Electromyographic Activity in the Masseter Muscle Resulting From Stimulation of Hypothalamic Behavioral Sites in the Cat

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This study examined the relationship between hypothalamically elicited emotional behaviors and electromyographic activity in the masseter muscle of the cat. Electromyographic amplitudes resulting from stimulation at hypothalamic sites at which affective defense and quiet biting attack behaviors were elicited were compared with those recorded during stick biting that simulated mastication. The electromyographic activity elicited by hypothalamic stimulation was greater than that found from stick biting for all behavioral sites. At control sites, from which no behavior could be elicited, the electromyographic activity associated with stick biting exceeded that observed from the hypothalamic sites. These findings suggest a relationship between hypothalamically elicited behaviors and increased levels of jaw muscle activity.
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There is significant evidence that muscle hyperactivity and emotional stress are important cofactors in the initiation and maintenance of temporomandibular joint disorders (TMD) for specific groups of patients.[1] Such evidence includes studies of populations diagnosed with TMD that have reported increased levels of electromyographic (EMG) activity at rest,[2,3] during sleep,[4,5] and in the performance of stressful activities during the day.[6,7] Observations by Clark and Rugh,[8] Rugh,[9] and Leifer[10] have suggested that the emotional stress connected with lifestyle changes, such as death of a spouse, family conflict, and job problems, may have a significant association with both TMD and increased levels of jaw muscle activity. Jaw muscle hyperactivity can be produced experimentally by placing subjects in planned stressful environments.[11,12] These observations suggest that regions of the central nervous system mediating the expression of emotional behavior may be associated with the muscle hyperactivity that often appears to accompany TMD.

Support for this hypothesis is derived from experiments showing that electrical stimulation of the hypothalamus and amygdala facilitate the jaw closing reflex and cyclic jaw responses in the cat.[13,14] The hypothalamus and the amygdala are capable of modulating jaw muscle activity[15–17] by virtue of their connections to the motor nuclei of nerve V and adjoining regions.[18,19]

Landgren and Olsen,[13] who observed facilitation of the jaw closing reflex during electrical stimulation of the lateral hypothalamus in the cat, suggest that facilitation of this reflex may serve as a mechanism underlying bruxing behavior. These findings are of par-

ticular interest in that the hypothalamus and associated limbic structures have also been identified as critical for the expression of emotions such as rage and anger.

Over the past 25 years, a number of animal studies have examined the role of the hypothalamus in the regulation of emotional behavior.[20] In the cat, stereotyped forms of emotional responses, including "affective defense" and "quiet biting attack," have been employed. Both of these behaviors appear to have significant emotional components.

Affective defense behavior is associated with noticeable affective signs including piloerection, retraction of the ears, arching of the back, marked pupillary dilation, vocalization (hissing), and unsheathing of the claws. This response can be evoked by electrical stimulation of sites located throughout the rostrocaudal extent of the medial preopticohypothalamus and the dorsal aspect of the midbrain periaqueductal gray.[20]

Quiet biting attack is predatory in nature and is characterized by stalking of the prey object (usually an anesthetized rat) followed by the biting of its neck. This behavior can be elicited by electrical stimulation along an anatomic region that includes the lateral and perifornical hypothalamus as well as the ventral aspects of the periaqueductal gray.[20]

Qualitative increases in jaw muscle EMG activity during stimulation of lateral hypothalamic sites in the cat[21,22] and monkey[23] have previously been reported. In these studies, however, the technology was not yet available for signal processing techniques such as rectification, integration, and quantification of the signal[24] for statistical treatment and replication. The baseline measurement used as a reference in these studies was resting EMG activity. No comparison was made between EMG activity during hypothalamic stimulation and that observed during normal masticatory activities. Comparison of EMG amplitudes, frequency responses, and power spectra of hypothalamically elicited behaviors with those obtained from masticatory activity may provide further understanding of the role of the hypothalamus in modulation of jaw muscle activity.

As a preliminary investigation of the effects of hypothalamic modulation of jaw muscle activity, the purpose of this study was to examine the amplitude of the EMG of masseter muscle activity resulting from electrical stimulation of sites associated with hypothalamically elicited affective defense and quiet biting attack behaviors in the cat and compare this data with that obtained during activity that simulates mastication.

Materials and Methods

Twelve adult cats (weight 2.5 to 4.0 kg) of both sexes that did not spontaneously attack rats were employed in this study. They were maintained on ad libitum feeding and drinking schedules for the duration of the experiments. The animals were deeply anesthetized with sodium pentobarbital (intraperitoneally, 45 mg/kg), and six guide tubes (17 ga) were stereotaxically located overlying the medial and lateral hypothalamus and cemented into place according to the stereotaxic coordinates of Jasper and Ajmone-Marsan.[25] Moveable electrodes were subsequently lowered through these guide tubes into the hypothalamus to elicit a behavioral response.

Elicitation of Behavior

Monopolar electrodes, coated to within 0.5 mm of their tips, were incrementally lowered in 0.5-mm steps with electrical stimulation applied (0.2 to 0.6 mA) to identify sites from which either quiet biting attack or affective defense could be elicited. During these procedures, the cat was awake and allowed to move freely in a 70 × 70 × 60-cm wooden observation cage with a clear plastic front. Stimulation was applied with biphasic rectangular pulses (0.2 to 0.6 mA, 62.5 Hz, 1 millisecond per half-cycle duration). Stimuli, generated by two independent stimulators (S-88, Grass Instrument, Quincy, MA) were led through stimulus isolation units to the cat. Pairs of 40K ohm resistors (Grass) in series with the cat approximated constant current conditions. The peak-to-peak current was monitored by an oscilloscope (502A, Tektronix, Beaverton, OR). When a reliable response was elicited from a site, baseline threshold values for the response were determined using the Methods of Limits in which a series of 10 ascending and descending trials were employed. Current levels were raised or lowered in 0.05-mA steps in a counterbalanced A-B-B-A design to avoid order effects. The response threshold for behavior was defined as the current value at which the responses were elicited in 50% of the trials, and a stable threshold value was defined as one that remained constant over a 10-trial period. A current 0.05 mA above threshold was utilized in these experiments. Stimulation currents larger than this would have caused undue fatigue to the animal during the course of the experiments. The electrode was then cemented in place with dental cement. A similar protocol was used to identify behavioral sites from which both quiet biting attack and affective defense behaviors could be elicited.

EMG Recordings

The skin overlying the masseter muscle on both sides of the face was shaved and dried with alcohol. Silver-silver chloride disposable bipolar surface electrodes (Myotronics Research, Seattle, WA) were affixed to the skin (15-mm interelectrode distance) with collodion so that they were overlying the masseter muscle and lying parallel to the orientation of the muscle fibers. A ground electrode was attached to the skull. The EMG signals were recorded and preamplified with a Bioelectric Processor (EM2, Myotronics Research) that rectified and integrated the EMG signals, providing a digital readout of each 1 to 2 seconds of signal record. Recordings were made from the masseter muscle bilaterally.

Experimental Paradigm

During the experiment, the animal was awake and gently restrained in a loose-fitting body bag to prevent it from removing the electrodes attached to its face. The EMG signal was recorded under the following conditions: (1) while biting on a stick unilaterally to simulate mastication; and (2) during ipsilateral stimulation of hypothalamic sites from which either quiet biting attack or affective defense was elicited.

Each experimental session was 45 minutes in length. A series of 10 alternating trials of stick biting and hypothalamic stimulation were made using an A-B-B-A paradigm to avoid order effects. The recordings for each trial extended for a maximum of 20 seconds in length. Only one behavioral site was tested during each experimental session. Trials of electrical stimulation were separated by 2-minute rest periods. The EMG recordings for the 10 trials of each condition were averaged, and mean values and standard deviations were obtained. A one-way ANOVA was used to test for differences in EMG activity between stick biting and electrical stimulation at behavioral sites. Post-hoc comparisons between conditions were made using Scheffe's multiple comparison test.

In addition, a series of control experiments were conducted in which EMG activity recorded during electrical stimulation at sites in the hypothalamus from which no behavior could be elicited were compared with EMG values obtained during stick biting. After determining that no behavior could be elicited from a given site, a standardized current of 0.4 mA was utilized for electrical stimulation.

Following the completion of these series of experiments, each cat was killed by transcardial perfusion with saline followed by buffered saline. The brains were removed and blocked. Serial sections were cut, mounted on glass slides, and stained with cresyl violet to identify the loci of the electrode tips.

Results

Sixteen behavioral sites were identified in the hypothalamus (Table 1). Six of these were sites from which affective defense behavior could be obtained; five sites were located in the ventromedial hypothalamus, and the sixth was situated in the dorsomedial hypothalamus. Quiet biting attack behavior was elicited from 10 different sites. The loci of these sites included the perifornical lateral hypothalamus and the region immediately ventral to the fornix (Fig 1).

Electrical stimulation of affective defense sites in the dorsomedial and ventromedial hypothalamus resulted in significant increases in EMG values. The mean masseteric EMG voltage for all six affective defense sites was 97.9 μV for the side ipsilateral to the hypothalamic site stimulated and 74.0 μV for the contralateral side. These exceeded the mean masseteric EMG values for stick biting, which were 44.2 μV for the ipsilateral side and 29.7 μV for the contralateral side. Analysis of variance showed significant differences between groups ($F = 9.83$, $df = 3,20$; $P < .001$). Post-hoc comparisons further indicated significant differences in masseteric EMG values for conditions of hypothalamic stimulation vs stick biting for both ipsilateral and contralateral sides (Fig 2).

Electrical stimulation of quiet biting attack sites in the perifornical and lateral hypothalamus resulted in increased EMG values. The mean masseteric EMG voltage for all quiet biting attack behavioral sites was 74.3 μV for the side ipsilateral to the hypothalamic site stimulated and 46.7 μV for the contralateral side. These exceeded the mean masseteric EMG values for stick biting, which were 28.6 μV for the ipsilateral side and 16.9 μV for the contralateral side. Analysis of variance showed significant differences between groups ($F = 8.73$, $df = 3,40$; $P < .0001$). Post-hoc comparisons further indicated significant differences in masseteric EMG values for conditions of hypothalamic stimulation vs stick biting for both ipsilateral and contralateral sides (Fig 3).

Electrical stimulation of control sites from which no behavior could be obtained resulted in masseteric EMG voltages (5.4 μV on the ipsilateral

Table 1 Experimental Data

Experimental site	Cat no.	Stim*	H-EMG (SD)†	SB-EMG (SD)‡	Anatomic site
Affective defense					
1	1548	0.3	157.2 (15.8)	57.6 (11.8)	VMH
2	1542	0.4	55 (15.3)	30 (12.8)	VMH
3	1695	0.2	85.2 (11.9)	35.8 (7.9)	DMH
4	1712	0.2	53.8 (8.6)	46.2 (7.2)	VMH
5	39347	0.4	60.8 (17.1)	41.2 (9.4)	VMH
6	39347	0.2	175.2 (10.1)	54.6 (6.3)	VMH
Quiet biting attack					
1	1593	0.1	62.2 (9.4)	12.2 (2.6)	PFH
2	1593	0.5	183.8 (6.14)	77.2 (12.3)	LH
3	1597	0.1	63.4 (29.1)	17.4 (3.6)	PFH
4	1593	0.2	34 (5)	11.4 (8.3)	PFH
5	1554	0.4	74 (16.4)	27 (17.1)	LH
6	1701	0.55	29.8 (6.8)	21.6 (13)	PFH
7	1701	0.4	48.8 (3.6)	15 (4.8)	LH
8	8410E2	0.2	76.5 (2.7)	18.4 (5.5)	LH
9	1712	0.4	72.4 (9.1)	22.8 (6.9)	LH
10	39350	0.3	54 (9.5)	40.4 (12.7)	LH
Controls					
1	1542	0.4	5.7 (2.3)	19.2 (4.1)	AH
2	1554	0.4	6.3 (2.0)	23.7 (4.1)	MH
3	1548	0.4	8.2 (2.1)	52.6 (6.2)	LH
4	1701	0.4	3.1 (2.4)	35 (4.2)	LH

*Electrical current used to elicit hypothalamic behavioral response (mA).
†Mean voltages recorded from ipsilateral masseter muscle following hypothalamic stimulation (μV).
‡Mean voltages recorded from ipsilateral masseter muscle resulting from stick biting (μV).

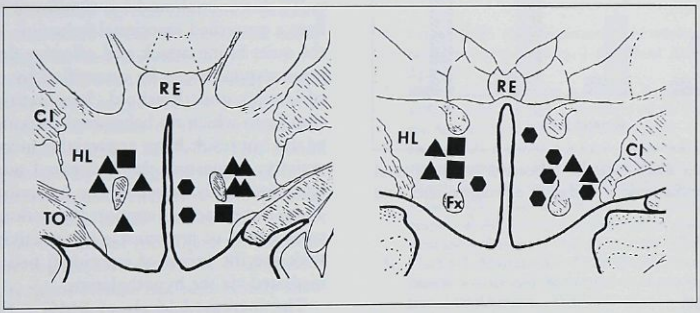

Fig 1 Distribution of electrically stimulated hypothalamic sites (▲ = quiet biting attack sites, ● = affective defense sites, ■ = nonbehavioral sites; CI = internal capsule, HL = lateral hypothalamus, RE = nucleus reuniens, TO = optic tract).

side, 4.3 μV on the contralateral side) that were significantly lower than those observed during stick biting (31.4 μV for the ipsilateral side, 24.6 μV for the contralateral side). This was in contradistinction to both quiet biting attack and affective defense sites where, as previously noted, electrical stimulation resulted in elevated masseteric EMG values. Analysis of variance showed significant differences among groups ($F = 12.19$, $df = 3,12$; $P < .001$). Post-hoc comparisons showed significant differences in masseteric EMG values for conditions of stick biting vs control "nonbehavioral" hypothalamic stimulation for both ipsilateral and contralateral sides (Fig 4).

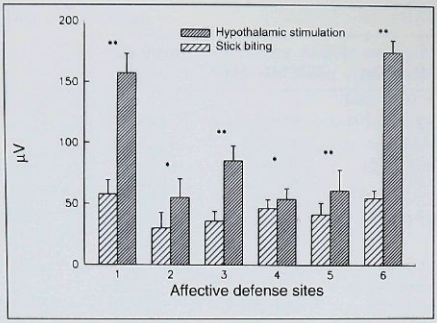

Fig 2 Mean EMG voltages recorded from 10-trial series of hypothalamic stimulation of affective defense sites and stick biting. Note that at all sites the EMG values associated with hypothalamic stimulation reached or exceeded that obtained from stick biting behavior (* = $P < .05$, ** = $P < .01$ for all Figs).

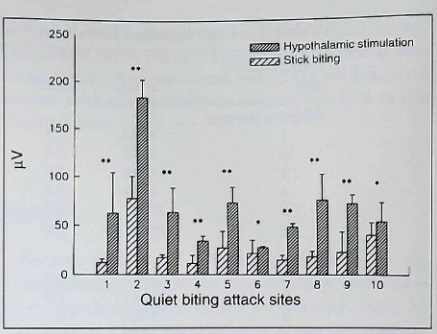

Fig 3 Mean EMG voltages recorded from 10-trial series of hypothalamic stimulation of quiet biting sites and stick biting.

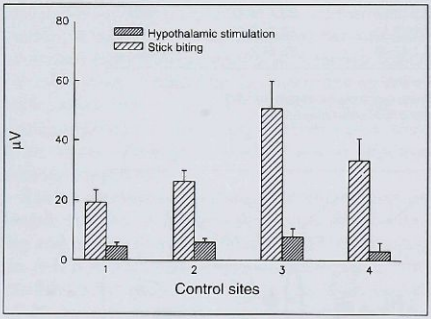

Fig 4 Mean EMG voltage recorded from 10-trial series of hypothalamic stimulation of control sites and stick biting.

Discussion

The purpose of this study was to examine EMG activity in the masseter muscle that was elicited by electrical stimulation of hypothalamic sites associated with stereotyped forms of emotional behavior. In particular, the muscle activity associated with emotional behavior was compared to that observed during activity that simulated mastication. Muscle activity resulting from hypothalamic stimulation exceeded that observed during simulation mastication activity that was elicited by gently inserting a stick into the animal's mouth. The results of this study therefore demonstrate that significant jaw muscle activity can be evoked by stimulation of regions of the hypothalamus associated with specific forms of emotional behavior, ie, quiet biting attack and affective defense. While there are several studies that have shown that stimulation of regions of the cortex and pyramidal tracts can modify masseteric reflexes and jaw muscle activity,[26,27] this animal model is significant in that it utilizes a patterned emotional behavior. Furthermore, the quiet biting attack and affective defense behavioral responses can be quantified by measurement of latency and threshold. Stimulation of control sites from which no behavioral response was elicited did not result in an appreciable increase in EMG activity, suggesting that increased muscle activity was specifically linked to the behavioral response. Thus, these findings support the notion that at least some forms of jaw muscle hyperactivity associated with specific forms of emotional behavior may be mediated via the hypothalamus.

The increased levels of EMG activity suggest that hypothalamic stimulation produces a more widespread and continuous activation of motoneurons that may result in a more rapid fatigue of the muscle. In support of this notion, preliminary analysis of the mean power spectrum shifts[28] during electrical stimulation at hypothalamic behavioral sites observed with this animal model indicates that there is a marked upward shift in the mean power frequency, suggestive of an increase in motor neuron-firing frequency and likely a more extensive recruitment of the motor neuron pool that includes a greater percentage of rapidly fatiguing fibers.

Furthermore, the results of this study support the possibility that muscle hyperactivity can be mediated by the central nervous system with little influence from peripheral receptors in the periodontal ligament surrounding the teeth. While it is true that during quiet biting attack the animal closes its jaws together and presumably has tooth contact that could modulate jaw muscle activity, this is not so in the case of affective defense. During this behavior, the animal hisses with the mouth open wide and the teeth separated. It is therefore possible that jaw muscle hyperactivity may be centrally initiated or mediated independently of periodontal ligament mechanoreceptors.

Early studies of TMD populations identified bruxing as an important etiology of this condition.[29] Further investigations, however, revealed that associated with and, perhaps, underlying bruxing behavior is hyperactivity of some or all of the jaw musculature, especially the masseter, temporalis, and lateral pterygoid muscles.[30] Human studies have shown that there exists a strong correlation between emotional stressors and jaw muscle hyperactivity. Rugh and Solberg[31] and Lindqvist[32] note that increased levels of stress and anxiety can be correlated with increased jaw muscle activity among TMD patients. While these studies do not establish a causal relationship between emotional behaviors and jaw muscle hyperactivity, it is generally agreed that both bruxing behavior and emotional stress are important cofactors in the etiology of these disorders.

The relationship between bruxing behavior and emotional stress was suggested by Rugh,[9] who showed that stressful events of the previous day are correlated with bruxing behavior. This notion is further supported by findings of increased levels of urinary catecholamine excretion in a bruxing population.[33,34] These levels presumably reflect increased levels of adrenal medullary secretion of adrenaline into the circulatory system. Endogenous medullary adrenaline release has been closely associated with stress-related responses.[35]

Parallel findings with regard to catecholamine release have been noted from experimental studies utilizing hypothalamically elicited emotional behaviors in the cat.[36] In particular, electrical stimulation of affective defense sites resulted in significant increases in plasma catecholamine levels within 1 to 4 minutes following electrical stimulation. These observations are significant in that they show a further similarity between the animal model of jaw muscle hyperactivity utilized in the present study and human subjects who demonstrate bruxing behavior. Clinical studies of depression, which is associated with a subclass of TMD patients,[37] have identified a diffuse activation of the sympathetic nervous system that was mediated by a corticotropin-releasing factor from the ventromedial nucleus of the hypothalamus, which is the region in the cat from which affective defense can be elicited.[38,39]

In this preliminary study, stick biting was the measure of "nonemotional" activity that simulated mastication. However, it would be useful if future studies were to employ a bite-force measurement apparatus that would permit quantification of the level and rate of masticatory activity. Further research should involve a patterned cyclic stimulation protocol to more closely parallel the in vivo TMD model in which the subject is under periodic emotional stress. Quantification of the EMG data utilized in this study also provides the intriguing possibility for comparing EMG recordings from humans diagnosed with TMD with an animal model of jaw muscle hyperactivity induced by stimulation at stereotyped behavioral sites within the hypothalamus. Such comparisons, however, will require careful analysis, since there are differences in fiber type composition[40,41] that may confound interpretation of such data.

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Resumen

Actividad Electromiográfica Masetérica Resultante del Estímulo Hipotalámico de los Sitios de Comportamiento en el Gato

En este estudio se utilizaron animales para examinar la relación entre los comportamientos emocionales evocados por el hipotálamo, y la actividad electromiográfica en el músculo masetero del gato. Las amplitudes electromiográficas resultantes del estímulo de los sitios en el hipotálamo de donde se obtuvieron respuestas de defensa afectiva y ataques caracterizados por mordeduras discretas, fueron comparadas a aquellas registradas cuando los animales mordían palos, lo cual simulaba la masticación. La actividad electromiográfica evocada por el estímulo hipotalámico fué mayor que la evocada al morder el palo, en todos los sitios de comportamiento. En los sitios de control, de los cuales no se pudo evocar ningún comportamiento, la actividad electromiográfica asociada con la mordedura de los palos excedió aquella proveniente de los sitios en el hipotálamo. Estos hallazgos indican que existe una relación entre los comportamientos evocados por medio del hipotálamo y los niveles elevados de actividad muscular en la mandíbula.

Zusammenfassung

Elektromyographische Aktivität im Masseter der Katze durch Reizung hypothalamischer Verhaltensregionen

Diese Studie untersuchte die Beziehung zwischen hypothalamisch evozierten emotionalen Verhaltensweisen und elektromyographischer Aktivität bei der Katze. Elektromyographische Amplituden aus der Stimulation von hypothalamischen Regionen, die die Verhaltensweisen der affektiven Verteidigung und der "quiet biting attack" evozieren, wurden verglichen mit denjenigen des Beissens auf einen Stab. Die elektromyographische Aktivität aus der hypothalamischen Stimulation war durchwegs grösser als diejenige aus dem Beissen auf den Stab. Kontrollstellen des Hypothalamus, die nichts mit typischen Verhaltensweisen zu tun haben, verursachten sogar kleinere elektromyographische Aktivität als das Beissen auf einen Stab. Diese Befunde weisen darauf hin, dass eine Beziehung besteht zwischen hypothalamisch evozierten Verhaltensweisen und vermehrter Kaumuskelaktivität.