the rats were held with their heads down, and with gentle paw contact with the ground, they did not ventroflex. However, they ventroflexed immediately upon releasing this paw contact. These observations suggest that there are tactile mechanisms which can also inhibit this exaggerated ventroflexion released by labyrinthectomy.

When an adult animal's body is inverted so that the head is displaced from its normal orientation in space, the head and neck will perform compensatory movements in order for the head to regain the appropriate orientation to gravity[6]. Such head righting is triggered vestibularly, and in some species (e.g. man, cat) can also be triggered visually[4,5]. This reflex can readily be demonstrated by holding an animal upside-down by the pelvis. The head and neck will dorsiflex, bringing the head towards a horizontal orientation to gravity[6]. In human infants this vestibular righting reflex is absent at birth and appears in the first 2–3 months[7]. This implies that the head righting response is absent until the vestibular system matures sufficiently to trigger and guide it. In addition to this positive vestibular control over head position, there also appears to be an inhibitory vestibular control over a proprioceptive whole body ventroflexion. In adult cats[14] and adult frogs[13] following labyrinthectomy and in infant mice with congenital vestibular dysfunction[3], the head is ventroflexed when they are held upside-down in the above described manner. Thus, in these cases the head does not merely hang downward, which would be the case if the labyrinths only exerted control over the head via the head righting reflex, but rather the head is actively ventroflexed. These findings indicate that there is an inhibitory vestibular control over such active head ventroflexion.

Our observations on labyrinthectomized rats with electrolytic partial transections at the level of the lateral hypothalamus (LH) suggest that there is a central neural inhibitory control over ventroflexion in addition to the inhibitory control by the vestibular apparatus.

Sixteen male Long–Evans Hooded rats weighing between 400–500 g were used. Eight rats were bilaterally labyrinthectomized. About a week later, all sixteen received bilateral damage to the LH. Details on these surgical procedures are presented elsewhere[1,2]. Prior to the LH damage, each rat was slowly lifted up from a standing position on the ground by the base of

* Present address: Department of Psychology, The University of Lethbridge, Lethbridge, Alberta, Canada, T1K 3M4.
Correspondence: S.M. Pellis, Dept. Psychology, The University of Lethbridge, Lethbridge, Alberta, Canada, T1K 3M4.

Fig. 1. Photographs of head and body postures adopted when held upside down by the tail. Labyrinths intact: A. Intact rat shown dorsiflexing the head and neck. This is typical of the vestibular head righting reflex from this position. C. LH-damaged rat on the day following surgery shown with the head sagging downward in the absence of vestibular head righting. Labyrinthectomized: B. Labyrinthectomized rat shown ventroflexing the head and neck. D. Labyrinthectomized, LH-damaged rat on the day following surgery is shown adopting an exaggerated ventroflexion including the shoulders and upper torso as well as the head and neck.

the tail until only gentle contact with the forepaws was left. Then the rat was lifted completely off the ground. The position of the head and body was recorded immediately before and after loss of contact. Each rat was tested 4 times. On the day following damage to the LH, each rat was tested again in the same manner. Photographs of head and body position were taken using a Canon AE programmable 35-mm camera. Details on the histological analysis of the LH damage in these rats are shown elsewhere[9].

Upon release of contact with the ground, intact rats dorsiflexed their heads (Fig. 1A), as is typical when the vestibular head righting reflex is tested in this manner[6]. In contrast, labyrinthectomized rats ventroflexed the head and the neck upon release of contact with the ground (Fig. 1B). On the day following LH damage, the vestibular head righting reflex was either weak or absent in rats with intact labyrinths (Fig. 1C). However, over the course of the next 2–3 days vestibular head righting reappeared in these LH damaged rats. Labyrinthectomy, in addition to damage of the LH, resulted in an exaggerated ventroflexion which included the upper torso, so that such rats bent ventrally into a tight U-shaped posture (Fig. 1D). These responses varied little among rats. All intact rats dorsiflexed their heads to the maximum amount possible — about 45° from vertical (Fig. 1A). Labyrinthectomized rats never dorsiflexed — all of them, to varying degrees ventroflexed. In the absence of LH-damage, labyrinthectomized rats tucked their heads ventrally no less than about 45° from vertical to a maximum of about 90° (as shown in Figure 1B). With the addition of LH-damage all the labyrinthectomized rats ventroflexed even more markedly, so that their snouts pointed skyward, making angular deviations of the snout from vertical of 135–180° (see Fig. 1D). LH-damage in the absence of labyrinthectomy never resulted in ventroflexion, but either showed no response (Fig. 1C), or they dorsiflexed. The absence of vestibular head righting in early post-recovery from LH damage may indicate that the disrupted pathways may have an excitatory function for this response. However, this is difficult to differentiate from the general somnolence which occurs following such damage[2].

These results suggest that supra-hypothalamic and possibly hypothalamic inhibitory controls exist, but that their impairment does not release the ventroflexion response while the labyrinthine inhibitory controls are present (Fig. 1C). However, when the labyrinthine controls are abolished by labyrinthectomy, then the impairment of these supra-hypothalamic controls becomes evident (Fig. 1D). Indeed, the weaker ventroflexion present with labyrinthectomy alone (Fig. 1B) suggests that the supra-hypothalamic systems are actively dampening the magnitude of such ventroflexion. Damage to the pontine reticular formation (PRF) also releases an exaggerated whole-body ventroflexion when such rats are held upside down or supine in the air[12]. Furthermore, electrolytic damage to the PRF produces a ventroflexion response which is as great or greater than that produced by combined labyrinthectomy and LH damage. Therefore, it is likely that such damage is disrupting the descending inhibitory pathways of both the vestibular and supra-hypothalamic systems.

In all cases, labyrinthectomized alone, labyrinthectomized with LH damage and PRF damage alone, contact of the forepaws on the ground when held upside down inhibited the ventroflexion. When such contact was interrupted, however, the head, neck and torso immediately ventroflexed. Such contact need not bear weight, as gentle digit contact was sufficient to inhibit the ventroflexion. Thus, forepaw contact on the ground appears to exert an inhibitory control over this proprioceptive whole-body ventroflexion. In the absence of such contact, inhibition of whole-body ventroflexion appears to depend upon the labyrinths and supraspinal neural systems.

Rats, like other mammals, adopt a ventroflexed posture in the uterus[11] which can persist for several days following birth[10]. It is possible that the ventroflexion reported in brain-damaged animals represents a release of this infantile behavior pattern. It appears that in rats this ventroflexed posture comes under inhibitory control in the first week postnatally[10]. The present paper indicates that several inhibitory controls may be involved, with tonic vestibular inhibition being the most important; the others are not evident unless in the absence of the labyrinths. Indeed, preliminary observations on neonatally bi-labyrintectomized neonate rats ($n = 4$) indicate that the whole body ventroflexion is exaggerated to the extreme seen in adult labyrinthectomized rats with LH damage (V. Pellis, S. Pellis and P. Teitelbaum, unpublished data). A developmental analysis of the onset of these systems would be enlightening in revealing their interactions in providing inhibitory controls over whole-body ventroflexion.

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1 Chen, Y.-C., Pellis, S.M., Sirkin, D.W., Potegal, M. and Teitelbaum, P., Bandage-backfall: labyrinthine and non-labyrinthine components, *Physiol. Behav.*, 37 (1986) 805–814.
2 Levitt, D. and Teitelbaum, P., Somnolence, akinesia and sensory activation of motivated behaviour in the lateral hypothalamic syndrome, *Proc. Nat. Acad. Sci. U.S.A.*, 72 (1975) 2819–2823.
3 Lyons, M.R., Hereditary absence of otoliths in the house mouse, *J. Physiol.*, 114 (1951) 410–418.
4 Magnus, R., *Körperstellung*, Springer, Berlin, 1924.
5 Magnus, R., On the co-operation and interference of reflexes from other sense organs with those of the labyrinths, *Laryngoscope*, 36 (1926) 701–713.
6 Monnier, M., *Functions of the Nervous System: Vol. II, Motor and Sensorimotor Functions*, Elsevier, Amsterdam, 1970.
7 Peiper, A., *Cerebral Function in Infancy and Childhood*, Consultants Bureau, New York, 1963.
8 Pellis, S.M., Pellis, V.C. and Teitelbaum, P., 'Axial apraxia' in labyrinthectomized lateral hypothalamic-damaged rats, *Neurosci. Lett.*, 82 (1987) 217–220.
9 Pellis, S.M., Pellis, V.C., Chen, Y.-C., Barzci, S. and Teitelbaum, P., Recovery from axial apraxia in the lateral hypothalamic labyrinthectomized rat reveals three elements of contact righting: cephalocaudal dominance, axial rotation and distal limb action, *Behav. Brain Res.*, 35 (1989) 241–251.
10 Pellis, V.C., Pellis, S.M. and Teitelbaum, P., A descriptive analysis of the postnatal development of contact-righting in rats (*Rattus norvegicus*), *Dev. Psychobiol.*, 24 (1991) (in press).
11 Robinson, S.R. and Smotherman, W.P., Chance and chunks in the ontogeny of fetal behavior. In W.P. Smotherman and S.R. Robinson (Eds.), *Behavior of the Fetus*, The Telford Press, Caldwell, NJ, 1988, pp. 95–115.
12 Sirkin, D.W., Schallert, T. and Teitelbaum, P., Involvement of the pontine reticular formation in head movements and labyrinthine righting in the rat, *Exp. Neurol.*, 69 (1980) 435–457.
13 Tait, J. and McNally, N.J., Some features of the action of the utricular maculae (Land of the associated action of the semi-circular canals) of the frog, *Physiol. Trans. Roy. Soc. Lond. (Biology)*, 224 (1934) 241–288.
14 Watt, D.G.D., Responses of cats to sudden falls: an otolith-originating reflex assisting landing, *J. Neurophysiol.*, 39 (1976) 257–265.