L_{min}), where L_{max} and L_{min} are the maximum and minimum intensities in the grating.
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21. We found that for receptive field positions more than about 5° from the area centralis, where the average resolution of neurons is slightly lower than in the center, cells were more commonly binocularly driven. However, this result is not simple to interpret since monocular units are also more common for the central visual field in normal cats [K. Albus, *Brain Res.* **89**, 341 (1975)].
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Chronically Decerebrate Rats Demonstrate Satiation But Not Bait Shyness

Abstract. *Taste substances applied to the oral cavity result in either ingestion or rejection, each with a characteristic muscular response pattern. These responses are the same in decerebrate and intact rats; the caudal brainstem appears to be the neural substrate of ingestion and rejection responses. The experiment determined whether decerebrates can alter these discriminative responses as a function of food deprivation or toxicosis. Food-deprived decerebrate rats, like intact ones, ingested a taste substance they had rejected when sated. However, these same decerebrates, in contrast to controls, neither rejected nor decreased ingestive reactions to a novel taste after that taste had been repeatedly paired with lithium chloride–induced illness. Although the forebrain may be important for integrating ingestion, some aspects of this control seem to be represented in caudal brain areas.*

Although many complex reflex sequences exist within the spinal cord and caudal brainstem (*1*), the integration and control of these sequences required for normal behavior has usually been attributed to higher levels of the brain. Bard (*2*) concluded that structures caudal to the hypothalamus are incapable of altering consummatory responses as a function of visceral or humoral variables. Bard's version of Jacksonian neurology provides the basis for most current interpretations of the effects of hypothalamic and other limbic system lesions and electrical stimulation on attack, copulation, grooming, thermoregulation, and food and water intake (*3, 4*).

Neural models for maintaining energy balance have hypothesized hypothalamic mechanisms for controlling both the initiation and cessation of feeding behavior (*3*). The metabolic monitors necessary to effect this control were also presumed to be located within the hypothalamus. Recently, the hegemony of the hypothalamus has been effectively challenged by experiments demonstrating the importance of systems outside the hypothalamus, and even outside the brain, in controlling feeding behavior (*5*). Nevertheless, the predominant assumption remains that the forebrain controls ingestive behavior even if some of the requisite information comes from the periphery.

In contrast, Garcia has suggested that the peculiar associability of gustatory and visceral stimuli evidenced in bait shyness (*6*) may reflect the intimate relationship of gustatory and visceral afferents within the nucleus of the solitary tract (*7*). Therefore, one might predict that a decerebrate animal could alter its ingestive behavior as a consequence of illness, but not of repletion. We have found just the opposite. The chronically

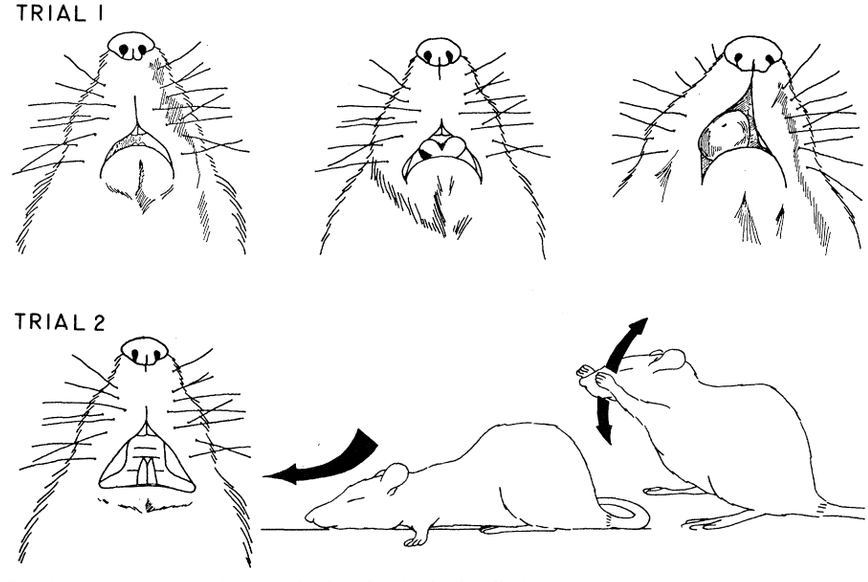

Fig. 1. Taste reactivity for unpaired and paired stimuli. Sucrose, NaCl, and HCl stimuli when presented intraorally in 50-μl presentations elicit an ingestion sequence composed of rhythmical movements of mandible and tongue and lateral tongue flicks (trial 1). After a single pairing of the taste stimulus with LiCl injection, the taste elicits a replica of the rejection response to quinine. This rejection response is composed of gaping, chin rubbing, and paw shakes (trial 2).

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decerebrate rats clearly altered their ingestive behavior as a result of repletion, but did not easily demonstrate learned taste aversion.

Decerebration at the supracollicular level was performed in two stages with a hand-held spatula (*8*). Despite loss of the forebrain, the chronically decerebrate rat maintains a righted posture, grooms spontaneously, and will walk, run, and jump when stimulated (*9, 10*). The decerebrate does not effectively thermoregulate and is permanently aphagic and adipsic (*11*). In order to assess ingestive behavior in aphagic, adipsic animals, we developed a taste reactivity test (*12*). We videotaped the oral-facial responses that occurred after we injected small, calibrated volumes (0.05 ml) of taste solutions directly into the oral cavity through permanent intraoral fistulas. The videotapes were examined frame by frame, so that each component of the response could be characterized, sequenced, and timed.

In intact rats, taste stimuli elicit one of two response patterns. Sucrose, NaCl, and HCl elicit low-amplitude mouth movements, tongue protusions, and lateral tongue flicks; the stimulus is ingested. Quinine elicits gapes followed by chin rubs, head shakes, face washes, paw shakes, and then paw wipes; the stimulus is rejected. Both responses are highly stereotypic in form, sequence, and timing of the components, both within and across rats. Since the response components associated with sucrose, NaCl, and HCl do not overlap those associated with quinine, the behaviors are easily differentiated once the videotape has been slowed somewhat. After a single pairing with an intraperitoneal injection of LiCl, a taste stimulus that normally elicits ingestion will then evoke an exact replica of the response to quinine. This altered taste reactivity is robust, discriminative, and persists for approximately 1 month in the intact rat (*13*).

In experiment 1, five decerebrate and two full surgical control rats (*14*) were examined for their capacity to retain the altered taste reactivity acquired during a pairing of a taste with LiCl before the transection was made and were reexamined after transection for the capacity to acquire the same association. To control for the possibility that the capacity to acquire or retain associations was present in the decerebrate but not operative because of a decrease in endogenous arousal, rats were also tested during exogenous arousal under conditions of tail pinch and amphetamine injection (*15*).

The decerebrate rats neither retained nor acquired an association of a taste with the LiCl even under conditions of exogenous arousal. While intact rats substituted rejection for ingestion after a single pairing (Fig. 1), decerebrates ingested the taste solution as if no pairing had occurred. Decerebrates were exposed to one bout of four daily taste-LiCl pairings before and two after transection, a total of 12 such pairings. Although each bout contained four times the pairings necessary to develop an association in a normal rat, it is possible that exposing these decerebrate rats to an even greater number of pairings might still demonstrate associative regulatory function. If evidence of an association did appear after, say, 25 pairings, when it requires only a single trial in control rats, we would need to determine whether the associative mechanism of the decerebrate was not qualitatively as well as quantitatively different from that of normal rats (*16, 17*).

Attempts to demonstrate classical conditioning of eye-blink and respiratory responses in chronically decerebrate rats and cats have suffered because it has been impossible to determine whether the associative capacity of the decerebrate is in any way comparable to that of intact animals (*16, 17*). The slower rates of acquisition, lower percentages of conditioned responses, and greater degree of intersession extinction argue strongly against complete association mechanisms restricted to the caudal brainstem. In fact, Lovick and Zbrozyna have suggested that the process of consolidation of newly acquired information is either absent or severely impaired in chronically decerebrate rats (*17*). Our findings seriously question the existence of associative neural mechanisms restricted to the caudal brainstem.

In experiment 2 we examined whether these same rats could respond to hunger and satiation, that is, alter how much of a constant taste stimulus they would ingest as a function of hours of food and water deprivation. Every 5 seconds, 50 μl of a 0.03*M* sucrose stimulus was injected intraorally. Taste reactivity to this constant stimulus was examined under two conditions: sated (1 hour after tube-feeding) and deprived (24 or 48 hours since last tube-feeding) (*18*). Termination of ingestion was operationally defined as an active or passive removal of the sucrose stimulus from the oral cavity that persisted through each of two 30-second no-stimulus pauses. In the sated condition, response termination was almost always characterized by a substitution of a quinine-like rejection sequence for the ingestion sequence. In order to control for the specificity of the response elicited by 0.03*M* sucrose, 1.0*M* sucrose and distilled water were also tested when the animals were sated.

The same decerebrate rats that failed to alter their response as a function of a previous, viscerally applied stimulus (LiCl, injected intraperitoneally) did change their responses as a function of an ongoing visceral stimulus (food in the gut). Table 1 depicts the volume of 0.03*M* sucrose ingested by decerebrate and full surgical control rats when they were sated or deprived of food and water for 24 to 48 hours. More data will be required in order to determine whether the decerebrates are more sensitive to the deprivation-repletion variable than the controls.

In all sated rats, the termination of weak sucrose ingestion was overcome by injecting a more concentrated (1.0*M*) sucrose solution. Since the test animals were maintained on a liquid diet, both food and water were withheld during deprivation. To test the possibilities that the weak sucrose stimulus was interacting with water deprivation to yield increased ingestion or that deprivation was

Table 1. Actual values of a 0.03*M* sucrose stimulus ingested by decerebrate and control rats when the stimulus was presented intraorally in 50-μl samples every 5 seconds. Ingestion of this taste stimulus was examined when the rats were sated and when they were deprived of food and water for 24 and 48 hours. The 48-hour deprivation was not complete in that rats ingested some fluid during the 24-hour deprivation session that immediately preceded it. Difference values compare each deprivation condition with the sated condition that most closely preceded it.

Subject	Amount consumed (ml)			
	Sated	Deprived		Difference
		24 hours	48 hours	
Chronically decerebrate rats				
65	2.8	13.4		10.6
	4.0	20.0		16.0
	1.6	12.0		10.4
			15.8	14.2
69	2.9	20.0		17.1
	0.8	17.4		16.6
	2.5	16.6		14.1
			20.0	17.5
66	2.2	6.8		4.6
	3.4	16.3		12.9
	2.9	15.4		12.5
			13.2	10.3
Control rats				
64	11.7	15.7		4.0
	9.8	12.5		2.3
	9.8	16.3		6.5
			10.6	0.8
68	2.0	8.3		6.3
	4.6	9.8		5.2
	2.5	8.0		5.5
			8.8	6.3

sufficiently arousing to increase the ingestion of any stimulus, the same experiment was repeated with distilled water substituted for 0.03*M* sucrose as the taste stimulus. Under these circumstances the amount consumed by both decerebates and controls during deprivation did not differ from the amount consumed when they had just been fed (*19*).

Hunger has been operationally defined as an increase in food consumption as a function of food deprivation; satiation is defined conversely (*20*). Our data support the hypothesis that at least some of the normal control mechanisms of hunger and satiation are restricted to the caudal brainstem. Previously these mechanisms had been assigned exclusively to hypothalamic-forebrain structures.

The two experiments reported here are parallel in that they examine whether the response to a constant stimulus is altered as a function of changes in internal state; they differ in that in one case the viscerally applied stimulus is ongoing (food in the gut) and in the other the stimulus was applied previously (LiCl in the gut). The decerebrate rat required that the visceral stimulus be ongoing or present in order for ingestion of a taste stimulus to be replaced by rejection. It is conceivable that if the decerebrate animal were tested during rather than after the period of LiCl stimulation, the formerly accepted taste would be rejected. Although the hypothalamus or forebrain may be instrumental in controlling ingestion, the data suggest that aspects of this control may also be represented at other, more caudal levels of the mammalian brain.

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10. Decerebrates have the same blood glucose concentrations (R. J. Di Rocco and H. J. Grill, in preparation), stomach emptying times (H. J. Grill, unpublished observations), and rate of weight gain as pair-fed controls (*9*).
11. Decerebrates were fed by gavage three 12-ml meals daily. The diet consisted of equal parts of sweetened condensed milk and water. Rectal temperatures were recorded three to five times daily; hyperthermia was treated by wetting the fur with water and, in extreme cases, by exposing the wet animal to a fan to facilitate evaporative cooling.
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14. Decerebrate rats survived 53, 66, 67, 107, and 115 days after complete transection. Full surgical control rats were subjected to all aspects of the two-stage decerebration procedure except for the actual lowering of the spatula into the brain.
15. The taste stimulus, 0.1*M* NaCl (four rats) and 0.03*M* HCl (three rats) injected intraorally in 50-μl volumes, elicited an ingestion sequence before LiCl pairing. The NaCl and HCl (rather than sucrose) were used as paired taste stimuli to eliminate the possibility that daily tube-feedings of a sweetened milk diet might serve as extinction trials for a sucrose stimulus. Taste and LiCl (0.15*M*, 1.5 meq per kilogram of body weight, intraperitoneally) were paired once on each of four consecutive days ending 10 days before complete transection; LiCl was injected immediately after the presentation of the taste stimulus. The same taste stimuli were used for pairings before and after transection. The retention test took place on day 9 after the complete transection, and retention with exogenous arousal (tail pinch during and immediately before the test) on day 11. Acquisition and acquisition with exogenous arousal were each examined during four consecutive days of taste-LiCl pairings. The acquisition experiment began on day 15, and acquisition with exogenous arousal [intraperitoneal injections of *d*-amphetamine sulfate (0.5 mg/kg) given 1/2 hour before testing] on day 55. All responses were videotaped.
Several studies have suggested that behavioral deficits accompanying neurological damage are not necessarily explained by the loss of the behavior's neural substrate. A more general process, reduction of a tonic activation system, may obscure interpretation. In certain instances, exogenous arousing stimuli have uncovered behavioral capacities that were presumed lost as a result of destruction of their neural substrates [D. L. Wolgin, J. Cytawa, P. Teitelbaum, in *Hunger: Basic Mechanisms and Clinical Implications*, D. Novin, W. Wyrwicka, G. Bray, Eds. (Raven, New York, 1976), p. 179; C. H. Beck and W. W. Chambers, *J. Comp. Physiol. Psychol.* **70**, 1 (1970); J. F. Marshall, D. Levitan, E. M. Stricker, *ibid.* **90**, 536 (1976); P. M. Meyer, J. A. Horel, D. R. Meyer, *ibid.* **56**, 402 (1963)].
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19. The distilled-water control experiment began on day 79; two decerebrates and both controls were subjects. Average water consumption (in milliliters) for control and decerebrates, respectively, was 2.68 ml (standard error, 1.12), 0.63 (0.08) when sated; 2.75 (1.90), 0.85 (0.25) when deprived for 24 hours; and 2.70 (2.10), 0.70 (0.15) during 48-hour deprivation.
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Ponto-Geniculo-Occipital (PGO) Burst Neurons: Correlative Evidence for Neuronal Generators of PGO Waves

Abstract. *A newly discovered class of neurons, ponto-geniculo-occipital (PGO) burst neurons, has PGO wave relationships of phase-leading, stereotyped discharge bursts, and the highest reported discharge specificity and coherence; these neurons thus fulfill correlative criteria for output generator neurons for PGO waves. The PGO burst neurons are recorded in a discrete dorsal brainstem area in apposition to the brachium conjunctivum.*

Ponto-geniculo-occipital (PGO) waves are electroencephalographic spikes recorded in pons, lateral geniculate nuclei (LGN), and occipital cortex just before and during desynchronized sleep episodes. Because of their possible role in information generation and transmission from brainstem to forebrain sites in a number of behavioral and developmental conditions, PGO waves have attracted the interest of workers in sleep physiology, visual system function, pharmacology, and developmental plasticity (*1*). Studies employing lesion, cooling, and macropotential recording techniques have outlined the projection pathways of PGO waves from pons to LGN and to visual cortex (*2*), thus opening the way for investigations at the cellular level to provide critical information on the localization and function of neurons involved in PGO wave generation and transmission.

As a first step in defining the neuronal network involved in the chain of events leading to PGO wave generation, it is important to identify the set of neurons forming the last link of this chain, that is, to identify a set of output neurons for PGO wave generation. Once such neurons have been identified one can work backward in the PGO generation network, tracing the inputs to these final stage or output cells. We reason that criteria for identification of such output generator cells for PGO waves should in-

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