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Haloperidol, Catalepsy, and Equilibrating Functions in the Rat: Antagonistic Interaction of Clinging and Labyrinthine Righting Reactions

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SCHALLERT, T. AND P. TEITELBAUM. *Haloperidol, catalepsy, and equilibrating functions in the rat: Antagonistic interaction of clinging and labyrinthine righting reactions.* PHYSIOL. BEHAV. 27(6) 1077–1083, 1981.—The neuroleptic drug haloperidol, which interferes with catecholaminergic activity in the brain, was used to produce akinesia and catalepsy in rats. Such akinesia and catalepsy can be viewed as one in which a subsystem of integrated motor functions (e.g., righting, standing still, clinging, and bracing), is operating to achieve and defend static stable equilibrium, whereas other movement subsystems such as scanning, orienting, and quadrupedal locomotion are meanwhile inoperative. Thus, the catecholamine-deficient rat is a useful preparation to study the isolated components of static stable equilibrium and their interaction, without the complexities encountered by the operation of alternative movement subsystems. Righting in the air was completely inhibited by clinging, no matter how ineffective such clinging was in actually providing physical support. High speed film analysis and lightweight wire-mesh frames of varying stability were used to determine the minimal stimuli that inhibit righting. We suggest that an antagonistic relationship exists between clinging and righting, in which the inputs involved in clinging are dominant over (and may be potentiated by) the vestibular inputs involved in labyrinthine righting. In addition, the bandage-backfall phenomenon, a hitherto bizarre reaction pattern that can be elicited in cataleptic adult animals and normal infants, can be explained by a related antagonistic interaction.

Catalepsy Haloperidol Catecholamine deficiency Motor subsystem Vestibular righting Supporting reactions Bandage-backfall Parkinsonism

AKINESIA is a symptom of brain dysfunction and is a common feature of Parkinsonism and related disorders in people [3, 4, 5, 6, 11, 13, 17, 20, 23]. A possible model of akinesia is readily induced in animals by brain damage (especially involving the nigrostriatal system) or by drugs that interfere with catecholaminergic activity [1, 8, 9, 19, 22, 27, 35, 36]. An akinetic animal stands awake but unmoving for long periods and, depending on the test, may maintain awkward postures in which it is placed by the experimenter. For example, a rat treated with the neuroleptic drug haloperidol, which in low doses blocks dopaminergic receptors, will long tolerate an upright leaning posture with its forepaws placed up on a horizontal bar (catalepsy) [1, 2, 7].

Such behavior may seem quite bizarre, but in recent work on drugged or brain-damaged animals, we have described movement subsystems that can be experimentally isolated from each other, and we have pointed out that the independent action of these subsystems, or their interaction in various combinations, can help account for symptoms such as akinesia, stereotyped behavior, and aberrant locomotion [7, 10, 24, 25, 26, 31]. For instance, we proposed that dopamine-deficiency catalepsy is a condition in which the

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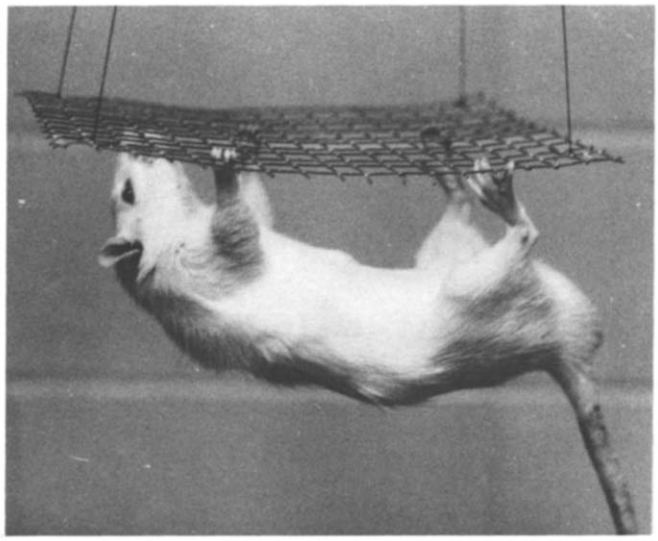

FIG. 1. A haloperidol-treated rat clings upside down to a rectangular grid. When the grid was released, the rat failed to right.

movement subsystem involved in maintaining stable static postural support and equilibrium is operating, whereas other movement subsystems (involved in locomotion, orienting, scanning, and mouthing) are inoperative. We suggested that such catalepsy represents an extremely simplified behavioral state, in which one can more easily examine the variables controlling the subsystem that remains active, without the complexities introduced by its interaction with other subsystems. Thus, if a haloperidol-treated akinetic rat is held supine but well supported in the air, it remains unmoving. However, as soon as the support provided by the experimenter's hands is removed, and the animal begins to fall, it immediately rights itself by lateral rotation of head and body, it extends its legs, and achieves in the air a "readiness-to land" prone posture [7, 18, 29], in which it falls to the ground. Once on the ground, where stable support is again provided by its legs, it remains standing, motionless, as long as the drug remains fully effective. Thus it appears that the motor program for labyrinthine (otolithic) righting, which may be organized in the mid-brain at the level of the oculomotor and red nuclei [18], or paramedian pontine reticular formation [29], is catecholamine-independent in the rat (although in primates catecholaminergic activity in the basal ganglia may play a more critical role [16]).

In the present paper, using haloperidol-treated akinetic rats, we show that righting in the air while falling is completely inhibited if the animal is given something rigid to cling to, no matter how ineffective such clinging actually is in providing physical support. We suggest that an antagonistic interaction exists between clinging and righting, in which the tactile-kinesthetic stimuli involved in clinging are dominant over the vestibular stimuli involved in righting in the air. In addition, we suggest that the bandage-backfall reaction, seen in cataleptic adult animals and normal infants [7, 30, 32, 33], may be explained by a similar inhibitory interaction.

EXPERIMENT 1

We initially discovered this phenomenon when we were studying the clinging reaction of haloperidol-treated rats (n=10). Following the administration of 2–5 mg/kg haloperidol, all rats reliably clung, upside down without moving, to a rectangular wire-mesh grid (22×15 cm, weighing 38 g), held 50 cm above the ground (Fig. 1). Undrugged normal animals (n=10), or rats treated with less than 0.5 mg/kg haloperidol, scanned, oriented, let go of the grid or quickly climbed forward and upward until they reached the upper horizontal surface of the grid where they moved around. Thus, only the sufficiently drugged rats were easily tested. If the entire grid was dropped, the haloperidol-treated rats remained supine, clinging to the grid all the way down until they landed on their backs (a soft, 6 cm thick foam rubber pad was used to cushion the impact and prevent injury). Each haloperidol-treated rat also was dropped from a height of 2 meters while being filmed using an 8 mm movie camera set at 40f/sec, and all were caught in a supine position just before hitting the ground. Without the grid present, they righted immediately (within 20 cm of free fall), and were caught in a prone position.

EXPERIMENT 2

In this experiment, we attempted to quantify the effects of clinging or non-clinging on labyrinthine righting in saline-treated versus haloperidol-treated rats.

METHOD

Animals

Twenty-four male Sprague-Dawley rats from the Animal Resource Center of The University of Texas were used. They weighed 310–450 g, and were housed individually in wire-mesh hanging cages in a temperature-controlled room (26°C) under a 12 hour light-dark cycle. All testing was carried out during the light period.

Drug

Haloperidol (McNeil) was dissolved in distilled water with an equal weight of tartaric acid (Sigma) in solutions of 0.5 mg/ml, 2 mg/ml, 4 mg/ml, and 8mg/ml.

Procedure

One group of 6 animals was injected intraperitoneally with 2 mg/kg of haloperidol. A control group received an equal volume of saline. Fifteen minutes later, animals from both groups were turned upside down and either allowed to grasp the 22×15 cm rectangular wire-mesh grid (Experiment 1) with all four paws (grid condition), or not (no grid condition). The order of conditions was balanced. The animals were then dropped a maximum of 100 cm before they were caught. The distance (in cm) needed to right the entire body 180° was recorded. The results were analyzed using a two-factor mixed ANOVA.

Following this first phase of the experiment, a preliminary dose-response procedure was carried out in which the animals in the saline control group received 0.5 mg/kg of haloperidol, and the animals in the haloperidol group received an additional 2 mg/kg, which brought their cumulative dose to approximately 4 mg/kg. Both groups were re-tested 15 minutes later using the above righting procedure. Next, the original control group was given an additional 0.5 mg/kg haloperidol, (which brought their cumulative dose to 1 mg/kg) and the original haloperidol group was given an additional 4 mg/kg of haloperidol (which brought their cumulative dose to 8 mg/kg). Both groups again were tested 15 minutes

TABLE 1

FREE FALL DISTANCE UNTIL RIGHTING, OR NON-RIGHTING AT MAXIMUM HEIGHT OF 100 cm IN RATS TREATED WITH SALINE OR HALOPERIDOL (2 mg/kg) WITH OR WITHOUT THE OPPORTUNITY TO CLING PRIOR TO BEING RELEASED SUPINE IN THE AIR

	Saline (n=6)	Haloperidol (n=6)
No Grid	16.7 ± 0.4 (17.0/15–18)	20.0 ± 1.2 (20.5/16–24)
Grid	50.5 ± 15.8 (30.5/19–100)	100 ± 0* (100/100–100)

Data are in cm (means ± SE and, in parentheses, median/ranges).
*Significantly different from No Grid condition in ANOVA post-hoc test ($p<0.01$).

later using the above righting procedure. Finally, the original haloperidol group was given an additional 8 mg/kg (which brought their cumulative dose to approximately 16 mg/kg).

RESULTS

As shown in Table 1, under haloperidol (2 mg/kg) air righting was greatly inhibited in the grid condition compared with the no grid condition ($p<0.01$).The opportunity to cling supine to a grid just prior to and during free fall was associated with an inhibition of righting. Although the drug × grid condition interaction was not significant, $F(1,10)=3.04$, n.s., it was clear that without haloperidol the effect was not reliably obtained. In some animals, however, clinging and immobility readily occurred. In these rats, righting was reliably inhibited during free fall in the presence of the grid.

At all other doses of haloperidol, clinging occurred which significantly ($p<0.05$) inhibited righting. The dose-response data, summarized, for the no-grid vs grid conditions were, respectively: 0.5 mg/kg, 17.0±0.5 vs 75±9.0; 1 mg/kg, 19.8±0.8 vs 100±0; 2 mg/kg, 20.0±1.2 vs 100±0; 4 mg/kg, 24.8±2.0 vs 100±0; 8 mg/kg, 27.3±0.4 vs 100±0; 16 mg/kg, 33.1±1.2 vs 100±0. In the no grid condition, haloperidol significantly slowed (but did not block) righting at 8 and 16 mg/kg ($p<0.01$).

EXPERIMENT 3

Details of behavior during free fall, particularly the righting reaction itself, were too rapid to analyze adequately even at 40 f/sec. To "magnify time" [14], we used a 16 mm LO-CAM high speed movie camera (with Estar base black and white film) to record the animals' reactions under various experimental conditions. Filming was done at speeds ranging from 24 to 493 f/s. A stop-frame flicker-free electro-optical motion analyzer (L & W No. 224 MK IV) was used for frame by frame analysis of the animals' movements.

To analyze the essential elements of the stimuli provided by the wire-mesh grid, we devised a set of fractional wire frames of various size and configuration, and tested their effectiveness in inhibiting righting by eliciting clinging while falling. In the first situation, four 4×4 cm squares constructed of 6 mm wire-mesh were interconnected by rigid hollow wires (3 mm in diameter) to form a simple 10×16 cm rectangular frame weighing 35 g (Fig. 2) which was suspended by threads attached to each square. Each paw of the animal clung to one of the grid mesh squares (F-F=H-H). This apparatus was sufficient to block righting in every

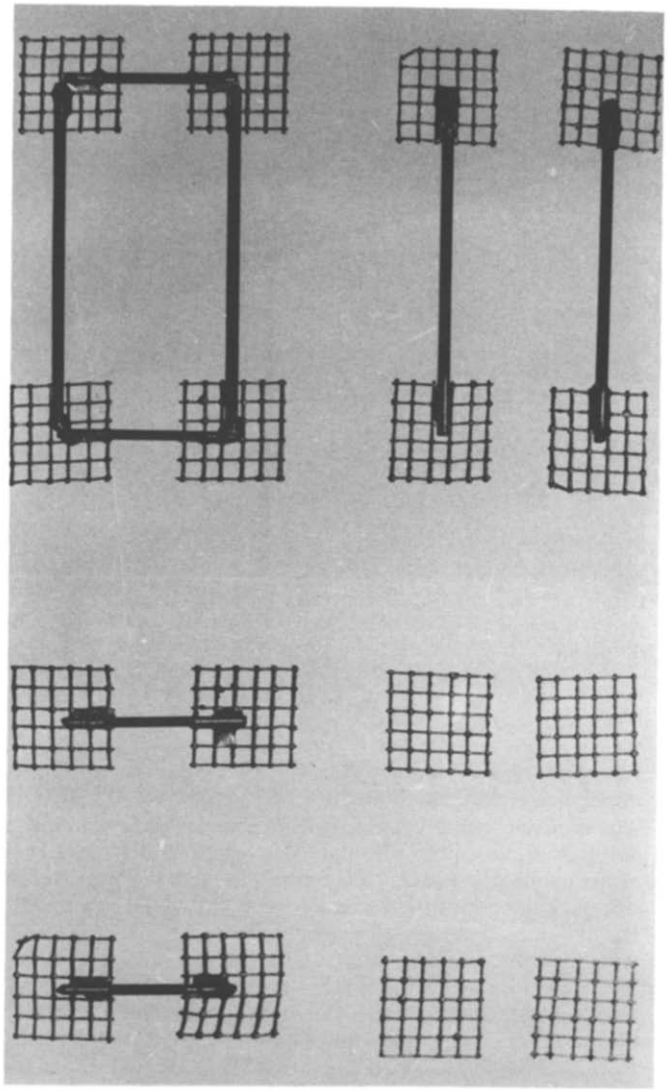

FIG. 2. Four sets of 4 lightweight wire-mesh squares, with different interconnections, could be grasped by haloperidol-treated animals, permitting systematic manipulation of stability among the limbs during free fall. Upper left: all four squares are interconnected by rigid wires to provide maximum interlimb stability and false support (F-F=H-H). Upper right: the squares are connected longitudinally to provide ipsilateral (LF-LH/RF-RH) or crisscrossed (LF-RH/RF-LH) stability between the fore and hind limbs. Lower left: the squares are connected to provide stability between each of the forelegs (F-F), between each of the hindlegs (H-H), or both (F-F/H-H). Lower right: the squares are not connected, providing no interlimb stability (F/F/H/H).

haloperidol-treated rat (n=6) in all ten trials (5 mg/kg). As soon as the frame was released and began to fall, each animal pulled the frame close to its body (e.g., Fig. 3), which demonstrates that the animal actively clung as it fell in its inverted posture.

Having demonstrated that a small and lightweight rectangular frame was sufficient to inhibit righting, we systematically varied the degree of stability of the frame by removing one or more of the hollow interconnecting wires (Fig. 2). The short horizontal segments of the rectangle (perpendicular to the longitudinal axis of the rat's body) were

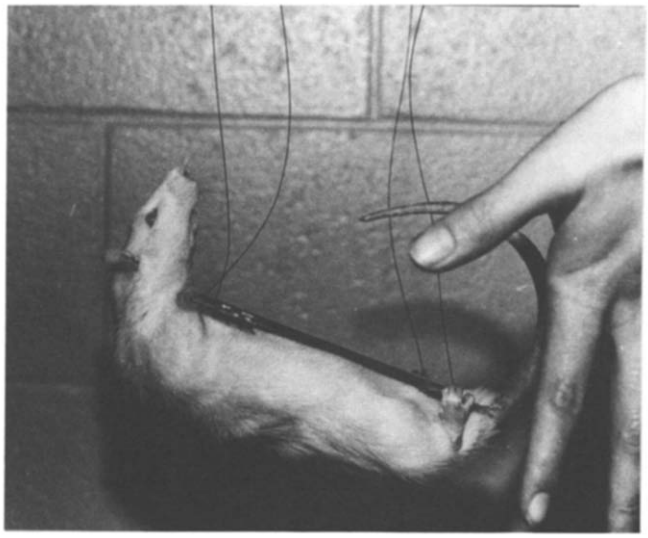

FIG. 3. A haloperidol-treated rat, which otherwise rights normally, fails to right when it is dropped while clinging to four interconnected grid squares (F-F=H-H). Note the exaggerated flexion in the limbs during free fall.

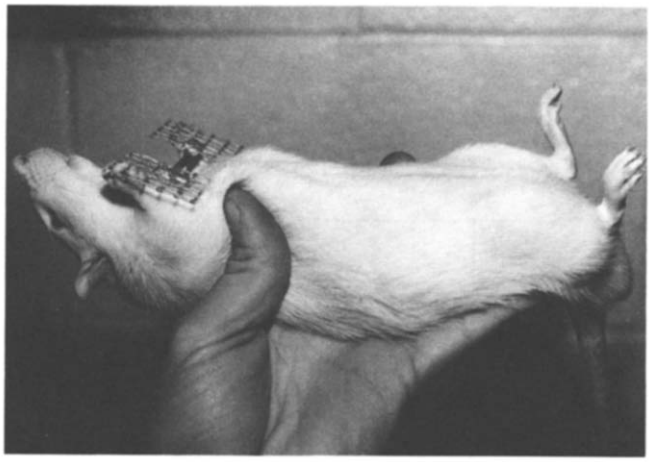

FIG. 4. A haloperidol-treated rat grasps two interconnected squares with its forepaws (F-F) and stays immobile while being held supine.

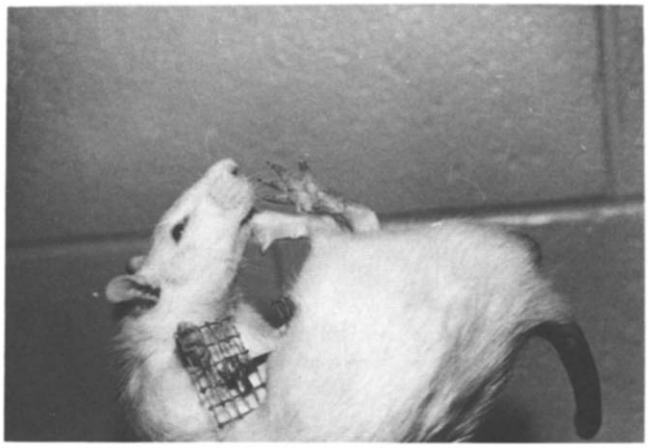

FIG. 5. A haloperidol-treated rat fails to right when it is dropped while clinging to two grid squares interconnecting the forelimbs (F-F). Note the exaggerated ventroflexion, which in this case leads to putting a hindfoot in the mouth.

removed, and the animal was left clinging to four squares connected rigidly only longitudinally between front and back legs on each side (LF-LH/RF-RH). Despite the reduction of stability, righting was still inhibited (tested in all 6 animals 10 times each). However, as soon as the animal began to fall, not only were the frame fractions instantly pulled up against the abdomen, but they were also pulled medially so that they were in tight contact with each other during the entire fall. Thus, in contrast to the unbroken rectangular frame condition, the animal fell with its forelegs together and its hindlegs likewise. This suggests that flexion is actually increased toward the midline when the animal falls, an effect not observed in the unbroken rectangular frame condition. A similar effect occurred when these longitudinally-connected frame fractions were crisscrossed so that each foreleg was connected to the hindleg of the opposite side of the body (LF-RH/RF-LH).

Next, the longitudinal segments were removed instead of the horizontal segments. The front paws were left clinging to an identical pair, with no connection between front and back legs (F-F/H-H). In this case also, righting was inhibited; however, as soon as the animal began to fall, flexion occurred at the trunk (a behavior that resembled the initial component of normal righting [7]), which carried the two pairs of grid squares into contact or near contact with each other. As shown in Fig. 4, if the hindleg pair of squares was then removed (F-F), the animal was left clinging only with its front paws to two rigidly connected wire-mesh squares (total weight=10 g). When the animal was released, it still failed to right. Instead it flexed sharply ventrally, sometimes so tightly that its hindlegs and face contacted each other. Figure 5 shows an instance in which a released animal put its foot in its mouth on the way down.

All of the above conditions, which triggered clinging involving the forepaws, no matter how insubstantial the actual support provided, were sufficient to inhibit righting in the air while falling. We also tested whether righting would be inhibited if only the hindlegs were interconnected (H-H). It was not. The animal righted normally, beginning with a rotation of the head and forequarters, followed by the hindquarters [7, 15, 18, 21]. The fractional frame was simply flung away during the righting sequence. However, on one occasion an animal's starting posture was too far flexed, and immediately upon release it managed to grasp the frame that interconnected the hindlegs. This abruptly halted righting and the animal landed on the dorsal surface of its hindquarters.

Finally, if none of the four squares were interconnected (F/F/H/H), clinging did not occur. One square was put onto each of the four paws of the rat, which was held supine. Even if the toes of each forepaw were curled around the wire-mesh, when the rat was dropped normal righting occurred and the grid squares were flung away.

EXPERIMENT 4

In this experiment we attempted to quantify the interfering effects of the various configurations of small grid squares on labyrinthine righting using a range of doses of haloperidol.

TABLE 2

FREE FALL DISTANCE UNTIL RIGHTING, OR NON-RIGHTING AT MAXIMUM HEIGHT OF 100 cm, IN RATS TREATED WITH DIFFERENT DOSES OF HALOPERIDOL AND DROPPED SUPINE WHILE CLINGING TO VARIOUS CONFIGURATIONS OF SMALL GRID SQUARES (MEANS ± SE; RANGE)

	Haloperidol Dose (mg/kg)			
	0 (n=6)	1 (n=6)	2 (n=6)	4 (n=6)
(F-F)=(H-H)	44.0 ± 14.8 (19–100)	87.9 ± 13.0* (22–100)	100 ± 0*† (100–100)	100 ± 0*† (100–100)
F/F/H/H	18.8 ± 1.0 (16– 23)	19.7 ± 0.8 (17– 22)	21.5 ± 1.3 (16–25)	26.0 ± 1.8† (18–30)
F-F	40.3–12.5 (18–100)	56.7 ± 9.8* (32–100)	100 ± 0*† (100–100)	100 ± 0*† (100–100)
H-H	19.8 ± 1.4 (18– 26)	34.5 ± 13.2 (16–100)	20.8 ± 1.9 (17–30)	27.0 ± 6.7 (16–60)
F-F/H-H	42.3 ± 12.8 (19–100)	74.3 ± 12.2* (31–100)	100 ± 0*† (100–100)	100 ± 0*† (100–100)
LF-LH/RF-RH	37.2 ± 12.9 (18–100)	58.5 ± 9.3* (33–100)	100 ± 0*† (100–100)	100 ± 0*† (100–100)
LF-RH/RF-LH	22.3 ± 10.6 (17–100)	53.6 ± 8.7* (24–100)	87 ± 6.5*† (28–100)	100 ± 0*† (100–100)

Forelimb contacts square (F); hindlimb contacts square (H); rigid interconnection (-); unconnected (/); left (L); right (R).
*Significantly different from F/F/H/H ($p<0.05$).
†Significantly different from 0 mg/kg ($p<0.05$).

METHOD

Animals

Twenty-four Sprague-Dawley rats from The Animal Resource Center at The University of Texas were used. They weighed 324–465 g, and were group housed, four to a cage.

Drugs

Haloperidol was dissolved in distilled water with an equal weight of tartaric acid in solutions of 0, 1, 2, and 4 mg/ml.

Procedure

Rats were injected intraperitoneally with either 4 mg/kg haloperidol (n=6), 2 mg/kg (n=6), 1 mg/kg (n=6), or 0 mg/kg (n=6). Fifteen minutes later, the animals were turned upside down and, by gently rocking them, they were induced to grasp in counterbalanced order one of seven configurations of the small grid squares (described in Experiment 3 and shown in Fig. 2). The animals were then dropped a maximum of 100 cm before they were caught. The distance needed to right was recorded.

RESULTS

As shown in Table 2, righting was inhibited most effectively when the animals were permitted to cling to a stable configuration which provided a rigid connection between the forepaws (F-F), or between the forepaws and the hindpaws in various combinations as described in Experiment 3. This effect was significant at all three doses of haloperidol. Without haloperidol, the effect was seen in some rats but not in others (note large SEs and ranges in Table 2 at 0 mg/kg).

GENERAL DISCUSSION

To our knowledge, this is the first demonstration of the inhibition of righting in the air by clinging. In terms of our previous analyses of the functional adaptiveness of the postures of akinesia or catalepsy in catecholamine deficient animals [7, 24, 25, 26, 31] it makes sense: the support subsystem operates by standing, bracing, or clinging reactions, which are integrated to achieve stable static equilibrium [3, 7, 15, 20, 21, 25, 26, 28, 33, 34]. When falling from the supine position, righting is a labyrinthine reaction that leads to a readiness-to-land posture preparatory for support [7,18]. If clinging is triggered, however, this signals that support is present (falsely, if the substrate being clung to is also falling), and the vestibular signal produced by falling no longer is effective in triggering righting—the animal does not inhibit its clinging reaction, nor is head and body rotation initiated. (Head rotation is physically possible, but this does not occur). Therefore clinging inhibits righting.

In the undrugged animal, the effect is difficult to obtain, perhaps because there is more variability in its behavior, which includes movements not directly involved in maintaining static stable equilibrium. However, if the animal is firmly clinging with its forelegs and hindlegs, and if its head is not turned at the moment it is dropped it often does not right and falls to the ground on its back while clinging as does the akinetic/cataleptic animal (clinging is an antigravity response [18], and clinging while falling may be an adaptive reaction in that it simulates grasping an unstable object such as a thin branch of a tree, which ordinarily would gradually decelerate and break an animal's fall). There appears to be a difference in the minimal stimulus that will trigger clinging and thus inhibit righting in normal animals. The smaller and more un-

stable stimuli with little inertia are less likely to promote clinging. However, in the haloperidol-treated animal, which lacks alternative response subsystems, even lightweight, relatively unstable objects (e.g., a piece of soft foam rubber 3 cm^3) induces intense clinging readily. In other words, the increased readiness for tonic clinging, not the inhibition of righting by support, may be the primary dose-related effect of haloperidol. The inhibitory interaction of clinging and righting seen in the cataleptic animal may be a normal reaction, but is one that is more readily studied in the simple isolated support subsystem which alone remains active with catecholamine deficiency.

Schallert (unpublished data, 1981) has since analyzed the interaction of supporting and righting reactions in water, another medium in which a rat depends on its vestibular apparatus [18]. For instance, haloperidol-treated animals were turned upside down in warm water and were given various mesh frames to cling to. The frame was positioned such that only the very tip of each animal's snout was above the water line to allow breathing. As in free fall, water righting reactions, which are otherwise intact, were totally inhibited by clinging, and the animal stayed immobile upside down in the water. Undrugged rats let go of the frame immediately and righted themselves.

In a preliminary analysis, we tested whether clinging can directly inhibit the readiness-to-land reaction that is characteristic of free fall in the prone position [7, 15, 18]. The basic posture includes tonic extension of the neck and all four limbs, with the toes spread. Haloperidol-treated animals stand motionless on the rectangular frame, and when dropped, cling rather than adopting the readiness-to-land posture (they showed only a very slight upper limb extension). Using the fractional frames, we found that extension in each foreleg could be inhibited independently, which was not true for the hindlegs. If a flat board is used instead of the wire grid, clinging does not occur and therefore no support is present when the board is dropped. In this case, the readiness-to-land posture instantly appears [7]. Indeed, limb extension (pushing) is so striking that the flat board always becomes separated from the animal and accelerates downward faster than the animal falls. Thus, to inhibit both righting and the related vestibular reaction preparatory to landing, it seems necessary that support stimuli be detectable in the animal's forequarters.

The bandage-backfall reaction, seen in cataleptic adult animals and normal infants, may now be understood in the same way: if an adult cataleptic animal is placed on a vertical surface to which it can cling(for cats, the back of a chair; for rats, or cats, a pair of vertically spaced horizontal bars or a vertical wire mesh grid), it will cling unmoving for long periods, keeping its head and neck erect [34]. However, if its head and neck are bandaged, then the head falls slowly backward, the neck becomes hyper-extended, the clinging reaction is inhibited, and the animal falls backward to the ground without righting as it falls [7, 30, 32, 33, 36]. The reaction does not depend on lack of vision; it occurs even if the eyes are not covered by the bandage. Pressure on the face by the bandage inhibits the upright posture of the head and neck—the reaction does not occur if the sensory fibers of the trigeminal and the first three cervical nerves are cut. We suggest that the bandage pressure on the face simulates the support that would be provided if the face were actually resting its weight on a surface. Such pressure signals support, thereby inhibiting the vestibular control of support of head and neck that would otherwise operate to keep the head erect [15].

As with the antagonistic interaction of clinging and air righting, bandage pressure on the head and neck can inhibit righting or produce the backfall reaction sometimes in normal undrugged rats and cats (Schallert, DeRyck and Teitelbaum, unpublished data). However, the interaction likewise is difficult to study in the normal animal because the animal usually scans, orients, climbs or jumps away.

In conclusion, we suggest that a hierarchical relationship exists between clinging and righting, in which the sensory inputs or motor feedback involved in clinging are dominant over (and appear to be potentiated by) the vestibular inputs involved in labyrinthine righting. We were able to analyze this relationship by administering the catecholaminergic blocking agent haloperidol, which relatively selectively suppresses behaviors that are not directly involved in achieving or maintaining static stable equilibrium. Dopamine blocking agents of the postsynaptic type may prove to be useful for studying the interactions of catecholamine-independent equilibrating functions, or dysfunctions, in general.

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REFERENCES

1. Costall, B. and R. J. Naylor. Neuroleptic and non-neuroleptic catalepsy. *Arzneimittel-Forsch.* **23:** 674–683, 1973.
2. Costall, B. and R. J. Naylor. On catalepsy and catatonia and the predictability of the catalepsy test for neuroleptic activity. *Psychopharmacologia* **34:** 233–241, 1974.
3. De Jong, H. H. *Experimental Catatonia. A General Reaction-Form of the Central Nervous System and Its Implication for Human Pathology.* Baltimore: Williams and Wilkins, 1945.
4. Delay, J. and P. Deniker. Drug-induced extrapyramidal syndromes. In: *Handbook of Clinical Neurology, Vol. 6, Diseases of the Basal Ganglia,* edited by P. J. Vinken and G. W. Bruyn. Amsterdam: North Holland, 1968, pp. 248–266.
5. Denny-Brown, D. *The Basal Ganglia and Their Relation to Disorders of Movement.* London: Oxford University Press, 1962.
6. Denny-Brown, D. Clinical symptomatology of diseases of the basal ganglia. In: *Handbook of Clinical Neurology, Vol. 6, Diseases of the Basal Ganglia,* edited by P. J. Vinken and G. W. Bruyn. Amsterdam: North Holland, 1968, pp. 133–172.
7. De Ryck, M., T. Schallert and P. Teitelbaum. Morphine versus haloperidol catalepsy in the rat: A behavioral analysis of postural support mechanisms. *Brain Res.* **201:** 143–172, 1980.
8. Duvoisin, R. C. Parkinsonism: Animal analogues of the human disorder. In: *The Basal Ganglia,* edited by M. D. Yahr. New York: Raven Press, 1976, pp. 293–303.

9. Fog, R. On stereotypy and catalepsy: Studies on the effect of amphetamines and neuroleptics in rats. *Acta neurol. scand.* **50:** 1–66, 1972.
10. Golani, I., D. L. Wolgin and P. Teitelbaum. A proposed natural geometry of recovery from akinesia in the lateral hypothalamic rat. *Brain Res.* **164:** 237–267, 1979.
11. 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.
12. Jung, R. and R. Hassler. The extrapyramidal motor system. In: *Handbook of Physiology, Vol. 2, Section 1: Neurophysiology,* edited by J. Field. Washington, DC: American Physiological Society, 1960, pp. 863–927.
13. Kolb, B. and I. Q. Whishaw. *Fundamentals of Human Neuropsychology.* San Francisco: W. H. Freeman, 1980.
14. Landis, C. and W. A. Hunt. Magnification of time as a research technique in the study of behavior. *Science* **85:** 384–385, 1937.
15. Magnus, R. Some results of studies in the physiology of posture. *Lancet* **211:** 531–536 and 585–588, 1926.
16. Martin, J. P. *The Basal Ganglia and Posture.* Philadelphia: J. B. Lippincott, 1967.
17. Matthysse, S. Schizophrenia: Relationships to dopamine transmission, motor control, and feature extraction. In: *The Neurosciences, Third Study Program,* edited by F. O. Schmitt and F. G. Worden. Cambridge: MIT Press, 1974, pp. 733–737.
18. Monnier, M. *Functions of the Nervous System,* Vol. 2. Amsterdam: Elsevier, 1970, pp. 369–431.
19. Pedigo, N. W., T. D. Reisine, T. Schallert, D. H. Overstreet, N. C. Ling, P. Ragan and H. I. Yamamura. Possible role of opiate receptors and des tyrosine-γ-endorphin in schizophrenia. In: *Advances in Biochemical Psychopharmacology,* Vol. 22, edited by E. Costa and M. Trabucchi. New York: Raven Press, 1980.
20. Peters, F. Pharmakologische Untersuchungen uber Corydalisalkaloide. *Arch. exp. Path. Pharmak.* **51:** 130–174, 1904.
21. Rademaker, G. G. J. *Das Stehen.* Berlin: Julius Springer, 1931.
22. Richter, C. P. and A. S. Paterson. Bulbocapnine catalepsy and the grasp reflex. *J. Pharmac. exp. Ther.* **43:** 677, 1931.
23. Sacks, O. *Awakenings.* New York: Vintage Books, 1976.
24. Schallert, T., M. DeRyck and P. Teitelbaum. Atropine stereotypy as a behavioral trap: A movement subsystem and electroencephalographic analysis. *J. comp. physiol. Psychol.* **94:** 1–24, 1980.
25. Schallert, T., M. DeRyck, I. Q. Whishaw, V. D. Ramirez and P. Teitelbaum. Excessive bracing reactions and their control by atropine and L-Dopa in an animal analog of Parkinsonism. *Expl Neurol.* **64:** 33–43, 1979.
26. Schallert, T., I. Q. Whishaw, M. DeRyck and P. Teitelbaum. The postures of catecholamine-depletion catalepsy: Their possible adaptive value in thermoregulation. *Physiol. Behav.* **21:** 816–820, 1978.
27. 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.
28. Schaltenbrand, G., Beitrage zur Pharmakologie der Korperstellung und der Labyrinthreflexe. XIV. Mitteilung. Die Wirkung des Bulbocapnins aud unverletzte Katzen. *Pflügers Arch. ges. Physiol.* **209:** 623–642, 1925.
29. Sirkin, D. W., T. Schallert and P. Teitelbaum. Involvement of the pontine reticular formation in head movements and labyrinthine righting in the rat. *Expl Neurol.* **69:** 435–457, 1980.
30. Teitelbaum, P. The physiological analysis of motivated behavior. In: *Psychology and Life,* 9th ed. Glenview, IL: Scott, Foresman, 1977.
31. Teitelbaum, P., T. Schallert, M. DeRyck, I. Q. Whishaw and I. Golani. Motor subsystems in motivated behavior. In: *Neural Mechanisms of Goal-Directed Behavior and Learning,* edited by R. F. Thompson, L. H. Hicks and V. B. Shvyrkov. New York: Academic Press, 1980, pp. 127–143.
32. Teitelbaum, P., D. L. Wolgin, M. DeRyck and O. S. M. Marin. Bandage-backfall reaction: Occurs in infancy, hypothalamic damage, and catalepsy. *Proc. natn. Acad. Sci. U. S. A.* **73:** 3311–3314, 1976.
33. Van Harreveld, A. and J. E. Bogen. The clinging position of the bulbocapninized cat. *Expl Neurol.* **4:** 241–261, 1961.
34. Van Harreveld, A. and D. J. Kok. A propos de la nature de la catalepsie experimentale. *Archs néerl. Physiol.* **20:** 411–429, 1935.
35. Whishaw, I. Q., T. E. Robinson, T. Schallert, M. DeRyck and V. D. Ramirez. Electrical activity of the hippocampus and neocortex in rats depleted of brain dopamine and norepinephrine: Relations to behavior and effect of atropine. *Expl Neurol.* **62:** 748–767, 1978.
36. Wolgin, D. L. and P. Teitelbaum. Role of activation and sensory stimuli in recovery from lateral hypothalamic damage in the cat. *J. comp. physiol. Psychol.* **92:** 474–500, 1978.