Spatio-temporal Patterns of Pre- and Postsynaptic Inhibition Induced by Primary Afferent Activation in the Trigeminal Sensory Nucleus in Cats

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Summary. Spatio-temporal patterns of pre- and postsynaptic inhibition were studied in the trigeminal spinal nucleus oralis of cats by means of systematic electrical stimulation of the facial skin.

Stimulation of the facial skin induced an EPSP-IPSP sequence in trigemino-thalamic relay cells (TRC). The IPSP was depressed by bicuculline but was resistant to strychnine. The largest IPSP was evoked from the center of the excitatory area, where stimulation induced the largest EPSP and spike potentials at the lowest latency in the same TRC. The amplitude of the IPSP decreased with increasing distance from the center in parallel with that of the EPSP.

In the great majority of trigeminal primary afferent fibers, the largest primary afferent depolarization (PAD) was not evoked from the center of the excitatory area, where the threshold for spike generation was lowest, but from the adjacent points on the face. Spike activity in a trigeminal primary afferent fiber did not evoke any detectable PAD in itself.

The duration of the PAD was definitely longer than the IPSP in TRC. However, the temporal distribution of the peak of PADs was very similar to that of the EPSP in TRC.

Inhibition was evoked in glutamate-induced spike discharges of TRC by stimulation of the points on the face, which were located close to the center of the excitatory area of the TRC. However, the afferent inhibition of both spontaneous and peripherally induced spike discharges of TRC outlasted the postsynaptic inhibition. Thus, the late phase of the afferent inhibition is most probably due to presynaptic inhibition. Postsynaptic inhibition, together with postsynaptic inhibition, would be involved also in the early phase of afferent inhibition through its mutual inhibitory organization.

Key words: Afferent inhibition - Trigeminal sensory nucleus - Postsynaptic inhibition - Presynaptic inhibition

Aff erent inhibition is considered to play an important role in sharpening the point to point localization of stimuli. In the trigeminal sensory nuclei, afferent inhibition of the trigemino-thalamic relay cells was demonstrated to be arranged in a surrounding fashion (Darwin-Smith 1965). It was assumed that presynaptic inhibition contributed largely to the surround inhibition based on the general similarities between the spatio-temporal patterns of the surround inhibition and that of the excitability increase of the trigeminal primary afferent terminals (i.e., an indirect manifestation of primary afferent depolarization), Wall (1975) evoked by stimulation of the facial skin (Darwin-Smith 1965; Baldescu et al. 1965; Stewart et al. 1967; Rowe 1970; Rowe and Carmody 1970; Djuat and Sondel 1971). By studies of the manner in which presynaptic inhibition contributes to sensory discrimination in the trigeminal sensory nuclei, it was shown that presynaptic inhibition reduced not only the variability but also the sensitivity (a ratio of output increment to input increment) of the responses of trigemino-thalamic relay cells to peripheral inputs, but did not affect the dynamic range of these secondary neurons (Rowe 1970, Rowe and Carmody 1970, Carmody and Rowe 1974). Compared to these analyses of the effects of presynaptic inhibition on the transmission characteristics of sensory afferents in the trigeminal sensory nuclei, the spatial distribution of presynaptic inhibitory

0014-4619/00/06001545$ 2.00
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inputs from the facial skin has not been analyzed in detail. In addition, no evidence has been provided for the contribution of postganglionic inhibition to trigeminal afferent inhibition, although such a contribution is highly likely.

In this study, evidence is presented for a contribution of postganglionic inhibition in trigeminothalamic relay cells to the afferent inhibition evoked from the facial skin. Secondly, the spatial and temporal patterns of intrasynaptically recorded primary afferent depolarization (PAD) in trigeminal primary afferent fibers and the FSPs in trigeminothalamic relay cells are revealed by means of systematic mapping stimulation of the facial skin of cats.

A part of this study appeared in abstract form (Ishimine et al. 1974).

Methods

Experiments were performed in 40 cats anesthetized with pentobarbital sodium (40 mg/kg i.p. initially). The animal's head was tramed close to the skin. After tracheal cannulation, the animal's head was fixed to a stereotaxic apparatus. The posterior part of the buccal skin was exposed and the upper and lower incisors were exposed to reveal the facial nerve. A covered silver wire was fixed to the facial nerve to allow recording of the compound action potential from facial nerve fibers. The facial nerve was stimulated by rectangular pulses of 0.1 ms duration and 0.5 mA intensity. The cutaneous afferent fibers were stimulated by stimulating the buccal skin with a rectangular pulse of 0.1 ms duration and 0.5 mA intensity.

Summary

Afferent inhibition was observed in the primary afferent fibers of the facial nerve. The afferent inhibition was mediated by a disinhibition of the superior cervical ganglion. The afferent inhibition was observed in the primary afferent fibers of the facial nerve.
level about 300 ms after stimulation and then a post-
inhibitory rebound was seen between 300 and 400 ms
after stimulation.

Figure 1B shows a sample of the time course of the
afferent inhibition of the evoked discharge of a
TRC. Test stimuli were applied to a point on the face
marked T in Fig. 1B. Conditioning stimulation was
applied to an adjacent point marked C. Each dot in
this graph represents the average discharge number
recorded during five conditioned trials expressed in
percent of the average discharge number during five
control trials. Complete inhibition continued up to a
conditioning-test interval of 130 ms. It took more
than 250 ms for the discharge to recover completely
from inhibition.

Fig. 2A-D. Effects of facial stimulation on glutamate-induced spike discharges of a TRC. Lower-case letters in each panel indicate
the histological layer at which stimulation (b, 0.1, 0.2 mA) was
induced in inset of D. a and b represent stimulation applied
across 10 mm b and c are the cathodal points, respectively.
Alternatively, stimulation in the ms after facial stimulation reduced, number
of spike discharges recorded in each successive 10 ms period over
30 trials of facial stimulation for the period of 200 ms.

All the records were displayed on a cathode-ray oscilloscope
photographed. Interevent and intrarticular potential levels
were monitored by a slow-swing pen recorder to check the mobility
of membrane potential during the study of the responses to
successive stimulation of various points on the face.

Results

Time Course of Trigeminal Afferent Inhibition

The time course of afferent inhibition was studied on
both spontaneous and evoked discharges of
trigeminothalamic relay cells (TRC) in the
spinal nucleus oralis.

Figure 1A illustrates the time course of afferent
inhibition of the spontaneous discharge of a TRC. By
studying the excitatory receptive field of this cell, the
point with the lowest threshold for spike generation
was found at the point marked by an open circle in
Fig. 1A. The afferent inhibition of the spontaneous
discharge was evoked by single shocks delivered to
the point marked by a filled circle in Fig. 1A. The
time course in Fig. 1A was obtained by summing the
number of spike potentials during each 10 ms period
for 400 ms over 300 consecutive trials. Spontaneous
discharge activity was facilitated for 10 ms after
stimulation. The discharge activity returned to the control

Spatial Distribution of EPSP- and IPSP-inducing
Input in Trigeminothalamic Relay Cells

Innervational recording from TRCs revealed that
stimulation of the appropriate point on the facial
skin evoked EPSPs and subsequent prolonged hyper-
polarizing potentials (Fig. 2A-C, top records).
The EPSPs showed a latency of 1.2–1.7 ms and
reached a peak at 0.5–1.5 ms after stimulation.
Spike potentials were induced on top of the EPSPs
(Fig. 2Aa). The value of the shortest latency of the
EPSPs indicates that they were monosynaptically
evoked by impulses in Aβ fibers, as the conduction time of spike potentials in Aβ fibers was found to be 0.6-0.8 ms in the present study. Aβ fibers might be involved in the EPSPs with longer latencies, though it is equally possible that they were polysynthaptically evoked by Aβ afferents.

The hyperpolarizing potentials reached a peak of 13-50 ms after stimulation (Fig. 5B, open histogram). In 60% of the recorded TRCs, the peaks were reached 25-40 ms after stimulation (Fig. 5Ba, Ch). An increase in membrane conductance was detected during the hyperpolarization (Fig. 3A). The hyperpolarizing potential was abolished by intracellular CF injection through KCl-filled electrodes (Fig. 3Bb, g). The results indicate that at least the major part of the hyperpolarization is composed of IPSPs.

Intravenous injection of picROTOxin (1.0 mg/kg) greatly reduced or abolished the IPSP in all 3 TRCs tested (Fig. 3C), while the IPSP was usually enhanced by intravenous injection of strychnine (0.1-0.2 mg/kg). Although the number of TRCs in which the IPSP was abolished by intracellular stimulation was tested both before and after application of these constrictants was not large, it was our experience that the hyperpolarizing potentials were not depressed but rather enhanced after strychnine administration compared with those before application. In contrast, the hyperpolarizing potential was never produced after application of picROTOxin. Peripherally evoked trigeminal FAD was reported to be depressed by picROTOxin (Nakamura and Wu 1970). Thus, it is indicated that both pre- and postsynaptic inhibition of TRCs are susceptible to picROTOxin.

The time course of the IPSP was usually similar: regardless of the stimulated points. However, at the same intensity, the amplitude of IPSPs was largest when the stimulation was applied to the point where the threshold for spike generation was lowest (the lowest threshold point, LTP). Figure 4A and B illustrates diagrammatically the amplitude of the EPSPs and IPSPs which were evoked in a TRC by stimulation of various points on the face. EPSPs and IPSPs are represented respectively by open and filled semicircles whose diameter is proportional to the amplitude. This TRC was identified by antidromic spike potentials evoked by stimulation of the contralateral VPM. A spike potential was evoked from LTP.
The amplitude of EPSPs and that of IPSPs was found to be 0.77 with regard to 83 EPSP/IPSP sequences recorded in 10 TCRs.

Figure 4C shows graphically the amplitude of EPSPs (open histogram) and of IPSPs (shaded histogram) evoked in 8 TCRs by stimulation of various points on the face. The ordinate represents the averaged amplitude of the synaptic potentials expressed in percent of the maximum amplitude which was obtained by stimulation of LTP; the abscissa shows the distance of the stimulated points from LTP. In all the 8 TCRs tested, the maximum IPSPs were evoked by stimulation of the points that evoked the maximum EPSPs (i.e., LTP) and there was a significant trend for the amplitude of the synaptic potentials to decrease with increasing distance from LTP (p < 0.001, Friedman rank correlation test).

The duration of the IPSPs in TCRs was measured after stimulation of various points on the facial skin. The results from 120 IPSPs in 11 TCRs are shown by the shaded histogram in Fig. 5B. In 75% of the IPSPs the duration was shorter than 130 ms and it never exceeded 160 ms. On the other hand, the duration of the depression of EPSPs evoked from a point on the face conditioned by stimulation of another point lasted as long as 250 ms (Fig. 5A, filled circle). In 2 TCRs the test stimulation evoked EPSPs of a relatively simple shape with a monosynaptic latency (Fig. 5A), reminiscent of the EPSPs monosynaptically evoked in spinal motoneurons by group Ia afferents. This would imply that the test EPSPs in these cats might be monosynaptically evoked by a directly synchronized volley in a rather homogeneous group of Aβ fibers. The half-width of these EPSPs was measured as an index representing the duration of the monosynaptic EPSPs (Rall et al. 1967). The half-width of the conditioned EPSP was definitely shorter, compared with that of the control EPSP during the conditioning test interval of up to about 100 ms (Fig. 5A, triangle), while the amplitude of the conditioned EPSP was still smaller than that of the control up to about 200 ms. This suggests that monosynaptic inhibition probably underlies the late phase of the trigeminal afferent inhibition.

Spatial Distribution of PAD-inducing Areas in Trigeminal Primary Afferent Fibers

In the present study primary afferent depolarization (PAD) was used as the indicator for presynaptic inhibition.

Trigeminal primary afferent fibers impaled in the spinal nucleus oralis were identified by their directly conducted spike potentials evoked at a constant drift.
latency by stimulation of the ipsilateral semilunar ganglion. Spike potentials were evoked directly in some of the fibers by electrical stimulation of certain points of the facial skin with a latency of 0.6-0.8 ms (Fig. 6B, E).

It is likely that the recorded fibers are fast conducting Aβ fibers most probably originating from mechanoreceptors in the facial skin, because the conduction time of afferent impulses via Aβ fibers from the facial skin to the rostral parts of the brain stem auditory nuclei was shown to be less than 1.0 ms either by orthodromic (Davies et al. 1973) or antidromic activation (Baldnera et al. 1987), while the afferent volley in Aβ fibers from the tooth pulp was shown to reach the same brain stem nuclei about 2 ms after stimulation of the tooth pulp (Broekaart et al. 1953, Davies et al. 1971). In addition, the majority of the mechanosensitive afferents in the trigeminal system was reported to belong to Aβ fibers (Dartnall-Smith 1965, Gottschaldt et al. 1973; Duhrer et al. 1974) and the thermosensitive and pain afferents to Aβ and C fibers (Iggo 1969; Sumino et al. 1973). In each fiber studied we could usually locate the facial point with the lowest threshold for spike generation (LTP) and it was used as the standard point for comparing the effects of stimulation of other face areas.

When various points near or at LTP were stimulated transcutaneously, PADS of various amplitudes were induced in each fiber (Fig. 6C, E, F). PAD amplitude ranged from 1.0 to 3.8 mV in all primary afferent fibers recorded intra-aurally. The features of the PAD were consistent with the results previously reported (Nakamura et al. 1974, 1977; Ya and Avery 1974) in their latency (1.5-6.8 ms), time to peak (2-22 ms), and duration (300-500 ms). The PAD was very easily depressed by repetitive stimulation at relatively low frequency. Thus, PDNs evoked by 10 Hz stimulation were less than 30% of amplitudes of PDNs induced by 1 Hz stimulation at the same intensity; they were completely abolished by 20 Hz stimulation. The amplitude of PDNs increased in an almost linear relationship with the increase in intensity of stimulating current in a certain range. At each intensity, however, the amplitude of the PAD varied depending on the points of stimulation. It seems reasonable, therefore, that the relative effectiveness of impulses in the primary afferent fibers, which innervate the neighborhood of each point for PAD production in the fiber under study, can be estimated by the relative amplitude of PDNs evoked by stimulation at a certain intensity.

The spatial distribution of PAD-inducing areas was investigated in 14 primary afferent fibers with stable membrane potentials greater than -40 mV. In 11 of 14 fibers, as shown in Fig. 6B and C, stimulation of LTP (Fig. 6A, point B) at the threshold intensity for spike generation evoked a spike potential followed by a slight depolarizing potential (Fig. 6B) which was much smaller in amplitude than the PAD evoked from an adjacent region (Fig. 6A, point C) at the same intensity of stimulation (Fig. 6C). When stimulation was applied to LTP at a subthreshold intensity for spike generation, the small depolarizing potential was not detected, although PADs were still induced from surrounding spots. With an increase in stimulus intensity in the range which is suprathreshold for spike generation, the prolonged depolarizing potential following the spike potential increased in amplitude, which was depressed by stimulation at a frequency of more than 20 Hz. This would indicate that the prolonged depolarizing potential observed with stimulation at just above the threshold for spike generation was in fact an afterdepolariation of the spike potential (Eccles and Kujirai 1959) and the large depolarizing potential induced by intense suprathreshold stimulation resulted from superimposed PAD.

In the remaining three primary afferent fibers, as shown in Fig. 6E and F, stimulation of LTP at subthreshold intensity for spike generation evoked PDNs which were larger in amplitude than those evoked by stimulation of adjacent points at the same intensity. In the fiber shown in Fig. 6E and F, stimulation of LTP (Fig. 6D, point E) with 0.8 mV either induced a spike followed by a prolonged depolarizing potential or only PAD (Fig. 6Eg). This
Fig. 7. Spatial distribution of PADS in trigeminal primary afferent fibers. Absets, distance of stimulus point from LTP; ordinate, amplitude of PAD expressed as percent of that evoked from point 10 mm apart from LTP. The amplitude of PADS evoked from various points on the face at equal-intensity stimulation threshold for spike generation was measured in five fibers and the spatial distribution is shown by squares, triangles, and circles connected with solid lines. The mean of the amplitude of these PADS is illustrated by filled circles connected with a dashed line.

PAD (Fig. 6E) was larger in amplitude than that evoked from an adjacent point (Fig. 6D, point F) with stimulation at the same intensity (Fig. 6F).

If we regard LTP as the excitatory point for a particular primary afferent fiber and the LTP-inducing points as the presynaptic inhibitory points for the same fiber, the excitatory and the inhibitory points were located side by side in the eleven of fourteen primary afferent fibers (lateral type), while in the other three fibers the largest presynaptic inhibition was obtained from the excitatory point (non-lateral type). Thus, the majority of the trigeminal primary afferent fibers belonged to the lateral type.

The LTPs of the 11 lateral type fibers were distributed widely over the cheek and the infraorbital region of the face, while all of the three non-lateral type fibers had their LTPs in the infraorbital region adjacent to the nose.

Although the threshold intensity for spike generation of the lateral type fibers ranged from 0.1 to 1.2 mA, in their majority it was either 0.2 mA (5 fibers) or 0.3 mA (3 fibers), that was much lower than that of the non-lateral type fibers, i.e., 0.8 mA (2 fibers) and 1.0 mA (1 fiber). The remaining three lateral type fibers had their threshold of 0.5, 0.7 and 1.2 mA, respectively. In each of two particular points where LTP for both lateral and non-lateral type fibers were found, the threshold intensity for spike generation of the non-lateral type fibers (0.8 or 1.0 mA) was notably higher than that of the lateral type fibers (0.3 mA).

Figure 7 illustrates the spatial distribution of PADS in five fibers of lateral type. Stimulus intensities threshold for spike generation were used. In all of these fibers PADS were not evoked from LTP but from surrounding points. The amplitude of PAD in each fiber (solid line) and the spike generation (dashed line) are plotted on the ordinate in percent of the PADS evoked from points 1 cm apart from LTP. There was a statistically significant trend of decrease in PAD amplitude with the increasing distance in the range from 1 to 5 cm from LTPs (p < 0.002, Friedman's rank correlation test).

In agreement with the above results suggesting the predominance of the lateral type, intra-nasal injection of depolarizing pulses through the recording electrode evoked a spike potential followed by a small but definite prolonged depolarizing potential, which was not depressed as all by stimulation at frequencies higher than 20 Hz. This suggests that the depolarizing potential following a spike potential evoked by intra-nasal current injection is not a PAD but an afterdepolarization.

Discussion

Pre- and Postsynaptic Components in Trigeminal Afferent Inhibition

The present study has shown that the duration of afferent inhibition of spike potentials in TRCs definitely outlasted the hyperpolarizing potentials and the inhibition of glutamate-induced spike potentials of TRCs which were evoked by facial stimulation. The results indicate that the late phase of trigeminal afferent inhibition probably would be due to postsynaptic inhibition and/or an antidromic inhibition.

The depression of the amplitude of monosynaptic EPSPs without change in the time course was adopted as the sign of presynaptic inhibition in spinal as well as trigeminal motorneurons (Cook and Cagniato 1972; Chandler et al. 1980). In the peripherally evoked monosynaptic EPSPS in TRCs, the afferent inhibition consisted of a decrease in both amplitude and duration during the early phase where hyperpolarizing potential was seen following conditioning stimulation, while in the late phase, which was uncompromised by any hyperpolarization, the decrease in amplitude only was found without shortening of the duration of EPSPS. The results suggest
that postsynaptic inhibitory processes are probably participating at least partly in the late phase of trigeminal afferent inhibition.

By intra-axonal tracing (Nakamura et al. 1974) as well as measurement of the excitability increase in postsynaptic terminals (Darley-Smith 1965; Baldiasseri et al. 1967) of trigeminal primary afferent fibers, the duration of peripherally evoked PAD of trigeminal afferent fibers was estimated to be 200-300 ms, which well corresponded to the duration of trigeminal afferent inhibition. This may support the above notion of participation of postsynaptic inhibition in the late phase of the afferent inhibition.

Kellerm and Szumski (1966a, b) reported a strychnine-resistant, picotoxin-sensitive postsynaptic inhibition in the trigeminal sensory nucleus. As an example, the duration of postsynaptic inhibition was almost 100 ms or more, which was acting at remote dendritic sites. Graslot et al. (1964) showed that the repetitive spike discharges in spinal monosynaptic Proxid by tonic depolarizing current was an index sensitive enough to detect the remote inhibition, i.e., the postsynaptic inhibition that was not revealed by any of other signs of postsynaptic inhibition (e.g., hyperpolarization, synaptic activation noise and monosynaptic EPSP depression). In fact, picotoxin-sensitive inhibition was detected by inhibition of spike potentials directly evoked by injection of tonic depolarizing current into motoneurons, even in the case where synaptic activation noise was weak and the membrane potential level was unchanged (Kellerm and Szumski 1966b). The peripherally evoked hyperpolarizing potential in TRCs was shown to be sensitive to picotoxin. The fact that the hyperpolarizing potential was depressed or abolished but not reversed to a depolarizing potential after intracelluar CT injection supports the view of Kellerm and Szumski (1966a, b) that picotoxin-sensitive EPSPs originates from distal dendrite region. Therefore, it is implied that glutamate-induced spikes might be a fairly satisfactory indicator to detect distal dendritic EPSPs. In the present study, however, no inhibition was found in glutamate-induced spikes in TRCs during the late phase of the afferent inhibition. Thus, no positive evidence could be obtained for remote dendritic inhibition involved in the late phase of trigeminal afferent inhibition.

Based on these results it can be concluded that the late phase of trigeminal afferent inhibition at intervals longer than about 150 ms (i.e., the maximum duration of EPSPs in TRCs) is not wholly due to remote dendritic EPSPs but rather is attributed mainly to presynaptic inhibition, whereas both presynaptic and postsynaptic inhibitory processes participate in inhibition at short intervals.

Spatial Distribution of Pre- and Postsynaptic Inhibition in Trigeminal Sensory Nucleus

The essential features of the possible neural mechanisms involved in pre- and postsynaptic processes for surround inhibition have been schematically represented by two alternative models (see Bokel at 1967). In the one model (Type A) surround inhibition is achieved exclusively or at least mainly by mutual inhibition, while in the other (Type B) it is due not only to mutual but also to autogen inhibition.

The present study has shown that the transmission of sensory messages in the trigeminal sensory nucleus is subjected to postsynaptic and, most probably, presynaptic inhibition, both contributing to the afferent inhibition. Furthermore, it has been revealed that pre- and postsynaptic inhibitory processes carried out their parts by two different ways respectively corresponding to Type A and B in the model of von Bokel at.

In this regard, primary afferent fibers classified as the non-lateral type should be mentioned. In this group the threshold for inducing spike potentials (Te) was invariably higher than that for PADs (TePD) at LTDs in contrast to fibers classified as the lateral type. Considering the relatively wide spacing (10 mm) of stimulating electrodes on the facial skin, the high Te relative to the TePD implies that the LTD tentatively determined might in fact be displaced from the real LTD (i.e., the center for the excitatory field of the fiber) near to the surrounding inhibitory fields. With a proper placement of stimulating electrodes, sufficiently weak stimulation at the real LTD might be activated exclusively the primary afferent fiber under study, since all the fibers sampled in this study were considered to belong to AP group in which no systematic difference is expected to exist in the threshold for spike generation. It is possible, therefore, that the classification as the non-lateral type was fictitious.

Furthermore, the present study has shown that intra-axonal application of depolarizing solutions evokes spike potentials but they are not followed by any detectable PAD in the fiber itself. This implies that spike activities in a particular fiber do not contribute in any appreciable amount to PAD production in the same fiber, either due to lack of autogenous PAD production or due to the reason that possible recurrent inputs from axon collaterals are too small to produce an appreciable PAD in the parent axon terminals. The present results have confirmed the report of Schmidt et al. (1967) that natural stimulation of a single cutaneous receptor did not induce any PAD in the afferent fiber innervating the receptor. All or almost all the PAD in a primary
afferent fiber may be due to spike activity in fibers other than the one being recorded from.

It is known that the maximum presynaptic inhibition is exerted at the peak of PAD (Schmidt 1974). The present study has shown that PADS in the majority of trigeminal primary afferents reach their peak during the peripherally induced excitatory phase of TRCs. This would localize that presynaptic inhibition exerts its maximum effect during this phase. Thus, an important role of presynaptic inhibition may be the selective blocking of converging excitatory inputs to TRCs from the peripheral region of the stimulated area via mutual inhibitory processes.

This spatial pattern of presynaptic inhibition suggests that high frequency afferent impulses in the fiber strongly activated from the center of stimulated area on the face exerts a stronger presynaptic inhibitory effect on low frequency afferent impulses in the fiber weakly excited from the periphery of the stimulated area. Thus, presynaptic inhibition may contribute to such a spatial pattern of afferent inhibition that responses evoked from the peripheral part of the excitatory receptive field are more susceptible to inhibition than responses elicited from the center of the receptive field (Jaing et al. 1977).

In contrast to presynaptic inhibition, IPSPs in TRCs were largest in amplitude when evoked from points inducing the largest EPSPs in the same TRC and the amplitude of the IPSPs decreased with increasing distance from those points in parallel with the decrease in the EPSP amplitude. The same spatial pattern (Type B) of excitatory and inhibitory receptive field was reported in thalamo-cortical relay neurons in the VB complex of the thalamus (Tsumura and Inubushi 1974a). By high frequency continuous stimulation of the peripheral parts of the receptive field, however, a pattern of excitatory center-inhibitory surround (Type A) became visualized (Tsumura and Inubushi 1974b). It is possible that IPSPs in TRCs contribute to sharpening the spatial information of the stimulated points on the face by means of a mechanism similar to that in the VB complex.

Acknowledgments. We thank Professor H. Shiraishi for support of this study as well as review of the manuscript. We also wish to express our appreciation to Dr. Y. Inoue and B. Chang for comments to the manuscript and to Professor A. Sakuma for advice on statistical analysis.

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