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Motivational–Sensorimotor Interaction Controls Aphagia and Exaggerated Treading After Striatopallidal Lesions

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This study examined the relationship between sensorimotor and motivational functions of the corpus striatum. In rats, excitotoxic lesions of the corpus striatum (neostriatum and globus pallidus) caused by kainic or quisqualic acid can produce both aversive aphagia and a "choreic" sensorimotor syndrome: an exaggerated treading of the forepaws that is triggered by oral sensory stimulation. Experiment 1 used a recovery-of-function approach to show that (a) aphagia induced by ventroposterior striatopallidal lesions was accompanied by an enhancement of aversion (a specific motivational process) to sweet stimuli, which was expressed in taste reactivity measures of affective evaluation; (b) aphagia and enhanced aversion recovered together; and (c) exaggerated treading did not disappear with aphagia–aversion but narrowed the range of its eliciting trigger to sour and bitter stimuli. Experiment 2 used a partial lesion approach to show that this dissociation of enhanced aversion and exaggerated treading could be reproduced by smaller lesions immediately after striatopallidal damage. Experiment 3 used a conditioned aversion procedure to show that the stimulus for exaggerated treading was aversion (natural or conditioned) and not a simple sensory feature of oral stimulation. Three conclusions were made: (a) Exaggerated treading after a small lesion or after partial recovery from a larger one results from a restructuring of a sensorimotor relations that is nested within a system of aversive reaction, (b) exaggerated treading is elicited only by tastes that elicit natural or conditioned aversion, and (c) more extensive lesions potentiate aversion to tastes that are normally palatable and expand the range of treading elicitors to include those tastes. In other words, affective and sensorimotor systems interact in a hierarchical manner in the production of choreic treading. These results demonstrate a specific hierarchical link between motivational and sensorimotor functions mediated by striatopallidal circuits.

Motivational and sensorimotor concepts are used in behavioral neuroscience to refer to different aspects of behavior. *Motivational concepts* generally refer to aspects such as affective displays, the homeostatic coordination of diverse inputs and outputs, dynamic spontaneity, and flexible means–ends readiness to achieve goals (e.g., Epstein, 1982; Fentress, 1983; Gallistel, 1980; Miller, 1982; Teitelbaum, 1977; Teitelbaum, Schallert, & Whishaw, 1983; Toates, 1986). *Sensorimotor terms,* on the other hand, typically denote integrations that create linkages between specific sensory modalities and motor systems. These integrations serve specific roles in coordinating action that are not restricted to either purely sensory or purely motor functions but are narrower in scope than motivational (or other high-level) integrations (Gallistel, 1980; Lidsky, Manetto, & Schneider, 1985; Marshall, 1980; Schallert et al., 1982; Stricker & Zigmond, 1986; Teitelbaum, 1986; Whishaw, Kolb, & Sutherland, 1983; White, 1986).

Although motivational and sensorimotor terms have separable meanings, it often happens that both are needed to describe the behavioral consequences of even a single brain manipulation. Damage to any one of a number of different forebrain or brainstem structures can produce behavioral consequences that involve both sensorimotor and motivational aspects. This study was concerned with interactions between sensorimotor and motivational processes.

The globus pallidus provides a prime example of a brain structure in which damage can produce behavioral changes that have both motivational and sensorimotor components. Deficits produced by striatopallidal damage that have been viewed as motivational (at least in part) include aphagia and adipsia (Dunnett & Iversen, 1980; Levine & Schwartzbaum, 1973; Morgane, 1961; Sorenson & Ellison, 1970), which may be as severe as the aphagia and adipsia produced by lesions of the lateral hypothalamus (Teitelbaum & Epstein, 1962). Changes that have been construed as sensorimotor include numerous passive deficits in posture control, oromotor coordination, and reaching to a target (Labuszewski, Lockwood, McManus, Edelstein, & Lidsky, 1981; Levine & Schwartzbaum, 1973). Striatopallidal damage or pharmacological manipulations also can induce positive or *release symptoms* of hyperkinesia, which generally are construed as sensorimotor (e.g., Schneider, 1984; Villablanca, Marcus, & Olmstead, 1976).

In rats, striatopallidal lesions can induce a hyperkinesic

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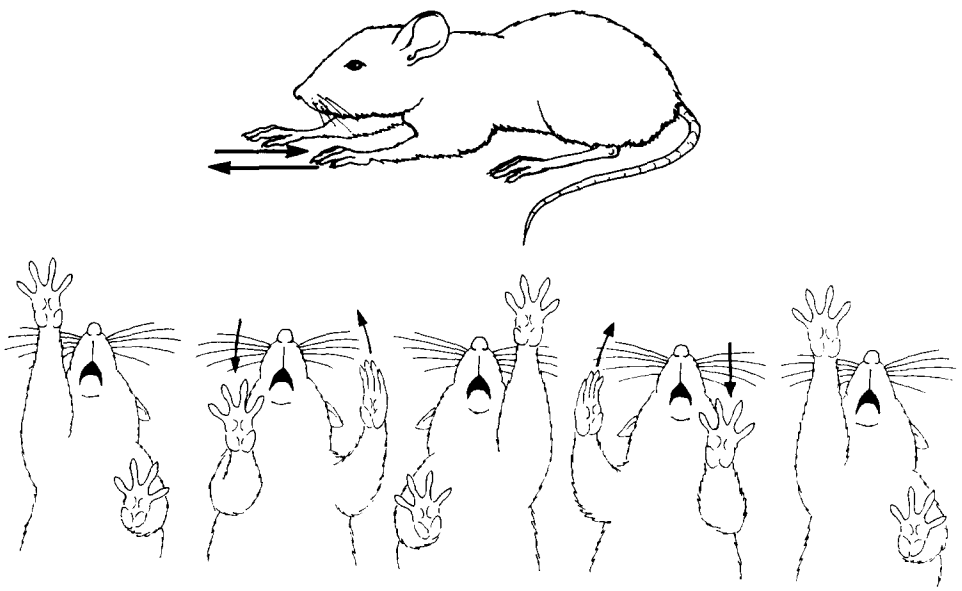

Figure 1. Exaggerated treading. (Side and ventral views of the exaggerated treading pattern triggered by tastes after striatopallidal lesions. Forepaws are extended rhythmically to at least 1 cm apart and move forward and backward at a rate of 3.5 Hz. Each paw remains 180° out of phase with the other.)

paw treading syndrome (Figure 1; Berridge, Fentress, & Treit, 1988) that resembles human chorea in certain respects: defined as "a brisk, graceful series of successive involuntary movements of considerable complexity which resemble fragments of purposeful voluntary movements" (Carpenter, 1978, p. 253). The fragment of movement induced in rats appears to be an exaggeration of natural movements used both to bury noxious objects and occasionally to respond to aversive tastes (Grill & Norgren, 1978; Pinel & Treit, 1983; first described by Teitelbaum & Epstein, 1962). Treading is normally very rare, contributing less than 5% to the aversive behavior elicited by an unpalatable taste, and normal rats never show exaggerated treading in any situation. However, after striatopallidal lesions, treading is exaggerated, in incidence, amplitude, and duration (Berridge et al., 1988). Although it is difficult to directly translate the symptoms of clinical sensorimotor syndromes across different mammalian orders, exaggerated treading by rats does fit the description of human chorea just quoted. To a clinical neurologist accustomed to the diagnosis of human sensorimotor disorders, exaggerated treading by rats may appear to combine features of the complex stereotyped motor tics of Tourette's syndrome with the incessant serial motion aspect of chorea, although it is both more fluid and coordinated than the former and less random than the latter (R. L. Albin, personal communication, January 31, 1990).

Exaggerated treading by striatopallidal rats is not simply a general motor disinhibition (see Figure 2A). Instead, it occurs only in particular situations. Exaggerated treading appears to be at least a sensorimotor phenomenon that is stimulus-triggered by orofacial sensation (see Figure 2B). It occurs as a rapid, vigorous paw treading action, which rarely is emitted spontaneously or in response to nonoral stimuli, but which is elicited reliably by the oral stimulation provided by infusion of a sucrose or quinine solution into the mouth. A rat with this syndrome will typically burst into exaggerated treading within seconds of onset of an oral infusion, continue without interruption for up to 1 min, and display intermittent bouts of intense treading for as long as 5 min afterward (Berridge et al., 1988).

A distinct relationship has been noted between the production of aphagia by a striatopallidal lesion and the production of the exaggerated treading disorder (Berridge et al., 1988). In fact, the appearance of aphagia after a striatopallidal lesion provided a reliable predictor that a rat would show exaggerated treading to taste solutions. Other types of sensorimotor deficits produced by neostriatal lesions, such as failures of sequential coordination, were not accompanied by aphagia in this way (Berridge & Fentress, 1987). Conversely, other neural lesions that cause aphagia (e.g., excitotoxic lesions of the lateral hypothalamus caused by ibotenic acid, 6-hydroxydopamine lesions of the substantia nigra, trigeminal deafferentation) were not accompanied by taste-elicited choreic treading (Berridge, 1989; Berridge & Fentress, 1985; Berridge, Venier, & Robinson, 1989). Only large electrolytic lesions of the lateral hypothalamus (e.g., Teitelbaum & Epstein, 1962) or large excitotoxic lesions of the nucleus basalis and globus pallidus (Whishaw, O'Connor, & Dunnett, 1985) share with striatopallidal lesions the capacity to potentiate treading. Although lesion studies of these sites have not elicited prolonged bouts of exaggerated treading to oral infusions as studied here, they have reported shorter bouts of treading that occurred spontaneously or at the food dish. These large lesions may overlap anatomically, and all are likely to disrupt striatopallidal and related circuits.

Why should a distinctive correlation exist between aphagia

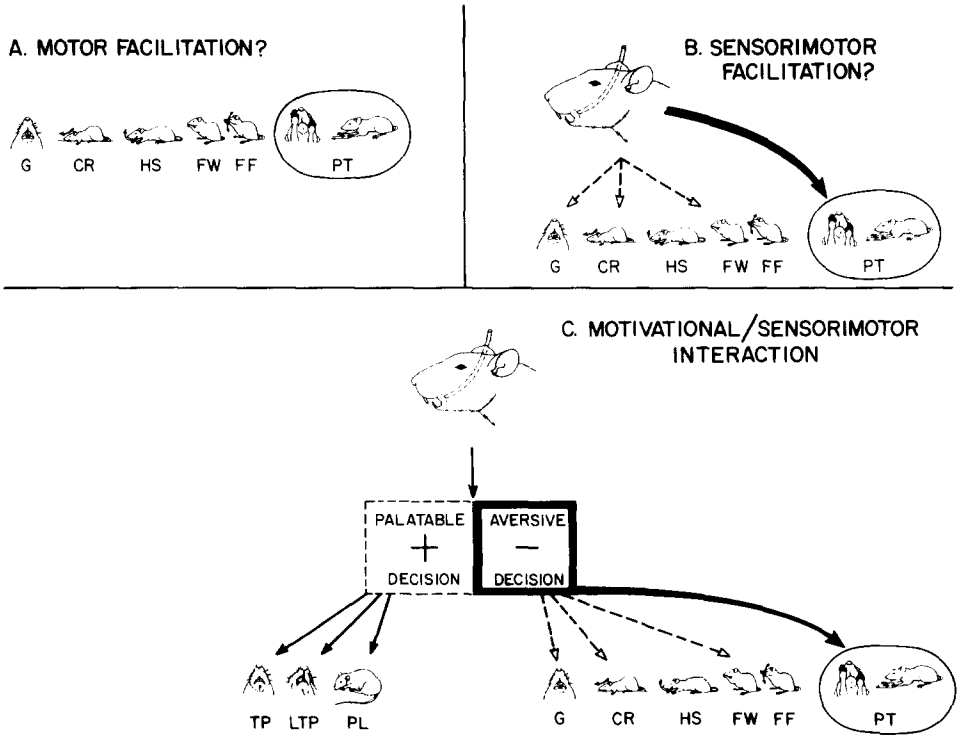

Figure 2. Motor, sensorimotor, and motivational interaction hypotheses of exaggerated treading. (A: Simple motor facilitation of paw treading (PT) in all contexts. B: Sensorimotor facilitation of treading in response to a particular sensory feature of oral stimulation. C: Interaction between potentiated aversion and aversion-triggered treading, which produces a motivational gating of the exaggerated treading. Circled paw treading and bold arrows portray a facilitation of that action either directly or in response to either a sensory event or an aversive evaluation. Broken line and arrow portrays an inhibition of competing responses or affective evaluation. G = gapes; CR = chin rubbing; HS = head shaking; FW = face washing; FF = forelimb flails; TP = tongue protrusion; LTP = lateral tongue protusion; PL = paw licking.)

and a positive sensorimotor disorder in the case of striatopallidal or related damage? There are at least three conceptual alternatives that have been used to account for why sensorimotor and motivational changes in behavior can result simultaneously from a single brain lesion. These three alternatives could be labeled single-factor, adjacent-systems, and hierarchical-levels-of-function interpretations.

Single-factor interpretations argue that behavioral changes that are ostensibly different can actually be subsumed under a single cause. For example, in the case of damage to hypothalamic dopamine pathways, single-factor hypotheses have suggested that an arousal or responsiveness factor might explain both aphagia and akinesia (Stricker & Zigmond, 1976, 1978, 1986; White, 1986). Similarly, in the case of striatopallidal damage, a single-factor hypothesis might account both for aphagia and for reaching, treading, and other deficits by positing either that a single sensorimotor change disrupts the activation and coordination of forepaw movements needed for feeding and other activities (e.g., White, 1986; Labuszewski et al., 1981) or that a global motivational change alone could explain all aspects of the behavioral changes caused by striatopallidal lesions. If evidence were found that demonstrated that motivational changes were produced in addition to sensorimotor changes, however, then a single-factor account would no longer suffice. It would be necessary to explain simultaneous changes in more than one functional system.

Adjacent-systems interpretations recognize that any single anatomical region is likely to contain a number of separate functional circuits; damage within that region is therefore likely to affect more than one function. An adjacent-systems interpretation could account for simultaneous motivational and sensorimotor changes after striatopallidal damage by supposing that both regulatory feeding circuits and sensorimotor control circuits were disrupted by the lesion. The motivational and sensorimotor changes would be separate and independent, according to an adjacent systems hypothesis, because they would depend on damage to separate anatomical circuits.

Finally, *hierarchical-levels-of-function* interpretations can account in a different way for the simultaneous existence of both sensorimotor and motivational changes in behavior. By a hierarchical view, motivational and sensorimotor functions constitute different levels of a single integrated system. Changes can occur simultaneously at these two levels after a single brain manipulation because hierarchical organization

allows a sensorimotor system to be "nested" within a motivational one. In addition, hierarchical interpretations can explain interactions between two behavioral functions. Interaction would occur when the expression of changes at one functional level depended on factors that were especially relevant to the other level. The explanation of interaction is possible for hierarchical theories of neurobehavioral organization because these theories generally posit that motivational and sensorimotor functions are linked together causally in two directions (Dawkins, 1976; Fentress, 1983; Gallistel, 1980; Teitelbaum, 1986; Teitelbaum et al., 1983). In an ascending direction, emergent motivational processes are created partly out of assemblies of lower sensorimotor ones. In a descending direction, motivational processes modulate the operation of elemental sensorimotor functions. A result of this linkage is that motivational and sensorimotor processes are not truly separate according to hierarchical interpretations. After a brain lesion, they could be expected to interact in complex ways. For example, changes in sensorimotor functions might depend for their expression on the existence of particular motivational conditions.

This study examined the relation between the production of aphagia and exaggerated treading in three experiments to discover which interpretation best described the relation between sensorimotor and motivational changes that result from striatopallidal damage. The first experiment documented a motivational change truly involved in aphagia, namely, an enhancement of aversion, in addition to sensorimotor treading. The experiment used a recovery-of-function approach (Teitelbaum & Stellar, 1954) to discover whether the affective enhancement of aversion disappeared simultaneously with exaggerated treading as rats recovered from aphagia after large bilateral lesions. The results demonstrated a dissociation between motivational and sensorimotor changes and ruled out a single-factor account of the effect of striatopallidal lesions. The second experiment used a partial lesion approach to confirm this conclusion. Small excitotoxic lesions showed that interactions seen between sensorimotor and motivational changes during recovery could be reproduced as an immediate consequence of a partial lesion. The results of Experiments 1 and 2 allowed rejection of the single-factor interpretation but were consistent with both the adjacent-systems interpretation and the hierarchical interpretation and did not permit a confident choice between them. The hierarchical possibility implied that exaggerated treading was controlled by an interaction between sensorimotor and motivational systems. The third experiment tested and confirmed a prediction derived from this interactive hypothesis: that aversion acted to gate sensorimotor treading in a hierarchical fashion.

Experiment 1

Method

Large bilateral lesions of the globus pallidus and posterior neostriatum were made using the excitotoxin kainic acid (1.0 μg in 0.5 μl phosphate buffer) in 10 anesthetized (100 mg/kg ketamine + 10 mg/kg xylazine) Sprague-Dawley rats as described previously (Berridge et al., 1988). Ten additional control rats received intrastriatal injections of only the phosphate buffer vehicle.

With bregma and lambda in the same horizontal plane, bilateral skull holes were drilled 0.8 mm posterior to bregma and 3.5 mm lateral to the midline. A 30-gauge cannula was lowered to 6.5 mm beneath the skull surface. The injection was made over a 5-min period, and the cannula was left in place for an additional 10 min. Diazepam (8 mg/kg) was given 30 min before and 30 min after injections to control convulsant activity.

Each rat was implanted with bilateral chronic oral cannulae (Grill & Norgren, 1978) to allow infusion of taste solutions into the mouth. These cannulae (heat-flared PE-50 tubing) entered the mouth lateral to the first maxillary molar and exited the head near the dorsolateral borders of the skull, where they were anchored with skull screws and acrylic cement.

Postsurgical Maintenance

All rats were allowed free access to fresh cereal mash (commercial baby cereal mixed with water), chow pellets, and water. Aphagia and adipsia may follow striatopallidal lesions (e.g., Morgane, 1961). It was important for the purposes of this study that behavioral changes be due directly to the lesion and not to dehydration or caloric depletion, secondary to adipsia and aphagia. For this reason, if body weight dropped after surgery, then rats were intubated with a liquid diet (sweetened condensed milk, water, and vitamins). A rat received one 12-ml intubation for every 5 g of body weight lost, with up to 3 intubations per day. Rats were considered to be aphagic if they ate neither chow pellets nor cereal mash and received all their calories by intubation. Rats were considered to be hypophagic if they ate any cereal mash but no chow pellets. Rats that ate chow pellets were considered to be nonaphagic.

Behavioral Testing

Each rat was tested for taste-elicited reactions during the 1st week after surgery, and once again during the 3rd week. Taste solutions were chosen to present a range of palatability: sucrose (1.0 M), which elicits primarily ingestive responses in normal rats; HCl (0.01 M), which elicits mixed ingestive and aversive responses; and quinine HCl (3×10^{-4} M), which elicits primarily aversive responses. For aphagic rats that required tube feeding, at least 2 hr elapsed between intubation and testing.

On each trial, a rat was placed in a transparent test chamber, which was suspended over a mirror. The mirror reflected a view of the rat's mouth and ventral surface into a videocamera. A delivery tube was connected to the oral cannulae of the rat. After 5-min habituation period 1 ml of the taste solution was infused into the mouth over a 1-min period. One stimulus was administered per day, and the order of presentation was random. One additional trial, in which 1.0 M sucrose was administered after a 24-hr food-deprivation period, was added to the end of the series in both the 1st and 3rd weeks of testing. This was done because food deprivation has been reported to reverse the enhanced aversion to tastes produced by electrolytic lesions of the lateral hypothalamus (Fluharty & Grill, 1981), and it was of interest to know whether changes in taste reactivity produced by striatopallidal excitotoxic lesions would show a similar dependency on caloric state.

Taste Reactivity Scoring

Videotapes were scored in slow motion (frame-by-frame to one tenth normal speed) for the occurrence of aversive reactions and for positive hedonic reactions as described by Grill and Berridge (1985).

A more stringent criterion for scoring exaggerated paw treading than was used in earlier studies of aversive treading was used here, however, because the focus in this study was on the exaggerated treading produced by striatopallidal damage. The purpose of using this stringent criterion was to clearly discriminate normal treading from the gross exaggeration of treading induced by lesions. For this study, a *unit of treading* was considered to be at least 5 continuous s of rhythmic forward and backward extension of the forepaws 180° out of phase, with the two paws extended at least 1 cm apart in every cycle, at a frequency of roughly 3.5 Hz. Only exaggerated choreic treading meets this criterion, and so most normal control rats have zero scores for treading even in response to aversive tastes.

Other aversive reactions. The following aversive reactions were also scored: *gapes*—large openings of the mouth and jaw lasting about 125 ms; *chin rubbing*—bringing the chin in direct contact with the floor and projecting the body forward; *face washing*—either a single wipe with the paws over the face or a bout of several wipes; *forelimb flails*—shaking of the forelimb back and forth; and *rapid head shaking.*

Positive hedonic reactions. The following positive hedonic reactions were scored: *lateral tongue protrusions* (nonrhythmic) lasting about 160 ms; *rhythmic tongue protrusions*—along the midline with a cycle length of roughly 160 ms; and *paw licking.* Each action was scored and counted using criteria described in Grill and Berridge (1985).

Histology

At the conclusion of the experiment, each rat was deeply anesthetized and perfused. Brains were frozen, sectioned, mounted on slides, and stained with cresyl violet. Sections were examined microscopically to assess the extent of gliosis and the loss of neuronal cell bodies. Lesions were mapped using the atlas of Paxinos and Watson (1982).

Criteria for Analysis of Palatability

Taste reactivity provides a sensitive measure of the affective evaluation of taste palatability (Grill & Berridge, 1985). The analysis of taste reactivity as a measure of palatability was complicated by lesion-induced exaggerated treading, however, for two reasons. First, because treading itself is an aversive taste-elicited action (although normally of low frequency and small amplitude), its inclusion as an aversive action after striatopallidal lesions could bias the results of palatability analyses toward aversive scores that were artificially high. For this reason, treading was specifically excluded from analyses of aversive palatability. For normal rats, treading occurs with such low frequency in relation to other aversive actions that exclusion of treading from aversive scores leaves the scores essentially unchanged (e.g., Berridge, Grill, & Norgren, 1981). Second, because the treading emitted by rats with striatopallidal lesions to tastes that are normally aversive (e.g., quinine) can be so extreme as to exclude other actions (i.e., by time-sharing competition or competitive inhibition), reactivity analyses of such naturally aversive tastes could be misleading. Exclusion by competition would reduce the number of aversive actions other than treading, but this reduction would reflect limitations of motor production or of time-sharing rather than a diminution of affective aversion. To avoid such a distortion of aversive response production, this analysis of palatability focused on 1.0 M sucrose, which is highly palatable to normal rats.

Results

Histology

Cell loss and gliosis (Figure 3, top row; see also Figure 4 and compare with Figure 5) produced by kainic acid was extensive throughout the globus pallidus and posterior neostriatum. The lesion extended ventromedially into the substantia innominata and nucleus basalis in every rat. Damage was also found in the anterior hypothalamus, neocortex, and amygdala of several rats.

Aphagia

Each rat that received striatopallidal lesions showed a period of aphagia followed by hypophagia. Periods of complete aphagia ranged from 5 to 11 days. All rats ate at least several grams of cereal mash per day by Day 14, and 7 of 10 rats were able to maintain their body weight by this time. Although most of the daily intake of these rats was in the form of mash, 6 of them also began to nibble chow pellets by Day 14, and 9 of the 10 rats did so by Day 21.

Enhancement of Affective Aversion

Aversive responses (gapes, head shakes, forelimb flails, and chin rubs) to sucrose by control and lesion rats (Figure 6) were tested both when the rats were fed (ad lib or 2 hr after tube feeding) and when they were deprived of food during the 1st and the 3rd weeks after surgery. A three-way Lesion × Week × Caloric State analysis of variance (ANOVA) was used. This analysis showed a pronounced enhancement of aversion in the lesion group compared with control rats, $F(1, 17) = 7.44, p < .02$, with a significant diminution of enhanced aversion over recovery, $F(1, 17) = 14.6, p < .01$, and a significant interaction between lesion and week of recovery, $F(1, 17) = 14.6, p < .01$. Rats with pallidal damage showed more aversive responses to sucrose during the 1st week after the lesion than did controls ($M = 7.2$ for lesion vs. 0.4 for control, $p < .05$, Newman–Keuls). By the 3rd week of recovery, however, the aversion of the lesion group had dropped from its initial level ($p < .05$, Newman–Keuls), and lesion and control groups no longer differed in aversion to sucrose (Figure 6). Nine of the 10 rats with lesions were nonaphagic by this time (having begun at least to nibble at chow pellets), and all of them were eating some cereal mash. In other words, aphagia appeared to recover as the enhancement of aversion receded. Food deprivation had no effect on either treading or aversion to sucrose in either the 1st or the 3rd week. The failure of food deprivation to modulate lesion-induced changes indicates that the aversion produced by striatopallidal lesions is not strongly gated by caloric state, in contrast to the state-dependent enhancement of aversion that has been reported to follow electrolytic lesions of the lateral hypothalamus (Fluharty & Grill, 1981). The cause of this difference is not clear: differences in the size, placement, or type of neuronal damage between our excitotoxic lesions and the electrolytic lesions of Fluharty and Grill (1981) all might contribute.

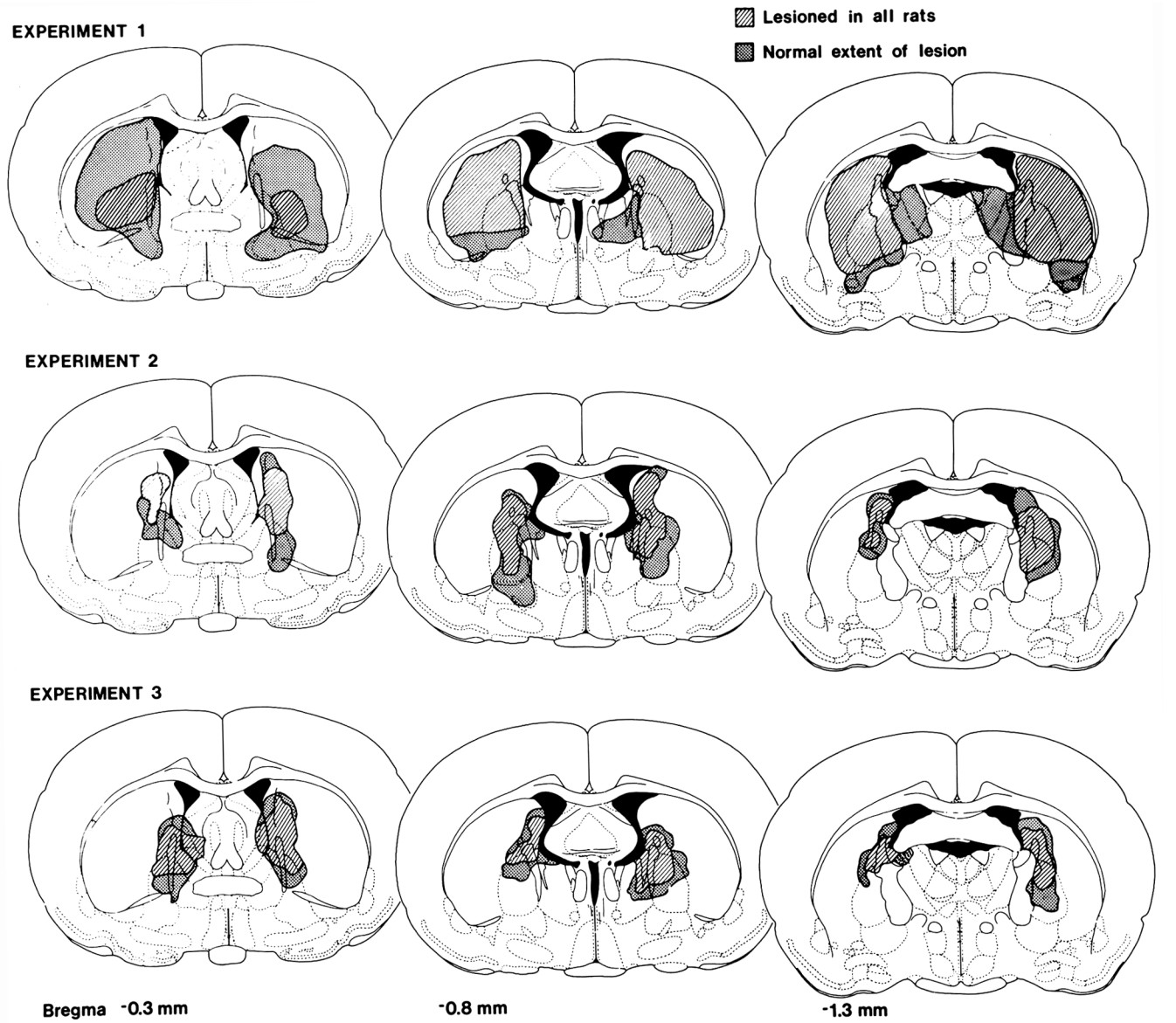

Figure 3. Extent of excitotoxic lesions. (Striped area shows region in which cell loss and gliosis was found for every rat. Stippled area shows the maximal extent of cell loss and gliosis found in any rat. Top row: Experiment 1. Middle row: Experiment 2. Bottom row: Experiment 3.)

Exaggerated Treading: Selective Recovery Over Time

Exaggerated treading was elicited from rats with striatopallidal lesions by all tastes (sucrose, HCl, and quinine) during the 1st week (Figure 7). In contrast, 9 of 10 control rats never met the treading criterion for any taste. Rats with lesions showed treading to each taste with durations averaging 15 times greater than control levels (M = 30 s for lesion vs. 2 s for control, $p < .01$ for each) and there was no effect of taste composition on the level of treading elicited from rats with lesions at this time. Treading also was elicited in the 3rd week after the lesion but was no longer independent of taste composition (Figure 7). A three-way ANOVA (Lesion × Taste × Week) showed a significant difference between lesion and control groups, $F(1, 17) = 47.5$, $p < .001$, a significant change over weeks, $F(1, 17) = 9.6$, $p < .01$, a significant effect of taste composition, $F(2, 34) = 11.2$, $p < .001$, and significant effects for every possible interaction ($p < .05$ for each). Specifically, treading was elicited with undiminished intensity by both bitter quinine and sour HCl in the 3rd week compared with the 1st week after lesions (Figure 7). Exaggerated treading to sucrose, however, was reduced to 20% of the 1st week level and no longer differed significantly from control values. Eight of 10 rats with lesions showed no treading at all to sucrose in the 3rd week in contrast to their high levels of treading during the 1st week.

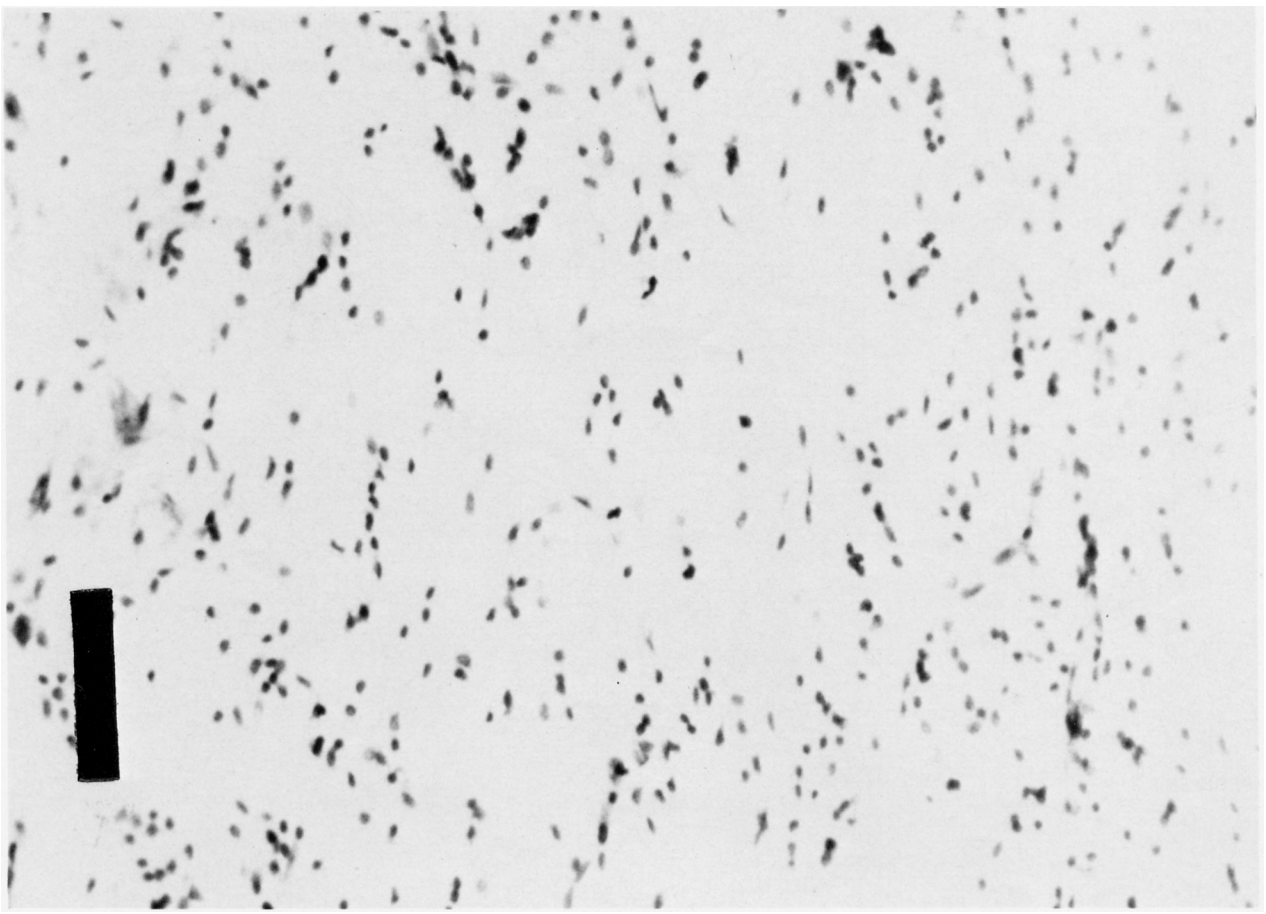

Figure 4. Micrograph showing gliosis and cell loss in striatopallidal tissue after excitotoxin lesion. (Lesion produced by kainic acid in Experiment 2. Bar = 250 microns. Magnification 10×. Compare with Figure 5.)

Discussion

The aphagia produced by striatopallidal lesions was accompanied by an enhancement of aversion, which distorted the affective evaluation of tastes. Sucrose, a preferred taste that normally elicits positive hedonic reactivity, instead elicited strong aversive reactivity after striatopallidal damage. Enhanced aversion to sucrose has been reported to result also from electrolytic lesions of the anterolateral hypothalamus (Fluharty & Grill, 1981; Schallert & Whishaw, 1978; Teitelbaum & Epstein, 1962; White, 1986) but is not a necessary component of all neurally induced aphagia. Neither mesotelencephalic dopamine depletion, trigeminal deafferentation, excitotoxic lateral hypothalamic lesions, nor certain electrolytic lateral hypothalamic lesions (perhaps posterior lateral hypothalamus; see Schallert & Whishaw, 1978) produce an enhancement of aversion, even though all can result in severe aphagia (Berridge, 1989; Berridge & Fentress, 1985; Berridge et al., 1989; Schallert & Whishaw, 1978). In the case of striatopallidal lesions (as in the case of very large or anterior electrolytic lesions of the lateral hypothalamus; Fluharty & Grill, 1981; Schallert & Whishaw, 1978; Teitelbaum & Epstein, 1962), however, enhanced aversion is a causal factor in producing aphagia. It is possible that lesions of the basal forebrain that enhance aversion all damage the same neural system within the ventral pallidus–basal nucleus–substantia innominata region.

Both aversion and aphagia recovered together over the weeks after the lesion, as would be expected if aphagia were driven by an aversive evaluation of food. The existence of this distinct change in affective evaluation, in addition to the sensorimotor potentiation of treading, indicates that no single factor can adequately describe the functional consequences of this lesion. Enhanced aversion is one distinct consequence. Exaggerated treading, a sensorimotor restructuring of stimulus–response relations that transforms taste-elicited treading from a very rare and low-amplitude event into a frequent and amplified one, is a separate consequence. It remains to be seen whether these consequences are best described by an adjacent systems or a hierarchical interpretation.

Does triggered choreic treading recover with aphagia and aversion after a striatopallidal lesion? In one sense, the answer is no. There is no general recovery of treading to parallel

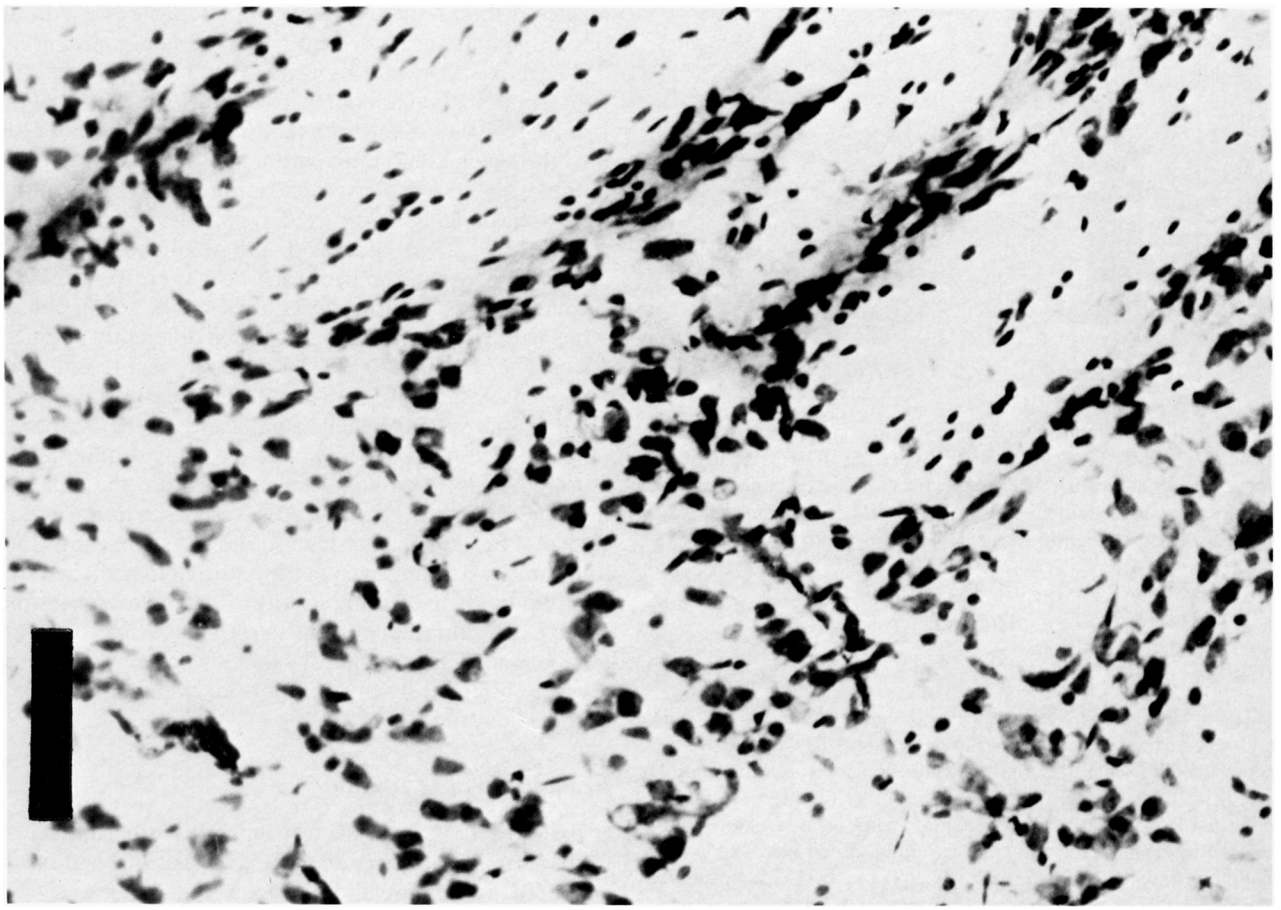

Figure 5. Micrograph showing healthy striatopallidal tissue. (Bar = 250 microns. Magnification 10×. Compare with Figure 4.)

the general recovery of aphagia and aversion. Stimuli such as quinine or HCl elicited treading with no lesser intensity in the 3rd week than in the 1st week after the lesion, and in this sense, no recovery could be judged to have occurred. But in a different sense, recovery was considerable. Sucrose elicited treading comparable to quinine during the 1st week after the lesion but showed a dramatic drop in both the probability and intensity of elicited treading to levels that were normal by the 3rd week. In other words, as aphagia and aversion gradually declined after pallidal damage, the recovery of exaggerated treading appeared to be an all-or-none phenomenon: Recovery could be both all and none simultaneously to different stimuli!

One possible explanation for this dissociation of recovery may lie in the fact that the stimuli that failed to show recovery (quinine, HCl) are tastes that are naturally aversive to normal rats. Bitter quinine elicits highly aversive reactivity from normal rats, and sour HCl elicits a mixture of strong aversion and positive reactivity. Sucrose, on the other hand, is a taste that normally elicits highly positive reactivity with no aversive component. The normal palatability of a stimulus might serve to predict whether it will show a recovery as a treading elicitor after striatopallidal lesions. If so, this would suggest that taste aversion is the effective trigger of exaggerated treading (Figure 2C). If this hypothesis were true, then it would strengthen and extend the conclusion that two separable effects of large lesions are revealed by the selective recovery of exaggerated treading. These separable effects would be (a) an enduring sensorimotor potentiation of treading to any taste that is evaluated as aversive and (b) a temporary expansion of the range of tastes that receive this aversive evaluation.

Experiment 2

The hypothesis that exaggerated treading involves two distinct changes would be more credible if the existence of the two changes could be confirmed in another fashion besides recovery from a large lesion. This might possibly be done through the use of partial lesions, smaller than those used in Experiment 1, which might immediately dissociate the two changes that were exposed during recovery. This second experiment examined whether treading and enhanced aversion could be distinguished using small lesions, which varied slightly in extent and placement, in a way that paralleled their relation during recovery from a large lesion.

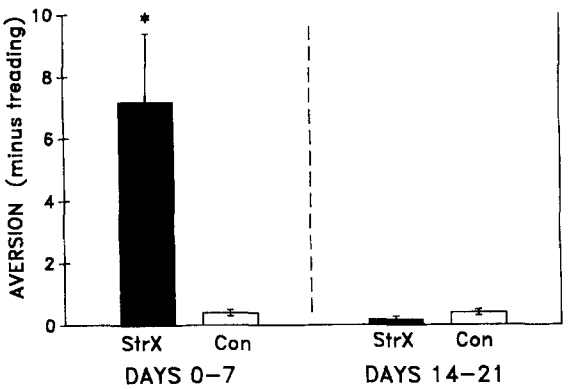

Figure 6. Enhancement and recovery of aversion to sucrose (Experiment 1). (Bars show the number [$M \pm SE$] of aversive actions [gape, chin rub, face wash, headshake, forelimb flail] elicited by oral sucrose from striatopallidal lesion [StrX] and from control [Con] groups in the 1st week and in the 3rd week of recovery.)

Method

Surgery

Small bilateral lesions were made in the globus pallidus and surrounding area in 16 anesthetized male Sprague-Dawley rats (250–350 g). A different neurotoxin, quisqualic acid, was used instead of kainic acid in 7 of these rats to be sure that lesion effects could be produced by other excitotoxins besides kainic acid. Lesions were produced by intrastriatal infusions of either kainic acid (0.5 μg in 0.5 μl phosphate buffer) or quisqualic acid (21 μg in 0.5 μl phosphate buffer). Each 0.5-μl infusion was made over a 1.5-min period. Placements were 1.0 mm posterior to bregma, 2.3 mm lateral to the sagittal fissure, and 8.3 mm ventral to skull surface. Six additional rats received control infusions of only the phosphate buffer vehicle. Each rat was implanted with oral cannula at the time of surgery.

Behavioral Testing

Taste reactivity tests were conducted over Days 1–14 after surgery. The selection of stimulus solutions was expanded to sucrose (1.0 M and 0.03 M), quinine HCl (3×10^{-4} M and 3×10^{-6} M), and citric acid (0.05 M and 0.01 M; citric acid elicits both aversive and positive responses at these concentrations from normal rats) to confirm that it was taste quality, and not a particular concentration of a taste, that determined trigger effectiveness. Behavioral testing and analysis procedures were the same as in Experiment 1.

Histology

Brains were removed, frozen, sliced, mounted, and stained as in Experiment 1. Maps of the extent of cell loss and gliosis were completed by light microscopy.

Results

Choreic Treading: Taste Selectivity

Two groups of rats could be distinguished after small posterior lesions were produced (Figure 8), based on whether they showed exaggerated treading to all tastes (nonselective treaders) or selectively to only some tastes (selective treaders). In a three-way ANOVA (Group × Taste × Concentration), treading was controlled both by lesion group, $F(2, 19) = 17.16$, $p < .01$, and by taste, $F(2, 38) = 4.79$, $p < .02$, with a marginal interaction between the two effects, $F(4, 38) = 2.10$, $p = .099$. The group that treaded to all tastes resembled the lesion group of Experiment 1 in their 1st week; rats in this group ($n = 4$; all with lesions produced by kainic acid) were aphagic and showed equally high levels of treading to sucrose ($M = 21$ s), citric acid, and quinine, $F(2, 6) = 1.08$, and to all concentrations of each taste, $F(1, 3) = 1.08$. The second group ($n = 12$; 7 rats with quisqualic acid lesions and 5 rats with kainic acid lesions) treaded differently to different tastes, $F(2, 22) = 5.80$, $p < .01$, in a way that resembled the "recovered" stage of Experiment 1; these rats did not tread at all to sucrose but treaded significantly to both citric acid ($M = 6.25$ s, $p < .05$, Newman–Keuls) and quinine ($M = 3.8$ s, $p < .05$, Newman–Keuls), which are the only tastes used that naturally elicit aversive responses in normal rats. Rats that treaded to all tastes also showed significantly longer durations of treading even to taste stimuli that elicited treading from the aversive-only group ($p < .05$ for each, Newman–Keuls). The durations of the selective aversive-only group were still greater than control values, which always were zero (even to quinine) by the criterion for pathological treading used in this study.

Enhancement of Aversion

Taste reactivity analysis of palatability was conducted as for Experiment 1. Aversion elicited by sucrose solutions was enhanced by striatopallidal lesions (Figure 9), even when

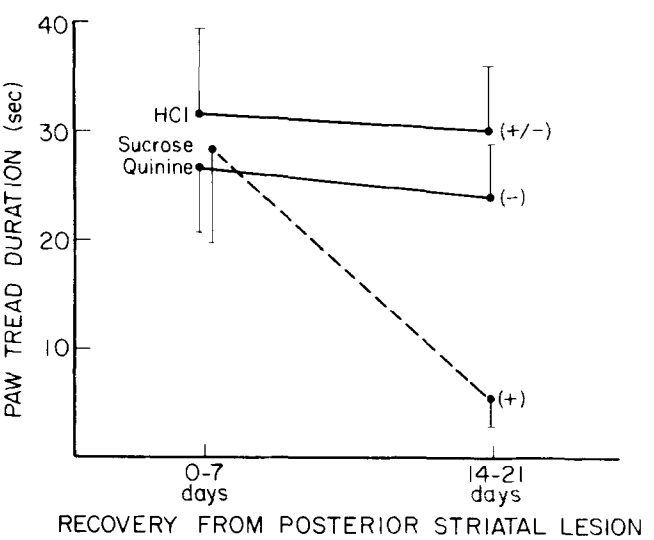

Figure 7. Selective recovery of exaggerated paw treading (Experiment 1). (Duration [$M \pm SE$; in seconds] of paw treading elicited by each taste in the 1st week versus the 3rd week after striatopallidal lesions. Plus and minus signs reflect the taste reactivity shown by normal rats to each taste; bitter quinine and sour HCl [solid lines], which have an aversive component, did not show recovery, whereas sucrose [broken line], which elicits entirely positive responses from normal rats, did.)

treading was not included as an aversive response, $F(2, 19) = 3.84$, $p < .05$. This enhancement of aversion was seen only in the group that was aphagic and that treaded to all tastes. When aversion was averaged across the different sucrose concentrations, rats from this group alone showed higher levels of aversion to sucrose compared with controls ($p < .05$, Newman–Keuls). The extraordinarily high levels of treading elicited by quinine or citric acid from rats that treaded to all tastes served actually to suppress the emission of other aversive actions below control levels ($p < .05$, Newman–Keuls). This competitive inhibition of other responses by high levels of treading testifies to the pathological restructuring of sensorimotor relations produced by striatopallidal damage. Competitive inhibition also reinforces the point that aversion can be gauged most accurately after these lesions only by minimizing the degree of competition from treading (i.e., by using sucrose as the eliciting taste).

Lesion Analysis

Gliosis and necrosis was centered in the ventral posteriomedial striatum and globus pallidus after lesions produced by kainic acid or quisqualic acid (Figure 3, middle row; see also Figure 4). Cell loss was consistently greater for lesions produced by kainic acid. Rats that showed enhanced aversion and that treaded to all tastes appeared often to have larger lesions. These rats also had damage that extended to the medial border of the globus pallidus and generally showed additional penetration into the nucleus basalis and occasionally into the substantia innominata (see Cromwell & Berridge, 1989, 1990, for detailed anatomical mapping of lesion effects).

Discussion

These results provide support based on partial lesions for the hypothesis of two functional effects, which was formed in Experiment 1 on the basis of recovery of function. The two consequences of pallidal lesions are reflected in the two lesion groups found in this experiment. First, moderate damage within the globus pallidus and posterior neostriatum appeared to potentiate exaggerated treading as a sensorimotor response to tastes such as quinine or citric acid. This was seen both after recovery from striatopallidal lesions in Experiment 1 and to a lesser degree in the selective-treading group in Experiment 2. The pathological potentiation of treading competes with the emission of other actions and can suppress them. Second, more extensive damage (especially around the medial border of the striatum and globus pallidus) potentiated the aversiveness even of tastes that are hedonically positive to normal rats, such as sucrose. The lesions that were most effective at enhancing aversion were those that penetrated beyond the medial border of the ventral pallidum into the nucleus basalis or ventrally into the substantia innominata. These may have overlapped with the area of damage produced by the aversion-enhancing electrolytic lateral hypothalamus lesions of Teitelbaum and Epstein (1962), Fluharty and Grill (1981), and others, and with the anterior lateral hypothalamus lesions of Schallert and Whishaw (1978). It is possible that all of these lesions enhance aversion by acting on the same system. Striatopallidal lesions that enhanced aversion in this study had the additional effect of adding sucrose to the set of treading elicitors. These results provide conclusive evidence that striatopallidal lesions alter both sensorimotor and motivational functions in distinctive ways.

Why are bitter and sour tastes the only effective elicitors of treading for rats that exhibit the sensorimotor change alone? And why is it that rats that show enhanced aversion also expand their range of treading elicitors to include sucrose? One possible answer is given by the adjacent-systems interpretation: that the sensorimotor and motivational effects of striatopallidal lesions are functionally separate and unrelated.

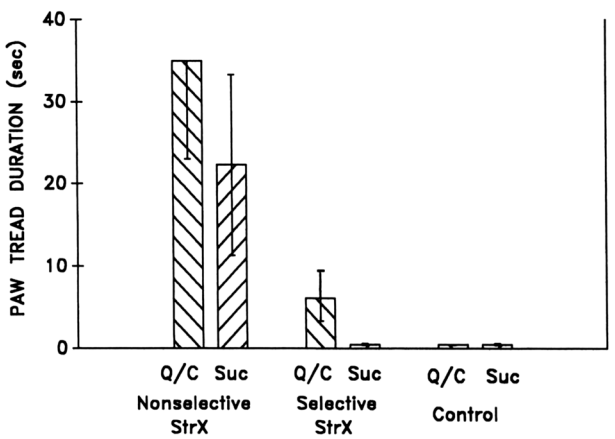

Figure 8. Selective potentiation of treading by partial lesion (Experiment 2). (Rats that tread nonselectively to all tastes showed highest levels of choreic treading [$M \pm SE$]. Rats that tread selectively to quinine [Q] or citric acid [C], but not to sucrose [Suc], had levels that were lower but still elevated over control baseline [which is zero].)

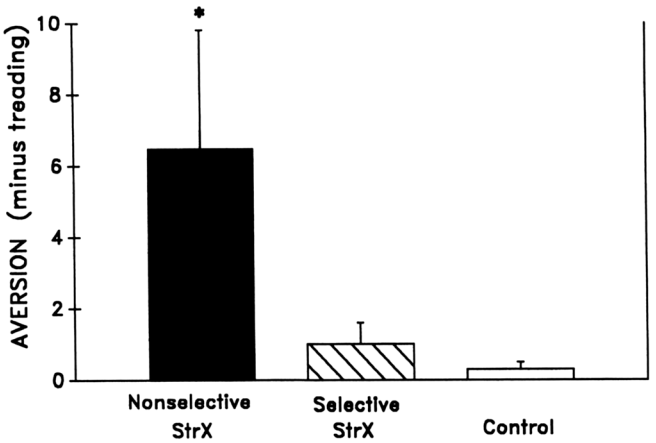

Figure 9. Enhancement of aversion to sucrose by lesions that produced nonselective treaders (Experiment 2). (Mean [$\pm SE$] number of aversive actions [gape, chin rub, face wash, head shake, forelimb flail] elicited by sucrose in the 1st week from the two groups of rats that received small striatopallidal [StrX] lesions and from the control group.)

In other words, these two changes might occur independently and be controlled simply by different thresholds of striatopallidal damage. If this were true, then sensorimotor treading would be potentiated by moderate striatopallidal damage to particular sensory features shared by quinine, HCl, and citric acid (Figure 2B; although it is difficult to specify the sensory features that might be shared by these tastes, based on either electrophysiological or psychophysical studies of taste coding; Pfaffmann, Frank, & Norgren, 1979; Schiffman & Erickson, 1980). The motivational potentiation of aversion would not be produced by this moderate degree of neural damage, and palatability would be left unchanged in taste-selective treaders. Further damage (or more recent damage in Experiment 1) might further potentiate the sensorimotor pathology, thereby expanding the sensitivity of the sensory trigger so that it could be activated by any taste. As a separate consequence, extensive damage might also enhance aversion, leading to elevated aversive reactivity. This affective consequence would contribute to produce aphagia by distorting food palatability. According to this hypothesis, however, the enhancement of aversion would not play a role in the expansion of treading by severe striatopallidal damage.

A hierarchical alternative to this adjacent-systems possibility was suggested in the discussion to Experiment 1: that motivational and sensorimotor changes interact in the production of exaggerated treading (Figure 2C). The interactive hypothesis posits that aversion is the trigger of the exaggerated treading and that the sensorimotor effect of severe neural damage is nested within the motivational effect. Even in rats that tread only to quinine and citric acid, it would be the normal evaluation of these tastes as aversive that triggered exaggerated treading—not shared sensory features of the tastes. In addition, larger lesions would potentiate the aversive evaluation that serves as trigger. This would not only contribute to aphagia and to taste-reactivity changes but would also cause tastes that are normally palatable to become effective elicitors of treading.

The crucial feature of the interactive hypothesis is that it posits an affective state of aversion to be the trigger that activates exaggerated treading. Aversive evaluations of taste palatability are not reducible to the sensory features of a taste (Grill & Berridge, 1985). Other factors interact with taste to control palatability. These include specific types of Pavlovian associations (e.g., Parker, 1988; Pelchat, Grill, Rozin, & Jacobs, 1983) and physiological states relevant to homeostasis. Formation of a Pavlovian association between a palatable sweet taste and the visceral illness caused by LiCl, for example, can convert the positive hedonic reactivity to that sweet taste into strong aversion (Berridge et al., 1981). Experiment 3 used this ability of taste aversion conditioning to reverse the affective palatability of sweet tastes to distinguish between the two hypotheses just described. To answer whether the selective treading of rats with partial lesions to bitter and sour tastes was triggered by affective or by sensory features, taste–illness aversion conditioning was used to reverse the affective evaluation of a sweet taste without changing its basic sensory features. This was done by conditioning a discriminative taste aversion for a particular sugar (either fructose or maltose), by pairing it associatively with LiCl injections, whereas the other sugar remained "safe" and palatable. The adjacent-systems hypothesis of separate sensorimotor and motivational changes predicts that this purely psychological manipulation should leave selective treading unchanged: Rats with partial lesions should still show exaggerated treading to tastes such as quinine and citric acid but not to any sugar. The hierarchical hypothesis of nested interaction between exaggerated treading and aversion, on the other hand, predicts that an aversive taste should elicit treading, regardless of how its aversive palatability was acquired. This hypothesis predicts that rats that show selective treading to sour and bitter tastes should show exaggerated treading also to the sugar that was paired with LiCl (CS+) but not to the unpaired, safe sugar (CS−).

Experiment 3

Method

Presurgical Discriminative Conditioning

One week before surgery, a discriminative taste aversion to either fructose or maltose was formed in 14 male Sprague-Dawley rats using a procedure modified from Berridge et al. (1981). Food (rat chow) and water were available to the rats throughout conditioning. Seven rats received 0.15 M fructose as their CS+ and 0.15 M maltose as their CS−, whereas the other 7 rats received 0.15 M maltose as their CS+ and 0.15 M fructose as their CS−. On alternate days, 15 ml of CS+ or CS− and 15 ml of water were made available to each rat in its home cage for 30 min. After the 30-min period, rats were given injections of LiCl (1.5 mEq/kg ip), on days when they had received their CS+, or isotonic saline, on days when they had received their CS−. A rat was considered to have developed a discriminative aversion when it ingested at least 10 ml of the CS− but no more than 5 ml of the CS+ on two consecutive trials with each stimulus. After this criterion was met, the rats were anesthetized and small bilateral striatopallidal lesions were made with kainic acid (0.5 μg in 0.5 μl phosphate buffer) in 8 rats in Experiment 2. The remaining 6 rats received striatopallidal injections of only the buffer vehicle and were used as controls. Chronic oral cannulae were implanted in all rats as in Experiment 1.

Postsurgical Conditioning

Both lesion and control groups received intraoral discriminative conditioning training over Days 2–5 after surgery to strengthen the discriminative aversion. The CS+ and CS− were presented on alternate days. On each trial, a delivery tube was connected to the oral cannulae, and the rat was placed in the test chamber. After 5 min, 1 ml of the CS+ or CS− was infused into the mouth over a 1-min period. The rat received injections of either LiCl (after its CS+) or saline (after its CS−) as already described. Every rat displayed aversive responses to delivery of its CS+ by Day 5.

Behavioral Testing

Exaggerated treading was examined after oral infusions of sucrose (1.0 M), quinine (3×10^{-4} M), the CS+ sugar (0.15 M fructose for half of the group; 0.15 M maltose for the other half), and the CS− sugar (again maltose or fructose for different rats). Rats were tested with each stimulus every day (sucrose, quinine, and CS sugars) over Days 1–7 as in Experiment 1. At the completion of testing, rats were

anesthetized and perfused, and histology was conducted as in Experiment 1.

Results

Criterion for Analysis

As in Experiment 2, rats with lesions could be grouped into two categories 24 hr after surgery with respect to the exaggerated treading elicited by novel tastes: those that treaded only to a naturally aversive taste (quinine) on the day after their lesion and those that treaded to all tastes including sucrose on Day 1. The goal of this experiment was to compare exaggerated treading elicited by the CS+ and CS− from rats that treaded selectively to quinine but not to sucrose. For this reason, CS reactivity and treading comparisons began for each rat at the time that the rat showed selective treading: beginning the 1st day after lesion if the rat never treaded to sucrose or else beginning on the day of recovery that a rat stopped treading to sucrose (Days 2–7) if it treaded to all tastes on the 1st day after its lesion.

Exaggerated Treading

Rats with striatopallidal damage showed strong exaggerated treading both to quinine and to their CS+ sugar but never to either their CS− sugar or to sucrose (Figure 10), $F(1, 7) = 20.25$, $p < .01$. When rats with striatopallidal lesions reached the criterion of selective treading, they still emitted prolonged bouts of exaggerated treading to their CS+ sugar ($M = 22.5 \pm 4.99$ s) but never treaded to their CS− sugar ($p < .01$) once they had ceased treading to sucrose. Control rats, which had formed discriminative aversions but did not receive lesions, did not show exaggerated paw treading, by our criterion, to any stimulus.

Aversion Analysis

At the time when rats stopped treading to sucrose, rats with lesions did not differ from control rats overall in aversion, $F(1, 12) < 1.0$. The CS+ and quinine both elicited more aversive responses than did sucrose ($p < .05$, LSD each). The CS+ sugar that had been paired with LiCl elicited marginally more aversive actions than the safe CS− sugar only if paw treading was included in the aversive count, $F(1, 7) = 5.95$, $p < .05$.

Lesion Analysis

The pattern of cell loss and gliosis after striatopallidal lesions was similar to that found in Experiment 2 (Figure 3, bottom row; see also Figure 4). Necrosis was found in the posteriomedial striatum and globus pallidus along the border of the lateral ventricle. Rats that treaded to all tastes initially tended to have larger lesions.

Discussion

These results confirm the interactive prediction that an aversive CS+ sugar should be an effective elicitor of treading for rats that otherwise tread selectively to quinine but not to palatable sugars (sucrose, CS−). This confirmation supports the hierarchical hypothesis that the trigger of treading is an affective evaluation of aversive palatability. Exaggerated treading was manipulated in this experiment by associative conditioning, a psychological manipulation that changed the affective evaluation of the CS+ from hedonic to aversive without altering the physical properties of the taste. The creation of a conditioned aversion to the CS+ sugar, by associative pairing with illness, caused a simultaneous expansion of the class of treading elicitors to include that CS+ sugar for rats that would ordinarily tread only to naturally aversive tastes such as quinine (Figure 10). This result cannot be explained by the adjacent-systems hypothesis. It leaves no doubt that the crucial factor that determines whether treading will be elicited by a particular taste is the affective evaluation, rather than any sensory feature, of that taste.

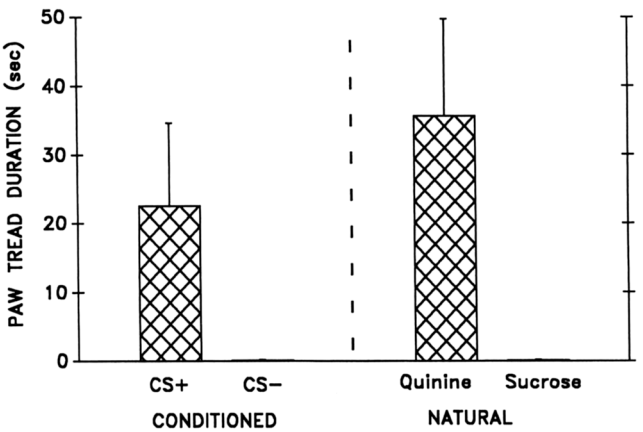

Figure 10. Exaggerated treading to an aversive CS+ sugar (Experiment 3). (Duration [$M \pm SE$; in seconds] of choreic treading elicited by the CS+ and CS− sugars, quinine, and sucrose after selective treading criterion was reached. Only rats with striatopallidal lesions are shown. Control rats did not show choreic treading to any stimulus.)

General Discussion

These experiments reveal an interaction between motivational and sensorimotor neurobehavioral systems of the corpus striatum. This interaction is expressed in the pattern of exaggerated treading after striatopallidal damage. Exaggerated treading is better described as a *sensory–affective–motor disorder* (Figure 2C) than as a sensorimotor disorder (Figure 2B). It is an affective evaluation of aversive palatability, not gustatory or other oral stimulation per se, that triggers this pathological action pattern.

Inadequacy of a Single-Factor Sensorimotor Interpretation

A single-factor sensorimotor account must attempt to explain all behavioral consequences of a lesion in terms of a sensorimotor change. Sensorimotor changes can explain treading and deficits in coordination after striatopallidal le-

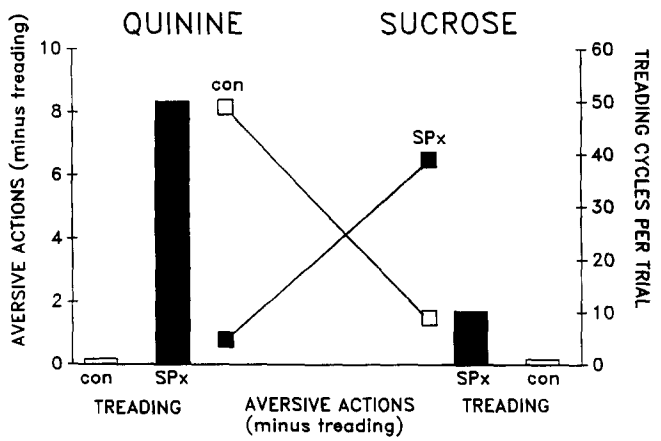

Figure 11. Reversal of sensorimotor relations. (Treading is transformed from a very rare aversive action into a frequent one by striatopallidal [SPx] lesions, and the potentiation of exaggerated treading suppressed the emission of other aversive responses. Exaggerated treading [bars] and other aversive actions [squares] emitted by rats that treaded to all tastes after lesions [filled symbols] and by control rats [open symbols] to sucrose [right side of figure] and to quinine [left side] are portrayed from Experiments 2 and 3. Sucrose elicited exaggerated treading plus elevated aversion [gapes, chin rubs, face washes, forelimb flails] from rats with lesions. Further potentiation of treading by quinine suppressed other aversive actions to below control levels. Cycle of treading = extension of right paw with simultaneous retraction of left paw, followed by extension of left paw with simultaneous retraction of right paw.)

sions. However, sensorimotor changes fail to explain the enhancement of aversion to sucrose during aphagia (the increase in aversive actions other than treading). The enhancement of aversion by striatopallidal damage is a distortion of affective processing itself. This change requires more recent or more severe striatopallidal damage than is needed to induce treading alone. The distortion magnifies the aversive evaluation of all tastes so that aversion is evoked even by stimuli that would elicit positive hedonics from a normal rat (e.g., sucrose). Finally, a sensorimotor account alone cannot explain how the conditioning of a learned aversion could affect treading.

Inadequacy of a Single-Factor Motivational Interpretation

Although our results show that aversion may be enhanced by striatopallidal damage, this motivational change alone can no more account for all behavioral consequences than can a single sensorimotor change. Aversive enhancement cannot explain the chorealike potentiation of treading. Exaggerated treading reflects a lesion-induced restructuring of sensorimotor relations among responses to tastes (Berridge & Fentress, 1986) in which the probability of treading in relation to other aversive actions is reversed by the lesion from very low to very high.

Although infrequent treading can be elicited by aversion in normal rats (albeit never with sufficient intensity to qualify as exaggerated), it should be noted that the enhancement of aversion by striatopallidal lesions could not by itself have produced the exaggeration of treading, in turn, as an enhanced aversive response. No known degree of aversion can elicit comparable treading from rats. Even an extremely bitter solution of supersaturated 1.0 M quinine HCl (40° C) elicits predominantly gaping, chin rubbing, and so on—not exaggerated treading—from normal rats (personal observations). Exaggerated treading reflects a true sensorimotor restructuring. If we compare the relative frequency of treading with other aversive responses shown by severe treaders and by control rats from Experiments 2 and 3 (Figure 11), we see that treading can suppress the emission of other competing actions. Treaders show more aversive actions, such as gapes, forelimb flails, and so on (even excluding treading), to sucrose than do control rats. But when the intensity of treading is enhanced further (by presenting quinine), it suppresses other aversive actions to below control levels. The potentiation of treading, in other words, is simply the most obvious aspect of a larger sensorimotor change that has restructured the emission probabilities of all aversive actions.

Inadequacy of an Adjacent-Independent-Systems Interpretation

An adjacent-systems interpretation must account for the simultaneous enhancement of aversion and the potentiation of treading by supposing that striatopallidal lesions damage independent motivational and sensorimotor circuits. But it is clear that the sensorimotor and motivational changes produced by our lesions were not functionally independent. If they were, then exaggerated treading should not have been affected by a learned aversion in Experiment 3. The ability of a learned aversion to activate exaggerated treading implies that sensorimotor and motivational factors were interactive rather than independent. They interacted in the sense that the motor pathology was seen if, and only if, a particular motivational–affective state (aversion) was present (Figure 2C). We need an interpretation that can account for this interaction.

Hierarchical Interaction

Of the three previously discussed alternatives for interpreting simultaneous sensorimotor and motivational changes, only theories based on hierarchical views of neurobehavioral function are able to account for this sensorimotor–motivational interaction (Figure 12). In a classic hierarchical formulation, Jackson (1958) used the term "re-representation" to denote the process by which psychological states could be formed out of elementary sensorimotor units. For affective states, "the emotional centre itself represents, although very indirectly, the parts of the body concerned in different emotional manifestations, and . . . the emotion arises during the central activities which, through subagency of the middle and lowest centres, produce the manifestations" (Jackson, 1958, p. 66). According to this hierarchical concept, sensory signals were re-represented at higher levels in new combinations. Processed and elaborated at these levels, they carried

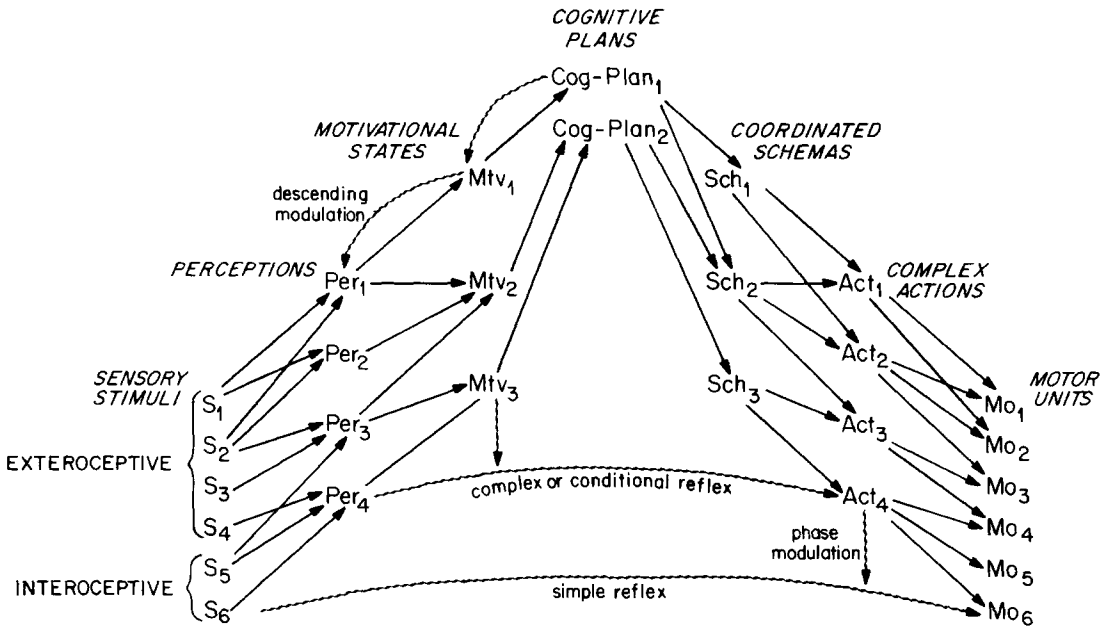

Figure 12. Hierarchical interaction between sensory–motivational–motor systems. (Connections between sensorimotor and motivational functions according to hierarchical theories of neurobehavioral organization. Each numbered unit represents an element of a functional hierarchy, such as a percept, motivational state, cognitive plan, motor command, and so on. Each level within the hierarchy represents a type of system [e.g., sensory], which has many elements. Interactions among different levels can be either hierarchical via signal inputs [ascending arrows on the right side of the hierarchy], commands [descending arrows on the left side], and modulation [descending "italic" arrows], or horizontal via reflex loops [horizontal italic arrows].)

properties and significance that they did not possess at lower levels; still, however, they retained a sensorimotor function that represented their "parts of the body." Jackson's view thus bridged the ascent from sensory to affective systems. In terms of motor control, a corresponding bridge downward existed in this hierarchy between motivational states and motor outputs. Motivational states were expressed only through the "final common path"; that is, through their modulatory influence on lower motor systems (see Gallistel, 1980). In modern hierarchical theories of neurobehavioral organization, sensory, motivational, and motor systems are linked together in a more elaborate but essentially similar fashion (e.g., Cools, 1985; Dawkins, 1976; Fentress, 1983; Gallistel, 1980; Teitelbaum, 1986). These functional levels are connected by the continuous flow of information upward from sensory perception to affective evaluation and higher systems and downward from affective evaluation to motor response (Figure 12). The sensory–affective–motor interaction seen in exaggerated treading can be understood, according to a hierarchical view, by supposing that striatopallidal lesions restructure the flow of signals up and down this hierarchy.

But it is important to note that this striatopallidal syndrome could not have resulted simply from changes within any level of a control hierarchy. It was not the case that lesions in these experiments changed the "motor level" of a control hierarchy in a fashion that simply enhanced the excitability of treading as a motor unit (Figure 2A) or even as a motor response to all kinds of noxious stimuli. Treading is not potentiated as a response to aversive stimuli in general, such as loud noises, air puffs, taps, or objects that deliver electric shocks (Berridge et al., 1988), but rather specifically to aversive palatability. The special relation of lesion-induced exaggerated treading to taste palatability recalls C. J. Herrick's (1926) prescient suggestion that "the globus pallidus . . . may be concerned chiefly with locomotor and facial reflexes involved in feeding" (pp. 113–114, cited in Mogenson, Jones, & Yim, 1980).

Vertical Structure

Why should exaggerated treading be elicited chiefly by a particular type of affective state? The key to explaining the specificity of this sensorimotor–motivational link may lie in the vertical connections between units that are at different levels of the control hierarchy (level and vertical connection are used here in a functional rather than anatomical sense). Vertical connections join specific elements at one level to elements at another (Figures 12 and 13). This means that a particular sensorimotor element, for example, may be controlled only by particular motivational systems and may be segregated from other elements even if those elements belong to the same sensorimotor level as itself. A consequence of this connectional specificity is that vertical "panels" or modules are formed that comprise connected units from different

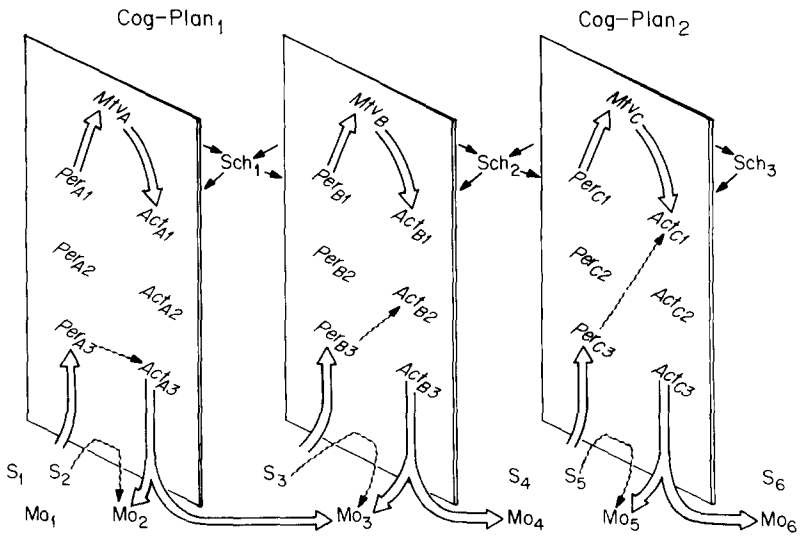

Figure 13. Vertical structure control hierarchy. (The same control hierarchy portrayed in Figure 11 but with the addition of vertical aspects of structure. Specific connections between elements from different levels allow integrated systems to be assembled out of these elements. These integrated systems are depicted here as distinct vertical panels or modules. The sensory and perceptual elements that contribute to a particular motivational state, such as aversive palatability, together with the motor patterns that are activated by that state, are represented by a single panel within the figure. Within this panel, all sensorimotor relations would be altered by a striatopallidal lesion: Taste aversion would become enhanced relative to taste hedonics, treading would become potentiated as a response to aversion, and other aversive responses would become suppressed. Different functional modules or panels may share certain motor outputs [e.g., Mo3; because treading, for example, can be activated naturally both by taste aversion and by defensive burying], but a striatopallidal lesion that altered the single panel would not affect the activation by another panel of the shared motor output. Information still flows hierarchically [large arrows] and in horizontal reflex loops ["italic" arrows], but units and flow paths are constrained within a vertical module. Interaction between modules is also restricted to particular routes, as between certain coordinated behavioral schemas that might inhibit, facilitate, or otherwise modulate one another.)

levels. Because they are connected, units within a panel act together as a single functional system but act separately from the units of other vertical modules that span across the same levels (Figure 13). In terms of this formulation, striatopallidal lesions reorganize the vertical relationship between evaluations of aversive palatability, at one level, and the set of species-specific motor patterns that are activated by aversion (treading, face washing, gaping, etc.) at a lower level. This reorganization does not apply to motor patterns activated by other higher states, even if those states are aversive in a different way and even if the same motor patterns are used (e.g., treading is not potentiated as an element of object burying elicited by an electrified object even in rats that tread to oral sucrose; Berridge et al., 1988). Instead of representing a change within the level that controls the motor activation of paw treading and other actions (i.e., a motor potentiation of treading), exaggerated treading results from a potentiation of the link from oral aversion to this movement together with a weakening of the links to all other aversive actions (Figures 2C and 13).

Rephrased in another way, the effect of this change in hierarchical vertical structure is to alter a contextually dependent set of sensorimotor relations (see Fentress, 1990). These sensorimotor relations persist so long as a particular motivational state remains but are replaced by a different set of relations (even involving the same elements) as soon as motivational state is changed. During states of aversive palatability, paw treading is highly potentiated as a sensorimotor response in rats that have striatopallidal lesions. This potentiation is so extreme that the enhanced treading suppresses the emission of other aversive actions: a restructuring of sensorimotor relations compared with normal animals (Figure 11). But treading is not potentiated as a sensorimotor response during other motivational states. Even other situations that produce aversive affect unrelated to ingestion fail to evoke exaggerated treading from rats that tread to sucrose (Berridge et al., 1988). If we accept that dynamic changes may occur in sensorimotor relations when motivational context is altered, we should recognize that a challenge is posed to our predominantly "static" models of neurobehavioral organization. Contextually dependent sensorimotor systems illustrate the need to incorporate dynamic vertical aspects of organization into our models of neurobehavioral hierarchy (Fentress, 1983, 1990).

The production of choreic treading by striatopallidal damage can be explained parsimoniously in terms of this view

of hierarchical vertical structure. It requires that lesions change only a single vertical panel in Figure 13, representing relations between aversive evaluations of palatability and the motor units that are activated by these evaluations. Within that panel, however, all relations are changed. The probability that a gape, head shake, flail, or other aversive action will be activated by an aversive evaluation of palatability is altered by the lesion because of the potentiation of treading. This explanation of striatopallidal treading in terms of vertical structure does not replace a hierarchical interpretation, of course, but merely shifts explanatory emphasis from the organization that exists within horizontal levels of the hierarchy to the organization that spans across them. The results of these experiments indicate that one function of striatopallidal circuits is to provide specific links across sensory, motivational, and motor levels.

The demonstration of hierarchical interaction sheds light on why the amplification of aversion to sucrose in Experiments 1 and 2 was accompanied by an expansion of the class of choreic triggers to include that taste. It also explains why both the enhanced aversion and the treading to sucrose disappeared together during recovery in Experiment 1, whereas treading to quinine and HCl remained. An enhancement of aversion was sufficient in both cases to produce aphagia and extend treading to normally palatable tastes. When the amplification of aversion declined for these tastes, after recovery in Experiment 1, the exaggerated treading also disappeared. But tastes that were naturally aversive remained fully capable of triggering exaggerated treading, because the vertical panel restructuring of affective–motor relations does not show an early recovery in parallel with the recovery of magnified aversion.

Related Evidence

This interpretation of exaggerated treading in terms of a vertical reorganization between sensory–affective–motor functional levels is consistent with a variety of anatomical, behavioral, clinical, and electrophysiological evidence concerning the "limbic striatum" and ventral pallidum (Alheid & Heimer, 1988; Mogenson et al., 1980; Nauta & Domesick, 1984). The area of the globus pallidus that was damaged in this study receives projections from the ventromedial limbic striatum and nucleus accumbens. The ventral border of the pallidum itself is interwoven with the "extended amygdala system" of the basal forebrain (Alheid & Heimer, 1988). This anatomical organization places ventral pallidal circuits in an excellent position to act "as an interface between the motivational and the more strictly motor aspects of movement" (Nauta & Domesick, 1984, p. 3).

Human clinical disorders of movement that are caused by basal ganglia dysfunction have often been observed to display an interaction with emotional states. For example, some symptoms of Parkinson's disease may be ameliorated during states of emotional stress, whereas other symptoms are enhanced by the same states (Marsden, 1982). The hyperkinesias of Huntington's disease and related disorders may show potentiation during states of psychological stress (Narabayashi, Chida, & Kondo, 1979). In animals, excitotoxic lesions of the ventral pallidum may block the self-administration of cocaine and heroin (Koob, Stinus, LeMoal, & Bloom, 1989), adding further evidence of a motivational role for striatopallidal neural systems.

Electrophysiological recordings from striatopallidal neurons have indicated that the sensorimotor functions of some striatal circuits are nested within motivational systems. Responses to visual or auditory stimuli may be controlled by the novelty or motivational significance of those stimuli (Aldridge, Anderson, & Murphy, 1980; Rolls & Williams, 1987; Schneider, 1987; West, Michael, Knowles, Chapin, & Woodward, 1987) rather than controlled merely by sensory features. The receptive fields of some striatal units, which respond to tactile oral stimulation, expand when a cat is looking at food (Manetto & Lidsky, 1984), in a manner reminiscent of Flynn's original discovery of hypothalamic expansion of receptive fields during motivated states (e.g., MacDonnell & Flynn, 1966). Finally, in accordance with the vertical structure connection between motor, sensory, and motivational functions indicated by our experiments, Lidsky and Manetto (1987) have reported a population of striatal units that appears to have sensorimotor functions that are nested within a specific motivational state. They have described neurons that fire in conjunction with oral movements but only when those movements are used for feeding and not when they are used in other contexts. A similar motivational nesting of sensorimotor function has been demonstrated to exist in a set of sensorimotor orientation deficits produced by nigrostriatal lesions. These are behavioral deficits in orosensory responsiveness and oromotor control that appear only when a rat is engaged in ingestive contexts and not in behavioral contexts such as body grooming or resting (e.g., Hall & Schallert, 1988; Schallert & Hall, 1988). Context-dependent restructuring of sensorimotor relations also may be induced by pharmacological manipulations (e.g., Morrissey, Pellis, Pellis, & Teitelbaum, 1989). Recognition of this type of vertical structure in neurobehavioral systems allows such contextual or motivationally nested properties of sensorimotor neurons or of behavior to be seen as natural instead of as paradoxical.

Conclusion

These experiments show that sensory–affective–motor functions are combined in the operation of striatopallidal systems. Striatopallidal lesions produce both an enhanced aversion to tastes and a pathologically intense potentiation of treading as sensorimotor response. These two effects of striatopallidal lesions interact in a hierarchical way. Such interaction can be understood in terms of hierarchical linkage within the corpus striatum between specific sensorimotor and motivational systems.

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