Hypothalamic and Cortical Control of Jaw Reflexes

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HYPOTHALAMIC AND CORTICAL CONTROL OF JAW REFLEXES

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Avhandlingen baseras på följande rapporter:


The subject of the thesis is a study of the projections from low threshold oral and face afferents to the cerebral cortex and of descending motor control mechanisms originating in the cerebral cortex or the hypothalamus and influencing the jaw reflexes.

Cats anaesthetized with chloralose were used for the experiments. Ipsilateral and contralateral nerves from the oral cavity and the face were stimulated electrically. Cortical potentials were averaged and recorded. The location of the projections was related to the cytoarchitectonic areas of the cerebral cortex. It was found that the afferents projected to separate maximum points in areas 3a, 3b, 5a and 6aβ. The projections to areas 3a and 3b were somatotopically organized, but the layout of the projections on the cortex was not facelike.

The effect of monopolar anodal stimulation of the cerebral cortex on the monosynaptic jaw closing and the disynaptic jaw opening reflexes was investigated. A sequence of facilitation and inhibition of both reflexes was elicited by cortical stimulation. The effects were of short latency (2.5 ms) and could start with either facilitation or inhibition. The timecourse of the sequence was sinusoidal with a period of 10 ms. The largest effect originated in the "sensory" areas 3a and 3b and not in the "motor" areas 4γ and 6aβ. It is suggested, that a trigemino-cortico-trigeminal loop via area 3a may function in reflex modulation of jaw movements.

The hypothalamic effects on the jaw reflexes were evoked by electrical stimulation in those parts of the hypothalamus, which are known to generate defence, attack or feeding responses. A tenfold facilitation of the jaw closing reflex and a facilitation followed by almost complete inhibition of the jaw opening reflex were observed in the anaesthetized animal with intact cerebral cortex. The effects remained but were diminished in amplitude after cortical ablation. The descending path was located in the ventral midbrain tegmentum.

It is suggested that the observed hypothalamo-trigeminal mechanism may exercise a tonic influence on the trigeminal motoneurones, thereby controlling the set points of the biting force and the rest position. The implications of this hypothesis on the etiology of bruxism and the myofascial pain-dysfunction are discussed.


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by

Kurt A. Olsson

UMEA 1979
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The original articles are referred to in the text as Reports I, II and III.
INTRODUCTION

Pathological mechanisms, presented by patients in the clinic, are complex and difficult to analyze. An experimental approach is often necessary in order to reveal causes and developments of events. The functional disturbances of the masticatory muscles offers an example. They form a syndrome with a multifactorial pathogenesis. This has resulted in conflicting theories concerning its etiology and also a variety of therapeutic approaches. Clinical research has specified two diseases within this syndrome. They have been referred to as bruxism and as myofascial pain dysfunction (MPD). Their etiologies have been explained along two principle lines. One of them focuses the interest on peripheral causes. The other points to central nervous mechanisms as the origin.

In the present work we have attempted an experimental analysis of the descending central nervous mechanisms, which control the jaw movements. We have assumed, that psychological factors influence the cerebral cortex and the hypothalamic centre controlling agonistic behaviour. The question was asked whether electrical stimulation of these structures would affect the jaw reflexes. The results revealed indeed very effective descending control mechanisms.

In the analysis of these mechanisms we have attempted to study them in the unanaesthetized freely moving animal as well as in the more rigorously controlled anaesthetized preparation. We have investigated ascending as well as descending components of the systems, and made a point of relating function to morphology.

Our documentation of a powerful facilitation of the jaw closers evoked from the defence attack area of the hypothalamus describes a mechanism,
which may evoke hyperactivity in the masticatory muscles in response to psychic stress (Report I). In this sense the findings support the hypothesis of a central nervous mechanism in bruxism and MPD. The clinical aspects of the findings are commented upon in the discussion of the Thesis.

The specific problems, analysed in the series of experiments, are presented in the beginning of chapters I - III, which summarize the results of the original reports. The organization of the oral projections to the cerebral cortex was investigated in Report II. Maximum points of the primary projections were localized and related to the cytoarchitectonic areas of the cortex. The origin of the cortical effects on the jaw reflexes was studied in Report III. The relevance of our findings to the sensory and motor mechanisms are discussed in the original reports, and will not be repeated in the discussion of the Thesis.

The present results shows that the functions of the jaw muscles are controlled by very effective CNS mechanisms and that large areas on the cerebral cortex receive oral projections with a detailed and interesting distribution. The trigeminal system may thus offer special advantages in the analysis of sensory and motor functions.
**LIST OF ABBREVIATIONS**

**Anatomy of brain stem nuclei**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
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<tbody>
<tr>
<td><strong>Thalamus</strong></td>
<td></td>
</tr>
<tr>
<td>AM</td>
<td>N anterior medialis</td>
</tr>
<tr>
<td>CI</td>
<td>Capsula interna</td>
</tr>
<tr>
<td>Cd</td>
<td>N caudatus</td>
</tr>
<tr>
<td>GP</td>
<td>Globus pallidus</td>
</tr>
<tr>
<td>Put</td>
<td>Putamen</td>
</tr>
<tr>
<td>RE</td>
<td>N reuniens</td>
</tr>
<tr>
<td>S</td>
<td>Stria medullaris</td>
</tr>
<tr>
<td>VA</td>
<td>N ventralis anterior</td>
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</tbody>
</table>

| **Hypothalamus** |           |
| Aa             | Area amygdaloidea anterior |
| Abm            | N amygdaloideus basalis |
| Acl            | N amygdaloideus centralis |
| Al             | N amygdaloideus lateralis |
| Al             | Ansa lenticularis |
| Am             | N amygdaloideus medialis |
| CA             | commissura anterior |
| Fx             | fornix |
| TO             | tractus opticus |

<p>| <strong>Nervi trigemini</strong> |           |
| V                 | n trigemini, tractus trigemini |
| NVmes             | N nervi trigemini mesencephalicus |
| NVmt              | N motorius n trigemini |
| NVmt dig          | N motorius n trigemini, subnucleus digastricus |
| NVmt mass         | N motorius n trigemini, subnucleus massetericus |
| NVspo             | N tractus spinalis trigemini oralis |
| NVsnpr            | N sensorius principalis n trigemini |
| NsV               | N supratrigeminalis |</p>
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<thead>
<tr>
<th>Dissected nerves</th>
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<tbody>
<tr>
<td>Alv inf</td>
<td>n alveolaris inferior</td>
</tr>
<tr>
<td>Alv sup</td>
<td>n alveolaris superior</td>
</tr>
<tr>
<td>Aur</td>
<td>n auricularis major</td>
</tr>
<tr>
<td>Ling</td>
<td>n lingualis</td>
</tr>
<tr>
<td>Mass</td>
<td>n massetericus</td>
</tr>
<tr>
<td>Ment</td>
<td>n mentalis</td>
</tr>
<tr>
<td>Mx nose</td>
<td>n infraorbitalis, branch to nose</td>
</tr>
<tr>
<td>Mx w</td>
<td>n infraorbitalis, branch to whiskers and lip</td>
</tr>
<tr>
<td>Ophth</td>
<td>n ophthalmicus</td>
</tr>
<tr>
<td>SR</td>
<td>n radialis superior</td>
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<tr>
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<tr>
<td>COR</td>
<td>G coronalis</td>
</tr>
<tr>
<td>ESYL</td>
<td>G ectosylvius</td>
</tr>
<tr>
<td>ORB</td>
<td>G orbitalis</td>
</tr>
<tr>
<td>PRO</td>
<td>G proreus</td>
</tr>
<tr>
<td>PRSYL</td>
<td>G presylvius</td>
</tr>
<tr>
<td>SIG A</td>
<td>G sigmoideus anterior</td>
</tr>
<tr>
<td>SIG L</td>
<td>G sigmoideus lateralis</td>
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<tr>
<td>SIG P</td>
<td>G sigmoideus posterior</td>
</tr>
<tr>
<td>SSYL</td>
<td>G suprasylvius</td>
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<tr>
<td>SYL</td>
<td>G sylvius</td>
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<tr>
<td>cor</td>
<td>S coronalis</td>
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<td>lat</td>
<td>S lateralis</td>
</tr>
<tr>
<td>orb</td>
<td>S orbitalis</td>
</tr>
<tr>
<td>pcd</td>
<td>post cruciate dimple</td>
</tr>
<tr>
<td>prsyl</td>
<td>S presylvius</td>
</tr>
<tr>
<td>rhin</td>
<td>S rhinalis anterior</td>
</tr>
<tr>
<td>syl</td>
<td>S sylvius</td>
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</table>
### Other abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tbody>
<tr>
<td>AD</td>
<td>antidromic</td>
</tr>
<tr>
<td>co</td>
<td>contralateral</td>
</tr>
<tr>
<td>CNS</td>
<td>central nervous system</td>
</tr>
<tr>
<td>CSR</td>
<td>caudal superficial radial projection</td>
</tr>
<tr>
<td>Cx</td>
<td>cortex cerebri</td>
</tr>
<tr>
<td>H</td>
<td>horizontal</td>
</tr>
<tr>
<td>H-C</td>
<td>Horsley-Clarke</td>
</tr>
<tr>
<td>ip</td>
<td>ipsilateral</td>
</tr>
<tr>
<td>L</td>
<td>lateral</td>
</tr>
<tr>
<td>OD</td>
<td>orthodromic</td>
</tr>
<tr>
<td>Pt</td>
<td>pyramidal tract</td>
</tr>
<tr>
<td>RSR</td>
<td>rostral superficial radial projection</td>
</tr>
<tr>
<td>SIIf</td>
<td>SII forelimb area</td>
</tr>
</tbody>
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GENERAL METHODOLOGICAL CONSIDERATIONS

A. GENERAL PROCEDURES

The experiments were performed on cats. Chloralose anaesthesia was used in acute experiments. In a few chronic experiments (Report I) the animals were anaesthetized with an inhalation anaesthetic (Fluothane, ICI). Further details concerning anaesthesia, premedication, surgical procedures and postoperative care are given in the reports.

The animals were mounted in a stereotaxic apparatus. Microelectrodes and stimulating electrode grids were carried in manipulators moving in the Horsley-Clark (H-C) system. The accuracy of the movements of the micromanipulators and the reliability of the correlation between electrophysiological and morphological data was analysed and discussed by Landgren and Olsson (1976).

The amplification and recording technique was described by Landgren and Silfvenius (1969). Averaging was used when recording the evoked potentials. A preset number of sweeps (20 - 40) were sampled by an average computer (Intertechnique, Didac 800, Physioscope). The averaged potentials were displayed on the oscilloscope and photographed.

B. PRIMARY PROJECTIONS PROJECTION FIELDS AND MAXIMUM POINTS

The present investigation is mainly concerned with primary projections to the cerebral cortex. These projections are subserved by afferent paths with a minimum of synaptic delay. They consist of three neurones: the primary afferent and the two relay cells at the trigeminal and the thalamic level. The latency of the cortical potentials evoked by stimulation of a primary
projection path is short i.e. about 3 ms in our preparations. Due to the relative simplicity of the path, electrical stimulation evokes a synchronous volley, which gives rise to a presynaptic thalamo-cortical spike potential in the specific cortical afferents. These afferents terminate in layer IV of the cerebral cortex, where they excite interneurones, which form excitatory synapses on the trunks of the apikal dendrites of pyramidal cells. The depolarization of the pyramidal cells evokes a characteristic potential field positive at the cortical surface and reversing to a negative wave in the deeper cortical layers. The presynaptic spike followed by a reversing evoked potential is typical for the primary projection.

A projection field refers, unless otherwise stated, to the cortical extent of a primary projection. Within this field stimulation of the primary afferents evokes a positive surface potential of shortest latency. The position on the cortex, where this evoked potential was of maximal amplitude, was localized and referred to as the maximum point. The border of the projection field was located at a distance from the maximum point, where the amplitude of the potential had decreased to 20%.

The maximal amplitude of the potential was not necessarily confined within a point. The maximum point thus could have a bandlike extent. The evoked potentials of shortest latency were always of large amplitude. Shortest latency and maximal amplitude were, however, not always observed in the same but in nearby points.

Electrical stimulation of peripheral nerves was used to activate afferent fibres in the present studies. This method has the advantage of exciting the afferent fibres synchronously, and an accurate timing of evoked potentials is achieved. The method may be used as a first approximation in defining central actions evoked by afferents from different types of receptors as discussed
by Silfvenius (1972). An advantage is that the method allows the collection of enough data from each animal to give a coherent picture of the projections, if many inputs are used in the same experiment (cf. Report II).

Stimulus strength and threshold of evoked potentials are given in multiples of the strength required to evoke a threshold cortical response (T), which was also the threshold for the afferent volley in the trigeminal root.

C. CONDITIONING-TEST EXPERIMENTS

The effect of electrical stimulation in the hypothalamus (Report I) or the cerebral cortex (Report III) on the trigeminal motoneurones were studied in conditioning-test experiments. The monosynaptic jaw closing reflex and the disynaptic jaw opening reflex were used as tests of the evoked effects.

Test reflexes

The physiology of the jaw reflexes contributes an important part of basal knowledge, which is a prerequisite of this study. Due to technical difficulties, met within the trigeminal experiments, the analysis of the trigeminal reflexes has lagged behind that of the spinal cord segment. The pattern of the jaw reflexes has therefore often been copied on the reciprocal segmental stretch reflex. Recent observations (for reviews see: Landgren and Olsson, 1977; Dubner et al. 1978) do, however, indicate that there are important differences between spinal and cranial nerve reflexes.

A close analogy exists between the organization of the monosynaptic jaw-closing reflex (Fig. 1 A) and the monosynaptic stretch reflex. The only difference between the segmental and the trigeminal pattern, is the location of the cellsoma of the muscle spindle afferent. The trigeminal muscle spindle
MONOSYNAPTIC JAW CLOSING REFLEX

Fig. 1. Diagrams illustrating the monosynaptic jaw closing reflex and the arrangements for evoking and recording the reflex response.
A. Peripheral and central components of the reflex drawn on cat's cranium and transversal histological sections.
B. Block diagram showing experimental set up for evoking the test reflex by electrical stimulation of the mesencephalic trigeminal tract (NVmes). The response is recorded from the masseteric nerve (Mass).
C. Antidromic (AD) and ortodromic (OD) signals recorded from Mass (top circle). Z-modulated response and integration beam adjusted for integration of the area of the reflex response (lower circle).

afferent has its cell soma in the mesencephalic nucleus of the trigeminal tract i.e. within the central nervous system. The segmental afferent, on the other hand, has its soma in the dorsal root ganglion.

The simplest jaw-opening reflex (Fig. 2 A) differs fundamentally from the segmental stretch reflex. The digastricus muscle lacks muscle spindles and
there is no monosynaptic digastricus reflex evoked by its muscle afferents. The jaw opening reflex is disynaptic and is thus considered to be evoked mainly from proprioceptors and exteroceptors outside the muscles, located in the alveolar processes, oral mucosa and the mandibular and maxillary dermatomes. Mechanical stimulation of a tooth may thus evoke the reflex in the cat. The interneuron of the reflex is suggested to be located in the oral or interpolar subdivisions of the nucleus of the spinal trigeminal tract (Cf. Fig. 2 A).

The afferents, which evoke the disynaptic jaw opening reflex, will also contribute disynaptic inhibition to the motoneurones of the jaw closing muscles. This inhibition is not reciprocal in the sens of the segmental stretch reflex, because muscle afferents are not involved and facilitation of the jaw closers is not accompanied by a disynaptic digastricus inhibition. The disynaptic inhibition of the masseteric motoneurones is further bilaterally distributed, which is at variance with the segmental reflex pattern.

The monosynaptic jaw closing reflex was elicited by electrical stimulation of the mesencephalic trigeminal tract. Single or double, submaximal shocks were delivered through one of three stereotaxically placed needle electrodes insulated to the tip (resistance: 10 - 20 kΩ, H-C coordinates: P 3, L 1.5, 3.0, 4.5, H -2). The reflex response was recorded from the proximal portion of the cut masseteric nerve mounted on bipolar silver wire electrodes (Fig. 1 B).

The disynaptic jaw opening reflex was evoked by graded stimulation of the inferior alveolar nerve through a pair of needle electrodes inserted into the nerve. The reflex was recorded from the anterior digastric nerve (Fig. 2 B).
In the first series of experiments (Report I) the peak amplitude of the conditioned reflex responses, expressed as percentage of the mean amplitude of the unconditioned test reflex, was measured and plotted as a function of the time interval between the first conditioning shock and the shock artefact of the test reflex. However, in later experiments (Report III) the recording method was further elaborated to get a more reliable measure of the digastric

DISYNAPTIC JAW OPENING REFLEX

Fig. 2. Diagrams illustrating the disynaptic jaw opening reflex and the arrangements for evoking and recording the response.
A. Cf. Fig. 1 A.
B. Block diagram giving experimental set up for evoking the test reflex by stimulation of the inferior alveolar nerve (Alv inf). Response recorded from the digastric nerve (Dig).
C. Test reflex response and integration signal. (top circle). Conditioned reflex response (lower circle). Magnitude of integrated area shown by a dot at the end of the record.
reflex response. A true integrator (MOS-FET operational amplifier) switched by reed relays (acc ± 0.5 ms) was then used for the plotting of complete conditioning test curves on single frames of film. The reflex response was Z-modulated and its area integrated (cf. Fig. 1 C and Fig. 2 C). The amplitude of the reflex in percent of the test was plotted on the ordinate, and the intervals between the first conditioning shock and the moment, when the test was calculated to reach the trigeminal motoneurones, were plotted on the abscissa.

Conditioning stimulation of the test reflexes was delivered to the ipsilateral cerebral cortex, to the hypothalamus and to the pyramidal tract. The arrangements of the set up of the conditioning test experiments are shown in Fig. 3.

Monopolar conditioning stimulation was applied to the surface of the cerebral cortex through a springmounted and chlorided silver ball electrode (diameter 0.5 mm). The electrode was made anodal or cathodal with reference to a chlorided silver plate which was enclosed in gauze and sewn to denervated skin area on the left side of the pool. An insulating sheet of plastic was inserted between the plate and the temporal muscle. In a few experiments (Report I) bipolar cortical stimulation was used as well. Glass coated platinumiridium needle electrodes (tip resistance 75 - 100 kΩ) were used for intracortical stimulation. The electrodes were mounted to penetrate perpendicular to the cortical surface at the stimulated point. They were made anodal or cathodal with reference either to the silver plate or to a chlorided silver ring (diameter: 5 or 8 mm) touching the cortical surface. The stimulating electrode penetrated in the centre of the ring. The conditioning stimulation in the hypothalamus was delivered through a grid of five glass coated PtIr electrodes (tip resistance: 15 - 50 kΩ). The electrodes were mounted in a row along a female connector with an inter-
Fig. 3. Diagram showing the experimental set up of the conditioning-test experiments. Test reflexes: cf. Figs. 1 B and 2 B. Conditioning stimulation of ipsilateral cerebral cortex (Cx) and hypothalamus.

electrode distance of 1.5 mm. The grid was introduced into the hypothalamus at coordinates selected according to the atlas of Ajmone-Marsan (1960). The five electrodes penetrated in the transversal plane at the Horsley-Clarke coordinates: Fr 11.5 - 13.5, L 0 - 8.0. Horizontal coordinates H 0 to -6 were investigated.

A grid of two pairs of tungsten electrodes, insulated to the tip, was used for stimulation in the pyramidal tract. The distance between electrodes was 1.5 mm and between pairs 2 mm. The rostral pair was adjusted to H-C coordinate P 3. The grid was introduced from the left side of the animal
at an angle of $20^\circ$ to the sagittal plane.

D. HISTOLOGICAL INVESTIGATIONS

The histological investigations played an essential role in this study, since it was necessary to correlate electrophysiological data with recording and stimulation positions in most of the experiments.

Maximum points of the somatosensory primary projections and the extent of the projection fields were thus correlated to the cytoarchitectonic areas as described in detail in Report II. The cortical origin of the effects on the jaw reflexes was also located and related to the somatosensory projections and to the cytoarchitecture (Report III). The effective electrode positions in the hypothalamus, the extent of lesions in the pyramids and mesencephalon and the extent of the cortical ablations were verified (Report I).

Information concerning details in the histological investigations are given in the individual Reports. Only some general procedures are mentioned here.

The brains were perfused with saline followed by fixation with a 4% buffered formaldehyd solution slightly modified after Holt and Hicks (cf. Grant et al., 1957). For orientation, and in order to facilitate the adjustment of the plane of sectioning, parallel india ink tracks, 2 mm apart were made rostral to the stimulating electrode grids. Maximum points of the projection fields were marked with india ink punctures. The appropriate parts were generally blocked in the stereotaxic apparatus. The parts of the hemispheres were, however, blocked in different planes (cf. Fig. 1, Report II). The blocks were localized on photographs. Cortical and hypothalamic blocks
were embedded in ceduol (Merk) and serial sections of 30 μm were prepared. Serial sections of 20 μm were cut on a freezing microtome from the blocks with the pyramid tracks and lesions.

India ink punctures (i.e. maximum points), cytoarchitectonic borders and electrode positions were determined in sections stained with toluidine blue. Sections showing the lesions were stained with Lyxol fast blue (Klüver and Barrera, 1953).

Cytoarchitecture was classified according to criteria given by Hassler and Muhs-Clement (1964).
THE EFFECT OF ELECTRICAL STIMULATION IN THE HYPOTHALAMUS ON THE MONOSYNAPTIC JAW CLOSING AND THE DISYNAPTIC JAW OPENING REFLEXES IN THE CAT
THE EFFECT OF ELECTRICAL STIMULATION IN THE HYPOTHALAMUS ON THE MONOSYNAPTIC JAW CLOSING AND THE DISYNAPTIC JAW OPENING REFLEXES IN THE CAT

This series of experiments were designed to investigate the effects on the jaw reflexes of electrical stimulation in the defence-attack area of the hypothalamus.

A tenfold increase of the monosynaptic jaw closing reflex and a facilitation followed by a complete inhibition of the disynaptic jaw opening reflex was observed in response to the hypothalamic stimulation.

METHODS

The experiments were carried out on 25 cats anaesthetized with halothane or chloralose.

In the first three experiments the effects of electrical stimulation in the hypothalamus on the unanaesthetized freely moving animal were studied. Surgical preparations were done under aseptic conditions and under halothane anaesthesia. A transversal grid of five stimulating electrodes was implanted according to Horsley-Clarke coordinates. The behavioural and electrocardiographic responses to stimulation was observed and recorded as described by Hess (1969). The optimal positions and stimulation parameters for evoking agonistic behaviour including increase in heart rate, attack or flight were noted. Attack responses were evoked from one or more of the medial electrodes in the grid in all three cats. It was realized that the Horsley-Clarke coordinates were reliable guides to the defence-attack area. In the following acute experiments they were then used for localization in combination with the observation of increase in blood pressure and heart rate.
The effect of conditioning stimulation in the hypothalamus was compared with the effect evoked by cortical stimulation in the oral and face projections to the somato-sensory cortex. Cortical conditioning stimuli were applied to the maximum points of the primary projection areas. These areas were defined as described in Report II. The cortical effects on the jaw reflexes are given in detail in Report III.

In four experiments the hypothalamic effects were studied after bilateral ablations of the sensori motor cortex. In another four cats the conditioning stimulation was delivered to the pyramidal tracts at the lower pontine level. The hypothalamic effects after electrolytic lesions of the pyramids and ventral midbrain tegmentum were analysed.

The brains were perfused and prepared for histological analysis. The effective electrode positions in the hypothalamus and in the pyramidal tracts, as well as the extent of the lesions in the pyramids and the ablated cerebral cortex, were verified in histological serial sections.

RESULTS

Electrical stimulation in the defence-attack area of the hypothalamus elicits a complex pattern of behaviour, which is similar to the natural defence and attack reactions in the cat. Both patterns include attention, hissing, piloerection, pupil dilatation, increase in heart frequency and attack or escape movements. These observations were verified in our un-anaesthetized freely moving animals. The typical jaw movements, seen as components of these patterns, were jaw opening occurring simultaneously with the hissing and jaw opening and closing in the biting attack. These electrically evoked jaw movements should be compared with the effects on the jaw reflexes recorded under anaesthesia.
The typical effects of hypothalamic stimulation on the jaw reflexes consisted of a strong facilitation of the monosynaptic jaw closing reflex and a facilitation followed by an inhibition of the jaw opening reflex. Optimal parameters for evoking the effect were, trains of 3 - 10 pulses, 0.5 ms duration, 0.5 mA, 500 Hz. the records in Fig. 4 show the effects obtained in an experiment, in which the rostral parts of both cerebral hemispheres were removed 4 months before the investigation of the effects on the jaw reflexes. The extent of the cortical lesions is shown in Fig. 4 C - F.

![Graphs and diagrams](image-url)

Fig. 4. Effects of ipsilateral hypothalamic conditioning on the monosynaptic jaw closing reflex (A) and the disynaptic jaw opening reflex (B). Diagrams C-F give the extent of the cortical ablation performed four months before experiment A and B. Dens stippling: Cortex removed. Thin stippling: Cortex undercut by lesion of the white matter (c.f. the transversal section D). In diagrams A and B and in the corresponding diagrams in the subsequent figures the mean and SD of the unconditioned test amplitude are indicated by the line drawn in full at 100% and by the broken lines. Conditioning stimuli, A: 9 pulses, 0.5 ms, 500 Hz, 0.2 mA. B: 14 pulses, 0.5 ms, 500 Hz, 0.6 mA.
The effect on the jaw closing reflex, as shown in Fig. 4 A, was a facilitation with a shortest latency of 7 ms, and a mean latency of 13 ms. It was maximal at approximately 30 ms and lasted for about 70 ms. The amplitude of the facilitation was 150 - 200% of the test in the preparation with ablated cerebral cortex.

The hypothalamic facilitation of the monosynaptic jaw closing reflex was much larger in animals with an intact cerebral cortex than in those with the cortex previously removed. Facilitation up to 1300% of the test amplitude was observed in the intact preparations. A train of shocks delivered to the hypothalamus can thus increase the amplitude of the jaw closing reflex more than 10 times that of the control.

The main effect on the jaw opening reflex was inhibition. The inhibitory phase may, however, be preceded by a facilitation giving the time course illustrated in Fig. 4 B. The mean latency of the initial facilitation was 11 ms. The facilitation was cut short by an efficient inhibition with a latency of 30 ms. The duration of the inhibition was of the order of 200 ms whether it occurred with or without initial facilitation. It's amplitude was considerable and could reach 100% i.e. complete inhibition of the test. The amplitude of the initial facilitation reached 200% of the test.

The time course of the hypothalamic effects on the jaw reflexes differed from those evoked by a conditioning train of shocks applied to the cerebral cortex, to the capsula interna or to the pyramidal tract. The cortical effects, described in detail in Report III, started with inhibition or facilitation with a mean latency of 3 ms, as compared to the longer latency of the hypothalamic response. This difference in time course was characteristic and offered a possibility to identify electrode positions in capsula interna already during the experiments.
As mentioned above the hypothalamic effects on the jaw reflexes could be evoked in preparations with the motor and somato-sensory cerebral cortices removed bilaterally. The cortical effects may also be eliminated by a lesion of the pyramids at the lower pontine level. A hypothalamic facilitation of the monosynaptic jaw closing reflex could still be evoked after such a lesion. A lesion mainly restricted to the pyramids thus left the hypothalamic facilitation of the jaw closing reflex in function. However, if the mesencephalic lesion was extended to include also the ventral part of the tegmentum mesencephali the hypothalamic effects were eliminated.

Fig. 5. Diagram showing the location in the hypothalamus of the electrode positions yielding the maximal effects on the jaw reflexes in six experiments. The rectangular frame indicates the area investigated by a grid of five electrodes (arrows) moved in 0.5 mm steps through the hypothalamus. Horsley-Clarke coordinates H and L in mm are given on the frame. Maximal positions from the same experiment are indicated by the same symbols in the center of a ring. The star indicates the location of the electrode position from which a typical affective attack behaviour was evoked in an unanaesthetized cat. All locations were verified on histological serial sections and transferred to a corresponding position on a tracing of one of the sections selected at Fr 12.5. For nomenclature of anatomical structures: see list of abbreviations.
The location of the optimal positions in the hypothalamus for evoking effects on the jaw reflexes was analyzed in six experiments. In these experiments the transversal grid of five stimulating electrodes was moved in steps of 0.5 mm between the horizontal coordinates 0 and -6. The tracks left by the electrodes were located on histological serial sections. The information gained in series of experiments covered the rectangular area shown in Fig. 5 including the coordinates H 0 -6 and L 0 -8. The rostro-caudal distribution of the investigated transversal planes was between Fr 11.5 and Fr 13.5. The locations of the maximal responses are indicated with symbols on the Figure. In five of the six experiments maximal responses were evoked from two or three electrodes. The responsive area thus covered a medio-lateral band from the fornix to the entrance of ansa lenticularis into the lateral hypothalamus. The loci of maximal responses were framed by positions giving smaller or no responses to the standard stimulus.

Further evidence of the medio-lateral location of the hypothalamic effects were obtained from a series of six other experiments, in which the position of the stimulating tracks were localized histologically, but in which only one horizontal coordinate (generally between H -3 and H -4) was investigated. The maximal responses obtained in this series were located within the area covered by the symbols in Fig. 5.

The effects on the jaw reflexes were elicited from those parts of the medial and lateral hypothalamus in which electrical stimulation evoked motivated agonistic and feeding behaviour. This was verified in our experiments on unanaesthetized freely moving cats. A fully developed attack response was thus evoked from a position just ventro-lateral to the fornix. The location is marked with a star in Fig. 5.
CONCLUSIONS

1. Hypothalamic conditioning with 3 - 10 pulses, 0.5 ms duration, 0.5 mA, 500 Hz evoked a strong facilitation of the jaw closing reflex and a facilitation followed by an inhibition of the jaw opening reflex.

2. The effects differed from those elicited from the cerebral cortex. The hypothalamic effects had longer latency (11 - 13 ms) and required a longer train of conditioning stimuli than was the case with those evoked from the cortex.

3. Bilateral ablation of the sensori-motor cerebral cortex or lesion of the pyramids at the lower pontine level diminished but did not abolish the hypothalamic effects. They did, however, disappear after lesions including the ventral midbrain tegmentum.

4. Stimulus positions eliciting the largest hypothalamic effects on the jaw reflexes were located in a region extending medio-laterally from the perifornical area to the entrance of the ansa lenticularis in the lateral hypothalamus. The region corresponds to those parts of the hypothalamus from which agonistic and feeding behaviour have been evoked.
II

LOW THRESHOLD AFFERENT PROJECTIONS FROM THE ORAL CAVITY AND THE FACE TO THE CEREBRAL CORTEX OF THE CAT
LOW THRESHOLD AFFERENT PROJECTIONS FROM THE ORAL CAVITY AND THE FACE TO THE CEREBRAL CORTEX OF THE CAT

The purpose of this investigation was to localize the projections of the low threshold afferents from the oral cavity and the face on the cerebral cortex of the cat, and to correlate their location with the cytoarchitecture of the cortex. The maximum points and the extent of the primary projections were mapped. The borders of the cytoarchitectonic areas were determined on histological serial sections of the individual brains.

Separate projection maxima of the afferent nerves were found in several of the cytoarchitectonic areas. A somatotopical, but not facelike, organization of the projections was observed.

METHODS

The projections to the cerebral cortex were mapped in experiments on 32 adult cats. The following ipsi- and contralateral nerves were dissected and prepared for electrical stimulation.

The inferior alveolar nerve (Alv inf), the mental nerve (Ment), the lingual nerve (Ling), the superior alveolar nerve (Alv sup), the infraorbital nerve: a, branch to whiskers and lip (Mx w); b, branch to nose (Mx nose), the ophthalmic nerve (Opht), the great auricular nerve (Aur), the superficial radial nerve (SR). The expressions ipsilateral (ip) and contralateral (co) are used with reference to the investigated right hemisphere.

Evoked potentials were recorded from the surface of the cerebral cortex and from penetrating microelectrode tracks. Recording positions were marked on photographs of the hemisphere and reference points were indicated with
India ink punctures. Maximum points and borders of the cortical projection fields were determined and mapped as described under General methodological considerations. The results from the individual experiments were summarized on standard diagrams in an attempt to represent the typical features observed.

Cytoarchitectonic borders were determined in histological serial sections stained with toluidine blue. The cytoarchitecture of the cortex was classified according to the criteria given by Hasseler and Muhs-Clement (1964). Cytoarchitectonic borders and maps of the projection fields obtained from the same animal were compared. The diagrams in Fig. 6 summarizes the method used for identifying the cytoarchitectonic areas. The patterns shown in Fig. 6 B attempts to give the typical cytoarchitectonic fields observed in the experiments. The framed area locates the enlarged diagrams which are used when summarizing the different projection fields shown in Fig. 7.

RESULTS

Maximum points of the projections to the cytoarchitectonic areas

Projections from the low threshold afferents (1.6 T) of the oral cavity and the face were found in the presylvian, coronal, anterior suprasylvian, lateral sigmoid, anterior sigmoid, anterior ectosylvian and the orbital gyri of the cat's cerebral cortex.

Primary projections defined by their short latency (3 ms) and typical reversal of the surface positive evoked potential in deep cortical layers were, however, only seen in the anterior suprasylvian, the coronal and the presylvian gyri. The low threshold afferents of the individual nerves showed
Fig. 6. Diagrams summarizing the method used for identifying cytoarchitectonic areas of the frontal part of the right hemisphere.

A. Nomenclature of sulci and gyri according to Hassler and Muhs-Clement (1964).

B. Cytoarchitectonic areas. The boundaries represent typical features observed in the individual experiments (cf. D). The frame locates the enlarged diagrams of Fig. 7.

C. Outline of a serial section (cf. sectioning plane I, section number 182 in D) with classified cytoarchitecture as determined according to the criteria of Hassler and Muhs-Clement (1964). The position of an India ink puncture is given by the arrow.

D. Photograph of the perfused hemisphere from an individual experiment. Borders of the cytoarchitectonic areas (thin lines) are given. Coarse lines (I, II, III) indicate the different planes of sectioning. Curved arrows denote India ink punctures made at points of interest.

separate maximum points located in the cytoarchitectonic areas 3a, 3b, 5a and 6aß. This finding is illustrated in Fig. 7 and is most clearly demonstrated by the projections form the maxillary whiskers shown in Fig.
7 C. The afferent impulses from the oral cavity and the face are thus distributed to several separately located foci in the sensory as well as in the motor cortices.

The projections to area 3a were partly hidden in the banks of the coronal sulcus. This explains, why the potentials evoked by the nerves from the maxillary whiskers and the nose were recorded as surface negative waves in area 3a near the coronal sulcus. A typical reversal was found when the banks of the sulcus were mapped with penetrating microelectrode tracks.

Short latency surface positive potentials were evoked by the oral, mental and maxillary afferents in the lateral sigmoid gyrus within area 4γ, thus in the motor cortex characterized by the giant Betz cells. The evidence of a reversal of these potentials were, however, inconclusive, possibly due to the complex enfolding of area 4γ in the coronal and cruciate sulci. Conclusive evidence of primary projections from the oral cavity and the face to area 4γ were therefore not obtained.

The postsynaptic latencies of the evoked potentials in the maximum points of the primary projections were found to be 3 ms. This latency does only allow for conduction time and for one synaptic delay in each of the trigeminal and the thalamic relay nuclei. The fastest path from the oral receptors to the cerebral cortex is thus subserved by a chain of three consecutive neurones.

Somatotopical organization

The projections of the low threshold afferents from the oral cavity and the face showed a clear somatotopical organization in area 3a as well as in area 3b. The organization is illustrated in Fig. 7 A - D. The diagrams show the coronal gyrus extending lateral to and along the coronal sulcus, from
Fig. 7. Diagrams showing the typical location of oral (A and B) and face projections (B, C and D) related to the anatomy of the sulci and gyri and to the borders of the cytoarchitectonic areas (thin lines drawn in full). Symbols of borderlines and maximum points of the fields are explained in the figures. Borders at 20% of maximal amplitude are given for the initially surface positive short latency potentials unless otherwise indicated (50% or > lat). > lat = responses of longer latency in projection to the orbital gyrus. Dense stippling: field of initially negative or diphasic positive-negative potentials, maximum point: ⨳. Thin stippling: field of diphasic negative-positive potentials. Oblique hatching (CSR, RSR, SIIif): projection fields of low threshold co SR afferents.
the anterior suprasylvian gyrus caudally to the presylvian gyrus rostrally (cf. Fig. 6 A). The cytoarchitectonic areas 3a and 3b occupy a medial and lateral longitudinal strip on the gyrus. The border between the two areas can be seen in the diagrams. It leaves the posterior sigmoid gyrus, crosses the coronal sulcus and continues rostrally along the gyrus.

The oral projections formed bands across the coronal gyrus extending over both area 3a and 3b. As mentioned above, they have separate maximum points in the two areas (cf. Fig. 7 A, ip Alv inf maxima). The somatotopical order began with a lingual band near the presylvian sulcus. Then followed in an overlapping fashion, an oral mandibular (Fig. 7 A) and an oral maxillary band (Fig. 7 B). Perioral mandibular and perioral maxillary projections then appeared in the direction towards the anterior suprasylvian gyrus. Ophthalmic and auricular nerve projections were found between the maxillary projection and the caudal superficial radial nerve area near the lateral ansate sulcus (Fig. 7 D).

The somatotopical organization presented above is in general agreement with the classical map of the face projections given by Woolsey (1958). We do, however, not find the facelike layout of his map with the mandible near the coronal and the maxilla near the orbital sulcus straddling the introral fields. According to our findings the projections are somatotopically organized but bodyisomorism is not an essential principle.

The ipsi- as well as the contralateral projection fields are illustrated in Fig. 7. It was observed that the ipsilateral oral projections were larger and here evoked responses of shorter latency than the corresponding features of the contralateral projection. The contralateral perioral projections were,
on the other hand, larger than the ipsilateral ones and the potentials evoked by the contralateral perioral afferents were of shorter latency.

The perioral mandibular projection differed from that of the perioral maxillary nerves. The latter formed a continuous band across area 3a and 3b. The mentalis nerve, on the other hand, had two fields, (cf. Fig. 4 in Report II) one in area 3b near the caudal end of the orbital sulcus, and thus between the oral mandibular and the perioral maxillary projections. Another mental field overlapped areas 3a and 6aβ near the rostral end of the coronal sulcus. The afferents of the perioral component i.e. the mental nerve, are included in the inferior alveolar nerve. The inferior alveolar field shown in Fig. 7 A therefore includes the mental nerve projections, which were analysed separately in other experiments (cf. Fig. 4 C, D in Report II).

The projections to the orbital gyrus (area 43) were more complex than the primary projections. The fields of all the investigated nerves overlapped in this area, suggesting a high degree of convergence. The evoked potentials had a longer latency and did not show a reversal in depth recordings.

The interpretation of our projection maps depends on the variations between observations from different animals. The variations were therefore investigated (cf. Fig. 4 A, B in Report II). The main features of the projections were seen in a majority of the animals. They may therefore offer significant information concerning the functional organization of the somatosensory cortex. Individual variations in the extent of the fields were however found. The interpretation of the variations in the course of the borders may be facilitated by determination of the cytoarchitectonic areas.
CONCLUSIONS

1. Low threshold afferents from the oral cavity and the face projected via fast, presumably three synaptic paths to separate locations in areas 3a, 3b, 5a and 6aβ.

2. The projections to area 3a and 3b of the coronal gyrus were somatotopically organized with the lingual field most rostrally near the presylvian sulcus followed by the inferior alveolar, superior alveolar, maxillary whisker and nose, ophthalmic and auricular fields. They were arranged in overlapping bands across and along the gyrus. The sensory projections occupied the full length of the coronal gyrus.

3. The mouth and the face had bilateral cortical projection fields. The ipsilateral oral projections were larger than the contralateral ones, which is typical for all the oral projections. The contralateral face projections, on the other hand, were always larger than the ipsilateral ones. The potentials evoked by the ipsilateral oral afferents had a shorter latency than those evoked by the contralateral ones. The opposite held true for the perioral afferents. The ipsi- and contralateral fields overlapped to a certain extent and their maximum points were found at adjacent although not identical locations.
III

FACILITATION AND INHIBITION OF JAW REFLEXES EVOKED BY ELECTRICAL STIMULATION OF THE CAT'S CEREBRAL CORTEX
FACILITATION AND INHIBITION OF JAW REFLEXES EVOKED BY ELECTRICAL STIMULATION OF THE CAT'S CEREBRAL CORTEX

The effect of electrical stimulation of the cerebral cortex on the monosynaptic jaw closing and the disynaptic jaw opening reflexes were studied. The cortical origin of the effects were located and related to the somatosensory projections and to cytoarchitecture.

It was found that the jaw reflexes were most effectively influenced from the inferior alveolar and the maxillary nerve projections to cytoarchitectonic area 3a. Fast excitatory and inhibitory connections between this region and the trigeminal motoneurones were discovered. The finding suggests the existence of a mechanism which is capable of rapid switching between excitation and inhibition with a period of 10 ms.

METHODS

The effect of electrical stimulation of the cerebral cortex on the jaw closing and the jaw opening reflexes were studied in 27 adult cats. Cortical conditioning stimulation was applied to the defined ipsilateral somatosensory projection areas of the mouth, face and forelimb. Projection fields and maximum points were mapped as described in Report II. Conditioning stimulation was delivered between a surface anodal electrode on the cerebral cortex and a distant reference, or between an intracortical needle-electrode and a surface reference (see: General methodological considerations). The location of the stimulated point was charted on photographs available during the experiments. Stimulus parameters were systematically varied. Histological localization of the borders of the cytoarchitectonic areas and of penetrating electrode tracks with lesions was done (for details, see Report II). The cortical origin of the effects on the test reflexes was located and re-
lated to the somatosensory projections, and to the cytoarchitecture.

RESULTS

The typical effect of cortical conditioning on both jaw reflexes was a rhythmic sequence of facilitation and inhibition. The sequence of events could start with an initial phase of facilitation or with an initial phase of inhibition. The time course of the effects is illustrated by the schematic diagrams of Fig. 8. The latency between the first cortical shock and the first sign of effect on the masseteric or digastric motoneurones was short. Minimum latencies of 2.5 ms were thus recorded. The initial facilitation or inhibition was followed by alternative peaks of inhibition or facilitation. The fully developed rhythmic pattern had a sinuslike time course with a period of approximately 10 ms between the 3 - 4 first peaks. The period was then gradually prolonged and the amplitude decreased with increasing intervals between conditioning and test shocks. Facilitation of up to 1000% of the monosynaptic jaw closing reflex was observed. The facilitation of the disynaptic jaw opening reflex was smaller but effects between 100 and 200% were found. Periods of complete inhibition (100%) were repeatedly recorded, when using either reflex as the test.

The conditioning effects on both test reflexes were evoked by stimulation of the cortical surface using a single pulse or a short train of 3 - 5 surface anodal pulses, 0.5 ms pulse duration, 400 - 600 Hz. The threshold of the facilitation of the monosynaptic jaw closing reflex was 0.3 mA and that of inhibition 0.6 mA. The corresponding observation on the disynaptic jaw opening reflex was 0.5 mA for facilitation and 0.2 mA for inhibition. Thresholds as low as 0.07 mA were, however, observed when the cortex was stimulated with a needle electrode as the cathode penetrating in the center of an anodal silver ring on the cortical surface.
Fig. 8. Schematic illustration summarizing the effects on the jaw reflexes evoked by electrical stimulation of the cortex. A-B. Sequences of events starting with short latency facilitation (A) or inhibition (B). C-D. Fully developed sequences of alternating facilitation and inhibition starting with facilitation (C) or inhibition (D). E-F. Sequences with dominating facilitation (E) and inhibition (F).

The conditioning effects on the jaw reflexes were evoked from an area covering the oral, facial and forelimb projection fields in the coronal, presylvian, orbital, anterior ectosylvian, anterior suprasylvian, lateral sigmoid, and anterior sigmoid gyri. Quantitative as well as qualitative differences were, however, observed between the effects evoked from different parts of this region.
The difference of the effects evoked by a standardized conditioning stimulus applied to selected points within the cytoarchitectonic areas are illustrated in Fig. 9. Effects on the monosynaptic jaw closing reflex are shown. Borders of the cytoarchitectonic areas, determined in the experimental animal are given on the photograph of the hemisphere.

Fig. 9. Effects on the monosynaptic jaw closing reflex with variation of the location of the conditioning cortical stimulus (anode, 3 pulses, 0.5 ms, 555 Hz, 0.6 mA). A-G. Diagrams obtained from the cortical positions A-G indicated on the photograph of the perfused brain of the experimental animal. Borders of cytoarchitectonic areas defined on histological sections of the individual brain and transferred to the photograph.
The largest and most complex effect was obtained after stimulation of point D. This point was located in the maximum point of the surface negative area of the contralateral maxillary whisker nerve projection to area 3a. As shown in Report II, this field is overlaying the projections to the lateral bank of the coronal sulcus. Large effects were also recorded from area 3b. Diagram B shows the response from the maximum point. The amplitude of the inhibition as well as the facilitation was, however, smaller when evoked from area 3b than from area 3a.

Records A and E from areas 5a and 6aβ showed smaller effects mainly consisting of inhibition. This was also true for the effects evoked from area $4\gamma$ (C), and area 2 (G). The point located in the caudal part of the orbital gyrus just caudal to area 43 (F) showed similar small effects.

The efficiency of area 3a in influencing the jaw reflexes is further illustrated in Fig. 10. Optimal effects were evoked from points C and E corresponding to the maxillary whisker and inferior alveolar nerve projections to area 3a. The forelimb projection to area 3a was less efficient (B) and no effect was evoked from the hindlimb region (A). The response elicited from area $4\gamma$ (G) was considerably smaller than those evoked from area 3a. Intracortical stimulation confirmed the observations made when surface anodal conditioning was used. The optimal location for the conditioning of both jaw reflexes was found in the white matter immediately below area 3a in the lateral bank of the coronal sulcus.
Fig. 10. Effects of a cortical conditioning train (anode, 3 pulses, 0.5 ms, 555 Hz, 0.6 mA) on the monosynaptic jaw closing reflex. The locations of the stimuli in relation to the defined cytoarchitectonic areas are given in the photograph of the individual brain. Same animal as in Fig. 9.
CONCLUSIONS

1. Cortical effects of facilitation and inhibition of the monosynaptic jaw closing and disynaptic jaw opening reflexes were evoked from the oral, facial and forelimb cortical projection fields. The effects had a short latency (2.5 ms) indicating a disynaptic path. The fully developed sequence of facilitation and inhibition showed a sinuslike rhythm with a period of 10 ms.

2. The largest reflex effects were evoked from the maximum points of the cortical projections of the nerves to the maxillary whiskers, the nose and the teeth in area 3a of the coronal gyrus. Considerable effects were also evoked from the maximum points of the whisker projections in area 3b. Effects from the projections to areas 4γ, 5a and 6αβ were less complex and of lower amplitude. The largest motor effects were thus evoked from the "sensory" areas 3a and 3b and not from the "motor" areas 4γ and 6αβ.
DISCUSSION

Functional disturbances

Functional disturbances of the masticatory apparatus are of great concern to odontology. Judging from the literature the interest in these problems has substantially increased during the last decade. Helkimo (1979) has reviewed the recent epidemiological studies of the dysfunctions. Their prevalence was reported to be between 12 - 88% of the investigated populations. About 20 - 25% of the patients were estimated to need treatment for their symptoms.

According to the literature the most common symptoms of the functional disturbance of the masticatory system are: temporomandibular clicking or crepitation, temporomandibular joint pain, tenderness or pain of masticatory muscles, impaired mobility of the mandible, irregular path of movement of the mandible, and jerkiness of certain movements of the mandible. Headache and facial pain are other symptoms often described as associated with the functional disturbances. Some patients have further been demonstrated to have a reduction in masticatory muscle force (Molin, 1972) and a prolongation of the silent period of the muscle following the jaw jerk or following tooth contact (Bassette et al., 1971; Widmalm, 1976).

Pain from the masticatory muscles and the temporomandibular joint region is usually the main symptom and main motivation to seek treatment. Because of the involvement of pain in relation to the muscle tissue the disease was labelled "the myofascial pain dysfunction syndrome" (MPD) by Laskin (1969).

Bruxism is another functional disturbance of the masticatory apparatus
It has been defined by Nadler (1966) as nonfunctional voluntary or involuntary mandibular movements, which may occur during day or night, manifested by the occasional or habitual grinding, clenching or clicking of the teeth. The clinical observation of habitual nonfunctional clenching in centric and eccentric jaw positions was named "centric" and "eccentric bruxism" by Ramfjord and Ash (1966). The nonfunctional gnashing and grinding in eccentric excursions is considered most common during the night, while pressing or clenching is more common in daytime. The prevalence of bruxism seems to vary. Estimates range from about 5-15%, based on subjective awareness (Reding, 1966), to 88% based on occlusal signs i.e. wear facets of the teeth (Bungaard-Jörgensen, 1950).

The symptoms most commonly associated with bruxism are those resulting from hyperactivity in the masticatory muscles. Here the symptomatology seems to be common with that of MPD. Besides the pain problem the bruxism may have significant influence on other oral tissues as well. Excessive tooth wear has thus generally been considered a consequence of bruxism (Ramfjord and Ash, 1966). Xhonga (1977) recently confirmed that tooth wear did indeed progress faster in bruxists than in a control group. Sharp, irritating enamel margins, fractured teeth or restorations are other possible dental sequelae. The effect on the periodontal tissues has also been much discussed (Svanberg and Lindhe, 1973; Svanberg, 1974).

The relationship between bruxism and MPD is far from clear. Several studies indicate that MPD patients are frequently found to be bruxists (Ramfjord, 1961; Ramfjord and Ash, 1966), but the opposite is not necessarily true, as pointed out by Molin (1973). The MPD syndrome and bruxism are considered to have many etiological factors in common and studies on one of them will therefore presumably be relevant to both.
Several hypotheses concerning the etiology and pathogenesis of MPD and bruxism have been presented. They were recently reviewed by De Boever (1979). Two main ideas have been discussed. One refers the cause to peripheral components of the masticatory apparatus, the other emphasizes factors in the central nervous system. Some brief comments on these two hypotheses will be given here.

According to the hypothesis of a peripheral etiology, pathological processes in the joints, in the muscles themselves, in nerves or in relation to the teeth, are the primary causes of the pain.

Because of the incidence of pain located in the region of the temporo-mandibular joint and of the audible clicking from the joints, the interest for many years focused on the joint itself as a source of the dysfunction. Pathological changes in the joints were, however, rarely seen nor was there any reliable evidence of changes observed with conventional radiographic techniques. In fact, Laskin (1969) excluded such changes in his definition given above.

Malocclusion of the teeth should be mentioned as another etiological factor according to the first hypothesis. Thomson (1971) did, however, not find any apparent difference in frequency of occlusal abnormality or deficiency between dysfunction patients and a control group. Pettersson and Andrén (1978) also reported that many patients showed interferences between teeth without signs of dysfunction symptoms. Schwartz (1968) considered malocclusion to play a secondary role, and Laskin (1969) also failed to explain the etiology of MPD on this ground. More recent investigations (Molin et al., 1976) have, however, concluded that balancing side interferences significantly correlated with symptoms of dysfunction.
The hypothesis of a peripheral etiology suggests that the stimulation of oral and facial afferents facilitates the motoneurones of the jaw closers. This would explain the hyperactivity of the jaw muscles, which is observed in bruxists. Neurophysiological suggestions of monosynaptic (Sessle, 1977) and polysynaptic (Funakoshi and Amano, 1974) afferent excitation of masseter motoneurones are available. Electrical stimulation of oral afferents in man can result in activation or synchronization of the electrical activity of the masseter and temporalis muscles (Goldberg, 1971). Amano and Funakoshi, (1976) have further reported a tonic periodontal-masseteric reflex. These responses were elicited only when the jaw closing muscles were voluntarily activated.

In the experimental animal, stimulation of receptors related to the teeth, the oral mucosa and the perioral cutaneous structures evokes the jaw opening reflex (Sherrington, 1917), which includes inhibition of the jaw closing motoneurones (Kidokoro et al., 1968). This inhibition is, however, followed by a polysynaptic facilitation. In man, high intensity electrical stimulation of the mucous membrane results in a brief pause in the closing muscle activity (Yemm, 1972; Yu et al., 1973), but no activation of the jaw opening muscles has been reported. The above considerations lead to the conclusion that neurophysiological evidence from reflex studies are compatible with the hypothesis of a peripheral etiology. There is, however, no evidence of a long lasting facilitation of the jaw closers, which could give rise to a tonic muscular hyperactivity.

The hypothesis of a central nervous etiology places the primary cause of MPD and bruxism within the central nervous system. Psychic factors are assumed to be important but an organic brain disease cannot be excluded as a cause (Photo, 1977).
The influence of psychological factors upon jaw function has long been recognized and was brought into the discussion by Moulton (1955) and Schwartz (1956). In recent years the role of these factors has gained increasing clinical emphasis. Laskin (1969) thus suggested a psycho-physiological theory of the pathogenesis. Molin (1973) has supported the hypothesis. He concluded that personality traits and emotional disturbances were of importance for the development of the MPD syndrome. The psychological basis of the hypothesis has recently been extensively reviewed by Rugh and Solberg (1979).

According to the hypothesis it is suggested that psychic and physical stress experienced by the individual tends to increase the activity of the jaw muscles. The discussion of the etiology is thereby directed towards central nervous mechanisms controlling the trigeminal motoneurones.

The clinical evidence of a centrally initiated hyperactivity was recently discussed by Yemm (1979). Yemm (1971, 1979) further tested the possibility that mental stress may provoke hyperactivity in the jaw closing muscles in man. By using a physical task and an objective method of measuring changes in activity of the masseter and temporalis muscles, he found this to be the case in normal subjects as well as in MPD patients. The former did, however, adapt to repeated experiments showing a decreased muscle activity. The patients on the other hand, did not adapt. The conclusion was drawn that not only does mental stress lead to an increase in activity of jaw closing muscles, but also there is some experimental support for the contention, that these patients may be more likely to develop and maintain such muscle tension.

The relation of emotional muscular tension and increased tooth clenching has been investigated outside the laboratory setting by Rugh and Solberg...
(1979). From studies with patients provided with portable electromyographic devices, they reported marked increase in masseter activity in certain situations. Stimulus situations which commonly elicited teeth clenching were free way driving, interactions with employers and children coming home from school.

Berry (1969), Molin (1973) and Gold et al. (1975) have reported that MPD patients are apt to suffer also from other psychosomatic diseases. Evaskus and Laskin (1972) have further reported that a group of MPD patients had significantly higher urinary concentrations of catecholamines and 17-hydroxy steroids than a control group. Agerberg and Carlsson (1977) also reported a significant correlation between frequency of headache, MPD symptoms and clenching of teeth. Olkinuora (1972) has reported that bruxists, are in general, emotionally more imbalanced than controls and have more headache and muscle pains.

A tenfold increase in the amplitude of the jaw closing reflex was observed in the present study as a response to electrical stimulation in the hypothalamus and the cerebral cortex. It is tempting to assume that mental stress activates the hypothalamic mechanisms that are involved in the organization of agonistic behaviour. If that is the case, then it would be hypothesized that mental stress may change the balance of motor control towards and increased activity in the jaw closing muscles through the powerful mechanisms observed by us. If present in man, they may be implicated both in bruxist behaviour and in the myofascial pain dysfunction syndrome.

The assumption that mental stress affects the hypothalamic agonistic centers is supported by the observations of Lipp (1978) and Lipp and Hunsperger (1978). They found that electrical stimulation of the medial hypothalimus
in the monkey evoked behavioural patterns that closely resembled the natural defence reaction of this animal when subjected to frightening stimuli. They concluded that the electrically elicited response "corresponds at least partially to the natural behaviour, sharing its functional properties". A comparison of stimulation sites yielding agonistic behaviour in the opossum with those observed in the cat and the monkey further revealed a similar organization in the different species. It is thus not unlikely that these mechanisms may be similarly organized also in man.

**Rest position**

The control mechanisms of mandibular posture is a problem, which is central to all practical considerations in clinical odontology. This is so, because reliable reference positions are needed in the examination and oral rehabilitation. The physiology of the mandibular positions are, however, not fully understood, as pointed out by Brill and Tryde (1974) and Matthews (1975).

The part played by the jaw muscles and the nervous control mechanisms in determining the mandibular rest position is one of the questions which have been much discussed. In the clinic, the rest position is used as reference and starting point when establishing the vertical dimension of the maxillo-mandibular relation for patients lacking occlusal stops. The position is then defined as the posture of the mandible in a relaxed patient, standing or comfortably upright seated with the head unsupported, and looking straight forward. In this position there is generally an interocclusal clearance or freeway space of 2 - 4 mm.

The early concept of a stable vertical dimension of the rest position throughout life (Thomson and Brodie, 1942) has not stood the test of time. It has become clear that far from being a fixed position, the mandi-
bular resting posture is variable over short periods and in the long term. In early reports of Atwood (1956) and Tallgren (1957) instability of the rest vertical dimension was thus shown following extraction of teeth.

Atwood (1959) and Berry and Yemm (1976) have summarized and discussed several other anatomical as well as physiological and pathological factors, which have been found to influence the rest position in any of the three dimensions.

When electromyography was applied to the study of postural activity in the muscles of mastication, it was expected that it would clarify and solve some of the clinical problems in assessing a "physiological rest position". Instead, the method seems to have added confusion by presenting conflicting evidence as to whether or not there is muscle activity when the mandible is in the rest position (Hickey et al., 1957; Garnick and Ramfjord, 1962; Möller, 1966). An improved analysis of the physiological mechanisms, which influence the position is therefore needed.

The mechanisms underlying the rest position may be described on the basis of two principles. One of them assumes that the position is the result of an equilibrium between gravitational forces and passive viscoelastic forces in the muscles, ligaments and connective tissues. According to the other principle the equilibrium is due to muscular activity controlled by reflex mechanisms. Some authors (Berry and Yemm, 1976) have advocated the first hypothesis. Wyke (1973) Goldberg (1976) and Taylor et al. (1978) have, however, convincingly supported the hypothesis of an active muscular equilibrium under reflex control.

The active muscular control of the rest position assumes that the length of the jaw closing muscles is subject to a servo-mechanism (Merton, 1951; Möller, 1976).
The monosynaptic jaw closing reflex may subserve this control. All mechanisms influencing the excitability of the trigeminal motoneurones will then also affect the servo control. Funakoshi et al. (1973, 1976) have demonstrated the effect of cervical and vestibular influence on the electromyograms of the jaw muscles. Our results show powerful facilitation and inhibition of the trigeminal motoneurones evoked from the hypothalamus and the cerebral cortex. These powerful mechanisms may therefore form part of the complex events underlying the variations of the rest position. If they can be proven to be triggered by anxiety and stress, as is likely for the effects evoked from the hypothalamic defence-attack area, they will become even more interesting to those studying the variability of the rest position or other maxillo-mandibular relations.

Set points of rest position and biting force

We have investigated two different descending mechanisms, which influence the trigeminal motoneurones. They originate in the hypothalamus and the sensory-motor cortex respectively. The observed difference between these two mechanisms was, that cortical conditioning via fast descending paths evoked a variable sequence of facilitation and inhibition of the jaw reflexes. Hypothalamic conditioning on the other hand, resulted in more stereotyped effects dominated by facilitation of jaw closers and inhibition of jaw openers.

Taking the lead of the previous discussion we assume that rest position as well as biting force are regulated around equilibrium positions with controllable set points. We further suggest that the hypothalamus controls the set points. This is in line with the role of the hypothalamus in the regulation of other functions subserving the homeostasis of the body.
SUMMARY

The subject of this thesis is a study of the projections to the somato-sensory cerebral cortex from low threshold oral and face afferents and of descending motor control mechanisms originating in the cerebral cortex or the hypothalamus and influencing the jaw reflexes.

Cats, unanaesthetized and freely moving or anaesthetized with chloralose, were used for the experiments. Ipsilateral and contralateral nerves from the oral cavity and the face were dissected and prepared for electrical stimulation. The evoked potentials were recorded from the cortical surface or perpendicular to the surface along microelectrode tracks. The extent and maximum points of the primary projections were localized and their location was related to the cytoarchitectonic areas and to the pattern of the gyri and sulci. The borders of the cytoarchitectonic fields were determined in the individual animals.

It was observed (Report II) that the low threshold afferents from the oral cavity and the face projected to separate locations in areas 3a, 3b, 5a and 6αβ. The latency of the evoked responses was short (2.7 - 4.0 ms) suggesting a three synaptic projection path.

The projections to areas 3a and 3b were somatotopically organized but the cortical map was not facelike, but consisted of overlapping bands across and along the coronal gyrus starting with the tongue near the presylvian sulcus and followed by oral mandibular, oral maxillary, perioral mandibular, perioral maxillary, ophthalmic and auricular bands in the direction towards the forelimb projections to the posterior sigmoid gyrus near the ansate sulcus.
The effect of monopolar anodal electrical stimulation of the cerebral cortex on the monosynaptic jaw closing and the disynaptic jaw opening reflexes was studied in Report III. It was found that cortical stimulation evoked a rhythmic sequence of facilitation and inhibition of both reflexes. The fully developed sequence had a sinuslike time course with a period of 10 ms for the first 3–4 peaks. The latency of the cortical effects on the jaw reflexes was short (2.5 ms) indicating a minimum of two synapses in the path. The largest and most complex effects were evoked from the oral and perioral projections to areas 3a and 3b. Less complex effects of lower amplitude were recorded in response to stimulation of areas 4γ, 5a or 6αβ. It is suggested, that a trigemino-cortico-trigeminal loop via area 3a may function in reflex modulation of jaw movements.

The hypothalamic effects on the monosynaptic jaw closing and disynaptic jaw opening reflexes (Report I) were evoked by electrical stimulation in a medio-lateral band extending from the fornix to the entrance of the ansa lenticularis in the lateral hypothalamus. Rostro-caudally the region was located at the level of the ventro-medial hypothalamic nucleus and the area just rostral to this nucleus. In the unanaesthetized animals defence or attack responses were evoked by electrical stimulation in the perifornical part of the stimulated area.

Hypothalamic stimulation resulted in a tenfold increase of the jaw closing reflex in the anaesthetized animal with intact cerebral cortex. The effect remained but was diminished after bilateral cortical ablation. The effect on the disynaptic jaw opening reflex was an initial facilitation followed by an almost complete inhibition. The descending path was located in the ventral midbrain tegmentum.

It is suggested that the observed hypothalamo-trigeminal mechanism may
excercise a tonic influence on the trigeminal motoneurones, thereby controlling the set points of the biting force and of the rest position. The implications of this hypothesis on the etiology of bruxism and myofascial pain dysfunction are discussed.
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