fected at those frequencies, while absolute sensitivity declined by 40 to 55 dB; we therefore conclude that the outer hair cells are unnecessary for normal intensity discrimination. Although the precise mechanism of intensity discrimination is not clear, the results suggest that those processes contributing to the discrimination are unimpaired at high SPL's even though cochlear mechanics may conceivably be altered by outer hair cell loss.

Although the stereocilia of inner hair cells remaining after kanamycin treatment may not appear normal when examined by scanning and transmission electron microscopy (*12*), the DL's we measured must have depended on activity of the inner hair cells. To our knowledge this is the first demonstration that the outer cells are not necessary for auditory intensity discrimination. Although several investigators have indicated that normally hearing human subjects and those with a moderate sensorineural hearing loss have DL's of the same magnitude (*13*), patterns of hair cell loss in the hearing-impaired patients could only be inferred without histological confirmation.

The contribution of the outer hair cells to frequency discrimination and frequency selectivity is equivocal (*14*). Assessing all data regarding differential functions of the two types of hair cells, we conclude that the outer cells, essential for normal absolute sensitivity, are not necessary for at least one aspect of suprathreshold auditory signal detection—intensity discrimination.

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## Reward-Induced Stereotypy: Modulation by the Hippocampus

Abstract. *In animals with hippocampal damage, the signaled administration of reward is sufficient to induce the sort of behavioral stereotypy and locomotion that heretofore has been observed only after drug administration. Haloperidol returns these behaviors to normal. The interaction of the hippocampus with reward helps to explain many well-known characteristics of animals with lesions in the hippocampus and may have relevance for catecholamine-based clinical disorders.*

Two of the most pervasive and probably least controversial features of the behavior of animals with extensive bilateral hippocampal lesions are excessiveness (*1, 2*) and invariability (*3, 4*). While these characteristics are sometimes exhibited in situations in which reward is not explicitly provided, they are especially visible when the brain-damaged animal is positively or negatively reinforced. Behavior established by these means is intense, rigid, and difficult to change (*2, 4*). An understanding of the interaction of the hippocampus with the dynamics of reward would help to explain much of the functional diversity that continues to be ascribed to the structure.

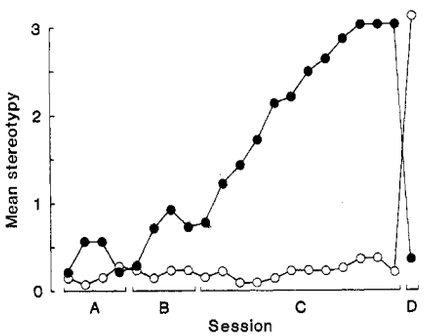


Fig. 1. Mean stereotypy scores of animals with hippocampal (closed circles) and neocortical (open circles) damage during free-fed (A), deprived (B), signaled reward (C), and drug (D) phases. Stereotypy scores: 0, asleep or stationary; 1, active; 2, predominately active but with bursts of stereotyped rearing and sniffing; 3, constant stereotyped activity over a wide area; and 4, constant stereotyped sniffing or head-bobbing in one place (6). Groups differed significantly in phase C [$F(1, 12) = 120.7$, $P < .001$]. Drugs affected both groups significantly [$t(6) = 13.0$ to $27.6$, $P < .001$].

We now report that rats with extensive bilateral hippocampal damage display an exaggerated reaction to reward. This reaction consists of behavioral automatisms that bear a remarkable resemblance to the peculiar stereotyped and locomotor acts ("stereotypies") that follow *d*-amphetamine administration. Reward-induced stereotypy is returned to control levels by the administration of a catecholamine antagonist.

These findings were obtained from seven rats with bilateral lesions of the hippocampus and seven with aspiration ablations of the neocortex (*5*). The animals lived in clear polystyrene observation units equipped with movement transducers (phonograph cartridges with digitized outputs) that provided activity data. These data were recorded from 1300 to 1400 hours, the midpoint of the lights-on period. After two habituation days, the rater, who was unaware of the animals' surgical and drug conditions, entered the experimental room at 1300 hours daily and scored the degree of stereotypy as specified by a rating scale


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(6) across conditions of free-feeding and then total deprivation (4 days each). Deprivation reduced body weights by about 20 percent.

Daily refeeding at 1400 hours—an amount of mixed, balanced animal ration sufficient to maintain body weights at 80 percent of predeprivation weight—then began and continued for the remainder of the experiment. The temporal arrangement of events permitted the entry of the rater and the time of day to signal food delivery, as the animals were otherwise undisturbed for the balance of each day. This established procedure (*7*) was expected to endow the rater with incentive reward properties and to avoid competition between stereotypy and eating behavior. Determinations of body weight and other maintenance procedures were carried out every 4 days just before feeding.

Drugs or vehicle were administered on the last two signaled reward sessions. This balanced procedure forced the rater to rely on observation, not guesswork, and it permitted assessment of the effects of the injection procedure. Haloperidol (the lactate, 0.06 mg per kilogram of body weight, in saline) was injected subcutaneously in animals with hippocampal lesions at 1230 hours, and *d*-amphetamine sulfate (1.5 mg/kg in saline) was administered intraperitoneally to control animals at 1240 hours. Vehicle (saline) was administered at corresponding times. Doses were selected on the basis of preliminary studies.

The signaled reward regimen drove stereotypy (Fig. 1) and locomotor activity (Fig. 2) of animals with hippocampal lesions far above control levels. This was in contrast to free-feeding and total deprivation, which had little or no differential effect on the two groups. Saline administration was without significant influence on the behavior of either group and is not illustrated. Haloperidol had a remarkable effect on the rats with hippocampal damage. By stripping away the superfluous behaviors induced by reward, the catecholamine receptor blocker rendered these rats superficially identical to animals with neocortical damage (*8, 9*). Conversely, the administration of *d*-amphetamine to controls produced behavior that—at least by our measures—was indistinguishable from the behavior of the rats with hippocampal lesions given saline.

When stereotypy was evident, regardless of group, it consisted of locomotion along relatively constant routes, rearing, orienting toward sources of stimuli that were not obvious to the observer, sniffing, and head-bobbing.

In view of these findings, it seems likely that the intact hippocampus moderates the effects of reward, probably by opposing catecholamine (*10*) mechanisms. It is probably in this way that the hippocampus promotes behavioral variability (*2*). Although we do not assert that unmodulated catecholamine activity accounts for all the reported effects of hippocampal damage, the findings do invite comparison between the behavior of intact rats administered amphetamine, another case of catecholamine-associated hyperactivity, and undrugged animals with lesions of the hippocampus. In both these cases extinction is retarded (*11*), spontaneous alternation is abolished (*12*), shuttle-box avoidance is improved (*13*), passive avoidance is impaired (*14*), tasks requiring lower rates of responding prove difficult (*15*), and "superstitious" behavior appears (*9*).

The catecholamine-hippocampus interaction has an importance beyond that of clarifying hippocampal function. The hypotheses that schizophrenic symptoms (*16*), affective disorders (*17*), and amphetamine psychosis (*18*) have their basis in catecholamine dynamics currently enjoy wide support. It would not be surprising—in fact it has been anticipated (*19*)—that such disorders could be exacerbated by the impairment of a system that opposes catecholamine function. Our findings point to an interaction that is in keeping with the central role of catecholamines, but which also accounts for the frequent implication of the hippocampus in these disorders (*18, 20*).

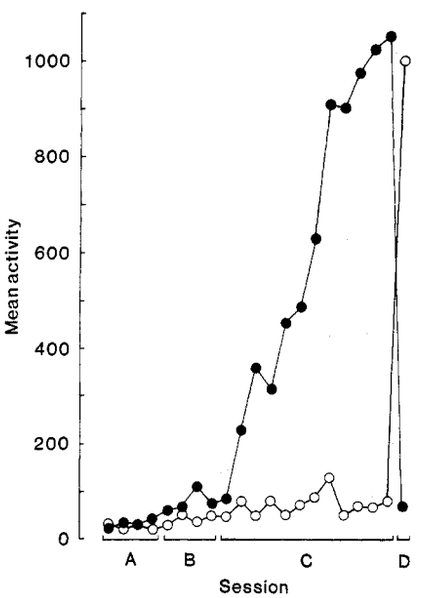


Fig. 2. Mean locomotor activity scores of animals with hippocampal (closed circles) and neocortical (open circles) damage during phases defined as in Fig. 1. Groups differed in locomotor activity during phase C [$F(1, 12) = 81.9$, $P < .001$], and these levels of activity were reversed in the drug phase [$t(6) = 12.8$ to $19.7$, $P < .001$].


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