Physiology & Behavior, Vol. 21, pp. 817–820. Pergamon Press and Brain Research Publ., 1978. Printed in the U.S.A.

BRIEF COMMUNICATION

The Postures of Catecholamine-Depletion Catalepsy: Their Possible Adaptive Value in Thermoregulation[1]

TIMOTHY SCHALLERT[2]

Psychology Department, University of Illinois at Urbana-Champaign, Champaign, Illinois

IAN Q. WHISHAW

Psychology Department, University of Lethbridge, Lethbridge, Alberta, Canada

MARC DE RYCK AND PHILIP TEITELBAUM

Psychology Department, University of Illinois at Urbana-Champaign, Champaign, Illinois

(Received 26 May 1978)

SCHALLERT, T., I. Q. WHISHAW, M. DE RYCK AND P. TEITELBAUM. *The postures of catecholamine-depletion catalepsy: Their possible adaptive value in thermoregulation.* PHYSIOL. BEHAV. 21(5) 817–820, 1978.—Our view of the cataleptic akinesia induced by the disruption of catecholaminergic systems is that it is a state in which the postural and motor subsystems organized to maintain static stable equilibrium (e.g., standing or bracing, and righting) are functioning, whereas other subsystems such as those involved in exploration (walking, scanning, or orienting) and eating are inoperative. In addition to their role in actively maintaining stable equilibrium, the postures of cataleptic akinesia appear also to be related to thermoregulation. Slight skin warming in animals made cataleptic and akinetic following lateral hypothalamic lesions or intraventricular 6-hydroxydopamine causes a dramatic inactivation of tonic support (the subsystem involved in standing and bracing) and the animal subsides into a prone position. So profound is the torpor induced by warming that if the animal is slowly rolled over onto its back, it no longer rights itself. Furthermore, otherwise untreated normal rats made severely hypothermic by a period of immersion in cold water assume the crouched immobile postures of cataleptic akinesia seen in the uncooled, normothermic catecholamine-depleted animal. After a few minutes, shivering is superimposed on these postures. We suggest that the seemingly bizarre condition of cataleptic-akinesia can be viewed as a very simplified neural state organized to permit not only the regulation of stable equilibrium, but also shivering thermogenesis.

Catalepsy Akinesia Thermoregulation Shivering Catecholamines Lateral hypothalamic damage Stable equilibrium Evolutionary adaptation Motor subsystems

CATALEPSY is presently regarded as a bizarre symptom common to many pathological states: it is a feature of catatonic schizophrenia and of some forms of brain damage (e.g., basal ganglia damage [4,10] and other structures related to parkinsonism [13,15]). Many drugs used to alleviate some types of psychoses or hyperkinetic syndromes—such as phenothiazines, butyrophenones, and reserpine—all inactivate catecholamine systems in the brain. In excess, they produce symptoms of parkinsonism, including catalepsy and akinesia [6]. Several animal models of such catalepsy/akinesia have been developed, all of which disrupt catecholamine systems, either by drugs [2, 5, 9, 22, 23], by electrolytic hypothalamic damage [7, 11, 14, 17], or by 6-hydroxydopamine (6-OHDA) applied intraventricularly [18,20] or locally to ascending catecholaminergic pathways [12,24].

We believe that the catalepsy/akinesia induced by catecholamine depletion or blockade can fruitfully be viewed as a state in which some independent postural and motor subsystems are functioning, while others are not [19,21]. The cataleptic akinetic animal can readily use postural subsystems involved in maintaining static stable equilibrium. It rights itself in the air and on the ground; it clings, and thereby resists falling from an upright position of stability [3, 18, 23, 25]; and, by exaggerated bracing and supporting reactions (negativism; [26]), it actively resists horizontal dis-

[1]Supported by National Research Council of Canada Grant A8273 to Ian Q. Whishaw, and by NIH Grant RO1-NS-11671, University of Illinois Biomedical Research grant, and William T. Grant Foundation to Philip Teitelbaum. We thank Dr. Evelyn Satinoff and Dr. Diane L. Schallert for helpful comments, Nancy Peshkin for technical assistance, and Debie Kassner-Whelchel for typing the manuscript.

[2]Present address: Psychology Department, University of Arizona, Tucson, AZ 85719.

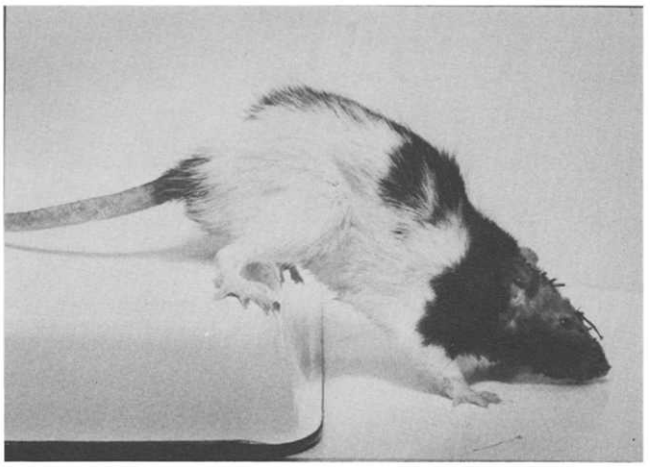

FIG. 1. Cataleptic posture in a rat. This rat was treated with intraventricular injections of 6-hydroxydopamine [18]. However, the posture is typical of that seen following disruption of catecholamine brain systems by other methods.

placement in space. At the same time, however, there is an inactivation of the neural subsystems involved in locomotion, head-orienting, head-scanning [7], and mouthing (biting and licking). In other words, in catalepsy/akinesia, the nervous system is organized to permit the animal to right, to stand still or brace against movement, but not to walk, explore, orient, or eat. As long as it is in a position of stable equilibrium the animal will tolerate bizarre postures without moving (see Fig. 1).

Might such postures be adaptive for other functions? The postures of catalepsy/akinesia serve not only to maintain stable support, they may also be useful in thermoregulation. We gained an insight into this relationship when we discovered that in lateral hypothalamic damaged animals that were akinetic and cataleptic, slight skin-warming caused a drastic loss of all support and righting reactions. For instance, after large (1 mA for 25–30 sec, anodal) bilateral lesions in the postero-lateral hypothalamus (see ref. [17] for histological analysis of lesions sufficient to produce the state of catalepsy/akinesia), the aphagic (Stage I [11]) animal may stand crouched and immobile (Fig. 2, top). In contrast to normal animals (which become more active in response to this stimulus), if the brain damaged animal (n=6) is warmed only slightly (1.5–2.0°C increase in subcutaneous temperature, with less than 1°C increases in its core temperature by a 250 watt radiant heat lamp 50 cm above it for 1–5 min), it loses all anti-gravity support and subsides into a prone position, inert and flat on the supporting surface (Fig. 2, middle). So profound is the torpor induced by warmth that if the animal is slowly rolled over onto its back, it no longer rights itself; instead, it lies motionless on the ground with its legs in the air for as long as 10 min (Fig. 2, bottom). Similar results were obtained in animals depleted of brain catecholamines after intraventricular application of 6-OHDA (n=6; see [18] for procedure). Thus, the residual systems remaining active during catalepsy/akinesia (i.e., those involved in righting and support) are drastically inactivated by warmth [19]. We can understand this phenomenon if we realize that the crouched immobile posture of catalepsy/akinesia closely resembles the thermoregulatory posture assumed by a normal rat as it huddles and braces for shivering in the cold. Indeed, after having been made severely hypothermic (body temperature as low as 27°C) by immersion for 4–6 min in cold water (10°C), the normal animal (n=6) is frozen in catalepsy—i.e., in the characteristic crouched, immobile postures of cataleptic akinesia seen in the uncooled, normothermic but catecholamine-depleted animal. It will also cling cataleptically (Fig. 3) (perhaps due to numbness of the limbs induced by the cold, some animals, particularly if they are heavy, do not support their entire weight well. Therefore, if the animal weighs more than about

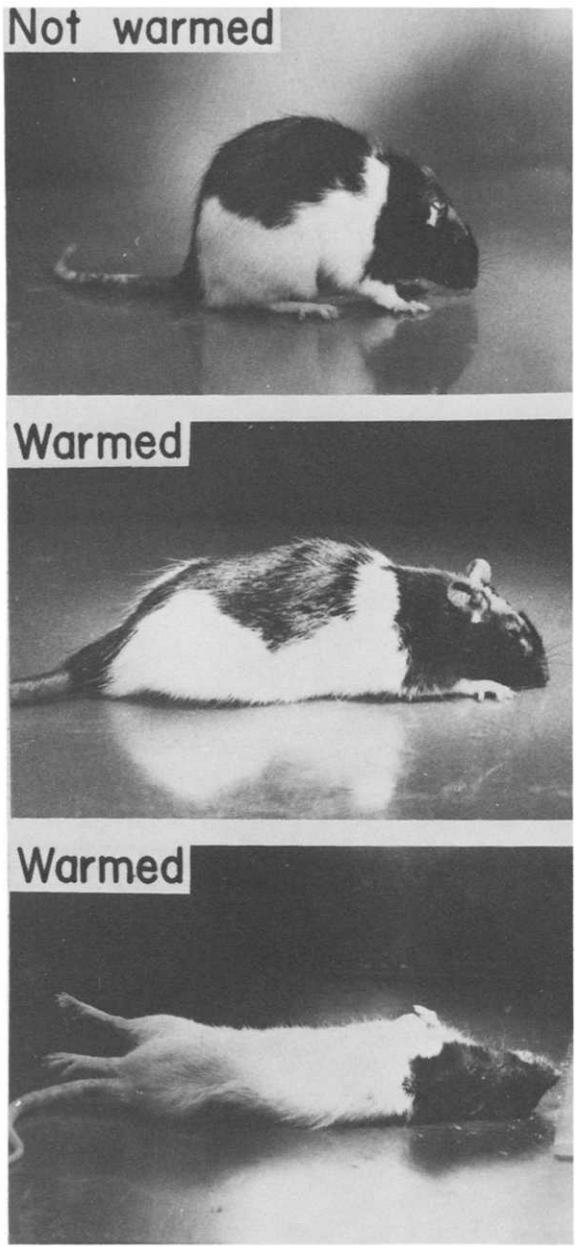

FIG. 2. (Top): The crouched, immobile posture of catalepsy/akinesia (in this rat, produced by electrolytic lateral hypothalamic damage). (Middle): Slight warming by a heat lamp causes it to lose support and subside into a prone posture. (Bottom): So profound is the torpor induced by such warmth that if the animal is slowly rolled over onto its back, it no longer rights itself.

FIG. 3. Prolonged clinging in a normal, hypothermic rat.

FIG. 4. Maintained cataleptic posture (similar to that shown in Fig. 1) in a normal rat made hypothermic.

250 g, its weight should be supported at the base of the tail while it is clinging, as shown in Fig. 3) and tolerate abnormal postures (Fig. 4) for long periods (more than 10 min) without moving. Shivering soon appears, superimposed on these postures.

We suggest that severe hypothermia (or the stress produced by it [1,27]) may inactivate brain catecholamine systems (involved in locomotion, orienting, and eating), reducing the animal to a profoundly simplified yet functionally adaptive state, in which the systems involved in crouching, bracing, standing, and righting predominate. These maintain stable equilibrium, but they are also involved in shivering—which generates the heat necessary to counteract the animal's hypothermia and thereby save its life. (At first consideration righting might not seem directly related to shivering. However, righting may be considered as "preparation for support;" and if support promotes more effective shivering, as we suggest here, then righting may be considered to be an allied reflex.)

In a masterful review of the hierarchical organization of the neural systems involved in temperature regulation [16], Satinoff has pointed out that such hierarchical control by levels of function must have evolved according to a principle of evolutionary co-adaptaton. For example, a posture that may have evolved [8] for one purpose (e.g., standing evolved for terrestrial locomotion and stability) later proved useful for shivering thermogenesis. The seemingly bizarre condition of catalepsy can therefore be viewed as a phylogenetically primitive state of neural organization primarily involving sensory and motor systems important for maintaining stable equilibrium and counteracting severe hypothermia.

REFERENCES

1. Bodner, R. J., D. D. Kelly and M. Glusman. Stress-induced analgesia: Time course of pain reflex alterations following cold water swims. *Bull. Psychon. Soc.* **11**: 333–336, 1978.
2. Costall, B. and R. J. Naylor. Neuroleptic and non-neuroleptic catalepsy. *Arzneimittel-Forsch.* **23**: 674–683, 1973.
3. De Jong, H. H. *Experimental Catatonia: A General Reaction Form of the Central Nervous System and its Implications for Human Pathology*. Baltimore: Williams and Wilkins, 1945.
4. Denny-Brown, D. Clinical symptomatology of disease of the basal ganglia. In: *Handbook of Clinical Neurology, Vol. 6*, edited by P. J. Vinken and G. W. Bruyn. Amsterdam: North-Holland, 1968, pp. 133–177.
5. Duvoisin, R. C. Cholinergic-anticholinergic antagonism in parkinsonism. *Archs Neurol.* **17**: 124–136, 1967.
6. Fog, R. *On Stereotypy and Catalepsy: Studies on the Effect of Amphetamines and Neuroleptics in Rats*. Copenhagen: Munksgaard, 1972.
7. Golani, I., D. L. Wolgin and P. Teitelbaum. A proposed natural geometry of recovery from akinesia in the lateral hypothalamic rat. *Brain Res.*, in press.
8. Heath, J. E. In: *Evolution and Environment*, edited by E. T. Drake. New Haven: Yale University Press, 1968, p. 259.
9. Hornykiewicz, O. Parkinsonism induced by dopaminergic antagonists. In: *Advances in Neurology, Vol. 9*, edited by D. B. Calne, T. N. Chase and A. Barbeau. New York: Raven Press, 1975, pp. 155–164.
10. Jung, R. and R. Hassler. The extrapyramidal motor system. In: *Handbook of Physiology, Vol. 2*, edited by J. Field. Washington, D.C.: American Physiological Society, 1960, pp. 863–927.
11. Levitt, D. R. and P. Teitelbaum. Somnolence, akinesia, and sensory activation of motivated behavior in the lateral hypothalamic syndrome. *Proc. natl. Acad. Sci. U.S.A.* **72**: 2819–2823, 1975.
12. Marshall, J. F., J. S. Richardson and P. Teitelbaum. Nigrostriatal bundle damage and the lateral hypothalamic syndrome. *J. comp. physiol. Psychol.* **87**: 808–830, 1974.
13. Martin, J. P. *The Basal Ganglia and Posture*. Philadelphia: J. B. Lippincott Co., 1967.
14. Robinson, T. E. and I. Q. Whishaw. Effects of posterior hypothalamic lesions on voluntary behavior and hippocampal electroencephalograms in the rat. *J. comp. physiol. Psychol.* **86**: 768–786, 1974.
15. Sacks, O. *Awakenings*. New York: Random House, 1976.
16. Satinoff, E. Neural organization and evolution of thermal regulation in mammals. *Science* **201**: 16–22, 1978.

17. Schallert, T. and I. Q. Whishaw. Two types of aphagia and two types of sensorimotor impairment following lateral hypothalamic lesions: Observations in normal-weight, dieted, and fattened rats. *J. comp. physiol. Psychol* **92:** 720–741, 1978.
18. Schallert, T., I. Q. Whishaw, V. D. Ramirez and P. Teitelbaum. Compulsive, abnormal walking caused by anticholinergics in akinetic, 6-hydroxydopamine-treated rats. *Science* **199:** 1461–1463, 1978.
19. Schallert, T., I. Q. Whishaw and P. Teitelbaum. Sources of spontaneity in the behavior of aphagic rats. International Conference on Physiology of Food and Fluid Intake. Jouy-en-Josas, France, 1977, Abstract.
20. Stricker, E. M. and M. J. Zigmond. Recovery of function after damage to central catecholamine-containing neurons: A neurochemical model for the lateral hypothalamic syndrome. In: *Progress in Psychobiology and Physiological Psychology,* edited by J. M. Sprague and A. N. Epstein. New York: Academic Press, 1976.
21. Teitelbaum, P., T. Schallert, I. Q. Whishaw, M. De Ryck and I. Golani. Motor subsystems in motivated behavior. U.S.A.–U.S.S.R. Symposium on the neurophysiological mechanisms of goal directed behavior and learning. Irvine, California, April, 1978.
22. Teitelbaum, P. and D. L. Wolgin. Neurotransmitters and the regulation of food intake. *Prog. Brain Res.* **42:** 235–249, 1975.
23. Teitelbaum, P., D. L. Wolgin, M. De Ryck and O. S. M. Marin. Bandage-backfall reaction: Occurs in infancy, hypothalamic damage, and catalepsy. *Proc. nat. Acad. Sci. U.S.A.* **73:** 3311–3314, 1976.
24. Ungerstedt, U. Brain dopamine neurons and behavior. In: *The Neuroscience Third Study Program,* edited by F. O. Schmitt and F. G. Worden. Cambridge: M.I.T. Press, 1974, pp. 695–703.
25. Van Harreveld, A. and J. E. Bogen. The clinging position of the bulbocapninized cat. *Expl Neurol.* **4:** 241–261, 1961.
26. Van Harreveld, A. and D. J. Kok. A propos de la nature de la catalepsie expérimentale. *Arch. Néerland. Physiol.* **20:** 411–429, 1935.
27. Weiss, J. M., H. I. Glazer and L. A. Poherecky. Coping behavior and neurochemical changes: An alternative explanation for the original "learned helplessness" experiments. In: *Animal Models in Human Psychobiology,* edited by G. Serban and A. Kling. New York: Plenum Press, 1976.