

Taste Reactivity as a Measure of the Neural Control of Palatability

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I. Introducing the Problem

A. PALATABILITY AS A RESPONSE MEASURE

Whether an animal will eat when the opportunity arises depends on a complex interaction of environmental, physiological, and associative factors. Three types of information are widely accepted as principal determinants of this decision to feed: taste signals initiated by contact with food, internal-state signals produced by food intake or starvation, and cues arising from previous associations with the taste of a food. Whether an animal will ingest or reject a food results from a CNS integration of these three factors, which we define as the palatability decision.

Palatability so defined is a response measure. The more common use of palatability is as a stimulus measure. It is generally used as a descriptor of the taste factor, as in "sucrose is a palatable taste." The problem with using palatability as a stimulus measure is that this usage generates an implicit prediction about the type of responses that palatable and unpalatable tastes will evoke. The implication of defining sucrose as a palatable taste and concentrated salt as an unpalatable one is the prediction that animals will ingest the former and reject the latter. We know, however, that this is not the case. Any given taste stimulus does *not* always elicit the same behavioral response. The very same taste may elicit ingestion or rejection or be rated as pleasant or unpleasant depending on the physiological state of the animal when it encounters that taste (Richter, 1956; Cabanac, 1971; Davis and Levine, 1977) and the existence of conditioned associations to the taste's postingestive consequences such as a previous illness or caloric repletion or a visceral illness (Booth, 1972, 1980; Garcia and Koelling, 1966; Rozin and Fallon, 1980).

The choice of a stimulus or a response measure of palatability has an impact on the study of the neural mechanisms of taste. A stimulus view of palatability fosters the hypothesis that the neural signals generated at the taste receptor and the initial processing of these signals within the caudal brain stem are unaltered by internal state and associative input. In contrast, the response view of palatability promotes investigation of whether the taste signal is integrated with internal-state and associative signals early in its central processing. A response measure of palatability even provides for the possibility of centrifugal influences on the taste receptors themselves. The available evidence supporting a caudal brain stem integration of taste and internal-state afferent signals is presently suggestive, as Norgren (1983) notes, but perhaps only because the stimulus view of palatability has dominated and this issue has seldom been addressed.

B. PALATABILITY AS AN INTERVENING VARIABLE

Physiological psychology has long recognized that diverse causal factors can act through a common mechanism (Miller, 1959). Some experimental manipulations may produce a constellation of effects on a variety of appetitive and consummatory behavior related to feeding that closely resembles the constellation of effects produced by other manipulations. For example, the postingestive cues arising from a meal, the addition of quinine to a food, and the previous association of a taste with visceral illness may all affect behavior in similar ways. All of these experimental manipulations reduce consumption of foods with the same taste in a preference test, reduce instrumental responding for that taste, produce a shift in taste-elicited fixed action patterns (FAPs) (in rats), and a shift in hedonic ratings of the food (in humans). All of these behavioral changes reflect the central nervous integration that we are calling palatability.

This association of physiological, environmental, and associative causes into groups defined by their similar behavioral effects has given rise to the psychological concept of central intervening variables. Palatability, hunger, and satiety are examples of central intervening variables. This concept suggests that all of the causal factors within such groups affect a single functional system whose output produces a similar constellation of effects on a set of behaviors. It also implies that the behavioral effect of activating this system does not depend on which particular causal factor activated it. That is, the same behavior is elicited by quinine-adulterated food, by sucrose that had been paired with visceral illness, and by concentrated NaCl when animals had received extra salt in their diet.

Palatability, hunger, and other central intervening variables can be of considerable use in understanding the relations that environmental events have to central neural events, and that central neural events have to behavioral events. These variables must be defined very carefully, however, in order to be useful. Confusion concerning the meaning of concepts like hunger and palatability has led to misunderstandings in the past and even to more recent suggestions that these concepts be dropped entirely from physiological analyses of behavior (Dethier, 1976, 1982; Zeigler, 1983). A number of points can be raised against these suggestions. From a merely descriptive point of view, central intervening variables such as palatability can order factors that would otherwise appear unrelated, such as gustatory stimulation, postingestive consequences, and Pavlovian associations, into comprehensible groups that share common relationships. These common relationships can then be used in two ways. First, they allow one to predict behavior more efficiently than can be done on the

basis of physiological knowledge alone. Second, and most important to physiological psychology, these relationships aid in the study of the central nervous system by helping to identify and locate those neural mechanisms that have important roles in the control of a particular behavior (Fentress, 1980; Gallistel, 1980a). The physiological mechanisms of behavior can be identified only by the correlations that exist between physiological and behavioral events. When manipulations of one are followed by a change in the other, we infer a causal connection.

If a central nervous manipulation, like stimulation or lesion, produces a change in a constellation of different behaviors that are also similarly affected by a group of independent variables, like alterations in taste quality or physiological state, then we may be justified in believing that we have found a single functional system that exerts control over a variety of behavioral outcomes. This argument derives from von Holst and von St. Paul's demonstration of the need for "level-adequate terminology," that is, for terms that adequately express the psychological complexity of the behavioral phenomena they represent. For example, if electrical stimulation of a particular brain site elicits neither a stereotyped motor response (e.g., walk forward) nor a behavior that could convincingly be described as a response to a particular sensation (pain, touch, etc.), then the activation of simple sensory or motor systems can be ruled out. If instead the stimulation evokes a set of functionally coordinated actions that bring the animal to a certain goal and are appropriately modified to attain that goal when the situation changes, then one can conclude that the stimulation has affected a motivational system that intercedes between sensory and motor systems (von Holst and von St. Paul, 1963). Likewise, if an experimental manipulation changes the consumption of a particular taste, its value as an instrumental reinforcer, its ability to elicit ingestive neuroendocrine responses, and the type of ingestive fixed action patterns (Timbergen, 1952) it can evoke, then we can conclude that the taste's central nervous evaluation has been altered.

It is the grouping of causal inputs and behavioral outputs that allows us to identify such functional control systems and helps us to identify the neural networks that embody them. Further, since behavioral studies that identify intervening variables by correlating inputs and outputs provide quantitative information about how different factors affect behavior, these behavioral data can specify the quantitative characteristics that must be shared by the neural mechanism responsible for the behavior. Behavioral data of this kind may include the range of effective stimuli, the quantitative input-output gain, the effective timing of inputs and patterning of outputs, and the values at which asymptotes begin. Such characteristics tell us what we need to know to identify the neural substrate for the

behavioral system of interest. This view of physiological psychology has much in common with Hebb's assertion (1949) that to proceed we "must find an anatomical and physiological understanding of what is known psychologically as a concept." This approach has been fruitfully applied to a variety of phenomena, from the resolution of visual Mach bands (the perceived gradations of brightness within an actually uniform band juxtaposed next to bands of differing brightness) into the physiological mechanisms of lateral inhibition in the retina (Schiffman, 1976), to the correlation of the temporal properties of elemental classical conditioning with sensory synaptic changes in *Aplysia* (Hawkins *et al.*, 1983), to the identification of the quantitative properties of reinforcing electrical brain stimulation with the particular neural pathways that share those properties (Gallistel *et al.*, 1981). Such successes illustrate the importance of complex behavioral concepts in the study of the nervous system.

C. FUNCTIONAL AND MECHANISTIC ANALYSES OF BEHAVIOR

In order for central intervening variables like palatability to be useful in studying neural mechanisms of behavior, however, the relationship that such variables bear to the neural mechanisms that mediate the behavioral outcomes must be clearly understood. Palatability evaluations or decisions are purely functional entities. A functional entity is not itself a neural mechanism, nor does it refer to a particular neural mechanism (Powley, 1977). A functional entity refers to a particular relationship between a set of experimental manipulations and a set of behaviors. A functional analysis of behavior therefore cannot replace a physiological or mechanistic analysis. It is equally important, however, to realize that a physiological mechanism cannot replace a functional intervening variable. Each refers to different things and neither is more "real" or "basic" than the other.

It has been argued that a complete physiological analysis of a behavioral system will show a functional one to be empty, misleading, or, at best, redundant (Dethier, 1982). This argument assumes that both kinds of analyses attempt to describe the same kind of thing and that one or the other must be true or best. But each approach is good for separate purposes. Functional analyses (meaning analyses of the functional controls of behavior, not analyses of an evolutionary function of behavior) will always provide the most efficient means of predicting the behavior of a complex system (Dawkins, 1976). And as the complexity of an animal, a neural preparation, or an array of competing stimuli increases, the relative predictive efficiency of a functional analysis will increase proportion-

ally. Further, the argument against functional constructs neglects the fact that, to the degree that a mechanistic explanation can be complete, it must explain the very relations that the functional analysis points out (Fentress, 1980; Gallistel, 1980b). Recognition of those functional relations is often what guides the successful experimenter in elucidating the neural mechanisms of a behavior whether he or she is explicitly aware of it or not.

The central neural integration whose outcome determines palatability is just such a functional entity. It highlights a particular relationship that exists between a set of behaviors that animals direct toward tastes and the set of causal factors that change those behaviors as a group, such as internal-state cues and classically conditioned associations, as well as taste properties themselves. This relationship immediately tells us a number of important things about palatability decisions. First, palatability does not reside in a taste stimulus itself nor in the gustatory receptors that are activated by that taste. The very same taste may be ingested or rejected, may activate positive or negative FAPs, and may be reported as pleasant or unpleasant depending on the present physiological state and classical associations that may exist between that taste and its post-ingestive effects. For example, while an infusion of sucrose into the mouth elicits a stereotyped set of ingestive FAPs in a naive rat, that ingestive pattern of responses is reduced as the animal becomes sated (Grill and Norgren, 1978d). This sequence of ingestive FAPs can even be switched to an equally clear sequence of distinct, aversive FAPs by previous pairings of that sugar stimulus with LiCl in a classical-conditioning paradigm (Grill, 1975; Berridge *et al.*, 1981). The sucrose taste stimulus is unchanged; yet the central evaluation of the taste that results in a particular consummatory behavior has entirely changed. Likewise, the taste of concentrated NaCl, which elicits aversive FAPs in normal rats, can be made to elicit only ingestive FAPs by sodium-depleting natriorexigenic treatment (Berridge *et al.*, 1984). This switch from aversive to ingestive evaluation can also be elicited from oral morphine addicts when the classical association between the bitter taste of morphine and its post-ingestive consequences changes the behavior that morphine evokes (Zellner *et al.*, 1984) from aversive to ingestive. The palatability evaluation elicited by taste stimulation is clearly not a constant, although the palatability evaluation of some tastes is more stable, under a broad variety of conditions, than that of others. [For example, taste aversion learning proceeds more rapidly and is less susceptible to latent inhibition when the conditioning stimulus (CS) is a familiar salt or acid taste than when it is a familiar sugar taste (Berridge *et al.*, 1981).] Palatability is a decision that arises within the central nervous system, not an inherent property of a taste itself.

We are not the first to suggest that palatability be defined in this integra-

tive way. Aristotle (in Ross, 1906) remarked that food smells are pleasant when one is hungry and unpleasant when sated. Young (1967) noted that while palatability is commonly attributed to the foodstuff itself, it actually depends on the integration of taste, inorganic conditions (internal state), and dietary history (taste associations). Davis and Levine (1977) defined palatability as the interaction of taste and internal state that generates an excitatory neural signal.

D. PALATABILITY IS DISTINCT FROM HEDONIC CONSCIOUSNESS

It is important to be clear that the palatability decision, as we have defined it, does not necessarily refer to the hedonic consciousness of taste that humans experience as they introspect. The palatability decision refers to the association of an entire constellation of behaviors. Human reports of conscious hedonic perceptions, or those perceptions themselves, are only one measure of this decision, and not necessarily the definitive one. It is entirely possible to conceive of a palatability decision, as defined here, existing in the absence of a conscious hedonic perception. Available evidence strongly suggests that this decision can be made in rats, cats, and humans (Miller and Sherrington, 1916; Grill and Norgren, 1978b; Steiner, 1973) that lack a neocortex or even an entire forebrain. The concept of palatability as an intervening variable must therefore be kept conceptually distinct from that of hedonic consciousness. For the sake of clarity, we will refer to the former as the palatability decision in order to emphasize its nature as a functional entity that integrates information about taste, physiological state, and prior associations, and to distinguish it from hedonic palatability as derived from introspection.

II. How Has the Palatability Decision Been Measured?

Measures of the palatability decision have included one-bottle or two-bottle tests, preference choice, and operant tests that provide either long-term (24–48 hr) or short-term (less than 1 hr) access to tastants (Richter and Campbell, 1940; Young, 1948, 1959; Guttman, 1954; Morrison, 1969; Young and Shuford, 1955; Cagan and Maller, 1974). Reflexological measures including integrated lick rate, electromyographic activity of masticatory muscles during licking, and taste reactivity (taste-elicited fixed action patterns) have also been used (Davis, 1973; Yamamoto *et al.*, 1982; Grill and Norgren, 1978a; Berridge *et al.*, 1981). While all of these measures use behavior that depends on the CNS evaluation of taste stimuli,

there is considerable discussion about which factors are integrated in this decision for each of these tests and which tests are the best measures of palatability.

Despite many provisos, the general consensus of the field has been to consider palatability as an attribute of the tastant that is invariant. For example, a distinction has been made between what is measured by short- and long-term access tests. Short-term tests, like recordings from peripheral gustatory nerves, show that responses are monotonically related to sucrose concentration (Pfaffmann, 1961). Conversely, with long-term tests there is a reversal in preference as indicated by a decreased intake at higher sucrose concentrations. Such data do not conform to either electrophysiological afferent recordings or short-term test results. For this reason, long-term tests have been regarded as confusing the immediate effects of the taste itself with the effects of a variety of sources of post-ingestive feedback.

While perhaps tempting, this identification of the palatability decision exclusively with the sensory characteristics of the taste itself is incorrect. First, it is not possible to dissociate taste from internal-state factors no matter how circumscribed the test may be. The logic that the best measures of palatability are those that do not allow changes in post-ingestive feedback from the meal to modify the behavior being measured has fostered the idea that surgically eliminating post-ingestive feedback (the sham-feeding preparation, e.g., Mook, 1963) yields a "true" measure of palatability. While the sham-feeding preparation clearly demonstrates that sucrose taste is a potent elicitor of ingestion as well as of gustatory nerve discharge, it must be remembered that this preparation attempts to hold internal-state factors constant rather than eliminating them from consideration entirely. Second, demonstrations of the cephalic reflexes of digestion (Powley, 1977; Berthoud and Jeanrenaud, 1982; Grill *et al.*, 1984) make it unlikely that the sham-feeding preparation can hold the internal state constant. Third, in short-term tests other than sham feeding, where access to tastes is limited to 15 min or less, not only must the internal state of the preparation at the beginning of the test be affected by cephalic reflexes, but there is evidence that glucose absorption occurs within 3 min of intake (Steffens, 1969). Post-ingestive feedback from mechanical or osmotic signals from the upper GI tract may be equally rapid.

Part of the resistance to modifying the assumption that taste input is the only factor operating in palatability has been the companion assumption that when post-ingestive feedback does modify the response to taste it does so in a way that does not transform the gustatory signal. For example, Pfaffmann (1982) notes that the reversal of sucrose preference in long-term tests is "only apparent and does not reflect a reduced prefer-

ence or aversion to the sugar per se." The position that such a reversal reflects other nongustatory, sensory, or physiological factors is shared by others. A variety of experiments, however, have demonstrated that this assumption may be incorrect. Electrophysiological studies suggest that taste signal itself can be transformed early in its central processing. Brush and Halpern (1970) found that inflating the stomach of the frog increased the glossopharyngeal response to NaCl on the tongue and decreased the response to oral quinine. Section of the vagus nerve eliminated the influence of gastric distension. Contreras *et al.* (1984) report that sodium deprivation sufficient to induce salt appetite without changing serum Na levels was correlated with a significant decrease in the suprathreshold response to NaCl in the chorda tympani nerve of the rat. In addition, despite meager evidence for primary afferent convergence of taste and visceral afferent neurons in the first central gustatory relay nucleus, electrophysiological experiments have described alterations in taste cell responses following gastric distension (Glenn and Erickson, 1976; see Norgren, 1983). In addition, many neurons in the rostral medulla that respond to electrical stimulation of peripheral taste nerves such as the chorda tympani also respond to stimulation of visceral afferent nerves such as the cervical vagus (Bereiter *et al.*, 1981). Behavioral data also support an integration among taste input, internal state, and associative factors. For example, measuring human psychophysical judgments of taste pleasantness, Cabanac (1971) has shown that the palatability of a constant glucose stimulus varied as a function of internal state. The same glucose stimulus that was judged pleasant when the subject was food deprived was judged unpleasant when the same subject had just consumed a concentrated glucose solution. Analogously, the palatability evaluation of sucrose taste (measured in a variety of ways) was systematically changed following treatments such as insulin and 2-deoxy-D-glucose injection, which change internal state by altering glucose homeostasis (Thompson and Campbell, 1977; Mayer-Gross and Walker, 1946).

Taste stimuli have a special capacity to be associated by classical conditioning with a variety of states (Garcia *et al.*, 1974; Rozin and Kalat, 1971), and these associations are also factors in determining palatability. For example, Booth (1977) has shown that rats and humans develop associations between tastes and their caloric consequences, and that these associations can produce conditioned preferences that are state dependent for the changes in internal state that occur within a single meal. When rats or humans are offered a choice between a taste that has been associated with the drinking of a rapidly absorbed, concentrated starch and another that has been paired with a dilute starch, the former is ingested at the onset of a meal but rejected at the end of the same meal,

while the latter taste is responded to in a reciprocal fashion (Booth *et al.*, 1982). This example clearly illustrates that internal state and classical associations are integrated with taste quality and intensity to yield a decision to respond to taste that we are calling the palatability decision.

Our operating assumption in this article is that the palatability decision represents an integration of taste, internal state, and associative factors and that when each of these variables contributes to modifying the response to a taste stimulus it can do so via a *common* mechanism. In assessing the relative weighting of these factors in other models of ingestive control, experimenters (e.g., Davis and Levine, 1977) have tended to characterize the nature of the internal-state factor as inhibitory and that of the taste factor, especially sugars, as excitatory. We take the perspective that the weighting of each of the integrated variables is not fixed (e.g., osmotic feedback is not solely inhibitory nor is the taste of sucrose solely excitatory), but rather their weighing can vary rapidly between excitatory and inhibitory valences. For example, the same internal-state variable (osmotic feedback) reduces the ingestive reaction to hyperosmotic tastes but increases the ingestive reaction to hypoosmotic taste stimuli (Stellar *et al.*, 1954). Put in other terms, the neural circuit whereby tastes that are hyperosmotic elicit ingestion may be inhibited by the same internal-state signals that excite the circuit whereby tastes that are hypoosmotic lead to ingestion. An additional example is that novel sucrose elicits a pattern of ingestive FAPs in rats. Yet, after a single pairing with the internal-state changes evoked by a LiCl injection, this same sucrose stimulus now elicits a quinine-like aversive pattern of behavior (Grill, 1975). The example of state-conditioned preferences and aversions (Booth, 1977) given in the last paragraph is also relevant here.

A. TASTE-ELICITED FIXED ACTION PATTERNS AS A MEASURE OF THE PALATABILITY DECISION

We take the position that all tests that measure palatability measure an integration of gustatory afferent signals, internal state, and classical associations. The fact all of these measures reflect the palatability decision, however, does not imply that palatability is the only thing they measure. Other central states might also be expected to affect these measures, and this poses a problem for the study of central-state changes. For example, fear might stop a rat from drinking sucrose, but does fear alter sucrose palatability? Pairing a taste with either footshock or visceral illness results in a subsequent reduction in intake of that taste. Using intake as the measure of palatability would lead to the conclusion that in both types of pairings, the palatability of the taste was decreased. However, this con-

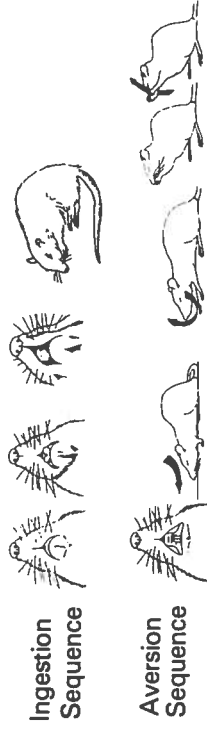


Fig. 1. Taste-elicited fixed action patterns. Ingestive responses are elicited by oral infusions of glucose, sucrose, and isotonic sodium chloride, and include rhythmic mouth movements, tongue protrusions, lateral tongue protrusions, and paw licking. Aversive responses are elicited by infusions of quinine, caffeine, and sucrose octaacetate solutions, and include gapes, chin rubs, head shakes, paw wipes, forelimb flailing, and locomotion (not shown). (Adapted by permission from Berridge *et al.*, 1981.)

clusion would be incorrect. There may be a degree of overlap in the constellation of effects produced by two functional variables such as fear and palatability; however, this overlap is not complete. When taste reactivity was used as a measure of palatability to compare the effects of taste association with footshock and visceral illness, the results were different. The taste reactivity measure of palatability analyzes the occurrence of nine separate FAPs that can generally be clustered into two patterns of responses: an ingestive sequence and a rejection sequence (see Fig. 1). Following taste-visceral illness pairing there is a shift in the pattern of taste reactivity responses elicited by the paired taste from an ingestion sequence to a rejection or aversion sequence. Taste-footshock pairing produces no such shift in taste reactivity responses (Pelchat *et al.*, 1983). These data suggest that the taste reactivity technique can be used to distinguish between the effects of separate psychological variables (e.g., palatability and fear) that are not distinguished by more traditional measures of palatability.

The taste reactivity measure has another advantage over traditional measures of the palatability decision that require spontaneous intake or appetitive behavior. A variety of neurological lesions and ablations produce aphagic preparations—preparations that do not seek food. A preparation that does not seek food may nevertheless be able to evaluate a taste that is placed directly into its mouth; appetitive and evaluative capacities should not be confused. One goal of our functional analysis of the palatability decision is to empower a neurological investigation of the sites of integration of taste, internal state, and associative factors. This has involved analyzing the palatability decisions made by a variety of aphagic neurological preparations, including chronic supracollicular decerebrate,

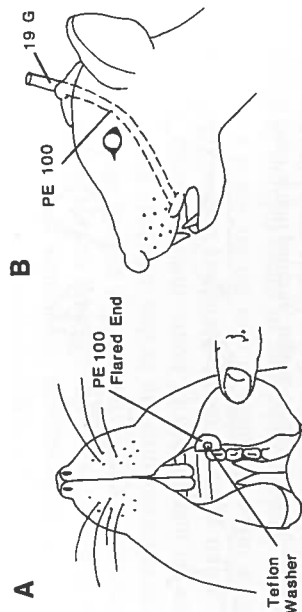


FIG. 2. Diagram of the intraoral catheter *in situ*. The intraoral end is placed just rostral to the first maxillary molar. The tubing is led out subcutaneously to the skull and secured to a short piece of 19-gauge (19 G) stainless-steel tubing with dental acrylic. (A) Ventral view; (B) lateral view.

thalamic, and decorticate rats. The taste reactivity test enables the palatability decisions of all such aphagic preparations to be examined.

The nonappetitive [in terms of Craig's (1918) original distinction between appetitive and consummatory phases of motivated acts] taste reactivity test makes use of chronic intraoral catheters to deliver calibrated amounts of a taste stimulus directly into the oral cavity, and subsequently to rinse it away without active initiation by or disturbance of the animal (see Figs. 2 and 3). Direct intraoral taste stimulation elicits responses that are videotaped and subsequently analyzed frame by frame. Analysis reveals nine different stereotyped response components that are generally grouped into two different response patterns corresponding to an ingestive and a rejection sequence (as seen in Fig. 1). The ingestive response sequence, seen in Fig. 1, is composed of four response components, while five other components comprise the rejection or aversion sequence. This method of response analysis can also be used in the context of an appetitive test in the neurologically intact animal, without the intraoral fistulae, as in Pelchat *et al.* (1983). Taste-elicited consummatory responses or FAPs share all the characteristics of classic consummatory responses. They are highly stereotyped and represent the final behavioral acts not only in the process of obtaining food, but also in the process of obtaining water and electrolytes.

Use of taste-elicited FAPs as a measure of the palatability decision requires that a number of criteria be met. If these taste-elicited responses do indeed reflect the palatability decision they should (1) represent a general decision to respond to tastes that agrees with other measures of this decision, (2) integrate information from a variety of sources (e.g., taste, internal-state cues, learned associations) to produce demonstrated

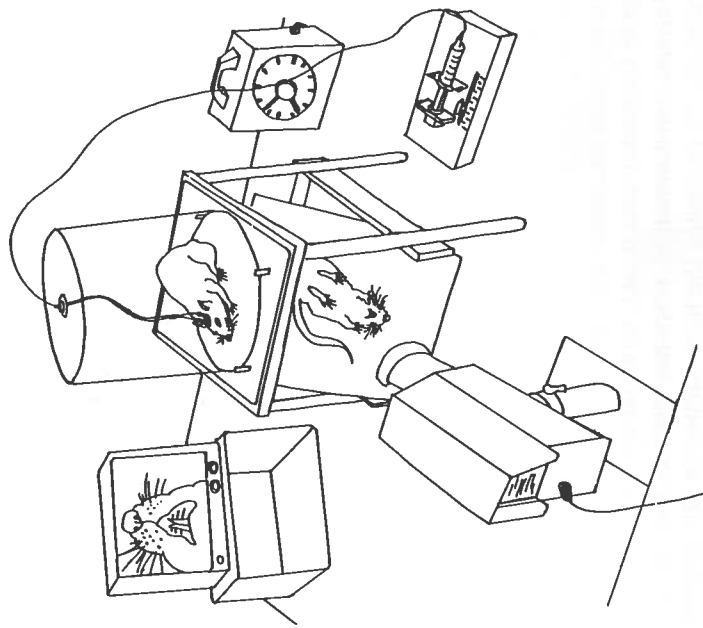


FIG. 3. Apparatus for videotaping taste reactivity responses to taste stimuli injected into the mouth via chronic intraoral catheters. Videotaping is done via a mirror located beneath the Plexiglas floor in the upper right-hand corner of the figure.

shifts or changes in the response to a constant taste, and (3) be organized as functionally related response clusters (e.g., ingestion and rejection sequences) that vary as a group with changes in palatability. This last point is stressed in order to be sure that individual FAPs are not controlled by individual systems and taste properties but rather vary together as functional sequences or clusters according to a more global palatability decision.

All of these criteria are met by existing data. Grill and Norgren (1978a) showed that these responses are elicited by taste stimuli in ways that correspond to other short-term measures of the palatability decision. Ingestive response components are elicited by oral application of sucrose in a graded, concentration-dependent manner. Low concentrations elicit only mouth movements and tongue protrusions. At higher concentrations, the number of tongue protrusions and lateral tongue movements increases as a function of increasing sucrose concentration (Schwartz and

Grill, 1984). Similarly in other short-term tests, intake volume and the number of tongue contacts with a spout have been shown to be dependent on sucrose concentration (Cagan and Maller, 1974; Davis, 1973). In palatability measures that use the amount ingested as a dependent variable, quinine HCl consumption falls off rapidly as concentration exceeds 0.01 mM, the absolute behavioral threshold for quinine (Koh and Teitelbaum, 1961). The threshold for the gape response (0.03 mM) is only one-half log step above this, reflecting the sensitivity of the taste reactivity test. Aversive responses to quinine are also elicited in a concentration-dependent manner. Gapes appear at threshold concentrations, chin rubs are added at slightly higher concentrations, and the three other aversive components are added to the gape and chin-rub sequence at higher concentrations of quinine HCl. Like the magnitude of the peripheral electrophysiological responses, the number of gape and chin-rub components elicited by quinine HCl increases with stimulus concentration (Schwartz and Grill, 1984). Parallel changes in the appearance of aversive components are seen in response to other "bitter" tastes, such as caffeine, sucrose octaacetate, and quinine sulfate (Schwartz and Grill, 1984). Short-term intake of bitter tastes decreases analogously. There is a strong correlation between gape frequency and intake termination for these tastes.

Characteristic consummatory response profiles can be generated for any taste stimulus by recording the total number of rats displaying each of the taste-elicited FAPs during a standardized 1-min intraoral taste infusion (see Fig. 4). These profiles are highly sensitive to the palatability decision. Glucose, isotonic NaCl, ammonium chloride, and quinine HCl can be ranked in that order as a linear palatability hierarchy on the basis of intake (Weiner and Stellar, 1951; Nachman, 1962; Pfaffmann, 1961). Figure 4 shows that taste reactivity profiles exactly parallel this ranking (Berridge *et al.*, 1981). Comparisons between different concentrations of the same taste are best made by quantifying the number of individual taste reactivity response compounds generated rather than by profiles (H. Grill and Spector, unpublished observations).

B. TASTE REACTIVITY RESPONSES REFLECT CHANGES IN INTERNAL STATE AND LEARNED ASSOCIATIONS

The second criterion, that the taste-elicited FAPs reflect an integration of taste with internal state and learned associations, is also met. Sucrose-elicited FAPs change as a function of changes in internal-state cues that signal both nutrient depletion and nutrient repletion (Grill and Norgren, 1978d; Grill, 1980; Flynn and Grill, 1983; Grill *et al.*, 1983). If a taste stimulus is continuously infused into a rat's mouth, the volume that is

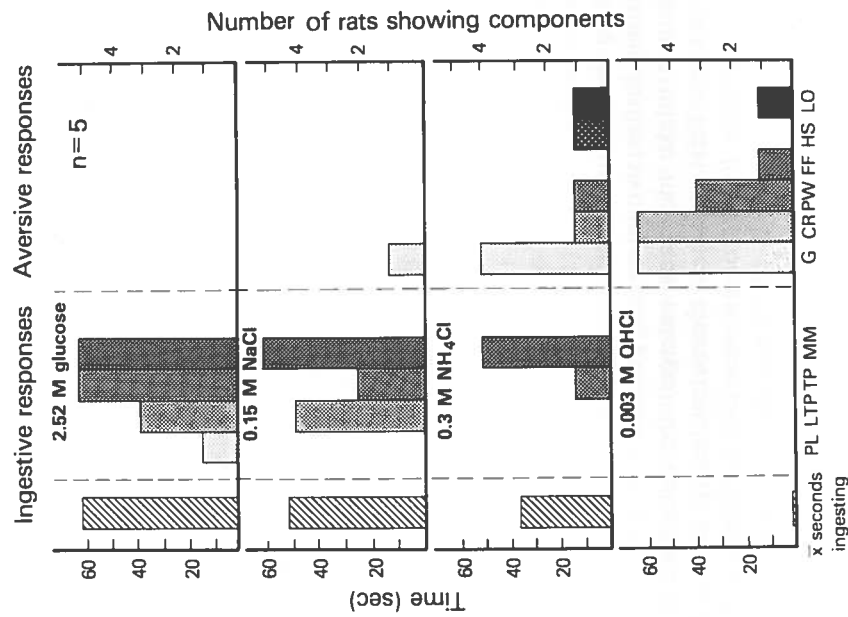


FIG. 4. Taste reactivity profiles. Taste-elicited responses to oral infusions of four different taste stimuli: glucose (2.52 M), NaCl (0.15 M), NH_4Cl (0.3 M), and quinine HCl (0.003 M). Response categories: PL, paw licking; LTP, lateral tongue protrusions; TP, tongue protrusions; MM, mouth movements; G, gapes; CR, chin rubs; PW, paw wipes; FF, forelimb flailing; HS, head shakes; LO, locomotion. Ingestion time is total seconds of stimulus consumption.

consumed can be measured along with the taste-elicited FAPs that it produces. Two different patterns of taste reactivity responses can accompany intake: a sequence of rhythmic movements or a pattern of tongue protrusions and lateral tongue movements interdigitated with mouth movements. Likewise, a taste stimulus can either be rejected passively by fluid dripping from the mouth, or actively by head shakes, chin rubs, paw wiping, or paw shaking. Oral intake is determined by measuring the volume of tastant consumed before it is rejected from the oral cavity. The

infusion is stopped at the time of rejection. After a 30-sec interval without infusion, the infusion pump is restarted. If the taste stimulus is rejected again within 30 sec of restarting the infusion, the intake test is terminated and the volume consumed computed. If, however, the rat continues to consume the taste for more than 30 additional seconds, the infusion proceeds until two successive fluid rejection responses occur. Table I demonstrates that when sucrose is continuously infused into the mouths of 24-hr food-deprived intact rats, the volume of sucrose ingested is much greater than when the same rat is tested while food replete (1 hr after a tube-fed meal). The same result is obtained if intraperitoneal insulin injection (regular insulin, 5 U/kg) is substituted for food deprivation in a different group of food-replete rats (see Table I). Water was substituted for the intraorally delivered 0.03 M sucrose in both paradigms, to control for whether alterations in internal state would give rise to a general facilitation of fluid intake. Rats did not increase their intake of orally delivered water when food deprived or insulin treated. It appears that the integration of internal energy-deficit cues and specific types of taste signals is necessary to enhance intraoral intake.

In analyzing the minute-by-minute changes in taste reactivity profiles during sucrose infusion, two interesting parallels between taste reactivity components and intake measures emerged for both food-deplete and -replete conditions. First and most characteristically, the number of sucrose-elicited ingestive FAPs (tongue protrusions, lateral tongue movements, and paw licks) declined with increasing infusion time (Grill, 1984). Second, the number of aversive components like head shakes, paw shakes, and gapes increased in frequency as meal termination approached. The difference between food-deplete and -replete conditions is that food deprivation greatly increases the number of ingestive FAPs emitted at the beginning of the infusion test. In both replete and deplete

TABLE I
EFFECT OF FOOD DEPRIVATION AND INSULIN INJECTION
ON SUCROSE TASTE REACTIVITY

Treatment	Sucrose intake (ml)	Water intake (ml)
Food deprivation		
1-hr deprived	2.74 ± 0.78	2.74 ± 0.82
24-hr deprived	4.45 ± 0.84#	1.97 ± 0.67
Insulin treatment		
Saline control	8.1 ± 1.1	4.7 ± 1.0
Insulin (5 U/kg)	11.3 ± 1.2	5.8 ± 1.9

conditions the number of ingestive responses declines as the test proceeds and is at minimum at the end of the test.

The change in NaCl intake that accompanies alterations in internal sodium state, called salt appetite, is a robust phenomenon in the field and laboratory (Richter, 1956; Denton, 1967). A variety of different explanations for this potent change in NaCl consumption are equally plausible, but traditional palatability measures cannot distinguish among them. Several experiments from our laboratory have addressed the issue of whether the heightened NaCl intake following sodium depletion is accompanied by a shift in the palatability of that substance, that is, in the reaction to its taste. To ensure that any observed shift was truly state dependent, rather than a permanent associative or habituation effect, rats were repeatedly brought in and out of sodium balance. We were thus able to examine NaCl taste reactivity a number of times in each state, and we discovered that FAPs elicited by concentrated NaCl did shift from aversive to ingestive during sodium depletion (Berridge *et al.*, 1984).

At 1.0-ml volume of 0.5 M NaCl was infused into a rat's mouth at a constant rate over 1 min. Taste reactivity responses were videotaped for subsequent frame-by-frame analysis. Each week rats were tested both sodium replete and 24 hr after artificial sodium depletion (simultaneous sc injections of 5.0 mg deoxycorticosterone and 7.5 mg furosemide, and 3 hr later an additional 7.5-mg furosemide injection). Each rat was tested five times in each condition and served as its own control. The results of biweekly two-bottle intake tests indicated that a sodium appetite, as traditionally measured, was induced. Sodium depletion dramatically affected taste-elicited FAPs as seen in the weekly data in Fig. 5. A significant increase in ingestive FAPs and reduction in aversive FAPs elicited by a constant NaCl taste is shown in the pooled data. The palatability of a constant 0.5 M NaCl taste stimulus was therefore controlled by internal-sodium-state cues.

Classically conditioned associations represent another factor that is integrated with taste and internal state to determine palatability. Of the associative paradigms used to affect the intake of taste stimuli, conditioned taste aversion (CTA) is the most widely used. Despite its name, however, aversive behavior *per se* is *not* normally measured in this paradigm. What is typically measured is the reduction in taste-elicited intake. The reduction of intake is the measure of taste avoidance. As noted earlier, however, intake tests cannot distinguish between reduced taste intake based on changes in palatability and reduced intake based on anticipated negative consequences (fear or danger). The taste reactivity analysis *does* measure aversive behavior. Using this test we have demonstrated that when a taste has been associated with a certain class of aversive

	INGESTIVE					AVERSIVE					
	PL	LTP	TP	MW	PD	G	CR	FW	FF	HS	LO
I	0.6 3.6	2.6 3.3	1.6 7.6	5.3 3.0	2.0 0	3.3 0	0 0	0 0	0.3 0	1.6 0	0 0
II	0.3 4.3	0.6 3.0	1.0 7.6	4.3 5.3	3.0 0	0 0	0 0	1.0 0	1.6 0	3.0 0	0.6 0
III	0 2.3	0.3 1.0	2.3 13.0	4.6 1.3	4.0 0	0 0	0 0	0 0	0 0	2.0 0	0.3 0
IV	0 3.3	0 0.6	0 8.3	2.3 4.0	4.0 0	0.6 0	0 0	0 0	0 0	3.0 0	0 0
V	0 4.0	0 1.0	0 9.3	4.0 3.6	4.3 0	0 0	0 0	0.6 0	0.6 0	3.3 0	0 0

FIG. 5. Mean number of taste-elicited FAPs per rat for three rats to 0.5 M NaCl when either sodium replete (upper rows) or deplete (lower rows) for weeks I-V. Ingestive responses are paw licking (PL), lateral tongue protrusions (LTP), tongue protrusions (TP), and mouth movements (MM). Failure to show any consummatory response, either ingestive or aversive, results in the passive drip of fluid from the mouth (PD). Aversive responses are gapes (G), chin rubs (CR), face washing (FW), forelimb flailing (FF), head shakes (HS), and locomotion (LO).

unconditioned stimuli (UCSs), its palatability is changed by even a single taste-UCS pairing (Grill, 1975; Berridge *et al.*, 1981; Pelchat *et al.*, 1983). This class of aversive UCSs is exemplified by the action of LiCl. Intoxication with lithium salts produces nausea and vomiting in humans (Baldezarini, 1980), and administration of antiemetic drugs blocks the expression of taste-LiCl paired taste avoidance in the rat (Coil *et al.*, 1978). Other classes of aversive UCSs (including footshock and lactose intolerance) that do not act on the upper gastrointestinal tract reduced taste intake but did not produce a shift in taste reactivity responses (Pelchat *et al.*, 1983).

Figure 6 demonstrates the changes in taste reactivity profiles produced by two taste-LiCl associations. Novel sugars or other tastes that elicit an ingestive response sequence will elicit a quinine-like aversive-response sequence following associative pairings with LiCl injections (Grill, 1975; Berridge *et al.*, 1981; Pelchat *et al.*, 1983). The first association of a taste

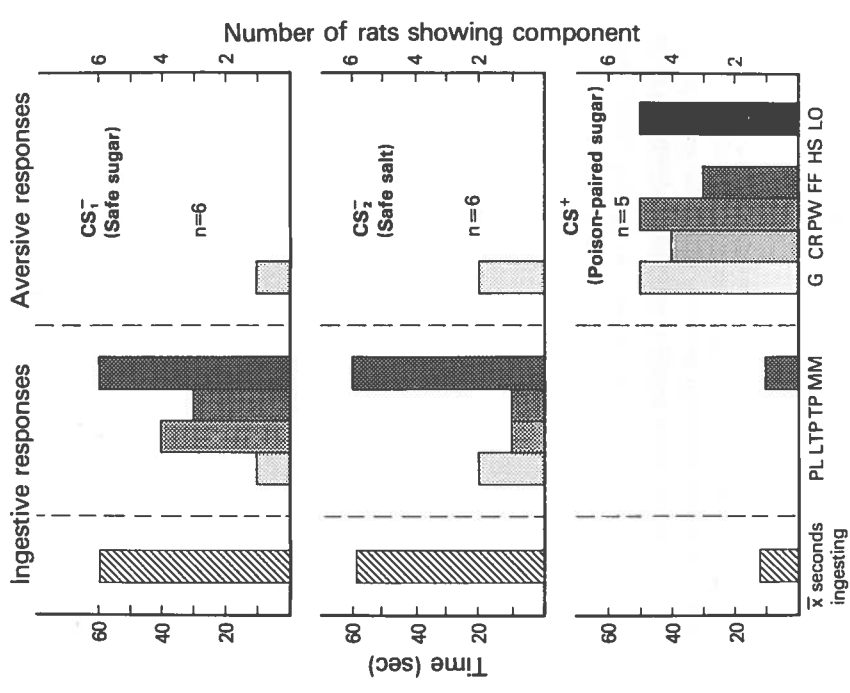


FIG. 6. Taste reactivity responses to discriminative taste CS. Taste-elicited FAPs to oral infusions of the CS₁ (15% maltose or fructose), CS₂ (0.15 M NaCl), and CS₊ (15% maltose or fructose). Response categories are as in Fig. 5. Ingesting time is total seconds of stimulus consumption.

with LiCl produces a shift in palatability that is most often typified by a mixed response, that is, a response containing components of both ingestive and rejection sequences. Following additional pairings, the palatability of the paired taste shifts even further to contain a greater percentage of aversive FAPs and a smaller percentage of ingestive FAPs until the response is exclusively aversive.

The CTA paradigm reveals that the association of taste with the internal consequences of certain aversive UCSs produces a change from ingestive to aversive in the palatability of the paired taste. Attempts to reverse taste palatability in the opposite direction, from aversive to ingestive, as a

periods of water access and during morphine withdrawal. In contrast, morphine-raised rats displayed ingestive taste reactivity responses (tongue protrusions, lateral tongue movements, and mouth movements) in response to morphine taste, and they swallowed the infused solution following either morphine home cage access or withdrawal. The percentage of ingestive components elicited by oral morphine was significantly greater for morphine-raised than water-raised rats. Sucrose elicited the same ingestive sequences in morphine- and water-raised groups. Quinine taste elicited aversive responses in both groups, and there were no significant differences in the number of aversive components produced by both groups.

In morphine-raised rats, quinine had a tendency to elicit mixed responses that included some ingestive components. These quinine data could therefore be interpreted as indicating that similarities in the tastes of morphine and quinine produced a generalization to the reinforcing qualities of morphine. Alternatively, the morphine-raised rats may simply have habituated to morphine's taste during their prolonged pretest exposure, and their response to quinine might then reflect generalization of the taste habituation. To control for this latter possibility, another group of rats was raised for a number of months on a quinine solution as their only source of water. Quinine is judged bitter by humans and has toxic or aversive post-ingestional consequences (Scalafani *et al.*, 1979). If habituation to a bitter taste occurs irrespective of the valence of the associated post-ingestive effects of the bitter taste, then quinine exposure should come to alter quinine taste reactivity similar to the shift in morphine taste reactivity. However, when subsequently examined, these rats did not display any ingestive components to quinine but passively rejected the taste even after a period of quinine withdrawal. It is concluded that the association of morphine taste with its internal consequences produces a change in its palatability.

As mentioned earlier, if the taste reactivity measure does indeed reflect the palatability decision it must satisfy several criteria. The examples just provided on the effects of internal state (e.g., food deprivation, insulin injection, sodium depletion) and classical associations (e.g., CTA, morphine addiction) on the palatability decision satisfy the second criterion, that the taste-elicited FAPs integrate information from a variety of sources to produce changes in the response to a constant taste stimulus. The third criterion, that clusters of taste-elicited FAPs vary together with changes in palatability, is also fulfilled. The use of the terms "ingestive sequence" and "aversive sequence" arose because in the sated intact rat some taste stimuli (e.g., sucrose) tended to elicit one set of FAPs, while another taste stimulus (e.g., quinine) evoked another response sequence

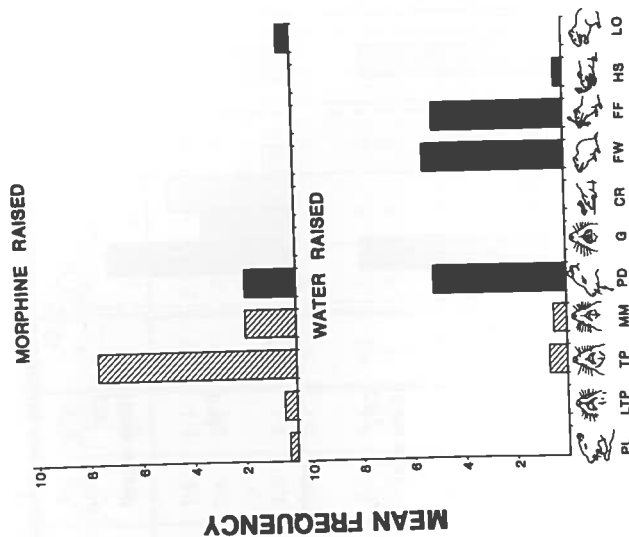


FIG. 7. Mean frequency of ingestive (hatched bars) and aversive (solid bars) FAPs elicited by intraoral infusions of 0.3, 0.6, and 1.5 mg/ml morphine sulfate for morphine-raised (group M) and water-raised (group W) rats maintained on either ad lib access to morphine sulfate or following 72-hr access to water. Response categories are as in Fig. 5.

function of association with the internal consequences of other UCSs have met with much less success (see also Booth *et al.*, 1982). We have made progress on this issue by using a morphine addiction paradigm. Morphine tastes bitter to humans. Rats avoid the taste of morphine when first exposed to it and consume water in preference to it in two-bottle tests. When rats are forced to drink morphine, they reverse their original preference and drink more morphine solution than water (Stolerman and Kumar, 1970; Ternes, 1975c). The question we addressed was whether the association of morphine taste with its potent opiate internal-state changes could change its palatability evaluation.

Taste reactivity to morphine and other tastes was examined in rats that had been raised for several months with morphine solution as their only source of water and in control rats raised with water (Zellner *et al.*, 1984). Taste reactivity was examined in two conditions: during home cage ad lib access to morphine or water, and during periods of its withdrawal. These data are shown in Fig. 7. Oral presentations of morphine elicited aversive FAPs, and the solution was spit out by water-raised rats both during

composed of different FAPs. During the sucrose response a pattern of mouth movements, tongue protrusions, and lateral tongue movements was elicited and the stimulus was ingested. During the quinine response a pattern of gapes, chin rubs, head shaking, paw wipes, face washes, forelimb shaking, and paw rubs was elicited, and the stimulus was rejected. Each component FAP of the quinine response appears to facilitate removal of the stimulus from contact with the animal and can therefore be defined as aversive responses based on the criteria of Craig (1918). While these data demonstrate that a given stimulus can elicit a characteristic group of FAPs, they do not indicate whether each FAP group is activated by a single mechanism. It is conceivable, for instance, that each FAP is elicited separately from all other FAPs and that each response is under unique control rather than under the control of a more global palatability decision.

Data that address this question come from an analysis of instances in which both ingestive and aversive FAPs are elicited (e.g., by mixtures of sucrose and quinine). Berridge (1983) analyzed the sequence of FAPs generated by tastes producing mixed response patterns. The FAP sequence or FAP transition analysis of these tastes is shown in Table II and reveals the frequency with which each FAP is followed by every other

TABLE II
TASTE REACTIVITY RESPONSES OF RATS TO A TASTE MIXTURE^a

	Ingestive					Aversive				
	PL	LTP	TP	MM	G	CR	FW	FF	HS	
Ingestive										
PL	1	—	—	—	—	—	—	1	—	
LTP	1	6	4	6	—	—	—	—	—	
TP	—	3	5	7	1	—	—	—	—	
MM	—	6	8	45	4	3	—	—	2	
Aversive										
G	—	1	—	6	8	3	1	—	3	
CR	—	—	—	—	2	3	3	—	4	
FW	—	—	—	—	—	1	2	5	2	
FF	—	—	—	—	1	—	7	3	—	
HS	—	—	—	1	2	5	—	3	3	

^a Mixture consisted of 0.03 M NH₄Cl, 1 M glucose, and 0.0003 M quinine HCl. Abbreviations: PL, paw licks; LTP, lateral tongue protrusions; TP, tongue protrusions; MM, mouth movements; G, gapes; CR, chin rubs; FW, face wash; FF, forelimb flail; HS, head shake. Vertical axis denotes first behavior; horizontal axis shows every instance in which a particular behavior followed. Note that transitions typically occur *within* ingestive or aversive subgroups rather than between groups, although some exceptions do exist.

FAP. This analysis reveals strong temporal bonds within ingestive and aversive response groups but very weak associations between these two groups. This temporal clustering suggests that an ingestive decision activates a number of ingestive FAPs together as a unit and that aversive groups likewise function as units. Schwartz (1983) extended the taste reactivity analysis of Grill and Norgren (1978a) by increasing the volume and duration of the oral infusion of taste from 0.05 ml in a single pulse to 1.0 ml infused over 1 min. An analysis involving FAP sequencing was performed. Ranks for every possible pair of taste-elicited FAPs were correlated using a Spearman rank correlation coefficient, and a coefficient of association was obtained. This coefficient represents the likelihood that any two FAPs were followed by the same third component. For example, a coefficient of 0.9 between mouth movements and tongue protrusions would mean that it is highly probable that the same third component, lateral tongue movements, will follow both mouth movements and tongue protrusions. A negative coefficient between gapes and tongue protrusions would mean that components that follow gapes are usually not those that follow tongue protrusions. As can be seen in Table III for sucrose, the same components that follow mouth movements usually follow tongue protrusions and lateral tongue movements. Conversely, the components that follow gapes are not usually followed by tongue protrusions and lateral tongue movements. Similarly, as shown in Table III for quinine, gapes, chin rubs, head shakes, and forelimb shakes (all part of the "aversive" sequence) are highly correlated with each other as a group and negatively correlated with mouth movements and tongue protrusions. These data, as well as those of Berridge, strongly suggest that each FAP is not under separate control but rather that functional clusters of FAPs do exist and can be referred to as ingestive and aversive sequences. It should be noted that in both sequence analyses, the mouth movement component is positively correlated both with gaping, an aversive component, and with tongue protrusions and lateral tongue movements, which are ingestive components. These and other data (e.g., Berridge and Grill, 1983) lead to the conclusion that mouth movements are not strongly weighted toward either ingestion or aversion, and may be best described as a sampling act whose function may be preliminary to evaluation.

III. Fine Structure of Palatability Decisions: A Two-Dimensional Model

An additional advantage of taste reactivity over traditional measures of palatability is that fewer assumptions are built into this measure. Traditional behavioral measures, such as intake, preference, or operants, all use responses that are themselves best described as varying along a single

TABLE III
TASTE REACTIVITY RESPONSES OF RATS^a

	LTP	TP	MM																
Ingestive ^b																			
LTP	—	0.65	0.70																
TP	—	—	0.82																
MM	—	—	—																
Aversive ^b																			
G	-0.90	-0.90	0.50																
	TP	MM	G																
Ingestive ^c																			
TP	—	0.50	—																
MM	—	—	—																
Aversive ^c																			
G	-0.70	0.50	—																
CR	-0.90	-0.80	0.80																
FF	-0.80	0.20	0.70	0.75						0.25									
HS	-0.90	-0.15	0.75	0.85						—									
LO	-0.75	-0.10	0.55	0.40	0.35	0.30				—									
R	-0.80	-0.70	0.65	0.50	0.43	0.40	0.90												

^a Abbreviations: LTP, lateral tongue protrusion; TP, tongue protrusions; MM, mouth movements; G, gapes; CR, chin rubs; FF, forelimb flail; HS, head shake; LO, locomotion; R, rearing. Correlation between the order of taste reactivity responses of rats receiving intraoral infusions of tastants. Each number corresponds to the direction of the relationship between the responses that follow any two taste reactivity responses; positive values mean that the relative frequency of occurrence of behaviors following any response on the horizontal axis is similar to that following any response on the vertical axis. For example, behaviors that follow lateral tongue protrusions have the same relative frequency distribution as behaviors that follow lateral tongue protrusions.

^b Responses to 0.3 M sucrose.

^c Responses to 0.0003 M quinine.

continuum, either from high to low or from positive to negative. Using these measures, the palatability evaluation of a given taste under a given testing condition is implicitly characterized as a single point along that continuum (e.g., Young, 1977). This characterization allows the response to a taste to be described as palatable, unpalatable, or neutral, but never as both positive and negative at the same time. The drawback of such measures and of the palatability description they provide is that they are likely to give the impression that palatability itself (apart from its measurement) is a single evaluation produced by the central nervous system. Even if the brain actually generated evaluations of palatability using a mechanism whose output could not be accurately described by a point falling along a single dimension, these measures would not detect it.

Taste-elicited FAPs, on the other hand, can vary along a number of dimensions—as many dimensions as there are FAPs. In fact, however, the FAPs do not appear to vary independently along these many (nine) dimensions. Instead, as just noted, they cluster into two distinct groups of ingestive and aversive responses, as noted earlier. By itself, this clustering appears consistent with the traditional single-dimension model of palatability. Each cluster could correspond to extreme palatability assessments and be plotted at either end of the continuum. A somewhat more difficult observation to reconcile with the one-dimensional model, however, is that the ingestive and aversive clusters occasionally intermesh, and that sometimes, especially during infusions of certain taste stimuli, there is a rapid alternation back and forth between ingestive and aversive FAPs (Berridge and Grill, 1983; Schwartz and Grill, 1984).

A. INTERPRETING ALTERNATING INGESTIVE AND AVERSIVE RESPONSES

Rapid alternation between one behavior and its functional opposite has often been interpreted as revealing conflict between underlying mechanisms (e.g., Tinbergen, 1952; Andrew, 1956; Sevenster, 1961; Hinde, 1970). Such alternation has been termed "ambivalent behavior" (Tinbergen, 1952). The rapid alternation we have observed between ingestive and aversive FAPs could imply an actual conflict between internal decisions to emit these two different kinds of acts. This would imply that the evaluation of palatability actually involves two separate analyses that correspond, respectively, to dimensions of ingestion and aversion (see Fig. 8). Low activation of both dimensions (point A) results in a relatively neutral response such as passive dripping of the infused solution or rhythmic

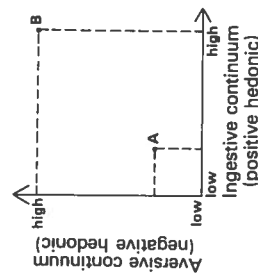


FIG. 8. A two-dimensional model of palatability. Point A represents a taste that weakly activates both ingestion and aversion and results in a passive response. Point B represents a taste that strongly activates both ingestion and aversion and results in active alternation of ingestive and aversive consummatory responses. The palatability of other tastes may be represented by points falling anywhere within the plane described by the axes of ingestion and aversion.

mic mouth movements occurring alone. High activation of both (point B) results in the simultaneous expression of the two extremes, namely the rapid alternation of mixed ingestive and aversive FAPs (Berridge and Grill, 1983).

The interpretation of FAP sequences is complicated, however, by the likely possibility that palatability decisions are not the only decisions that play a role in their production. Additional mediating systems must exist in order to translate palatability decisions into a particular sequence of behavioral responding. These mediating systems, or motor control systems, can be expected to involve rules of their own and are responsible for the actual patterning of movement (similar to the control systems invoked by MacFarland, 1974; Fentress, 1981). Before concluding that a particular pattern of behavior reflects the existence of two separate central nervous decisions about palatability, one must ask whether the same pattern could have been produced instead by a particular motor control system acting secondarily on a single palatability decision. In the case of alternation such a result is easy to imagine. A behavioral alternation between ingestive and aversive FAPs could result from a single palatability decision if the motor control system symmetrically activated responses on either side of the palatability decision itself: ingestive responses first and then aversive responses and so on.

B. THE USE OF ISOHEDONIC TASTES TO SUPPORT A TWO-DIMENSIONAL MODEL

To choose between the hypothesis of a motor control system whose output produced alternation operating with a single palatability decision and the hypothesis of a two-dimensional analysis of palatability, we need to know more than the mere fact that behavioral alternation exists. We need to know how the rat behaves under conditions in which the two hypotheses make different predictions. One such situation is when the negative palatability decision elicited by a taste shifts to become either more negative or more positive. According to the single-dimension view, a shift moves the evaluation along the palatability continuum, and one should therefore see a reciprocal change in the behavioral output: as one class of FAPs grows the other should decline reciprocally. According to the two-dimensional view, in contrast, it should be possible to produce an increase in the strength of aversion without changing the strength of ingestion, by moving the decision point in Fig. 8 upward along the aversive axis but keeping it stationary along the ingestive axis. Berridge and Grill (1983) found that the proportion of rats emitting aversive FAPs could be increased without changing the proportion of rats emitting ingestive FAPs

by adding quinine HCl together with sucrose to an original solution of ammonium chloride. Subsequent work in our laboratory has used a modified version of Young's isohedonic (equally preferred) taste pairs to show that the number of emitted ingestive and aversive FAPs can be independently manipulated (Berridge, 1983; Berridge and Grill, 1984). Young and Schulte (1963) determined the concentration of sucrose that must be added to a given quinine HCl solution to make the mixture equally preferred to a standard, unadulterated sucrose solution of lower concentration. The resulting taste mixture together with its sucrose standard constitute an isohedonic pair. When a number of different quinine-sucrose mixtures have been identified as isohedonic to a given sucrose standard, a curve may be plotted in a space where the axes denote sucrose and quinine concentrations, respectively. This curve is an isohedonic contour, and all points along it are assumed to be equally preferred (Young and Madsen, 1963).

Two isohedonic contours corresponding to 4 and 8% sucrose standards were chosen from Young and Schulte (1963) for FAP analysis. Both the FAPs that were elicited by these two standards alone and by their isohedonic mixtures that contained 0.01% quinine (7% sucrose plus 0.01% quinine for the lower concentration, and 18% sucrose plus 0.01% quinine for the higher) were examined. Since the primary purpose of the study was to replicate the independent increase in aversive FAPs found by Berridge and Grill (1983), the 8% sucrose standard was also compared to a mixture that should lie on the aversive side of its isohedonic contour, namely, a solution of 18% sucrose and 0.05% quinine. The number of FAPs emitted for each tastant was analyzed using the scoring procedure described in Berridge *et al.* (1984).

In the first isohedonic comparison, the sucrose-quinine mixture elicited both significantly more active aversive responses and significantly more ingestive responses than its 4% sucrose standard (this always refers to tongue protrusions, lateral tongue movements, and paw licking; see Fig. 9). Mouth movements are not included in this analysis, since the performance of other more strongly weighted ingestive components can break up and reduce mouth movement bins by simple intrusion (Berridge and Grill, 1983). This result by itself could suggest either that the taste and mixture evokes an increase in nonspecific activation in conjunction with a single palatability evaluation, or that the two separate evaluations of ingestion and aversion are both enhanced by the sucrose-quinine mixture. Interestingly, this effect was not seen in the comparison between the higher concentration isohedonic pair: FAPs remained the same. Since the level of FAPs makes a ceiling effect unlikely, this could reflect the presence of inhibitory relations between the central positive and negative

ingestive components. These data (Berridge and Grill, 1984) are clearly incompatible with the single-dimension model of palatability, as it has been considered so far.

A modification of the motor control system might be suggested here that would allow these data again to become compatible with a single-dimensional model. A change in aversive FAPs while ingestive components remain constant is compatible with a single palatability decision, only if one supposes that aversive FAPs are more sensitive to changes in the strength of the palatability decision signal than are ingestive FAPs, and that ingestive components have a flat sensitivity curve above a certain low threshold. Put another way, it takes a greater shift of the palatability evaluation to produce a change in ingestive FAPs than to change aversive ones. If this were true, however, aversive responses should always be at least as sensitive as ingestive FAPs to changes in palatability. Other evidence from our laboratory suggests that this is not the case. In the study of the effects of sodium depletion on taste reactivity described earlier, it was found that the FAPs elicited while the animal was sodium replete shift over repeated trials to become less ingestive. Pooled ingestive FAPs elicited from sodium-sated animals gradually declined with repeated trials, while the number of pooled aversive responses remained unchanged as rats were repeatedly brought into and out of sodium balance (Berridge *et al.*, 1984).

Together with the previously discussed experiments, these data suggest that both ingestive and aversive FAP groups can be made to change independently, and they provide further support for the hypothesis of two separate dimensions of palatability evaluation. A motor control system that is differentially sensitive to a single palatability decision will not account for these data unless one supposes that the ingestive production system is more sensitive to palatability shifts under some conditions, but that the aversive group is more sensitive under others.

C. ADDITIONAL SUPPORT FOR A TWO-DIMENSIONAL MODEL

The hypothesis that palatability processing involves two separate decisions finds additional support from other lines of evidence. Rat pups begin to show ingestive consummatory responses to tastes at an earlier age than they do aversive responses (Grill and Norgren, 1978c; Hall and Bryan, 1981). This could reflect a difference in the maturation rate of either gustatory receptors or the decision systems themselves. Studies by Weingarten and colleagues indicate that the well-known "finickiness" of ventromedial hypothalamic (VMH)-lesioned rats is asymmetrical (Weingarten, 1982; Weingarten *et al.*, 1983). These rats are hyperreactive

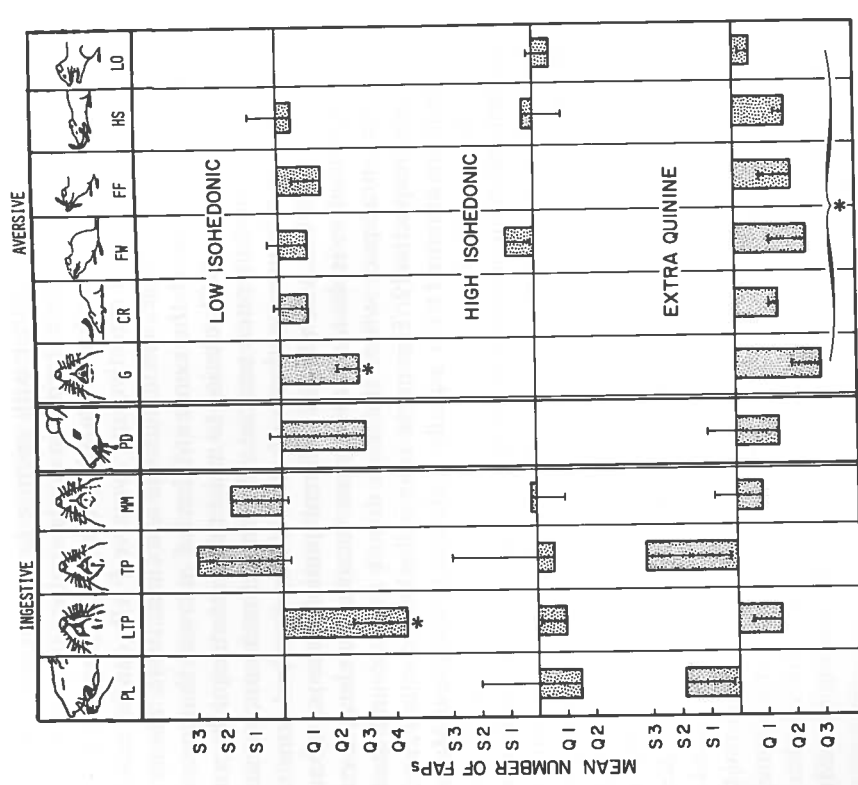


FIG. 9. Difference in the number of FAPs emitted. Bars represent the mean difference (\pm SEM) in the number of FAPs elicited by the constituents of each taste pair. Extension of the bar above the line represents a greater number of FAPs elicited by the sucrose standard; extension of the bar below the line represents a greater number of FAPs elicited by the sucrose-quinine mixture. Asterisks represent significant difference at $p < 0.05$. FAP symbols are as in Fig. 5.

decisions, or between their motor control systems, at high levels of activation (Fentress, 1973, 1983).

It is important to note that in the comparison between 8% sucrose and its "extra-quinine" mixture (18% sucrose plus 0.05% quinine), there was no significant change in ingestive components, but there was a significant increase in pooled aversive components (Berridge and Grill, 1984; see Fig. 9). This result demonstrates again that changes in the strength of aversive components need not be accompanied by reciprocal changes in

to sweet but not to bitter solutions in sham-feeding analyses compared to weight-paired controls, suggesting that only the positive palatability decision is affected by these lesions. In a similar dissociation of positive and negative responses to taste, Xenakis and Scalfani (1982) have reported that the consumption by nonfasted, obese VMH rats is overinhibited by quinine adulteration relative to normal-weight controls, but that the same animals respond as do controls to the positive reinforcing properties of sweet tastes, as measured by the effects of sweetness dilution. Finally, Ganchrow *et al.* (1981) have reported that rats that tend to self-administer high rates of intracranial electrical stimulation also tend to overconsume saccharin solutions compared to rats that self-stimulate at lower rates, but that they do not differ from low self-stimulators in quinine consumption. This again implies a separation between systems of positive and negative evaluation, and suggests further that the systems mediating the positive evaluations of different stimuli may overlap.

Thus, the hypothesis that palatability processing involves two separate decisions about a taste, corresponding to magnitudes of ingestion and aversion, appears to hold some promise of being an accurate description of how the brain analyzes gustatory information. It also serves as a reminder that new insights often can be gained by a more careful analysis of the stream of behavior, even behavior that at first sight appears quite simple.

IV. Neural Analysis of Palatability

The goal of a functional analysis of palatability is to empower a neurological investigation of the sites of integration of taste, internal state, and associative factors that would (1) suggest how the nervous system organizes its control of palatability and (2) improve the likelihood of reconstructing these events at the neurophysiological level. The approach that we (work begun by H. Grill and R. Norgren in 1974) applied to this neurological analysis was to examine whether a Jacksonian hierarchical organization would adequately explain the way the nervous system organizes the control of palatability. This hierarchical approach involved analyzing the palatability decisions made by classical neurological preparations including chronic decerebrate, thalamic, and decorticate rats. As an organizing principle for this section, the data generated by these neurological preparations will be compared to the palatability decisions made by intact rats.

As discussed earlier, palatability represents the CNS integration of taste, internal state, and associative factors. The relative weighting of

these factors, however, is not always the same. A functional hierarchy of palatability decisions is made by determining the number of factors that are integrated to produce the six different examples of palatability decisions discussed previously. Table IV organizes these examples into three levels of palatability decision complexity. Level 1, the simplest type of decision, integrates two factors: present taste and present internal state. In the intact rat, taste is *always* being integrated with state and compared with previous associations between taste and state; however, the weighting of the association and state factors can be reduced in certain instances. For example, in the intact rat, if the comparison of taste and state associations in memory, previously shown to be very rapid (Halpern and Tapper, 1971), yields no match to the present taste or state, then the associative factor would have no weight and would not be included in this particular decision. In contrast, when the comparison does yield a match, association then becomes a weighted factor in the palatability decision (see later). Second, it is possible that neural manipulations may reveal instances where palatability decisions are determined by taste alone. This type of decision will be called level 0 because it is not seen in the intact rat. The respective effects of food deprivation or sodium depletion (internal-state factors) on sucrose or NaCl taste reactivity and intake represent level 1 decisions. In these cases the association factor can be thought of as having no weight.

Association becomes an integrated factor when a previous association between taste and state has occurred. Both level 2 and 3 decisions involve

TABLE IV
THREE LEVELS OF PALATABILITY DECISION COMPLEXITY

Level	Integrated factors	Examples
1	Present taste, present internal state	Effects of food deprivation and insulin treatment on sucrose palatability; effects of sodium depletion on salt palatability
2	Present taste, previous association between what is now present taste and a previous internal state, present internal state	Effects of conditioned taste aversions; effects of morphine addition on morphine palatability
3	Present taste, previous association between what is now present taste and a previous internal state; correspondence between present state and state previously associated with present taste	Booth's conditioned satiety and hunger effects on palatability

the association factor but differ in their relative weighting of the present internal-state factor. Level 2 and 3 decisions include present taste, present state, and past taste-state associations as factors in the integration of the palatability decision. Level 3 decisions, however, require a correspondence between the present state and the particular state during the previous taste-state association for a change in palatability to be evidenced; in other words, level 3 decisions are state dependent. Level 2 decisions include present state *only* in a nonassociative context (as does level 1), while level 3 decisions include present state as an associative element. The conditioned taste aversion and morphine addiction paradigms represent examples of level 2 decisions, because a correspondence between present state and state previously associated with present taste is not necessary for an alteration in palatability to occur. For example, ingestive FAPs are elicited by the taste of "bitter" morphine when it is presented to an addicted rat or to one that is in morphine withdrawal. Likewise, aversive FAPs are evoked by the taste of a LiCl-paired glucose taste whether it is presented to a sated or to a mildly hungry rat. Present state is included as a factor in level 2 decisions, because its weighting can influence the behavioral outcome. For example, extremes of food or sodium deprivation will alter the degree of taste avoidance when either sucrose or NaCl taste has been a conditioned stimulus in a CTA. In contrast, level 3 decisions require a correspondence between present state and the state of the rat during its previous taste association. For example, a rat prefers a taste that has previously been associated with dilute starch ingestion only when it is tested in a sated condition (Booth, 1977). In fact, if this rat were presented with the dilute starch-paired taste when in a hungry condition, it would avoid the taste, that is, display a reversed preference. The functional hierarchy of the complexity of palatability decisions just noted may be useful in evaluating whether the neural control of palatability is hierarchically organized in the rat's nervous system.

A. CHRONIC DECEREBRATE RATS

1. Level 1 Decisions

The chronic supracollicular decerebrate rat is a caudal brain stem preparation that lacks its forebrain, as shown in Fig. 10. The gustatory system of this preparation contains only the first and second central gustatory relay nuclei, the nucleus of the solitary tract (NTS), and the parabrachial nucleus (PBN). The chronic decerebrate rat is a viable preparation despite its extensive neural damage. It maintains a righted posture, locomotes (albeit sluggishly), and grooms effectively, as shown in Fig. 11.

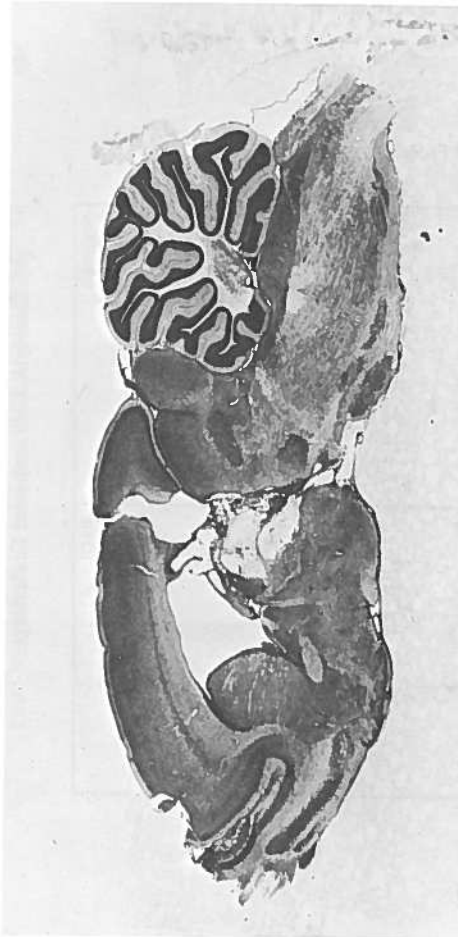


FIG. 10. Sagittal section stained with cresyl violet from the brain of a representative chronic decerebrate rat; survival time, 37 days. The supracollicular plane of section is highly similar for each rat. The tissue posterior to the transection appears normal in the light microscope. A cavity filling the space normally occupied by portions of the thalamus and hippocampus is present anterior to the transections of rats surviving 30 days or more.

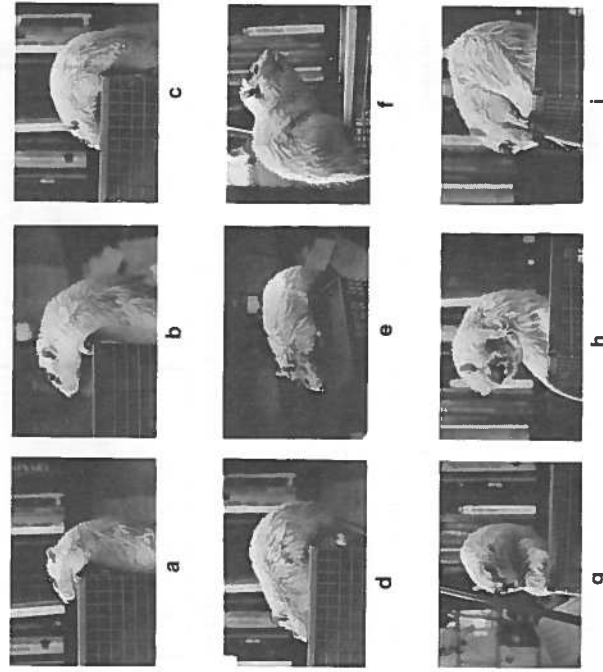


FIG. 11. Chronic decerebrate rats exhibit no spontaneous activity other than grooming but often overreact with well-coordinated movements to seemingly inappropriate stimuli. Tail pinch facilitates a brisk, well-coordinated sequence of cage climbing (a-e). Decerebrate rats maintain their fur; face washing (f) and grooming of the flanks (g and h) and anal (i) and genital areas involve complicated postures that are executed in a coordinated fashion by these rats.

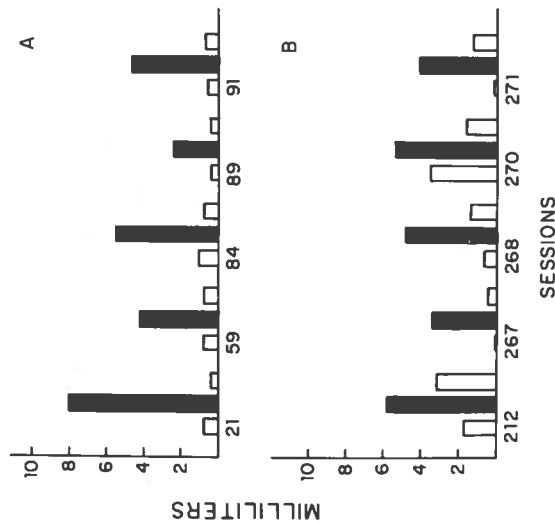


FIG. 13. (A) Internal sucrose (0.03 M) intake of chronic decerebrate rats and (B) pair-fed control rats for just-fed (open bars) and 24-hr food-deprived (solid bars) conditions. Rat code numbers are shown below the abscissa.

centrations result in greater numbers of lateral tongue movements for both chronic decerebrate and intact rats. The decerebrate rat's threshold for the gape response (0.03 mM quinine HCl) is identical to that of the intact rat, as is the number of gapes elicited by increasing concentrations of quinine (see Fig. 12).

Palatability decisions are directly affected by changes in the internal metabolic state of the decerebrate rat. Figure 13 displays the intraoral sucrose intake of decerebrate rats tested in sated and food-deprived conditions. When sucrose is infused into the mouths of 24-hr food-deprived decerebrate rats they, like their pair-fed intact controls, double their sucrose intake over sated levels (Grill and Norgren, 1978d; Grill, 1980). The specific types of internal-state factors that are integrated with gustatory afferent signals (present taste) to produce state-dependent alterations in decerebrate palatability decisions are an important issue. Simple 24-hr food deprivation will affect a great many internal-state factors, while other internal metabolic manipulations may be more specific in their action. For this reason we have examined the effect of insulin injection on the palatability decisions of sated decerebrate rats. Figure 14 demonstrates that insulin injection produces the same reduction in plasma glu-

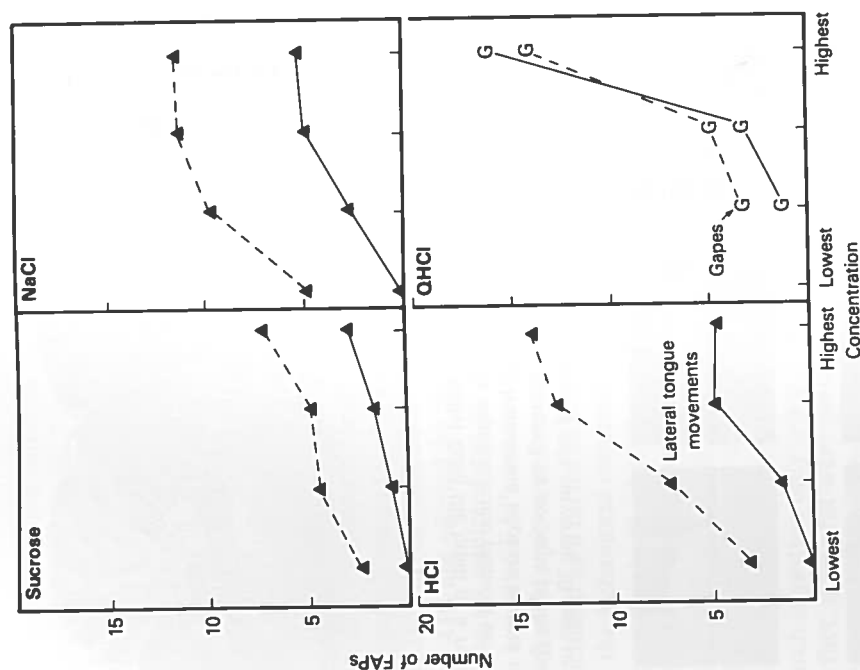


FIG. 12. The highest concentrations of sucrose, NaCl, and HCl elicited approximately the same number of lateral tongue movements from chronic decerebrates (—) as intact rats (---) performed in response to the lowest concentration of these stimuli. Nevertheless, the slope of the lateral tongue movement-concentration function was roughly parallel for both preparations. The number of gape responses to quinine stimuli were very similar for decerebrate and intact rats.

When tested in the sated condition (1 hr after an intragastric meal), the chronic decerebrate rat displays the same taste reactivity responses as the sated intact rat (Grill and Norgren, 1978b). For example, sucrose, NaCl, and HCl stimuli elicit an ingestive sequence composed of mouth movements, tongue protrusions, and lateral tongue movements, while quinine stimuli evoke an aversive sequence including gapes, chin rubs, head shakes, and forelimb shakes. As seen in Fig. 12, increasing sucrose con-

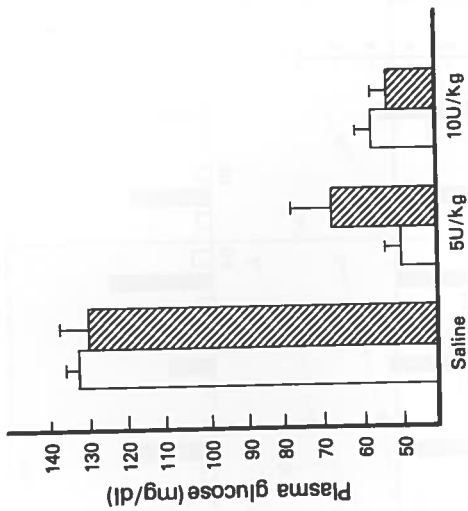


Fig. 14. Mean plasma glucose concentrations (\pm SEM) of control (open) and chronic decerebrate (hatched) rats at the time of intraoral intake tests, that is, 3 hr after treatment with saline and insulin (5 and 10 U/kg).

cose level for decerebrate and pair-fed intact rats. Figure 15 reveals that insulin's effect on plasma glucose, like the more general effects of 24-hr food deprivation, results in a significant increase in sucrose intake but not water intake for both decerebrate and intact rats (Flynn and Grill, 1983). Decerebrate and pair-fed intact rats increase their ingestion of sucrose but not water, in response to the internal-state deficits posed by insulin injection and food deprivation. Since both groups of rats discriminated between the taste of these solutions, the enhanced ingestion of sucrose following internal-state changes in fuel availability does not reflect a general facilitation of fluid intake. It appears that a combination of internal fuel-deficit signals together with specific types of taste signals is necessary to change the intake and palatability of the sucrose taste stimulus. The isolated, caudal brain stem mechanisms of the chronic decerebrate rat are therefore sufficient to integrate these signals into a level 1 palatability decision. But, does this preparation demonstrate its integration of other internal-state changes in the palatability decisions it expresses for other tastes?

We have examined two other examples of level 1 decisions in the chronic decerebrate rat: the effects of hyperosmotic signals on the palatability evaluation of water, and the effects of sodium loss on the evaluation of NaCl palatability. In both instances in chronic decerebrate rats integration had taken place; that is, they did not respond differentially to

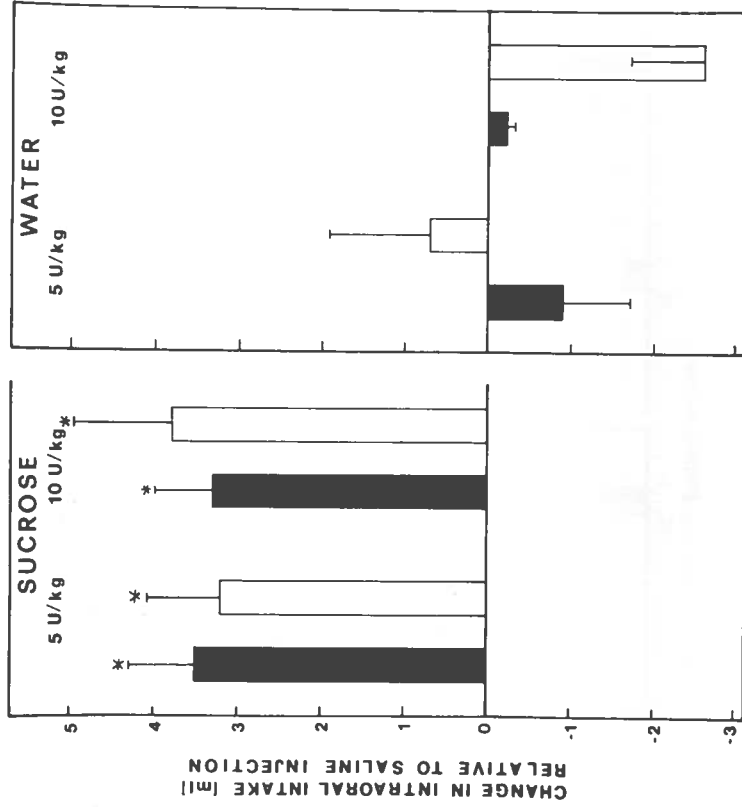


Fig. 15. Change in intake (milliliters) relative to intake during saline injection condition; solid bars, decerebrate; open bars, control. Asterisks denote $p < 0.05$; paired t -test comparisons, significantly different from saline of that group. Sucrose = 0.03 M.

water or NaCl taste as a function of internal-state changes. Figure 16A-C demonstrates that changing the internal osmotic state of decerebrate rats using a variety of treatments (subcutaneous injections of hypertonic NaCl, intragastric NaCl, or water deprivation) does not lead to an immediate or even a delayed increase in the palatability of water as measured by intraoral intake (Grill and Miselis, 1981). The same treatments dramatically elevated intraoral water intake in pair-fed intact rats. The chronic decerebrate, like the intact rat, conserves urinary sodium after sodium loss and excretes sodium during salt loading. Despite this physiological similarity, however, sodium loss, effected by the combination of a diuretic (furosemide) and high doses of a mineralocorticoid (deoxycorticosterone acetate), did not result in either an increase in 0.5 M NaCl intake or a change in the pattern of taste reactivity to NaCl (Grill and Schulkin, 1984). As noted before, pair-fed intact rats dramatically alter both their

decebrate rats can differentially respond to the taste of NaCl and water, their lack of level 1 capacity for integrating taste and internal-state information in response to water or sodium deprivation appears to derive from either a lack of internal-state information available to the caudal brain stem or a lack of an integration of these signals with taste afferent input at this level.

At present the sites of osmotic and other relevant interoceptors and the integration of these signals into the neural mechanisms for the achievement of water balance by ingestive responses are presumed to require the forebrain (Grill and Miselis, 1981; however, see also Rogers and Novin, 1983). With respect to sodium appetites, it is interesting to note that sodium deficit leads to an alteration in primary afferent discharge to NaCl (Contreras *et al.*, 1984). Important questions for the neurology of sodium appetite and the neural control of palatability are whether this change in peripheral nerve response is (1) sufficient to account for the altered behavioral response and (2) dependent or independent of centrifugal control. It is interesting to consider these questions in the light of the decerebrate data. If the peripheral nerve change is behaviorally sufficient and independent of centrifugal influence, then why does the decerebrate not alter either its intake of or taste reactivity to saline? It will be necessary to examine the chorda tympani's electrophysiological response to NaCl in sodium-depleted decerebrate rats to clarify this point.

2. Level 2 Decisions

Garcia *et al.* (1974) have suggested that the association of gustatory and visceral stimuli that results in conditioned taste aversions may reflect the intimate relationship between gustatory and visceral afferent neurons within the NTS of the caudal brain stem. Therefore, one might predict that a chronic decerebrate rat could alter its response to a taste that had been associated with visceral illness. We have found just the opposite (Grill and Norgren, 1978d). Intact rats display a palatability shift for a LiCl-paired sucrose stimulus after a single association (an aversive sequence replaces an initial ingestive one). In contrast, chronic decerebrate rats do not alter their response to sucrose following as many as 12 sucrose-LiCl pairings. Sucrose continued to elicit an ingestive pattern in these rats that was in no way modified by the 12 sucrose-LiCl pairings. Decerebrate and pair-fed intact rats were retested at 24-hr intervals in this paradigm. To examine the possibility that decerebrate rats require an ongoing visceral stimulus in order to display a palatability shift, in a manner analogous to the effects of food deprivation and insulin injection,

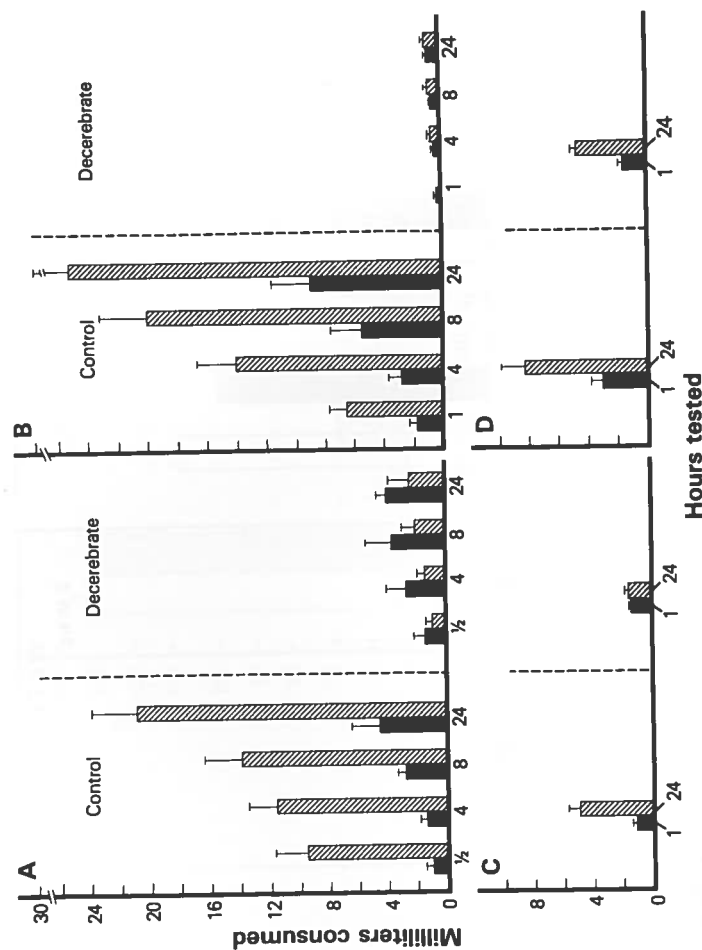


FIG. 16. (A) Intraoral water intake following a 2-ml sc injection of either isotonic saline (control test, solid bars) or 10% saline (experimental test, hatched bars) administered 30 min after intragastric meal. Water was administered remotely via fistulas at 0.5, 4, 8, and 24 hr after the injection; rats did not receive any additional meals during this period. Bars indicate cumulative water intakes from first ingestion test. (B) Intraoral water intake following intragastric administration of 12 ml of regular diet (control test, solid bars) or diet osmotically enhanced with 3% NaCl (experimental test, hatched bars). Testing paradigm was same as in (A) except that first administration of water was given 1 hr after intragastric meals. No additional meals were given during the testing period. (C) Intraoral water intake following intragastric diet deprivation of either 1 hr (solid bars) or 24 hr (hatched bars). (D) Intraoral sucrose (0.03 M) intake following intragastric diet deprivation of 1 hr (solid bars) and 24 hr (hatched bars). Testing paradigm was same as in (C) except that rats were infused with 0.03 M sucrose instead of distilled water.

taste reactivity and intake responses to 0.5 M NaCl as a function of internal sodium state (see Fig. 5).

The sodium-depleted or osmotically challenged decerebrate rat responds to intraorally delivered water or NaCl as it did when tested in the nondeprived state; that is, it consumes the same volume of these tastants irrespective of internal state (level 0 integration, see earlier). Since de-

we have retested these rats 1 hr after taste-LiCl pairing. While intact rats display a palatability shift in this paradigm, decerebrates do not (R. Norgren and H. Grill, unpublished observations).

Decerebrate rats display level 0 decisions for CTA, osmotic challenge, and sodium appetite paradigms, while they demonstrate level 1 decisions for meal and energy challenge paradigms. We have extended the list of the decerebrate's level 1 capacity by showing that when this preparation is treated with cholecystokinin after 24 hr of food deprivation, it (like the intact rat) will reduce both its intake of sucrose and the ingestive taste reactivity responses it elicits (Grill *et al.*, 1983).

B. CHRONIC THALAMIC RATS

1. Level 1 Decisions

The chronic thalamic rat is a diencephalic preparation whose telencephalon has been removed by aspiration and therefore whose thalamus is effectively disconnected from its reciprocal neocortical connections (see Fig. 17). Like the decerebrate, the thalamic rat is permanently aphagic. The gustatory system of the thalamic rat is more complete than that of the decerebrate. The brain of this preparation contains two more relay nuclei than the decerebrate's, the parabrachial nucleus' projection to sites in the ventral forebrain and the PBN's bifurcated dorsal projection to the ventral posteromedial thalamus (Norgren, 1976). In the sated condition (1 hr after tube feeding), however, thalamic rats respond aversively to 50- μ l intraoral applications of *all* taste stimuli and therefore are quite different from intact and chronic decerebrate rats. As shown in Fig. 18, tongue protrusions and lateral tongue movements are not elicited by NaCl, HCl, water, or sucrose taste stimuli in the sated thalamic rat. In contrast to the taste reactivity of sated decerebrate and intact rats, sated thalamic rats display a pattern of mouth movements, rearing, gaping, chin rubbing, and paw wiping in response to a variety of taste stimuli (Grill and Norgren, 1978b). During the execution of this response sequence the stimulus is actively or passively rejected rather than swallowed. The same pattern of response is also elicited by orally applied quinine taste stimuli. While the sated thalamic rat does not display an ingestive sequence of response as a

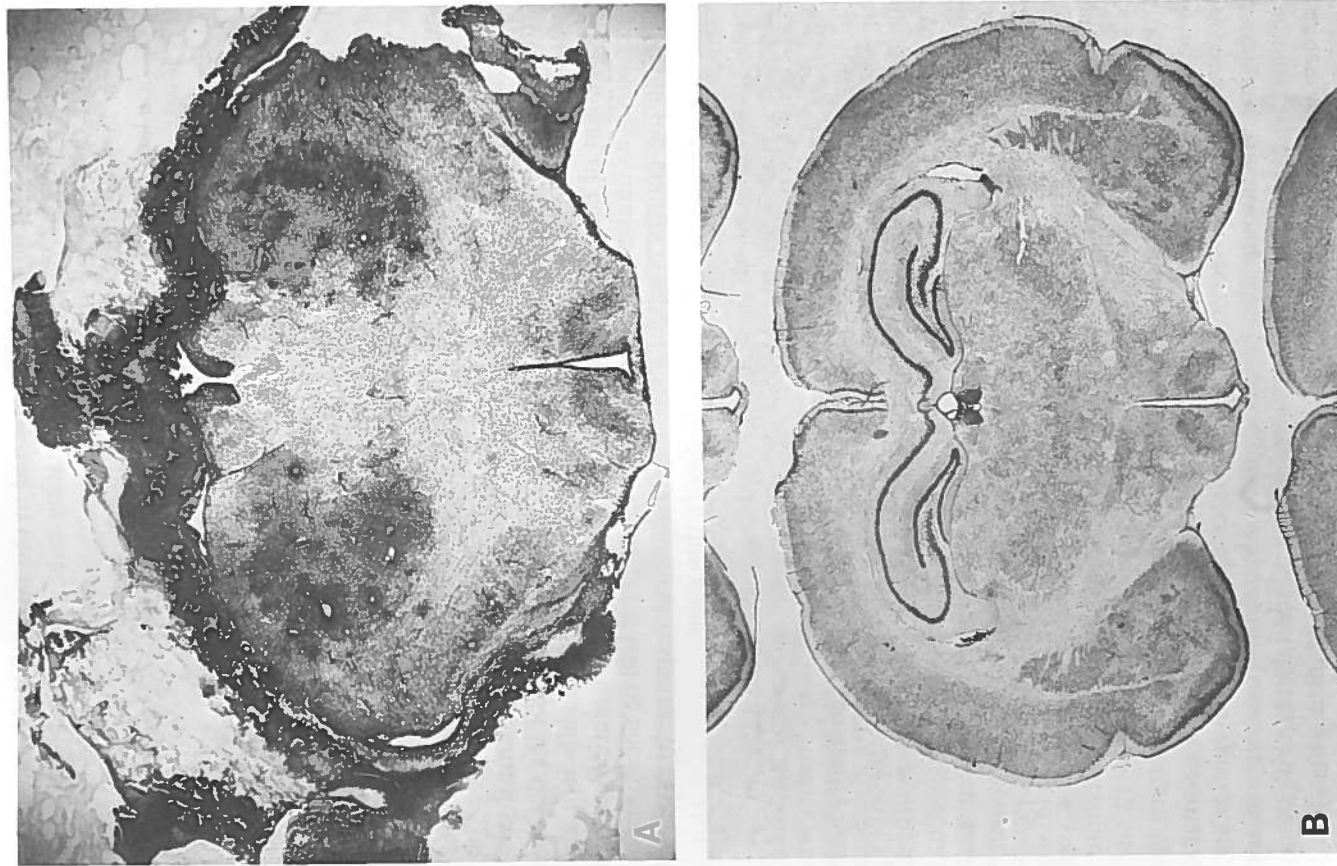


FIG. 17. Coronal section of a representative thalamic (A) and intact (B) preparation at the level of the ventromedial nucleus of the hypothalamus. Sections are stained with cresyl violet; survival time of the thalamic rat, 20 days. There is extensive retrograde degeneration within the thalamus in each case. In this brain, there is gliosis within the thalamus that is probably vascular in origin.

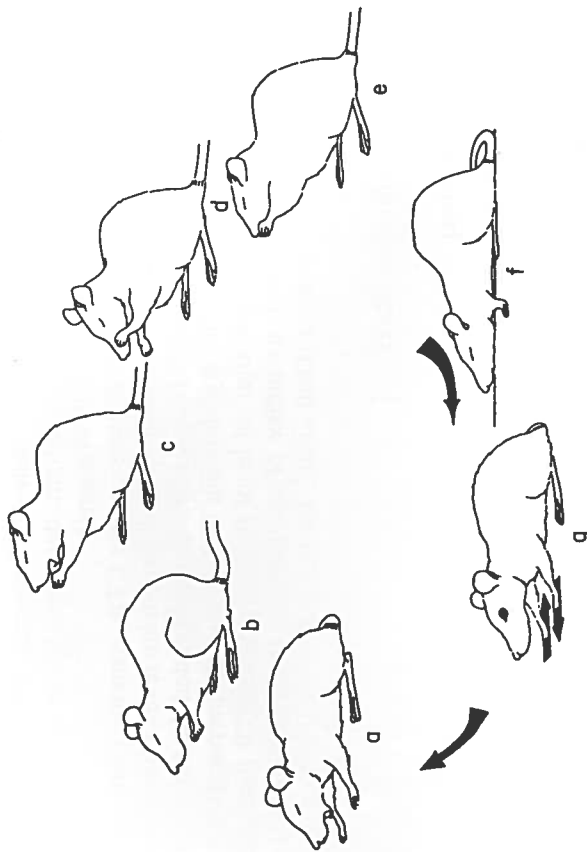


FIG. 18. Every taste stimulus tested elicited the same stereotyped response sequence from thalamic rats. The response began with gaping (a), which was coincident with rearing (a-c), and assuming bipedal posture. In this posture additional gapes were followed by paw wiping (d) and face washing (e). The thalamic rat then resumed the initial quadrupedal posture and performed chin-rubbing (f) and paw-pushing (g) responses. The entire response sequence was repeated up to four times as a function of stimulus concentration and category. Eyes were closed and facial muscles tightly contracted in all components except paw pushing.

function of taste quality, it does alter the number of repetitions of its basic aversive response sequence shown in Fig. 18. Therefore, the duration of its response to taste increases with increasing taste concentration (see Fig. 19).

The capacity of the chronic thalamic rat to alter its response to sucrose taste as a function of metabolic-state change is of great interest but has not yet been examined. Pfaffmann (personal communication) points out that the sated chronic thalamic preparation is the first example of a neurological lesion that completely eliminates positive responses to taste. Since chronic decerebrates have the capacity to execute ingestive response sequences, it seems that neural mechanisms rostral to the midbrain in some way suppress (or depotentiate) the production of ingestive responses of the sated thalamic rat.

One other neural manipulation appears to eliminate positive responses to food, if only temporarily: lesions of the lateral hypothalamus. Teitel-

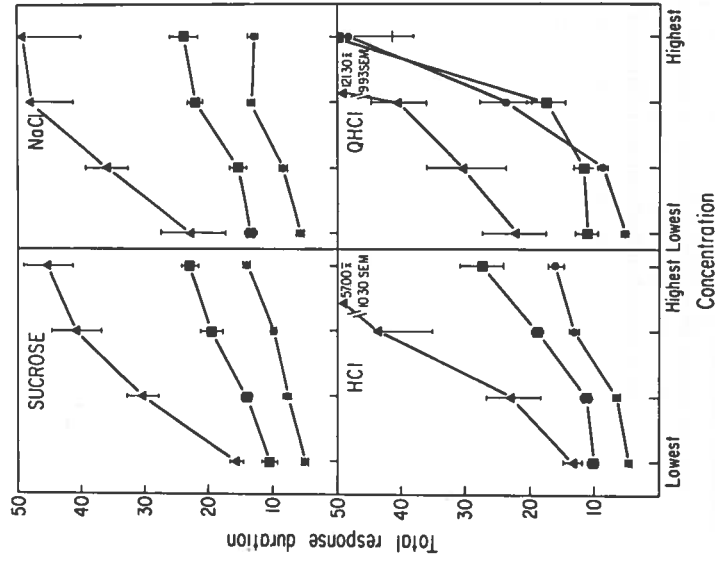


FIG. 19. The response duration for sucrose, NaCl, and HCl stimuli was longer for decerebrates (■) than intact rats (●) for all concentrations tested. Furthermore, the decerebrate's increased response duration was consistent, approximately 7.5 ± 2.1 sec within and between these stimulus categories. Thalamic rats (▲) response duration slope, for all taste stimuli, resembled the rapidly accelerating quinine slopes of intact and decerebrate rats. The quinine-like response duration slope was consistent with the general aversive response of the thalamic preparation. Brackets, SEM.

baum and Epstein (1962) mention that depositing a normally preferred taste into the mouths of stage I lateral hypothalamically lesioned (LHX) rats elicits an aversive pattern of response that includes chin rubbing and forelimb shaking. In an effort to document further and extend these observations, our laboratory has performed additional experiments (Fluharty and Grill, 1980). Assessment of sucrose, NaCl, and quinine taste reactivity and intraoral intake of 1.0 M sucrose and distilled water (1 ml infused during 1 min) began 2 days after rats received large bilateral LH lesions. When tested sated (1 hr after tube feeding), LHX rats, like sated thalamic rats, actively or passively rejected all taste stimuli. Presentations ($50 \mu\text{l}$) of sucrose, NaCl, and water stimuli, as well as quinine, were rejected. These taste stimuli elicited either an aversive pattern that included gapes,

chin rubbing, and paw wiping, or passive fluid rejection. In contrast, when the same LHX rats were tested in the deprived condition their taste reactivity was normal. When these rats were tested food and water deprived (24 hr after intragastric liquid diet), sucrose, NaCl, and water stimuli all evoked an ingestive sequence (like that of intact and chronic decerebrate rats) that included lateral tongue movements and tongue protrusions. Deprived LHX rats, like intact and decerebrate rats, responded to quinine HCl concentrations at or above 0.03 mM with an aversive sequence. Like taste reactivity, the intake measure of palatability was also affected by the internal state of the LHX rat. Intake of 1.0 M sucrose and distilled water was 0.98 ± 0.01 and 0.73 ± 0.10 ml (a total of 1.0 ml was presented) when tested deprived, and 0.36 ± 0.08 and 0.05 ± 0.02 ml, respectively, when the LHX rat was tested sated. To clarify which aspects of the tube-fed meal procedure contributed to the inhibition of ingestive responses, the effects of the tubing procedure itself as well as the volumetric, osmotic, and caloric effects of the liquid diet meal were separately analyzed. As can be seen in Fig. 20, the results of these experiments demonstrate that the suppression or depotentiation of ingestive responses in LHX rats following a tube-fed meal derives from an exaggerated sensitivity to the caloric consequences of the meal.

C. CHRONIC DECORTICATE RATS

The chronic decorticate rat is a striatal preparation whose neocortex has been removed via aspiration as seen in Fig. 21. The central gustatory system of the decorticate is complete except for its cortical component. Decorticate rats are not permanently aphagic, unlike the thalamic and decerebrates. While deficits in ingestive tasks are short lived or minimal, some transient and permanent deficits in tongue and forelimb use that could contribute to food consumption have been noted (Castro, 1975; Kirwin *et al.*, 1978; Whishaw *et al.*, 1981). Despite the loss of the entire neocortex and its cortical gustatory area, decorticate rats seem capable of displaying both level 1 and 2 palatability decisions (see Wirsig and Grill, 1982).

1. Level 1 Decisions

Using the intake measure of palatability, sated gustatory decorticate rats have been shown to display normal taste thresholds for sucrose, NaCl, HCl, and quinine tastes (Benjamin, 1959; Braun and Kiefer, 1975). Decorticate rats respond to alterations in internal metabolic, osmotic, and

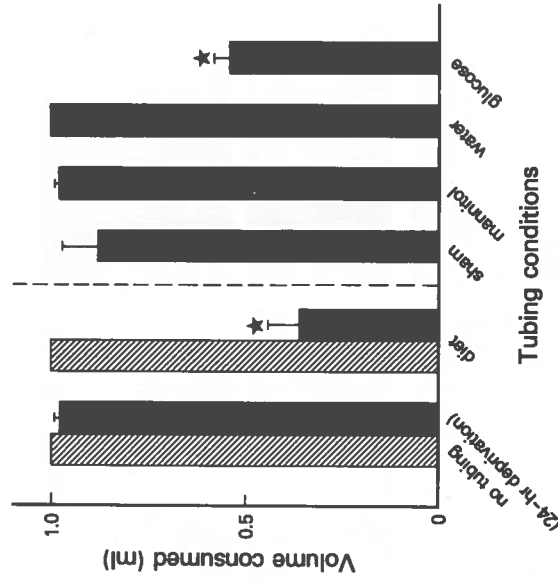


FIG. 20. The effect of six randomly presented intragastric intubation conditions on intragastric sucrose intake. The conditions are 12 ml of liquid diet, 0.2 M mannitol, 1.4 M glucose, 8 ml of distilled water (the portion of 12-ml diet that is water); passing the tube but not delivering any liquid (sham); or neither passing the tube nor any liquid (24-hr deprived). At 90 min after each intubation condition, 1 ml of 1.0 M sucrose (1 ml/min) was intragastrically infused into the mouths of lateral hypothalamically lesioned (solid bars) and yoked control (hatched bars) rats. The volume of the sucrose ingested is shown as the amplitude of each histogram. Analysis of variance reveals that sucrose intake following diet and glucose-tubed conditions is significantly lower ($\star, p > 0.01$) than other conditions for the lesioned rat.

sodium state by altering their intake of liquid diet, water, and NaCl, respectively (Wolf *et al.*, 1970; Schulkin and Grill, 1980).

2. Level 2 Decisions

While there is some disagreement over the effect of gustatory cortical ablation on the rat's ability to acquire and retain a conditioned taste aversion, this may be explained by a lack, until recently, of an adequate anatomical description of this region (Kosar *et al.*, 1984; Norgren, *et al.*, 1982). Using physiological feedback first to determine the boundaries of this region and then to ablate it bilaterally, Yamamoto *et al.* (1980) have determined that while these rats do not retain a prelesion CTA, they can acquire a postlesion CTA and therefore possess what appears to be level 2 decision capacity (Kiefer *et al.*, 1984). As a proviso to the data presented on the complete or gustatory decorticate rat, it should be noted that only

intake responses and not taste reactivity responses have thus far been measured. Since intake responses do not distinguish between taste avoidance and taste aversion (see earlier and Peichat *et al.* 1983), it will be important to reexamine these experiments using the taste reactivity measure of palatability.

Operating on the assumption that while the neocortex does not seem to contribute to level 1 or 2 palatability decisions, its preeminent position in the neural hierarchy should be revealed by deficits in more complex palatability decisions, Wirsig and Grill (1982) examined decorticate rats for their ability to express a salt appetite by latent learning. Using the paradigm of Kriekhaus and Wolf (1968), groups of water-deprived decorticate rats were trained to perform a bar-pressing task to obtain either water or isotonic NaCl. After daily bar-pressing rates had stabilized, groups of rats were first allowed ad libitum access to water (water deprivation ameliorated), then were treated with furosemide and deoxycorticosterone acetate to elicit salt appetite (sodium deficit facilitated), and then were returned to the operant situation and were allowed to press a bar that did not yield fluid reward (extinction). Decorticate rats that were trained with isotonic NaCl reward (for as little as 1 min!) resisted extinction, while water-trained decorticates extinguished rapidly. In this paradigm, present taste was eliminated as a factor by the extinction condition, but present state (sodium deficit) and the previous association between taste (NaCl) and the behavioral act of procuring that particular taste (bar pressing) were integrated to yield the resistance to extinction in the NaCl-trained group. These data indicate that decorticate rats are capable of demonstrating the output of complex palatability integrations that are even more complex than those comprising levels 2 and 3, suggesting a fourth level.

V. Neural Control of Palatability: The Adequacy of a Hierarchical Model

There are many ways in which the functional palatability decision could be theoretically mapped onto the brain. For instance, the various inputs (taste, internal-state cues, prior associations) could converge onto a single neural integrator that would decide on the appropriate response for the whole system. This single integrator could be an anatomically compact nucleus or region (e.g., hypothalamus, see Grill, 1980) or could be distributed as a network throughout the brain. The defining feature of a single-integrator model is that its complex integration would produce only a single decision that would direct all responses to a taste. A disruption of the system should likewise affect all responses equally. At the other theo-

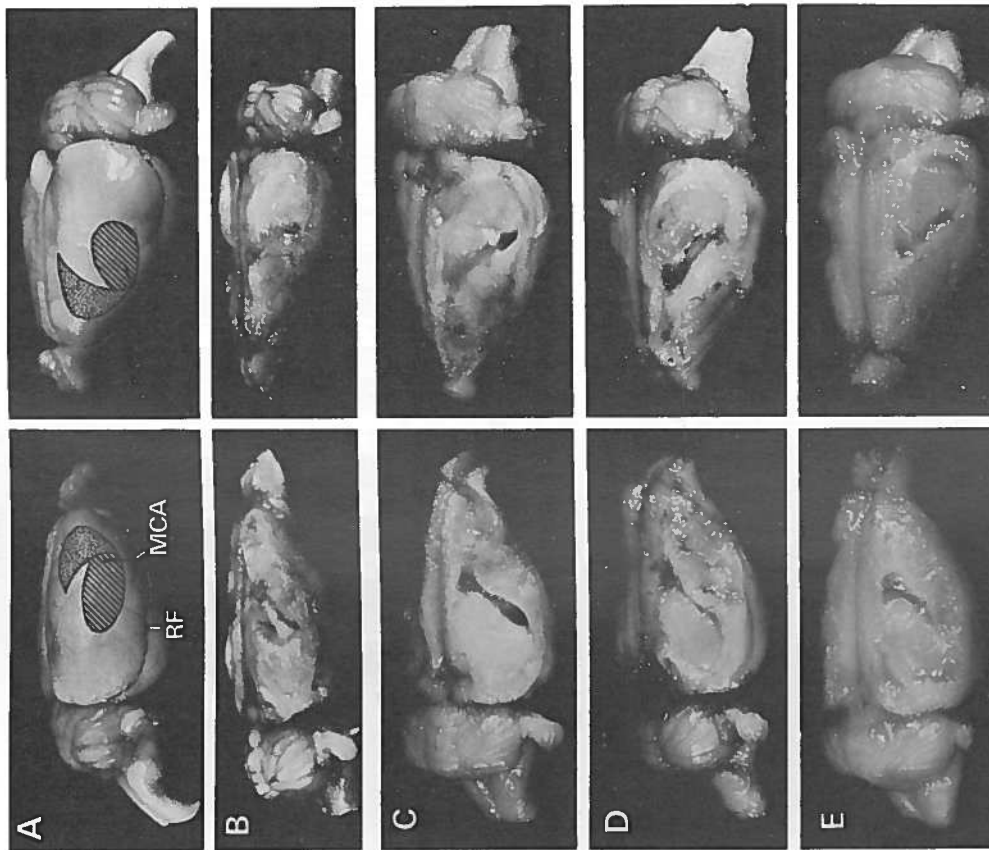


FIG. 21. Lateral views of each hemisphere of four representative decorticate and one intact brain. The upper pair of photographs (A) of an intact brain focuses the reader's attention on the position of the rhinal fissure (RF) and the fissure within which the middle cerebral artery (MCA) courses. The crossing of these two fissures just dorsal to RF is the approximate center of the gustatory neocortical representation. The two outlined regions of neocortex in (A) represent the approximate position of oromotor (dotted pattern) and orosensory (striped pattern) neocortical representations (Hall and Lindholm, 1974). The slightly different appearance of these regions in the two lateral views relates to the different rotations of the brain in the two photographs. As can be seen from comparing (A) with (B-E), oromotor and orosensory regions have been bilaterally removed in the brains shown.

retical extreme, there might exist myriad neural entities, each making separate decisions unrelated to one another, that sum together equally to produce an output (cf. Dethier, 1982). The unitary appearance of such an output might be misinterpreted according to this view; the behavioral output would not need to correspond to any one of the decision-making entities but only to their interaction in the final common path.

We believe that the data just described suggest that neither of these models is accurate, and that palatability is instead processed by the brain in a neural hierarchy characterized by a number of neural decision-making entities. The presence of these multiple decisions is revealed both by the data from surgical transections and lesions, and by the detailed analysis of the FAP sequences of intact animals discussed earlier in this article. These decision-making entities are not equal, however. Some of the decisions revealed by decerebrate transections, for example, are overruled by the forebrain in intact animals. For example, sucrose paired with LiCl injection results in the substitution of an aversive pattern for an ingestive pattern in intact rats but not in decerebrate rats. Likewise, the separate decisions that are displayed in the taste-elicited FAPs may be combined into a single one to direct instrumental appetitive behavior. This implies that the multiple components are organized hierarchically: some entities integrate more factors and are the "boss of" other decision makers (Dawkins, 1976), being able to overrule them.

As a caveat, it should be noted that this interpretation holds only if we assume that the caudal brain stem mechanisms that make decisions in the decerebrate and thalamic rats continue to make those same decisions in the intact rats but are overruled by the rostral mechanisms. In other words, in order to reject the hierarchical hypothesis of palatability processing, it would be necessary to show that the processing and integration of taste within the caudal brain stem is completely different in decerebrate and intact animals, and that the decisions shown by the decerebrate are simply not made in a caudal brain stem that has an attached forebrain. However, there is currently no reason to assume that the caudal brain stem functions differently when isolated from its forebrain connections. In fact, decerebration is often employed in physiological experiments concerned with demonstrating the physiological function of *intact* caudal brain stem function (e.g., Lund and Dellow, 1971; Kawamura and Yamamoto, 1978).

The hypothesis that palatability is organized hierarchically within the brain carries definite implications. First, discrete neural manipulations such as transections or lesions should not eliminate the intervening variable entirely but instead should either fragment it by destroying the hierarchical connections between components, or unbalance it by eliminating

certain components of the hierarchy or inputs to the components. The fragmentation of behavior is exemplified by the decerebrate, in which only a limited subset of the causal factors are processed and only FAPs and neuroendocrine responses are activated, and by the demonstrations of Teitelbaum and colleagues of the deterioration of goal-directed movement into its component (and often antagonistic) reflexes following lateral hypothalamic lesions. The discovery of such fragmentation has led to the suggestion that the purposiveness of directed behavior in intact animals and the apparent unity of intervening variables are "visual illusions" (Teitelbaum, 1982). This suggestion is based on the belief that when behavior can be broken into parts, the parts may be more real than the whole. It is important to recognize, however, that this is not true if the nervous system is organized hierarchically. The relations between the parts, the direction in which information flows, and the relative priority of each part's output are as important as the parts themselves. The importance of these interpart relations make the whole as real an entity as the parts themselves. A goal-directed action may utilize many movement subsystems. However, these systems are not allowed to operate independently in the integrated nervous system of an intact animal. They are selectively overruled and combined by higher rules of connection that are extrinsic and superordinate to the subsystems themselves. It is the reality and importance of these rules that is revealed by neural manipulations that fragment behavior, not their "illusory" nature.

A second implication of neural hierarchies concerns the procedures for validating intervening variables in behavioral analyses. An intervening variable is defined by a high degree of correlation between a constellation of different behaviors: they vary together within an animal and they are influenced in the same ways by the same things (Miller, 1959). The question is, how good must such a correlation be before one can conclude that a single decision is controlling a group of behaviors? If the single variable exerts a direct and exclusive control over each behavior, then the correlation between different behavioral measures should be perfect (Miller, 1959, 1982). In a hierarchical mechanism, a perfect correlation between outputs is possible as long as each component is controlled by only one higher component. But not all hierarchies are organized in this way. In many hierarchies, called branching (Dawkins, 1976) or lattice (Gallistel, 1980a) hierarchies, the descending networks of separate high-level components overlap. In these hierarchies, a given low-level component appears to have multiple controls. Two low-level components may share certain higher controls with one another but not share others, and therefore may not correlate perfectly with one another. Since the evidence for multiple controls over behavioral acts is quite strong (e.g., Premack,

1962; Fentress, 1968; Hinde, 1970; Gallistel, 1980b), we should expect that when the nervous system controls behavior in a hierarchical fashion the hierarchies involved will be lattice ones. Agreement between different behavioral outputs that are influenced by the same high-level controls should be probabilistic rather than perfect. This should be kept in mind when considering what constitutes a valid empirical test for intervening variables.

A. ANATOMICAL MODELS

The model of brain organization suggested by Hughlings Jackson incorporates a functional hierarchy into a neural framework (in Taylor, 1958). This hierarchy is a lattice hierarchy: the principle of "descending control" allows low-level motor elements to be activated by a variety of high-level entities. For example, tongue movements, whose final common path neurons are contained within the hypoglossal nucleus, may be utilized in different constellations of behavior such as speech and food intake. The cortical lesion accompanying aphasia interferes with tongue movements in speech without affecting the tongue movements of food ingestion (as noted by Jackson in 1878). That is, the final common path is preserved, but some aspects of its control by higher-level structures have been disabled.

Jackson's hierarchical model carries a very salient feature that has influenced our present conception of the nervous system: it is organized rostrocaudally. Higher functions are assumed to be the province of rostral entities, and the more complex the integration, the more rostral the corresponding structure is thought to be. Thus, the neocortex sits at the apex of this neurofunctional hierarchy. Caudal mechanisms can integrate and control simple behaviors but not complex ones, and even the simple integrations of caudal mechanisms are performed again in (and may be overruled by) more rostral structures.

To a large degree, the data we have described here are compatible with Jackson's longstanding model. Decisions about palatability are made at several levels of the nervous system. Even the supracollicular decerebrate can integrate taste with the internal-state cues arising from food deprivation or insulin injection, to produce the appropriate behavioral responses. The ability to integrate other sources of information, such as learned associations, emerges only in rats possessing more rostral structures. And these additional structures and integrations are capable of entirely reversing the decisions made by the isolated caudal brain stem. For example, while a single oral infusion of sucrose will always elicit ingestive FAPs in a decerebrate rat, even one that has repeated treat-

ments in which LiCl has been paired with sucrose, taste aversion conditioning can eliminate all traces of ingestion and replace it completely with aversive FAPs in the intact rat. Sodium depletion similarly changes the pattern of taste reactivity for intact but not chronic decerebrate rats.

B. EVALUATING JACKSON'S HIERARCHICAL MODEL

There are two observations from our data, however, that strongly contradict Jackson's model and suggest that modifications of his schema must be made. The first is that the behavioral competence of the decorticate rat in tasks involving higher integrations is nearly identical to that of the rat that possesses a neocortex (e.g., Braun, 1975; Oakley, 1979; Braun *et al.*, 1981; Wirsig and Grill, 1982). The decorticate is responsive to all of the internal-state variables that affect the responses of intact rats to tastes, and can associate tastes with their postingestive consequences and act accordingly. The deficits of decorticate rats, while undeniably real, appear to have more to do with low-level acts of sensorimotor integration (e.g., fine tongue movements and place orientation; Whishaw *et al.*, 1981) and with certain memorial or retentive processes (in taste aversion learning; Yamamoto *et al.*, 1980). The low-level nature of these deficits is striking considering that one has removed what ought to be the prime integrator of behavior (highest level). To date, the only thing the neocortex seems to give the rat is a number of relatively simple integrations. Vanderwolf (1983) has summarized a variety of work with his colleagues that suggests that decortication can produce more severe behavioral deficits in rats than we have indicated here; however, differences in surgical procedures, involving most notably their removal of pyriform cortex and hippocampus, and damage to subcortical structures may account in large part for the increased severity of the deficits they report.

A hierarchical model of the nervous system is compatible with these data only if one is willing to reject the assumption that the cortex dominates the functional hierarchy. Unless future tests can be developed to show that the decorticate rat lacks specifically high-level functions, it appears that the cortex plays a functional role of no greater complexity than the caudal brain stem in the control of ingestive behavior and that, instead, it is the subcortical forebrain that exerts the highest functional controls on palatability processes. Our view of the cortex as a functionally low- or middle-level component in the control of palatability is compatible with trends in the study of the controls of complex voluntary movement in primates (cf. Phillips, 1973; Evarts, 1975) and with human clinical data (Penfield and Rasmussen, 1950). A similarly non-Jacksonian role has been suggested for the entire forebrain of the less encephalized

teleost fish (cf. Aronson, 1970, for review). Forebrain ablation typically eliminates no single complex integration in these animals. Instead, it slightly impairs performance on a great many behavioral tests in ways that are not compatible with the loss of specifically high-level integrations.

A second difficulty for Jackson's model comes from a comparison of the thalamic and decerebrate preparations. The decerebrate rat is capable of producing both ingestive and aversive FAPs in response to the same tastes as the intact rat, and of modifying these responses on the basis of its caloric state. The thalamic rat, in contrast, shows only aversive FAPs (even to sucrose), and if it is capable of modifying its response at all, it certainly is less flexible than the decerebrate rat. The presence of the substriatal forebrain combined with a caudal brain stem appears to produce a less sophisticated behavioral product than the caudal brain stem alone, in direct contradiction to Jackson's predictions. The hierarchical model of the nervous system can be saved, however, if we assume (1) that the separate positive and negative decisions reflected in the FAPs of the intact rat are also separately embedded in the neural decision-making systems of the forebrain, and (2) that the mechanisms of the negative decision predominate in the substriatal forebrain. This argument will be convincing, and the Jacksonian hierarchical model can be retained, only if it eventually can be shown that all of the integrations and decisions (positive as well as negative) made by the decerebrate are also made by the thalamic rat, but that the positive ingestive decisions are simply overruled by the additional rostral circuitry in this preparation. If this is true, then the thalamic preparation can be viewed as unbalancing the hierarchy at the level at which the positive and negative assessments of palatability still persist as separate entities. This still contradicts Jackson's model to some degree (because an animal that possesses part of a forebrain remains less flexible than one that has none), but it does at least allow the nervous system to become functionally more complex as one ascends rostrally. It is simply that the functional lopsidedness of the remaining circuitry prevents some of its potential sophistication from being expressed behaviorally.

C. HIERARCHIES OF INFORMATION PROCESSING

The degree to which taste information may be transformed as it is conveyed rostrally along the functional hierarchy is a separate issue, concerning which little empirical evidence exists at present to guide us. Two extremes are possible: on the one hand, taste information might be preserved in its original form at every level. Each level would then sepa-

rately carry out all of the analyses relevant to its particular decision. This is the state of affairs envisioned by Jackson. In his model, the elementary integrations performed by lower levels are carried out again by higher mechanisms (the principle of rerepresentation; in Taylor, 1958). This requires sensory information to be unchanged by initial processing if it is to arrive at higher mechanisms in its original "raw" form. It also requires that a tremendous amount of redundancy be built into the system. Some elementary integrations may have to be performed many times on the same sensory data.

An alternative possibility is that of a two-way transfer of information between different levels of the hierarchy. In this model, not only are lower levels controlled by the decisions of their superiors, but also the outputs of those lower levels are themselves the objects on which higher levels base their decisions. If higher mechanisms can delegate simple processing to lower elements, then "raw" sensory data need not be conveyed unchanged to every level of the hierarchy. This is a two-way hierarchy in the sense that there is a hierarchy of information processing, as well as a hierarchy of control. In sensory systems there are numerous examples of reciprocal projections from rostral levels to the preceding one(s). For example, the whole gustatory system receives feedback from the cortical relay (Norgren and Grill, 1976), and this arrangement is not unique to the taste system but seems to be a feature of all sensory systems. In addition to this possibility of descending control, there are, as noted earlier, several suggestions that the "purity" of the taste signal is not preserved as it is processed successively by the central gustatory system. Rather, there is some evidence to suggest that the taste signal is altered in its early central processing by its integration with visceral afferent signals (see Norgren, 1983).

These two models are theoretical extremes, and intermediates could also exist. Higher levels could use unfiltered sensory information for some decisions but rely on the outputs of lower levels for others. It is also possible that functionally identical decisions could be made using different sources of information under different behavioral conditions. For example, Fentress (1972) has found that while tactile sensory feedback is crucial to sequencing decisions in normal mouse grooming, the same decisions are made without sensory feedback during displacement grooming under conditions of high behavioral arousal. The resolution of these issues will require a better understanding of the degree to which taste as well as visceral information is transformed and how it is used by different levels of the nervous system hierarchy. Both behavioral studies in conjunction with neural manipulations and electrophysiological data will be relevant to deciding between these possibilities.

D. ALTERNATIVES TO BRAIN HIERARCHIES

This discussion has focused entirely on a hierarchical model of nervous function, because it is most useful for predictions about how various manipulations should affect behavior, and because the data gathered by us so far are compatible with such predictions, given the modifications to the model discussed earlier. We wish to stress, however, that this need not always continue to be the case. Other models of neural function are conceivable, for example, the single-integrator and distributed equal systems models mentioned earlier. More sophisticated alternatives have also been suggested under the label of heterarchies or other systems of distributed control (Winston, 1972; Nelson, 1973; Mountcastle, 1978; Dethier, 1982). Dynamic systems in which the rules of control are not fixed but instead vary are also possible, so that some models obtain under certain conditions but others are more suitable in different behavioral situations (Fentress, 1983). As a general rule, however, these models are defined primarily in opposition to hierarchies or simpler models (cf. Dawkins, 1976). The precise details of their operation are unclear and not susceptible to definite prediction. Until specific versions of these models can be constructed that suggest testable predictions for ingestive behavior, it is difficult to think about them with the same degree of rigor that one can bring to bear on the hierarchical model. Advances in this direction therefore await theoretical rather than empirical development.

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