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Control of Orienting Gaze Shifts by the Tectoreticulospinal System in the Head-Free Cat. I. Identification, Localization, and Effects of Behavior on Sensory Responses

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SUMMARY AND CONCLUSIONS

1. The input-output connectivity, in cat, of tectoreticular (TRNs) and tectoreticulospinal (TRSNs) neurons [together called TR(S)Ns] suggests a role for these cells in the sensorimotor transformations necessary for controlling orienting behavior. Multimodal sensory information converges directly onto these tectal neurons, and they project to several brain stem and spinal cord centers involved in the control of eye- and head-orienting movements. In this and the following two papers, we describe the sensorimotor discharges of antidromically identified TR(S)Ns. Here we describe the process of localizing and identifying them, characteristics of both their antidromic and sensory responses, and effects of behavioral context on these responses.

2. In 13 alert, chronically prepared cats, a total of 293 neurons were antidromically identified from either the predorsal bundle (PDB) immediately rostral to abducens nucleus or the ventromedial funiculus of the spinal cord at the level of the first cervical vertebra (C_1). The cell bodies of all identified TR(S)Ns were confined to the intermediate and deep laminae of the superior colliculus (SC). The antidromic nature of the action potential evoked by stimulating either the PDB or C_1 was verified by the use of a number of established criteria, including collision testing.

3. The mean antidromic latency from the PDB (TRNs + TRSNs) was 0.84 ± 0.59 (SD) ms (n = 217). The conduction velocities of all cells activated by PDB stimulation ranged from 4 to 40 m/s. The mean latency from C_1 (TRSNs) was 1.03 ± 0.52 ms (SD) (n = 64), whereas conduction velocities ranged from 14 to 80 m/s.

4. One hundred thirty-eight TR(S)Ns were studied long enough to yield significant data regarding their involvement in visuomotor-orienting behavior. Ninety-eight percent (130/133) of the TR(S)Ns tested for visual responses could be induced to discharge action potentials in response to some form of visual stimulation. The other three neurons remained silent, even in response to the most provocative stimuli. These silent neurons nevertheless were shown to be depolarized by visual stimuli. TR(S)Ns were occasionally tested for auditory and somatosensory responses and some were multimodal.

5. TR(S)Ns had visual receptive fields that conformed to the retinotopic map of the visual field that is represented within the SC. Cells found in the lateral SC had receptive fields located in the lower visual field, whereas neurons that were situated medially had receptive fields in the upper visual field. Cells found in the rostral SC had small fields that included a representation of the area centralis. TR(S)Ns located in the more caudal regions of the SC had large fields that did not include a central representation. To distinguish between rostral and caudal cell groups, and for reasons that will become amply evident in the subsequent papers, we call them *fixation* [fTR(S)Ns] and *orientation* [oTR(S)Ns], respectively. The present paper considers only the latter.

6. oTR(S)Ns responded to the onset of a stationary light-emitting diode (LED) with a brief phasic burst, the latency of which was remarkably constant from trial to trial. The mean onset latency for 33 cells was 57 ms. For this same population, the mean of the earliest response latency of each cell was 47 ms.

7. oTR(S)Ns responded very well to moving visual stimuli. All 15 neurons tested were found to have a directional preference for stimulus motion away from the area centralis. For most cells, the preferred direction of stimulus motion was parallel to the horizontal meridian.

8. During the behavioral act of attentive fixation, 10 of the 13 oTR(S)Ns that were tested for fixation effects had significantly attenuated responses to an abstract stimulus moving in their receptive field.

9. By comparison, the sensory responses of oTR(S)Ns were enhanced when the stimulus became the target of an orienting response. In 9 of the 14 cells tested, response to the onset of an LED was significantly augmented when the cat oriented to the LED within 1 s of its onset. The amount of enhancement that occurred was linked to the latency of the animal's orienting response. Enhancement was greatest when the animal's response latency was very short, such that the phasic visual response overlapped movement onset.

10. The excitability levels of 15 oTR(S)Ns were evaluated in different behavioral conditions, using a *twin-pulse antidromic technique* to determine whether the behaviorally induced variability in oTR(S)N sensory responsiveness was the result of changes in either the afferent sensory input or the level of oTR(S)N excitability. oTR(S)Ns were found to be at significantly lower levels of excitability when the hungry animal attentively fixated the food target. When the animal was about to orient to a food target moving through a cell's visual receptive field, the neuron was at a significantly higher level of excitability. If the animal was about to orient to the food in the opposite direction, the level of excitability was significantly reduced. Because oTR(S)Ns project directly onto eye and head premotor circuits, the sensory responsiveness of these cells is presumed to influence the probability of occurrence of orienting movements.

INTRODUCTION

The input-output connectivity of the cat superior colliculus (SC) underlines its role in the transformation of visual information to the motor commands required to elicit orienting movements of the head and eyes. The superficial layers of the SC receive projections from the retina and visual cortex, thereby inducing visual responses in neurons that are organized into a retinotopic map subtending up to 80° of the contralateral visual field (Feldon et al. 1970).

Experiments performed in the head-restrained monkey have shown that the deeper layers of the SC are organized into a motor map that is in spatial register with the overlying retinotopic visual map (Robinson 1972; Schiller and Koerner 1971; Schiller and Stryker 1972; Van Opstal et al. 1990): a point in the superficial layers that is activated by an eccentric visual stimulus lies above the site in the deeper layers that, when excited, drives a saccadic eye movement to the stimulus. In the cat with unrestrained head (head free), the motor map encodes the vector error between current gaze and target positions and drives gaze (= eye-in-head + head-in-space) to the target (Crommelinck et al. 1990; Guitton et al. 1980; Munoz and Guitton 1989, 1991; Munoz et al. 1991; Roucoux et al. 1980).

A major descending efferent pathway emanating from the deeper laminae of the cat SC is the tectoreticulospinal tract. Neurons with axons that lie within this tract, tectoreticular neurons (TRNs) and tectoreticulospinal neurons (TRSNs), are situated in the intermediate and deep layers of the SC (Edwards and Henkel 1978; Grantyn and Grantyn 1982; Huerta and Harting 1982a,b; Kawamura and Hashikawa 1978; Moschovakis and Karabelas 1985; Murray and Coulter 1982). Their axons cross the midline within the dorsal tegmental decussation in the mesencephalon and descend through the brain stem. TRSNs project all the way to the upper cervical spinal cord, terminating predominantly in lamina VII (Huerta and Harting, 1982a,b; Nyberg-Hansen, 1964; Petras, 1967). Intra-axonal injection of horseradish peroxidase into these collicular efferent neurons has revealed their extensive pattern of collateralization as they descend toward the spinal cord (Grantyn and Berthoz, 1985; Grantyn and Grantyn, 1982; Moschovakis and Karabelas, 1985). They send out a multitude of axon collaterals to mesencephalic, pontine, and medullary centers, many of which are involved in the control of eye and head movements.

Many single-unit recording studies in alert monkeys have shown that neurons in the SC have sensory- and motor-related components in their discharge patterns (Goldberg and Wurtz, 1972a,b; Mays and Sparks, 1980; Mohler and Wurtz, 1976; Schiller and Koerner, 1971; Sparks, 1978; Sparks et al. 1976; Sparks and Mays, 1980; Wurtz and Goldberg, 1971, 1972; Wurtz and Mohler, 1976a). For example, the visual responses of many neurons located in the superficial layers of the monkey SC can be enhanced if the stimulus triggers an orienting response (Goldberg and Wurtz, 1972b; Wurtz and Mohler, 1976). Saccade-related burst neurons, located in the deeper layers of the monkey SC, discharge high-frequency bursts of spikes before saccadic eye movements (Schiller and Koerner, 1971; Sparks 1978; Sparks et al. 1976; Wurtz and Goldberg, 1971, 1972). If a visual stimulus fails to evoke a saccade, then these neurons may still be activated, but they lack the high-frequency saccade-related burst component from their discharge (Sparks, 1978). In the monkey, this latter population of neurons forms at least part of the descending collicular efferent projection to the brain stem saccadic premotor circuitry (Keller, 1979; Moschovakis et al. 1988a,b).

In this series of papers, we describe the discharge characteristics of TRNs and TRSNs in alert behaving cats to determine their role in controlling orienting movements of the eyes and head. In this paper, we provide information on the identification, localization, and sensory responses of these neurons. It will be shown that their sensory responses are enhanced when the stimulus triggers an orienting movement and suppressed when the behavioral situation promotes attentive fixation. Furthermore, these behaviorally related changes in sensory responsiveness are the result of changes in the level of excitability of the neurons. The functional significance of modulating the excitability of these neurons is discussed. In the next paper (Munoz and Guitton, 1991), we describe a sustained pattern of activity that is related, depending on the cell type, to mechanisms of motor preparation and fixation. In the third paper (Munoz et al. 1991), we describe spatiotemporal variations in TR(S)N phasic discharges that accompany coordinated eye-head orienting movements.

Preliminary reports describing some of the findings in this paper have appeared elsewhere (Munoz and Guitton, 1986, 1987).

METHODS

The sensorimotor discharges of TRNs and TRSNs were studied in 13 alert, behaving cats. Each animal was studied when its head was either held immobile (head fixed) or left unrestrained (head free). Procedures used in preparing the animals for chronic experiments and recording of eye and head movements have been described elsewhere (Guitton et al. 1984, 1990). Here we describe methods used in recording and analyzing single-unit data.

Single-unit recording procedures

Single-unit activity was recorded in the intermediate and deep layers of the SC. Conventional filtering, amplifying, and display techniques were employed. The reference electrode consisted of a silver wire attached to a skull bolt that was embedded in the acrylic skullcap. Two different extracellular recording electrodes were used: stainless steel microwires or glass micropipettes. The microwires were permanently implanted into the SC so that very stable recordings, lasting several days, could be obtained from the neurons in both head-free and head-fixed conditions. Glass pipettes were lowered through a recording chamber and driven into the brain. Unlike microwires, the position of the glass pipette was adjustable, thereby increasing the number of cells that were identified and recorded in a single cat. However, there was more instability when recording cell activity with pipettes, so that the duration of recording of a single cell, especially in the head-free condition, often did not allow the full battery of experimental paradigms to be exploited.

GLASS MICROPIPETTES. Glass micropipettes were driven into the SC of nine cats by use of a hydraulic microdrive assembly (Kopf). During construction of the skullcap assembly (Guitton et al. 1990), a bilateral craniotomy was performed (stereotaxic coordinates A5.0–P1.0 and R6.0–L6.0) and a stainless steel recording chamber was attached with dental acrylic onto the skull above the SC. The dura underlying the opening in the bone over one cerebral hemisphere was retracted to allow the pipette tip to enter the brain without breaking. The dura on the contralateral side was left intact until a later time. The exposed cortex was covered first with an absorbable gelatin sponge USP (Gelfoam) then by a silicone film (Silastic), and cotton. The sterile recording chamber was closed until experiments began. At the start of each experimental session the recording chamber was opened and cleaned. An XY micropositioner (Kopf), controlling rostrocaudal and mediolateral positions of the electrode track, was fitted to the chamber. A

water-filled hydraulic microdrive (Kopf), clamped to the XY micropositioner, drove the electrode vertically into the brain. Microfilament glass pipettes (A&M Systems) were prepared on a Narishege puller and filled with 4 M NaCl. Pipettes with a tip impedance of 1.0–2.0 MΩ were used. Because the dorsal surface of the SC was located 12–15 mm below the exposed surface of the cortex, the electrode tips were tapered for >2 cm to minimize damage to the cortex overlying the SC.

STAINLESS STEEL MICROWIRES. Stainless steel microwires were implanted into the SC to record single-cell activity in four animals. We modified the technique of Palmer (1978). Bundles of 10–12 stainless steel microwires (25-μm diam, tri-ML insulation, California Fine Wire) were held together in a specially designed rubber (Flexane 80, Devcon) connector, created within a stainless steel mold, that was embedded in the acrylic of the skullcap. The overall objective was to lower the tips of the microwires to the location within the SC that had the maximum antidromic field potential after stimulation of either predorsal bundle (PDB) or the ventromedial funiculus of the spinal cord at the level of the first cervical vertebra (C_1). To accomplish this, we lowered the bundle of microwires into position through a small hole in the bone and dura, with the aid of a rigid tungsten microelectrode blank that passed through the rubber mold. Wires were attached to the tip of the microelectrode blank with glucose. The mold was attached to the skull with dental acrylic while the glucose melted. The microelectrode blank was then withdrawn, leaving the fine, flexible wires in the brain. The orthodromic and antidromic spike shapes, antidromic latency, threshold current, and strength-duration relationships all remained constant for the duration of recording of all TR(S)Ns that we describe (Munoz, 1988).

Antidromic stimulation procedures

TRNs and/or TRSNs were electrophysiologically identified by antidromically activating their main descending axon at one of two sites: *1*) PDB immediately rostral to abducens nucleus or *2*) C_1. Stimulation consisted of mono- or biphasic current-regulated pulses that were isolated from ground (Grass S88, PSIU6). In the first experimental animals, C_1 stimulation was used to identify tectal efferent neurons, and therefore all neurons identified were TRSNs. In subsequent animals the site of identification was switched to the PDB to avoid damaging the dorsal neck muscles and spinal cord. Both TRNs and TRSNs were identified with PDB stimulation. These cells will be called tectoreticulo(spinal) neurons or TR(S)Ns, because we could not tell which of the recorded identified neurons projected to the spinal cord.

PDB STIMULATION. Axons of TR(S)Ns were antidromically identified from the PDB using a bipolar concentric stimulating electrode (Kopf, SNEX100). Under stereotaxic guidance, the electrode was lowered into the brain stem at an angle of 20–30° posterior to the frontal plane to a site just rostral to abducens nucleus (stereotaxic coordinates: P5.5, H −5.0, ML0; see Berman, 1968). The final electrode position was determined by eliciting a short-latency field potential with the largest amplitude in the intermediate and deep layers of the SC.

C_1 STIMULATION. A dorsal approach was used to expose the surface of the spinal cord at C_1. To minimize damage, we retracted neck muscles only along the midline. The only muscle cut and not resutured was rectus capitus dorsalis minor, a small muscle with origin at the dorsal arch of the atlas and insertion into the occipital bone. This muscle is involved in raising the snout. A small hole, bored through the first vertebra, exposed the spinal cord along the midline. Three or four 75-μm-diam stainless steel wires (Teflon insulated, Tensolite Insulated Wire Co.) were passed through a small incision in the dura and lowered into the ventromedial funiculus of the spinal cord, to a position where the optimal field potential could be recorded in the deeper layers of the SC. The final position of the wires was maintained by anchoring them to a small pedestal made of dental acrylic, which, in turn, was attached to the atlas with a small stainless steel screw (1-64) placed laterally in the bone. The wires were then led subcutaneously to the acrylic skullcap, where the connector pins were located.

Histology

At the end of an experiment, reference electrode positions within the colliculus, brain stem, and spinal cord were marked by passing positive DC current through stainless steel electrodes (30 μA for 10–20 s). To localize lesions, the Prussian blue marking technique was employed (Talbot et al. 1967). Cats were overdosed on barbiturate and, with the lower circulation clamped, perfused intracardially with 500 ml of saline, followed by 500 ml of a ferriferrocyanide, 10% Formalin solution that produced blue spots at locations where current was passed through the electrode tips. The brains were removed and stored overnight in the fixative and then transferred to a solution of 30% sucrose in 10% formal saline until they were cut into 40-μm sections with a freezing microtome. Sections were stained with cresyl violet.

Visual stimulation

TR(S)N visual response properties were studied with a number of different types of visual stimulation. First, a piece of food—placed on a small plastic spoon fitted with a search coil to measure horizontal and vertical positions (Guitton et al. 1990)—was moved by hand throughout the visual field to localize a cell's visual receptive field. Movements of the food target almost always elicited orienting responses from the cat (Guitton et al. 1990; Munoz et al. 1991). This stimulus produced a complex moving visual pattern consisting of the food and holder and the experimenter's hand, sleeve, and arm. Therefore, once the receptive field of a TR(S)N was localized, its visual response properties were studied with stimuli for which size, shape, onset time, and movement characteristics were more precisely controlled.

Stationary visual stimuli consisted of light-emitting diodes (LEDs) mounted on moveable arms that could be positioned anywhere in front of the cat (±90° horizontal; ±50° vertical; 50–60 cm away from the cat's eyes). In addition, LEDs could be attached to the edges of an opaque barrier that was placed in front of the animal. The trained cats had no obvious difficulties in discriminating the onset of the LED, as indicated by the very short response latencies of orienting movements to the LED.

Moving visual stimuli consisted of dark bars of various widths and lengths, oriented parallel or perpendicular to the direction of movement and back-projected onto a large translucent plastic screen (spanning 100° horizontal × 120° vertical) that was placed 60 cm in front of the cat. The direction, amplitude, velocity, and repetition rate of stimulus motion could be independently controlled. The visual stimulus was projected onto the screen via a mirror controlled by a galvanometer. A function generator drove the galvanometer with a single ramp that moved the stimulus across the screen at a constant velocity. At the end of the ramp, the visual stimulus was reset to its initial position. The resetting of the stimulus was very fast (~20 ms) and was equivalent to a very-high-velocity movement of the stimulus in a direction opposite to the ramp. Repetitive stimuli presented to the alert animal were necessarily projected onto different parts of the retina, depending on current gaze position. In the data analysis, stimulus presentations corresponding to specific eye positions could be extracted from the data set.

Data storage and analysis

Hardware and software (courtesy of Dr. R. M. Douglas, Ophthalmology Dept., University of British Columbia, Canada) capa-

bilities permitted the simultaneous monitoring and analysis of up to three neurons. Unit activity was recorded on FM tape, along with the positions of horizontal and vertical gaze, head, and food target and with trigger pulses marking the occurrence of sensory stimulation. At a later time, selected portions of the experiment could be played back and sampled by a computer. In one cat the experimental data were sampled directly by the computer. For playback from FM tape, or on-line sampling, unit activity was first passed through a time-amplitude window discriminator (BAK Electronics), which isolated single-unit activity and produced a transistor-transistor logic pulse for each action potential. Blocks of experiments were then digitized; individual spikes were counted into 1-ms bins and stored with gaze, head, and stimulus traces (low-pass filtered at 250 Hz), which were digitized at either 500 Hz or 1 kHz. During off-line analysis, TR(S)N discharge characteristics (e.g., number of spikes, duration of firing, and frequency of firing) were measured and stored in analysis files along with other data describing the characteristics of eye, head, or gaze movements (see Guitton et al. 1990) and the onset of sensory stimulation. Individual unit traces that were selected for analysis could be aligned with any event (e.g., LED onset) and rank-ordered according to any chosen criteria (e.g., latency or peak firing rate).

RESULTS

Antidromic identification of TR(S)Ns

The dorsal surface of the SC was easily identified in the alert cat. Strong multiunit visual responses were evoked from the dorsalmost (superficial) layers of the SC. Stimulation of the PDB or C_1 elicited no responses from these superficial laminae. Once the tip of the recording electrode reached ~1.2 mm (1.0–1.5 mm) below the dorsal surface, a short-latency field potential began to be observed after PDB or C_1 stimulation. This field potential subsequently increased and then decreased in size during the next 2–3 mm of vertical electrode travel. This electrode depth corresponded to the intermediate and deep layers of the SC. TRNs and TRSNs were identified within this zone; they were found ~1.2–4 mm below the surface of the SC. The lowest threshold currents required for evoking eye-head movements after microstimulation were obtained in this same vertical zone of the SC. Outside this zone there was a sharp rise in the threshold current necessary to evoke an orienting movement.

TR(S)Ns were located by repeatedly stimulating the PDB or ventromedial funiculus of the spinal cord at C_1 while lowering the recording electrode through the SC. As the recording electrode approached an efferent neuron, the antidromic action potential began to grow out of the field potential. The antidromic nature of this potential was verified using a number of well-established criteria (Lipski, 1981): *1*) constancy of the latency of the response, *2*) stability of the threshold, *3*) collision between the antidromic and orthodromic action potentials, *4*) fractionation of the antidromic spike into initial segment (IS) and somadendritic (SD) components, and *5*) ability of the response to follow high-frequency stimulation. The first three criteria were performed on all cells except silent neurons, in which case the collision test could not be performed. The last two criteria required twin-pulse stimulation, which, in general, was performed only after extensive testing of a cell's visuomotor responses.

The antidromic identification of one TR(S)N is illustrated in Fig. 1. This neuron was located in the statum griseum intermediale of the right SC and could be activated by electrically stimulating the contralateral PDB, just rostral to abducens nucleus. This cell responded to stimulation of the PDB with an action potential of short and invariant latency (Fig. 1*A*). Threshold current (1.0T) was defined as the current necessary to activate the neuron in 50% of the trials. When the current intensity was raised to 1.2T, the antidromic spike was always observed.

An orthodromic action potential from the same neuron is illustrated in Fig. 1*C*. The shape of the antidromic spike waveform depended on the position of the recording electrode tip relative to the structure of the neuron, notably its IS and SD compartments. In most cases, the antidromic spike waveform (Fig. 1*A*) had a notch in the initial phase (marked by the arrowhead) that was absent from the orthodromic spike (Fig. 1*C*). This inflection point on the initial phase of the antidromic action potential was presumably caused by the spike being delayed in its invasion of the SD compartment of the cell (Bishop et al. 1962; Brock et al. 1953; Lipski, 1981). This delay caused a partial separation of the IS and SD components of the antidromic spike. The presence of an IS-SD notch provided further strong evidence that the spike was antidromic and also verified that the recording was made in the vicinity of the cell body and not from an axon of passage.

The most conclusive proof of antidromic activation was the demonstration of collision between the antidromic and orthodromic action potentials (Fig. 1*B*). Orthodromic spikes were used to trigger PDB or C_1 stimulation. As the

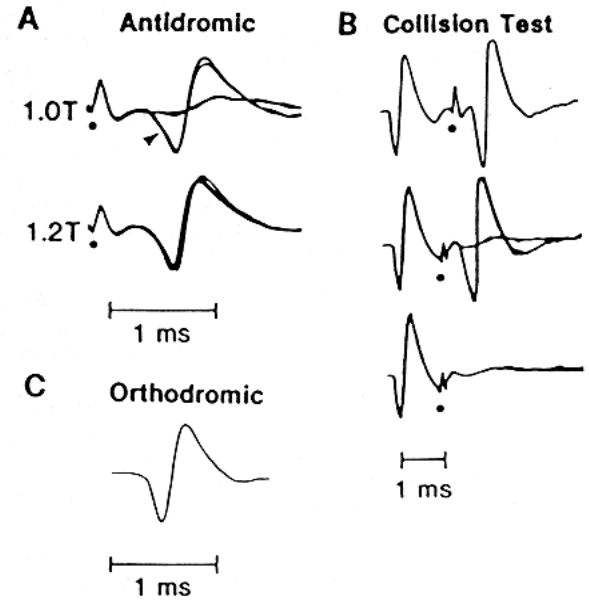

FIG. 1. Identification of a TR(S)N that was antidromically activated by stimulation of the predorsal bundle (PDB). Occurrence of stimulation pulses marked by filled circles *under* traces. *A*: antidromic activation at threshold (1.0T) and suprathreshold (1.2T) current. Arrowhead points to notch between initial segment (IS) and somadendritic (SD) components (see text). *B*: collision of antidromic responses with orthodromic action potentials. Blocking of the antidromic spike was dependent on the interval between the orthodromic spike and the stimulus pulse. *C*: orthodromic action potential, having a similar shape as the antidromic spike, but void of any IS-SD notch.

interval between the orthodromic spike and the stimulus pulse was decreased, the antidromic response was blocked due to collision of orthodromic and antidromic spikes along the axon. Any differences in the measured critical delay (time from orthodromic spike to stimulation pulse that gave 50% collision) and the theoretical critical delay (axonal refractory period plus antidromic latency) were well within established criteria (Fuller and Schlag, 1976).

Antidromic response to twin-pulse stimulation

The IS and SD components of the antidromic spike could be separated during high-frequency twin-pulse stimulation (Fig. 2*A*). For a specific interpulse interval (the refractory period of the SD spike), the IS-SD notch in the second antidromic spike was more pronounced, and the SD spike was occasionally absent (*2nd trace from top* in Fig. 2*A*). When the interval between the two stimulus pulses was decreased further, the SD component of the second antidromic spike was eliminated altogether (*3rd trace from the top* in Fig. 2*A*). We defined the SD refractory period to be attained when SD spikes were absent in 50% of the trials. We shall see subsequently that the SD refractory period of TR(S)Ns was very dependent on the cat's behavioral state. When measured in an animal that was not solicited by either food or visual targets, the mean SD refractory period for all tested neurons was 1.65 ± 0.29 (SD) ms (n = 33; note use of the same abbreviation for "standard deviation" and "soma-dendritic"). By comparison, the mean refractory period for the IS component was 0.75 ± 0.18 (SD) ms (n = 33, example in Fig. 2*A*, *4th trace from the top*). Figure 2*B*) illustrates, for five cells, the probability of observing the SD component of the second antidromic action potential as the twin-pulse interval was varied. For some neurons, a small change in this interval dramatically improved the probability of observing the second SD spike. For example, the probability curve of the neuron illustrated with filled circles in Fig. 2 went from a value of 0 to 1 within 0.2 ms.

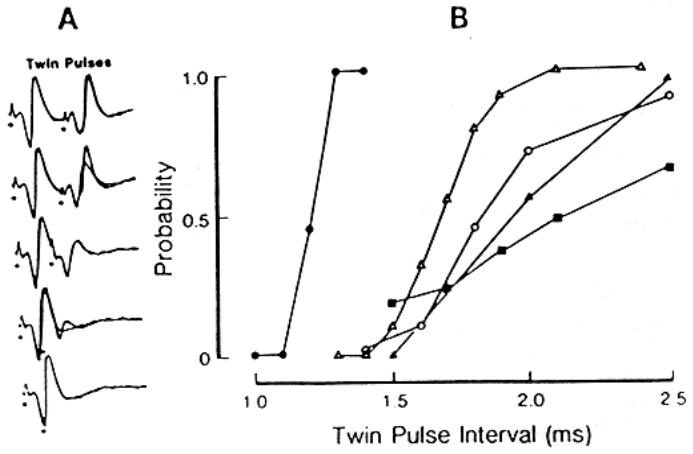

FIG. 2. Response of TR(S)Ns to twin-pulse antidromic stimulation. *A*: high-frequency stimulation, showing fractionation of antidromic response into IS and SD components as twin-pulse interval was varied. Occurrence of stimulation pulses marked by filled circles *under* traces. *B*: robability of observing 2nd SD spike as the twin-pulse interval was varied or 5 TR(S)Ns. SD refractory period was determined as the interval that generated the 2nd SD spike on 50% of trials when the alert cat faced a blank screen.

This neuron had a high safety factor (Lipski, 1981) for antidromic invasion of the SD compartment, which was uncharacteristic of most TR(S)Ns studied. The more typical response is illustrated by the other probability curves in Fig. 2*B*. For example, the curve illustrated with open circles went from a probability of 0 to nearly 1 in >1 ms. This cell had a more pronounced IS-SD delay, corresponding to a lower safety factor for antidromic activation of the SD compartment. For those cells with a lower safety factor, the refractory period of the SD spike was very dependent on the level of excitability of the neuron. This property will be exploited in a subsequent section to yield estimates of how TR(S)N excitability varies depending on whether the animal is attentively fixating or about to orient.

Latency of antidromic responses

A total of 293 neurons were antidromically identified and tested for latency in 13 chronically prepared, alert cats. The number of cells identified from any one cat ranged from 1 to 67. In each cat, only one locus of stimulation [either PDB (219 cells) or C_1 (74 cells)] was employed to identify neurons. In experiments in which neurons were identified after stimulation of the PDB, it was impossible to differentiate between TRNs and TRSNs. We found no differences in the discharge characteristics of neurons identified from either the PDB or C_1.

The latency of the antidromic response (i.e., the time from stimulus onset to initiation of the antidromic spike recorded in the SC) of all identified neurons is summarized in Fig. 3. The distribution of latencies was very similar for both PDB (Fig. 3*A*) and C_1 (Fig. 3*B*) stimulation: both histograms were skewed to the left. The mean antidromic latency from the PDB was 0.84 ms (SD ± 0.59 ms, n = 217), whereas the median, reflecting the skew of the distribution, was 0.58 ms. The mean latency from C_1 was 1.03 ms (SD ± 0.52 ms, n = 64), and the median was 0.76 ms.

The conduction velocities of these neurons were estimated after measuring the distance between the SC, PDB, and C_1 of two cats. Using a distance of 4 cm from C_1 to the SC and neglecting the time to initiate the antidromic spike, we estimated the conduction velocities from C_1 to be 14–80 m/s. We measured an axonal distance of 1.6 cm between the site of PDB stimulation and the SC. The estimated conduction velocities after antidromic activation at the PDB ranged from 4 to 40 m/s. The range measured for PDB might have been brought closer to that of C_1 had activation time (not evaluated) been incorporated in the calculation of velocity.

General discharge characteristics of TR(S)Ns

Of the 293 neurons we tested for latency (Fig. 3), 45% (133/293) of these had an adequate signal-to-noise ratio and were recorded long enough to yield significant data in tests that ranged from studies of a cell's visual responses to studies of its discharge during visuomotor behavior. The limited time we could record from a cell in the head-free condition did not allow each neuron to be tested with the full battery of experimental paradigms. Eighty-eight of the neurons were identified from the PDB, whereas the remain-

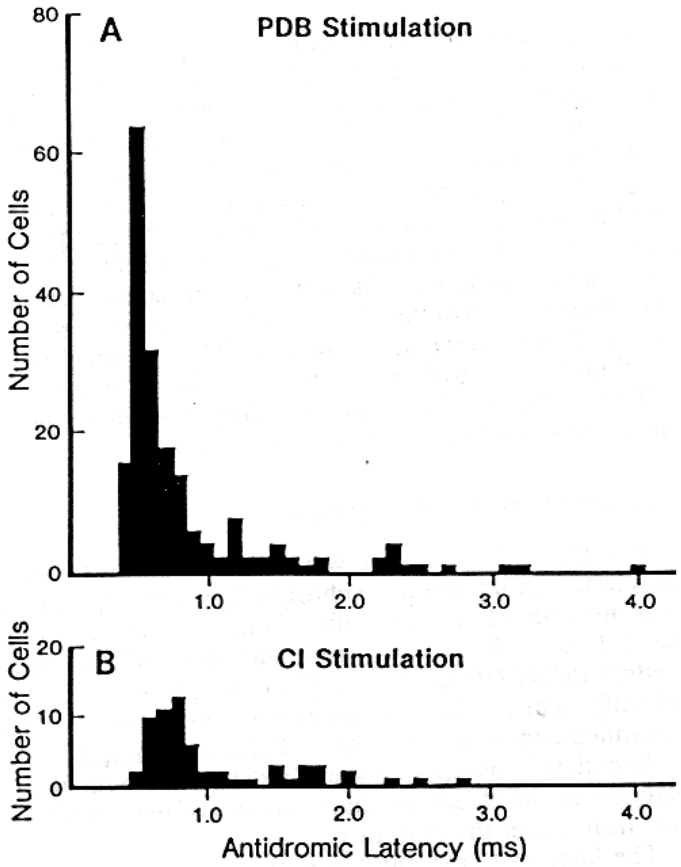

FIG. 3. Histograms illustrating antidromic latencies of all neurons identified from the PDB and the ventromedial funiculus of the spinal cord at the level of the 1st cervical vertebra (C_1). Note that PDB stimulation (*A*) activated TRNs and TRSNs, whereas C_1 stimulation (*B*) activated only TRSNs.

ing 45 were identified from C_1. The sensorimotor discharges of no more than 29 TR(S)Ns were studied in any one animal.

We will refer to TR(S)Ns as either *fixation* [fTR(S)Ns] or *orientation* [oTR(S)Ns] neurons. There is a strong rationale for this nomenclature. It is based on links between cell discharge and the animal's behavior, and it will be the subject of the next two papers (Munoz and Guitton, 1991; Munoz et al. 1991). Briefly, fTR(S)Ns were active when the animal fixated a target of interest. Their receptive fields included a representation of the central visual field (i.e., area centralis). By comparison, oTR(S)Ns had sustained and phasic discharges related to the preparation and execution of orienting gaze shifts. Their receptive fields excluded the area centralis.

Table 1 lists the percentage of cells showing a particular response type in their discharge pattern. An important characteristic observed in almost all TR(S)Ns was that they lacked spontaneous activity (Grantyn and Berthoz, 1985; Munoz and Guitton, 1985). The results presented in this and the following two papers show that the typical "average" TR(S)N carries three types of signals: *1*) responses to sensory stimulation, which are subject to the modulatory influences of attention, fixation, and orientation (133 cells were tested; described herein); *2*) sustained discharges, which were recorded during periods when the visual axis was not moving and which served to maintain relevant localized ensembles of SC output cells in a depolarized state during either attentive fixation and/or the preparation of an orienting gaze shift to a target of interest (99/133 were tested, 86/99 had sustained discharge, 74/86 were oTR(S)Ns, and 12/86 were fTR(S)Ns; described in Munoz and Guitton, 1991); and *3*) movement-related phasic discharges that contributed to determining a gaze movement's direction, amplitude, latency, and time course [65/74 oTR(S)Ns and 10/12 fTR(S)Ns were tested; described in Munoz et al. 1991]. Any given TR(S)N can be considered to carry all of the above signals, but with different "gains," such that one or more of the three signal categories becomes dominant. Of the sensory responses, to be described below, only those to visual stimuli were thoroughly studied; neurons were only occasionally tested for auditory or somatosensory inputs. Therefore a percentage of responsive cells was not reported in Table 1 for these stimuli.

Visual responses

The visual responses of TR(S)Ns were studied using a number of different experimental paradigms. Table 2 shows the fraction of cells tested that responded to different forms of visual stimulation. One hundred thirty-three neurons were tested for and influenced by visual inputs; 130 could be induced to discharge action potentials, whereas only 3 did not fire a single action potential, even in response to stimuli that were very provocative for the majority of other cells (e.g., moving food target). These silent neurons, nevertheless, had subliminal responses that will be described in a subsequent section.

TABLE 1. *Types of TR(S)N responses in alert cats*

Response Type	Fraction of Cells: No. responsive/no. tested	Fraction of Cells: %
Sensory responses		
Visual	130/133*	98
Auditory	19/20†	
Somatosensory	3/3†	
Sustained discharges, recorded during periods when visual axis was stationary, related to gaze position error	86/99	87
Preorientation-related [oTR(S)Ns]	74/99	75
Fixation-related [fTR(S)Ns]	12/99	12
Phasic discharges related to execution of movement		
oTR(S)Ns		
Visible target	62/65	95
Predicted target	35/46‡	76
fTR(S)Ns		
Visible target	10/10	100
Predicted target	4/4§	

TR(S)N, tectoreticular and tectoreticulospinal neurons. *Three of 133 neurons never discharged action potentials to visual stimulation. However, they could be depolarized by visual stimulation (see text). †Neurons tested in these conditions form a subset of the 133 tested for visual responses. ‡Neurons tested in this condition form a subset of the 65 oTR(S)Ns tested with the visible target. §Neurons tested in this condition form a subset of the 10 fTR(S)Ns tested with the visible target.

TABLE 2. *Visually responsive TR(S)Ns studied in different conditions*

Stimulus Type	Fraction of Cells: No. reponsive/ no. tested	Fraction of Cells: %
Moving food target	130/133	98
Stationary LED	48/50	96
Moving abstract stimulus	29/31	94
Directional selectivity	15/15	100
Attenuation of respones by fixation	10/13	77
Enhancement of responses by orientation	9/14	64

A total of 133 neurons were tested with the moving food target. Subsets of these were tested with other stimuli. TR(S)Ns, tectoreticular and tectoreticulospinal neurons; LED, light-emitting diode.

Stationary visual stimuli, consisting of LEDs positioned in front of the cat, were used to evaluate the response latency and receptive-field contours of TR(S)Ns. Ninety-six percent (48/50) of all cells subjected to this test responded to this form of visual stimulation. Responses to the onset and offset of the LED were usually weak unless the LED was used for the target of an orienting movement (see below). Thirty-one of the visually responsive TR(S)Ns were tested with a moving abstract stimulus (e.g., a dark bar, tongue, or edge) projected onto a lit tangent screen, and 29 cells responded.

RECEPTIVE-FIELD CHARACTERISTICS. It is important to note that precise borders of visual receptive fields were difficult to delineate in the alert, behaving animal because of frequent gaze shifts and the dependence of responses on the behavioral context within which a stimulus was presented. Nevertheless, by noting the position of the visual axis during each target presentation and avoiding situations during which the animal was attending to other stimuli, it was possible to determine a field's location and approximate its contours.

Several studies have described the retinotopic map of visual space that is represented within the SC (Berman and Cynader, 1972; Feldon et al. 1970; Lane et al. 1974; Sprague et al. 1968). TR(S)N visual receptive fields conformed to this map. Fixation TR(S)Ns were found at the "zero" representation in the rostral SC and had receptive fields that included the area centralis. Orientation TR(S)Ns were situated outside of the "zero" representation. The oTR(S)Ns had visual receptive fields that did not contain a representation of the area centralis: cells in the lateral SC had visual receptive fields located in the lower visual field, whereas those neurons in the medial SC had receptive fields in the upper visual field.

In the TR(S)Ns tested for multimodal responses (Table 1), visual receptive fields were coextensive with receptive fields of other sensory modalities. For example, Fig. 4 shows the relationship of the visual, auditory, and somatosensory receptive fields of an oTR(S)N, *cell M8*, that responded to sensory stimulation in all three modalities. The boundary of each receptive field encloses all points where at least one action potential was evoked by stimulus onset. The contours of the visual receptive field were first established by presenting a LED at various positions in the visual field while the animal was alert. Once we had gathered sufficient data on the visuomotor properties of the cell, the cat was lightly anesthetized with an intramuscular injection of diazepam (5 mg/ml) and ketamine (50 mg/ml) to obtain more precise information on the multimodal receptive-field structures of this neuron.

The shading in Fig. 4 covers the maximum area from which a response could be elicited from the cell in each modality. Auditory and somatosensory receptive fields were evaluated while the cat was in total darkness. The visual receptive field (Fig. 4*A*) is plotted in retinotopic coordinates, whereas the auditory receptive field (Fig. 4*B*) is plotted in head-centered (craniotopic) coordinates. The circular contours mark 30, 60, and 90° from the center position of the visual axis (Fig. 4*A*) or head axis (Fig. 4*B*). The visual receptive field was confined to the lower left quadrant of the visual field (Fig. 4*A*) and was coextensive with the cell's auditory receptive field (Fig. 4*B*), which extended peripherally to just beyond the interaural axis. The somatosensory receptive field extended over the length of the left forelimb (Fig. 4*C*). If the animal was standing and directing its head and visual axes straight ahead, then a sensory stimulus in any modality, confined to the shaded areas of Fig. 4, would trigger approximately the same direction orienting movement: left and down.

Of great interest was whether receptive fields in different modalities remained coaligned when gaze position varied in the alert animal (Jay and Sparks, 1984, 1987b). Unfortunately, because of the large size of the receptive fields, the lability in neuronal responses, and the effects of fixation on sensory responses (see below), we could not determine whether the auditory field moved with changes in eye position in the head-fixed condition or whether the somatosen-

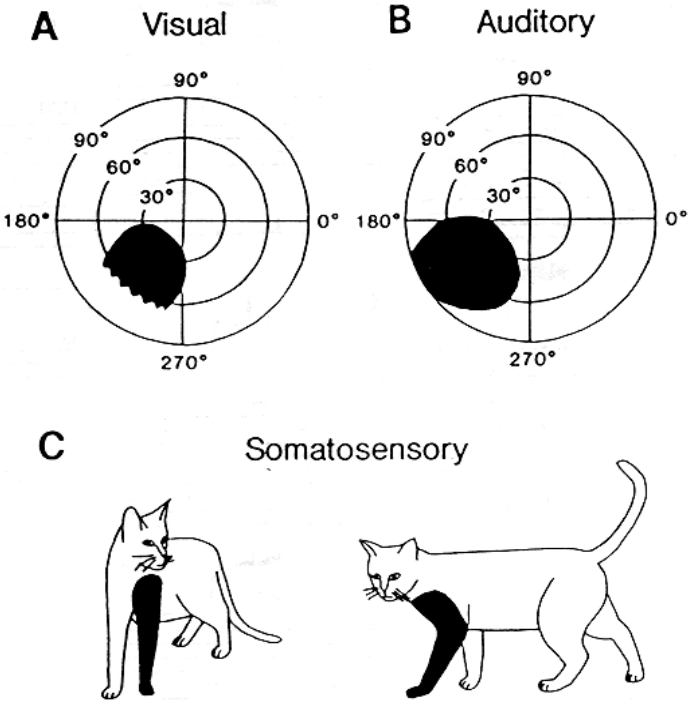

FIG. 4. Visual (*A*), auditory (*B*), and somatosensory (*C*) receptive fields of *cell M8*, an oTR(S)N located in the caudolateral right SC. Shading covers the maximum area from which a response could be elicited from the cell in each modality.

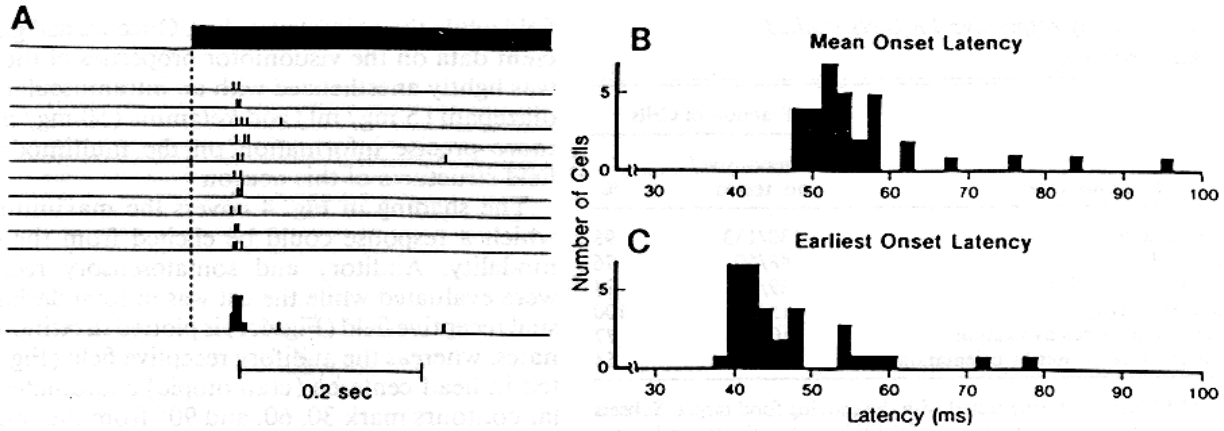

FIG. 5. *A*: responses of an oTR(S)N, *cell E9*, to onset of a light-emitting diode (LED) placed in the neuron's visual receptive field. *Top* to *bottom*: LED trigger pulse; individual raster displays of 10 trials; summary histogram. Vertical dashed line marks the onset of the LED. Animal did not orient to the LED. Each vertical tick on each raster corresponds to a single action potential. *B* and *C*: mean and earliest response latencies of 33 TR(S)Ns to onset of LED.

sory field remained independent of gaze position when the cat changed the position of its head.

LATENCY OF VISUAL RESPONSES. Figure 5*A* shows the responses to LED onset of an oTR(S)N, *cell E9*. The response to the onset of the LED consisted of a brief phasic burst, the latency of which was remarkably constant from trial to trial. For *cell E9* the mean response latency for LED onset was 53 ms (SD ± 6 ms, $n = 28$), whereas the earliest response latency recorded from this neuron was 40 ms.

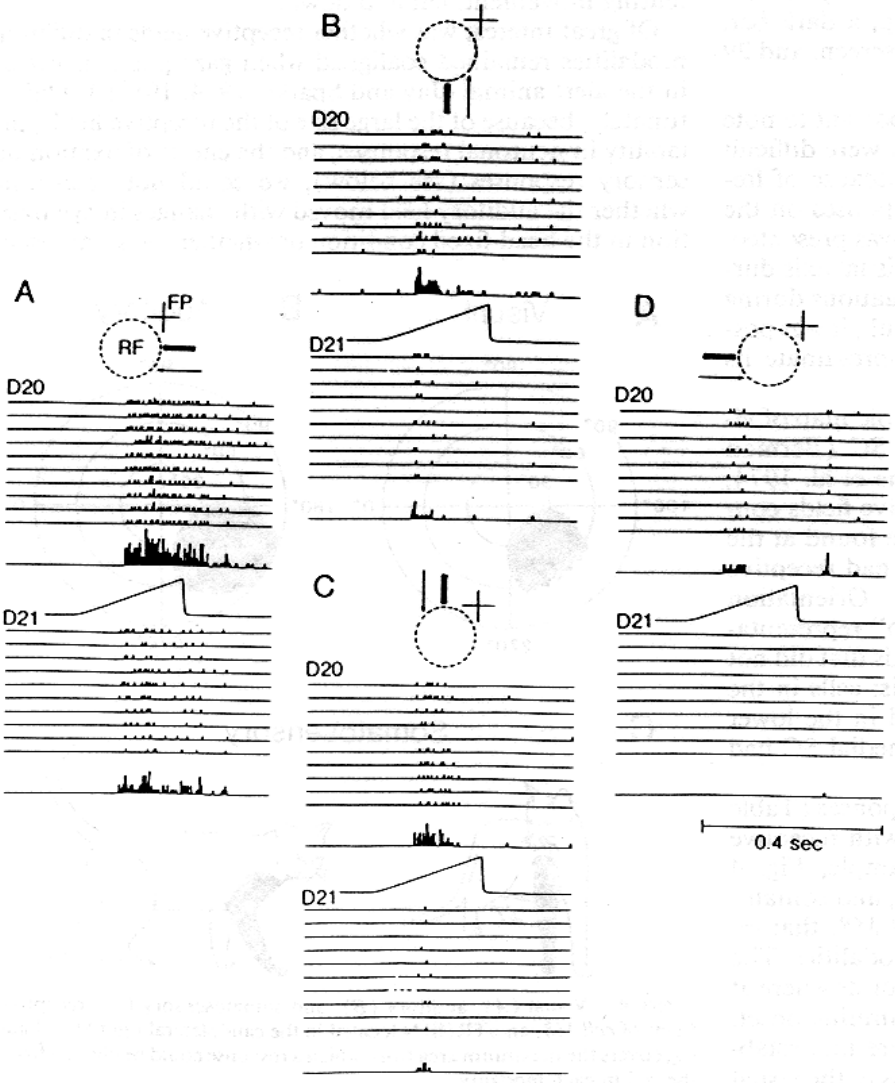

FIG. 6. TR(S)N visual responses are selective for direction of motion of visual stimulus. Data recorded simultaneously, on 2 adjacent microwires, from 2 oTR(S)Ns, *cells D20* and *D21*. Stimulus consisted of a dark tongue (projected onto the lit visual screen) moving at 200°/s in 1 of 4 orthogonal directions. *Top* to *bottom* in each panel: a schematic of the direction of stimulus motion through the visual field; rasters and histogram obtained from *cell D20* for 10 trials; a trace representing stimulus motion; and rasters and histogram obtained from *cell D21* for the same 10 trials. The 2 neurons had coextensive visual receptive fields (RF) located to the left and down of the fixation point (FP).

Enough data were collected to establish reliably the mean and earliest response latencies for 33 of the 48 LED responsive cells that we tested. The mean of the mean onset latency of each cell (Fig. 5*B*) for this population was 57 ms (SD ± 10 ms, $n = 33$). For the same population, the mean earliest response latency (Fig. 5*C*) was 47 ms (SD ± 9 ms, $n = 33$). These latencies are compatible with other studies of deeper layer neurons in the cat SC (Peck et al. 1980; Syka et al. 1979).

DIRECTIONAL SELECTIVITY. It has been reported previously that visually responsive neurons in the deeper layers of the cat SC have a directional preference for movement of a visual stimulus through their receptive fields (Gordon, 1973; Hoffmann and Cynader, 1976; Sprague et al. 1968; Stein and Arigbede, 1972a; Syka et al. 1979; Talbot et al. 1967). We tested 15 oTR(S)Ns for directional preference in the alert cat, by using a dark, square-edged, tongue-like shape moving at constant velocity across the illuminated screen in the head-fixed condition. This stimulus was chosen because it evoked particularly strong visual responses from TR(S)Ns without eliciting tracking eye movements. For any given neuron, stimulus velocity was kept constant at ~200°/s. We did not attempt to determine whether directional selectivity depended on movement velocity (Stein and Arigbede, 1972a).

Figure 6 illustrates the visual responses of two oTR(S)Ns recorded simultaneously but separately, on two adjacent microwires in the right SC, when the direction of stimulus movement through their receptive fields was varied. Responses in which eye movements were present are not included in these rasters. Shown in each section of Fig. 6 is a schematic depicting the direction of stimulus motion and rasters and summary histograms illustrating the discharge of both neurons for the same 10 trials. The fixation point (FP) is represented by the cross. The visual receptive fields (RF) of both cells appeared coextensive and were located in the lower left visual field. *Cell D20* always had stronger responses than *cell D21* for all directions. Movement of the visual stimulus away from center but parallel to the horizontal meridian (Fig. 6*A*) generated the best responses from both cells. When the direction of stimulus movement was orthogonal to the optimal direction, weaker responses were recorded from both cells (Fig. 6, *B* and *C*). The weakest responses were observed when stimulus movement was opposite to the optimal (Fig. 6*D*). Note that, for this direction of ramp movement, *cell D21* responded only to the very quick resetting movement that brought the stimulus back to its initial position, i.e., high-velocity movement of the stimulus in the optimal direction, and then only very weakly. *Cell D20* also responded to this steplike resetting movement.

In general, we attempted to test cells by the use of eight different directions of motion at 45° intervals. All 15 oTR(S)Ns tested in the alert animal were found to have a preferred direction of stimulus movement. Figure 7 illustrates the directional preference of 11 neurons that were sufficiently tested with at least four different directions of stimulus movement. The magnitude of each response (measured as the number of spikes, averaged from ≥10 trials) is expressed as a percentage of the maximum response and plotted as a function of the direction of movement through the receptive field. The reference direction of 0° corresponds to horizontal, contralaterally directed stimulus motion (H_c), away from the vertical meridian (such as shown schematically in Fig. 6*A*). Movement of the stimulus in the opposite direction (H_i), toward the vertical meridian (Fig. 6*D*), corresponds to +180° (same as −180°). All other directions of stimulus motion lie between 0 and ±180°. Downward (Fig. 6*C*) and upward (Fig. 6*B*) directions of movement were assigned positive and negative angles, respectively.

The directional responses of four cells, with visual receptive fields located on the contralateral horizontal meridian, are plotted in Fig. 7*A*. The optimal direction of stimulus motion for these neurons was contralaterally directed (away from the fixation point) along the horizontal meridian. Figure 7*B* shows the directional responses of seven TR(S)Ns, the visual receptive fields of which were situated in the contralateral hemifield below the horizontal merid-

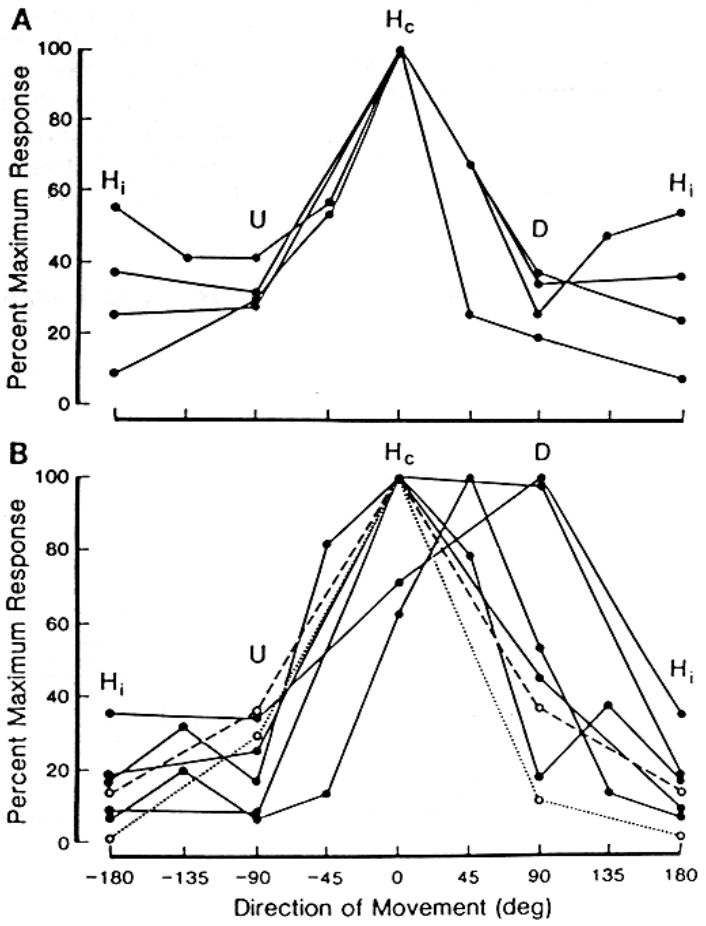

FIG. 7. Plots of percent maximum response vs. direction of visual stimulus motion for 11 oTR(S)Ns tested for directional selectivity. Stimuli moving horizontally, away from the vertical meridian, through a neuron's visual receptive field, and into the contralateral visual field (H_c) were assigned an angle of motion corresponding to 0° (see Fig. 6*A*). Stimuli moving horizontally from the periphery, through a neuron's receptive field, toward the vertical meridian (H_i) had an angle of ±180° (see Fig. 6*D*). Stimuli that were directed downward (D) and upward (U) were assigned positive and negative angles, respectively. *A*: directional preferences of 4 oTR(S)Ns, the visual receptive fields of which were situated on the horizontal meridian. *B*: directional preferences of 7 oTR(S)Ns having visual receptive fields located below the horizontal meridian. Dashed and dotted lines represent directional preferences of *cells D20* and *D21*, respectively.

ian. Two of the cells preferred oblique directions of movement, away from the fixation position; one cell preferred downward motion; and the remaining four cells preferred a direction of visual motion away from the vertical meridian, parallel to the horizontal meridian. The dashed and dotted lines in Fig. 7*B* represent the directional responses of *cells D20* and *D21*, respectively, both of which we have seen preferred contralaterally directed motion parallel to the horizontal meridian.

SILENT NEURONS. Three of the neurons tested for visual responses (see Table 1) did not fire even a single action potential in response to the food target moving through the contralateral hemifield, although this was a very provocative visual stimulus for the majority of cells. A characteristic feature of these "silent" neurons was the low probability that an SD action potential could be elicited after antidromic stimulation of their axons (i.e., low safety factor; see earlier section), suggesting that these neurons were under strong inhibitory influences. These silent neurons, nevertheless, could be influenced by visual inputs because presentation of visual stimuli in the contralateral visual field permitted the SD spike to be seen regularly when antidromic and visual stimuli were applied simultaneously. These observations suggest that the level of excitability of a TR(S)N is an important parameter in shaping a cell's visual response. This point will be considered in greater detail below.

Attentive fixation reduces TR(S)N sensory discharges

The sensory responses of oTR(S)Ns were substantially reduced when the cat fixated a target of interest. To quantify what effect attentive fixation had on the visual responses of these cells, we presented an abstract visual stimulus, moving in the optimal direction through a cell's visual receptive field, under two conditions: *1*) when the cat looked straight ahead at the visual screen or *2*) when the cat, assuming the same gaze position, attentively fixated a food target that was protruded through a small opening in the screen. In the latter condition the animal was rewarded with food for maintaining fixation of the target. In a typical experiment, the two behavioral conditions were alternated after every 5–10 stimulus presentations. Trials were selected for analysis only if the gaze axis was directed straight ahead and the cat made no orienting movements throughout the trial.

Figure 8 shows responses of *cells D20* and *D21* when the stimulus, a dark tongue moving at 250°/s, was presented while the cat's visual axis was directed straight ahead. There was a sharp decrease in the magnitude of the response recorded from both cells when the cat fixated the stationary food target and the identical moving stimulus was presented (Fig. 8*B*).

The effect of fixation on oTR(S)N visual responses was evaluated for 13 neurons. Recall that each of these cells had

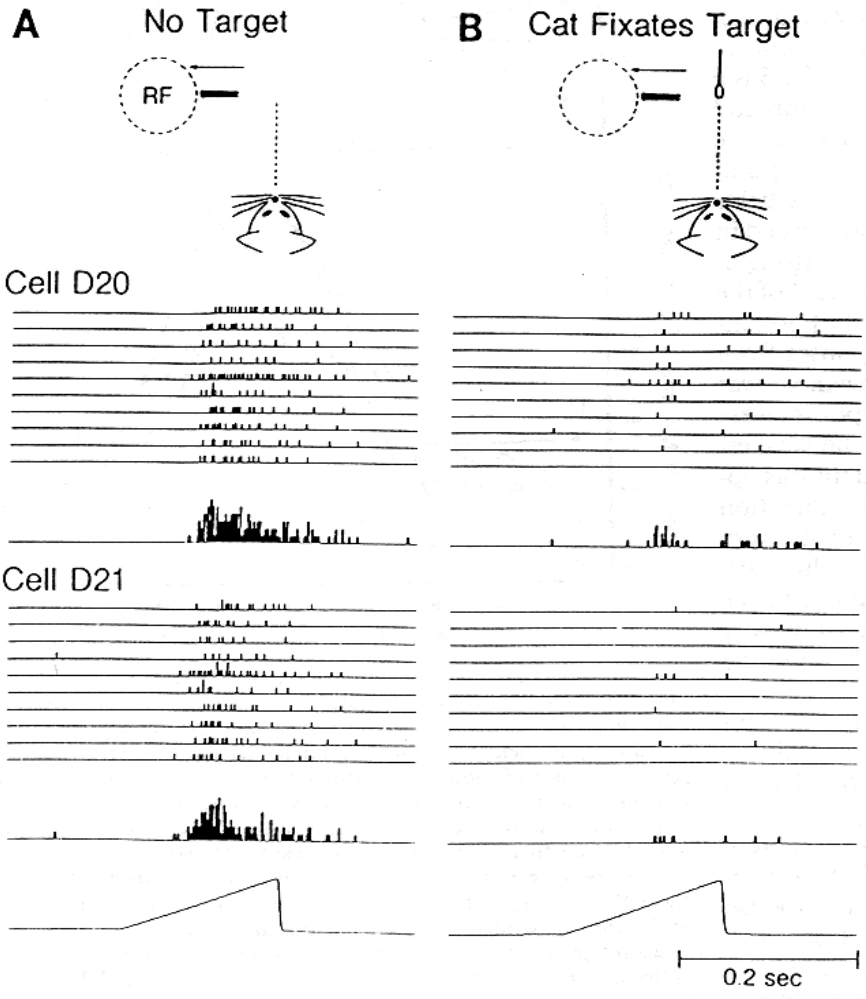

FIG. 8. Effect of attentive fixation on visual responses of *cells D20* and *D21*. Stimulus consisted of a dark tongue, moving at 250°/s on the visual screen. *Top* to *bottom* in each panel: a schematic illustrating the behavioral situation; rasters and histogram obtained from *cells D20* and *D21* for the same 10 stimulus presentations; and a trace representing stimulus trajectory on the screen. *A*: 10 trials, containing no orienting movements, recorded while the head-fixed animal's visual axis was directed straight ahead at the screen. *B*: 10 trials recorded while the head-fixed animal's visual axis was directed straight ahead at a food target protruded through hole in screen. Note that the position of the visual axis, and therefore positions of neurons' visual receptive fields, is the same in *A* and *B*.

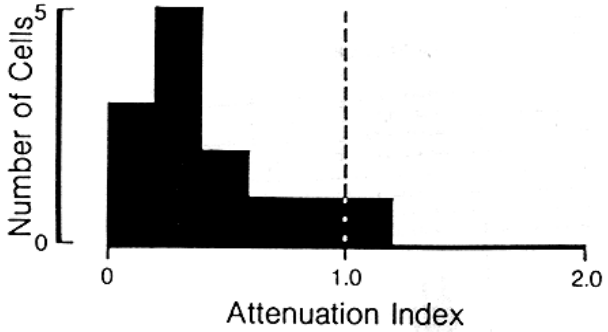

FIG. 9. Histogram summarizing the effect of attention on visual responses of 13 oTR(S)Ns. An attenuation index (see text) was calculated for each cell. This index was defined as the magnitude of the visual response (number of spikes, averaged from ≥10 trials) recorded during attentive fixation (see Fig. 8*B*) divided by the magnitude of the response recorded when the food target was absent (see Fig. 8*A*).

visual receptive fields for which the excitatory region did not include a representation of the area centralis. Therefore the food target was not located in the visual receptive fields of these neurons while it was being fixated by the cat. For each cell we calculated an attenuation index (Moran and Desimone, 1985), defined as the magnitude of the visual response (number of spikes, averaged from ≥10 trials) recorded during attentive fixation of the food target divided by the magnitude of the response recorded when the food target was absent. Any background activity was subtracted from the responses. The histogram in Fig. 9 shows the range of attenuation indexes obtained from the 13 oTR(S)Ns. Twelve of these cells had an attenuation index that was <1. In 10 cells this decrease in response magnitude was statistically significant (t test, $P < 0.01$).

In a few cells we also tested the effect of attentive fixation on sensory responses in other modalities. Figure 10 illustrates the activity of an oTR(S)N, *cell M8*, in response to auditory and somatosensory stimulation. This neuron presented a weak but consistent response (Fig. 10*A*) after presentation of an auditory stimulus—created by grating two objects together to the left of the head-fixed cat—below the horizontal plane, where its auditory receptive field was located (see Fig. 4*B*). During fixation of the food target, the auditory responses recorded from *cell M8* were abolished (Fig. 10*B*), even though the position of the eye, head, and pinna remained constant with respect to the location of the sound stimulus. This same neuron also had a large somatosensory receptive field on the left forelimb (see Fig. 4*C*). A light phasic tap on the left forepaw, which was hidden from the cat's view by an opaque barrier, generated a strong, sustained response from the neuron (Fig. 10*C*). When the cat attended to the food target, the responses of this oTR(S)N to the same stimulus were markedly attenuated (Fig. 10*D*). The reduction of both the auditory and tactile responses of this neuron during attentive fixation was highly significant (t test, $P < 0.005$).

Orienting movements enhance oTR(S)N visual responses

Sensory responses of oTR(S)Ns could be enhanced when the stimulus became the target of an orienting response. This is shown in Fig. 11, *A* and *B*, which illustrates the discharge of two oTR(S)Ns, *cells M7* and *M8*, that were recorded simultaneously but separately, on two adjacent microwires in the caudal right SC, in different behavioral conditions while the cat's head was unrestrained. The two neurons had coextensive visual receptive fields, located in the lower left quadrant of the visual field, so that the LED, when positioned left and down from the point of fixation, activated both cells simultaneously. The LED was attached to one side of a barrier that was positioned in front of the cat. The vertical dashed lines mark the onset of the LED, which then remained illuminated for the duration of each raster. The cat sometimes oriented to the LED (Fig. 11*B*) and sometimes did not (Fig. 11*A*). The filled arrowhead *above* each of the raster displays in Fig. 11*B* marks the onset of the head-free gaze shift to the LED.

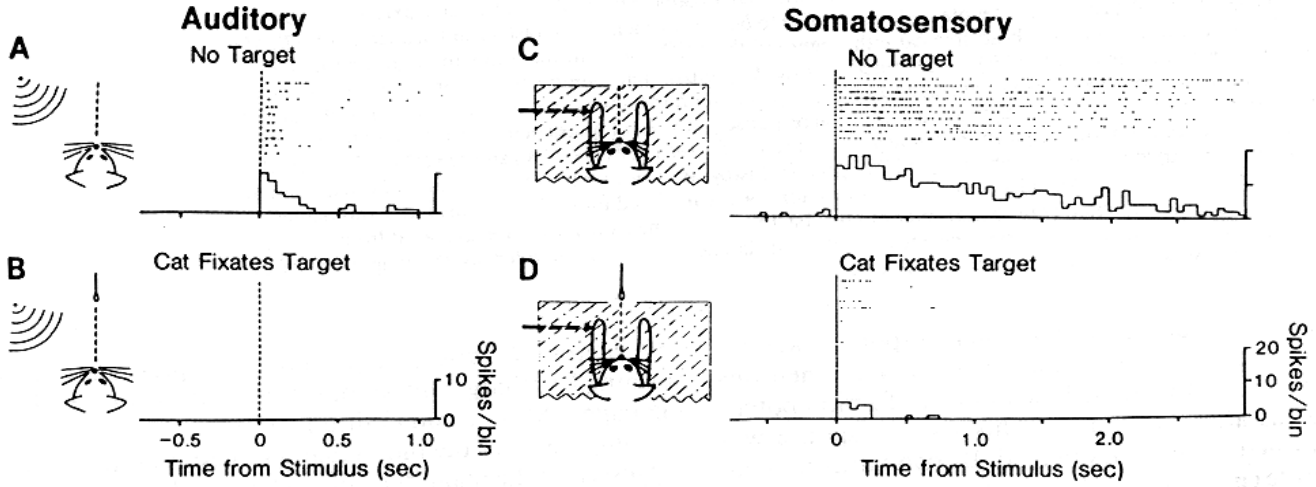

FIG. 10. Effect of attentive fixation on auditory (*A* and *B*) and somatosensory (*C* and *D*) responses of *cell M8*. This oTR(S)N was located in the caudolateral right SC and had its auditory receptive field (RF) located to the left and down of center (see Fig. 4*B*) and its somatosensory receptive field located on the left forelimb (see Fig. 4*C*). Shown in each section is a cartoon depicting the behavioral conditions and 10 rasters and a histogram obtained in the head-fixed condition. Each dot on the raster corresponds to a single action potential. Vertical dashed lines denote stimulus onset, estimated by manually activating a marker pulse while synchronously generating the stimulus. *A* and *C*: cat looks straight ahead at a screen. *B* and *D*: cat fixates food target protruded directly ahead through an opening in the screen.

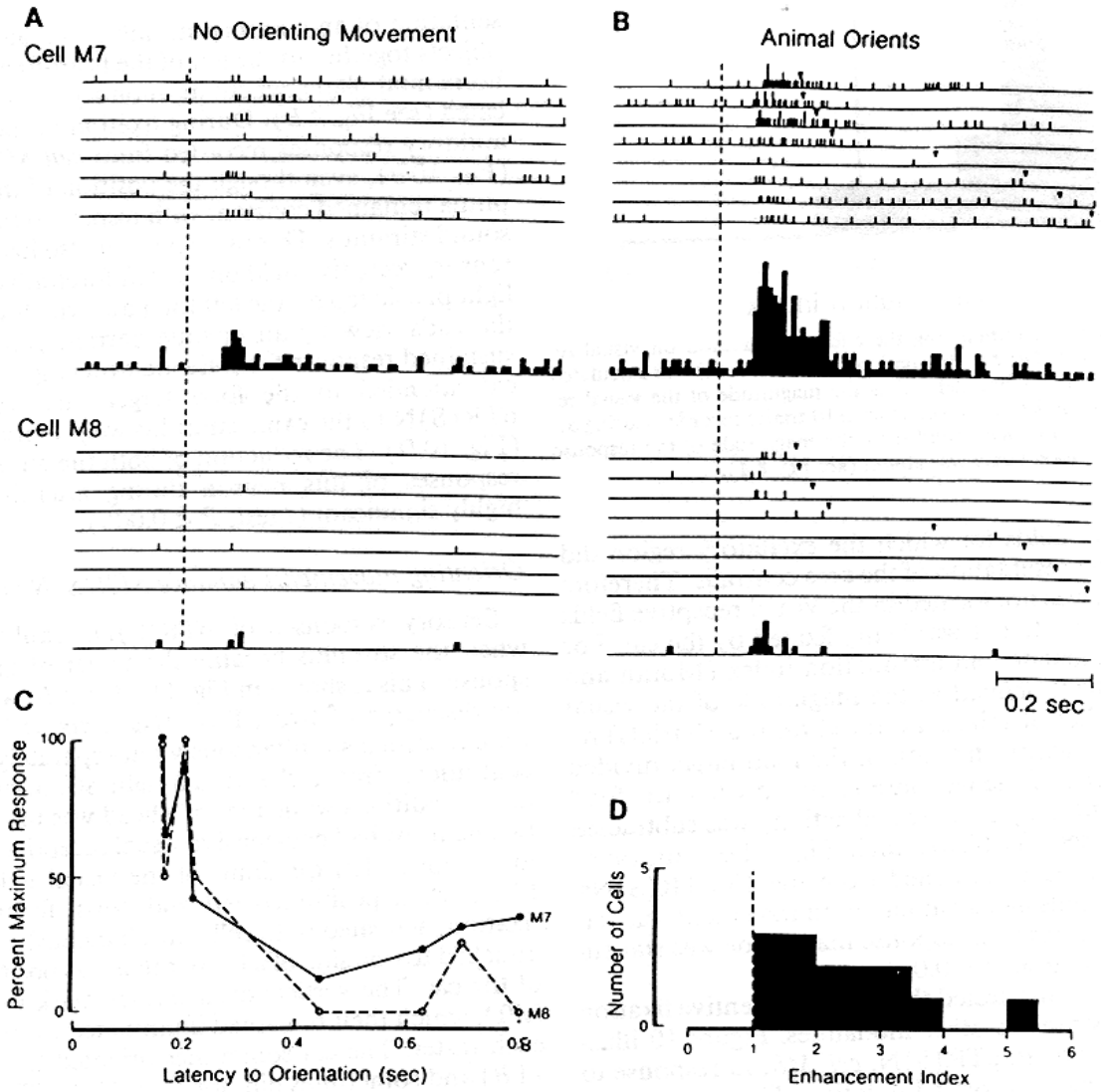

FIG. 11. Effect of whether or not the cat orients to the visual target on the visual responses of 2 oTR(S)Ns, *cells M7* and *M8*, recorded separately but simultaneously on adjacent microwires. Both neurons were located in the caudolateral right SC and had their visual receptive fields confined to the lower left quadrant of the visual field. Visual stimulus consisted of an LED attached to the lower left edge of a rectangular, obliquely oriented, opaque barrier. Animal's visual axis was directed to the upper right edge. Vertical dashed lines denote LED onset. *Top* to *bottom* in each panel: rasters and histogram of *cells M7* and *M8* for the same 8 trials while the head-free cat either ignored the LED and continued looking right and up (*A*) or oriented left and down to fixate the LED (*B*). Occurrences of orienting movements are marked by arrowheads *above* each raster in *B*. *C*: magnitudes of oTR(S)N visual responses were dependent on the animal's response latency. For the same 8 trials presented in *B*, there is plotted the magnitude of the visual response (number of spikes in the 1st 150 ms of the response, expressed as percentage of maximum) against the latency to orientation for *cells M7* (●——●) and *M8* (○——○). Note that response magnitude covaried for these neighboring oTR(S)Ns. *D*: histogram summarizing the influence of orientation on visual responses of 14 oTR(S)Ns. An enhancement index (see text) was calculated for each cell. This index was defined as the magnitude of the visual response (number of spikes in the 1st 150 ms of a neuron's response, averaged from ≥6 trials) recorded during trials accompanied by orienting movements with latencies <1 s (*B*) divided by the magnitude of the neuron's visual response when no orientations occurred (*A*).

The magnitude of oTR(S)N visual responses was affected by the animal's orienting behavior. Both neurons illustrated in Fig. 11 increased the number of action potentials ~55 ms after LED onset. This visual response was enhanced when the animal oriented. By comparison, there was very little change in the latency of the visual response of both cells; visual response latencies decreased by only some 5–10 ms when the animal oriented to the LED. Note that when the animal delayed its orienting response beyond 200 ms, *cell M7* frequently continued to discharge in a sustained manner until the movement was initiated (see *cell M7, bottom 3 traces* in Fig. 11*B*). The properties of this sustained discharge will be considered in detail in the following paper (Munoz and Guitton, 1991).

Comparison of the individual response trials in Fig. 11*B* suggests that the magnitude of a cell's initial visual response is linked to the latency of the orienting response. This is shown more quantitatively in Fig. 11*C*, where response magnitude is plotted against orientation latency. Response magnitude was measured as the number of discharges in the first 150 ms of the neuron's visual response minus background activity (taken as the mean firing frequency in the

300-ms period preceding LED onset), which was generally very weak. Each point represents one trial, the cell's discharge in that trial being expressed as a percentage of its maximum response. The visual responses of both *cell M7* (●——●) and *cell M8* (○——○) increased when the animal oriented to the LED with a short latency. Conversely, the initial responses to LED onset were weaker when the latency of the orienting response was long. Note that the response magnitude of both cells covaried. When the animal's response latency was very short, oTR(S)N visual responses often overlapped movement onset. In the last paper of this series (Munoz et al. 1991), we will show how these phasic visual responses are also, in fact, motor signals involved in shaping movement trajectories.

To compare cells and to further quantify the changes in their visual responses that resulted from orientation, an enhancement index was calculated (Bushnell et al. 1981). This index was defined as the mean response magnitude recorded during all trials accompanied by orienting movements to the LED with latencies of ≤1 s divided by the mean response magnitude recorded in those trials in which no movements were generated. Response magnitude was measured as for Fig. 11*C*. It appeared that orienting movements that occurred with a latency >1 s corresponded to a visual response as weak as when no orienting movement occurred at all. Cells for which fewer than six trials were available in either condition were not included in the calculation. Response enhancement was evaluated for 14 oTR(S)Ns. Figure 11*D* shows, in histogram form, the range of enhancement indexes obtained. All cells had an index >1. For nine cells this increase in response magnitude was statistically significant (*t* test, $P < 0.05$).

Fixation and orientation influence oTR(S)N excitability

The influence of fixation and orientation on the sensory responsiveness of oTR(S)Ns could have been achieved neurally by modulating either the level of TR(S)N excitability or the amount of sensory afferent input onto these neurons. To determine which of these mechanisms was responsible, we devised a simple technique to quantify changes in TR(S)N excitability in an alert behaving animal by studying the characteristics of the antidromically evoked spike in different behavioral conditions. We took advantage of two properties of TR(S)Ns: *1*) antidromic action potentials can be easily fractionated into IS and SD components (Fig. 2); and *2*) SD spike latency is variable (i.e., low safety factor for antidromic activation; see earlier section and Lipski, 1981), presumably due to the SD geometry of the cells (Grantyn and Grantyn, 1982; Grantyn et al. 1983; Moschovakis and Karabelas, 1985). We therefore used twin-pulse stimulation, applied to the PDB, to discriminate between a reduced excitability of a TR(S)N itself and a reduced level of its visual input. When the cat was neither orienting nor attentively fixating, we set the interval between the stimulus pulses to be within the relative refractory period of the SD component of the second antidromic spike (see Fig. 2). We then measured, during fixation and orientation behavior, an "all-or-none" event: the presence or absence of the SD spike in the second antidromic response. This was a much more robust measure of neuronal excitability than that obtained by measuring subtle shifts in SD latency in response to a single antidromic stimulus pulse (Lipski, 1981).

Figure 12 illustrates typical changes in the SD spike latency and refractory period as the excitability level of an oTR(S)N (*cell T2*) was determined in different behavioral conditions. Twin-pulse stimuli were delivered to the PDB at times indicated by the filled arrowheads *beneath* the traces in Fig. 12*E*. In the control state (Fig. 12*A*), the alert,

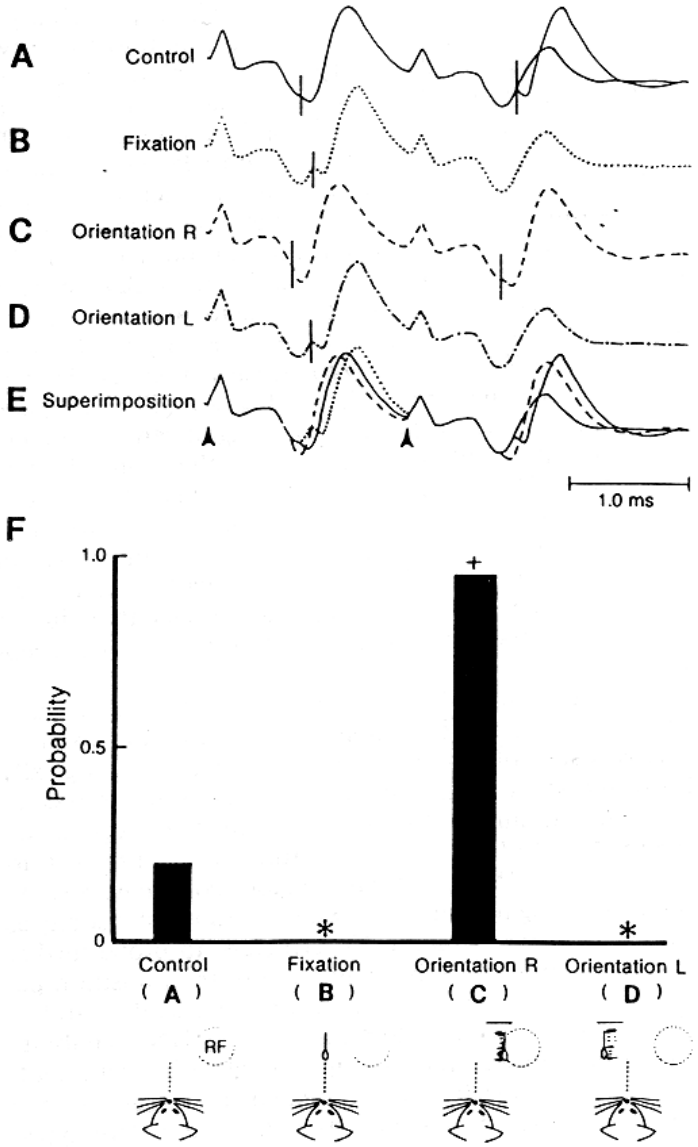

FIG. 12. Neuronal excitability of *cell T2* evaluated, in different behavioral conditions, by the twin-pulse antidromic technique. *A–E*: antidromic spike waveforms were recorded when the PDB was stimulated with twin pulses (see text for details). Arrowheads denote onset of stimulus pulses. Short solid vertical lines on each trace indicate the IS-SD notch. Each trace was obtained in a different behavioral condition. *A*: control; *B*: attentive fixation; *C*: orientation to right; *D*: orientation to left; and *E*: superimposition of *traces A–C*. *F*: quantification of excitability levels in different behavioral conditions, expressed as probability of occurrence of the 2nd SD spike. This probability was significantly increased above control levels when the head-free animal was about to orient right to a food target moving through the neuron's receptive field (denoted by +) and was significantly reduced when the animal was either attentively fixating the food target center or about to orient left to the moving food target (denoted by *).

head-free animal had its visual axis approximately centered on a small 5°-wide rectangular hole in a large blank screen. The first stimulus pulse generated a full action potential consisting of both IS and SD components. The IS-SD notch is indicated by the first vertical tick mark. The second stimulus pulse was delivered 1.7 ms later, an interval that equaled the SD refractory period of this neuron. When present, the second SD spike was delayed relative to the IS spike by a time longer than the IS-SD delay of the first antidromic spike. Thus the antidromic response that followed the second stimulus pulse had a more pronounced IS-SD notch (see 2nd vertical tick mark). For this cell, the probability of observing the second SD spike varied from 0 to 1 in a systematic manner as the interpulse interval was increased from 1.5 to 2.0 ms. In the control state, with a stimulus interval of 1.7 ms, the second SD spike was present on 20% (21/105) of the trials (Fig. 12*F*, column labeled Control).

When the hungry animal fixated a morsel of food protruding through the small hole in the screen, the IS-SD delay was increased after the first stimulus pulse (Fig. 12*B*). Because the first SD spike occurred later, the SD compartment of the cell was in a refractory state when the second antidromic spike arrived, and therefore only the IS spike was observed. The second SD spike was not observed in any of the 67 trials performed during fixation (Fig. 12*F*, column labeled Fixation), which was significantly different from the distribution obtained in the control state [$\chi^2(1,n = 67) = 13.4$, $P < 0.001$]. Similar decreases in neuronal excitability were clearly obtained in three cats from all nine oTR(S)Ns tested in this condition. All of these neurons had visual receptive fields with excitatory regions that lacked a central representation, such that, during fixation, the food target was not within their visual receptive fields.

It might be argued that the above results are due to a generalized increase in inhibition created by the animal's increased state of arousal when it sees food. Teleologically this would seem unlikely because it makes sense to specifically inhibit, during fixation, neurons that act—as do oTR(S)Ns—to break fixation (Munoz et al. 1991). Furthermore, oTR(S)Ns discharge before contralaterally directed orienting responses, for which the animal is surely as aroused as during fixation. Nevertheless, to probe further the changes in oTR(S)N excitability in the transition period between fixation and orientation, we used the antidromic twin-pulse technique to measure oTR(S)N excitability just before contralaterally and ipsilaterally directed orienting gaze shifts: the food target, which the hungry animal was fixating, was moved quickly with a very stereotyped trajectory (target motion was monitored with a search coil) either through the cell's receptive field or in the opposite direction. The latency of the animal's response was very stereotyped and we adjusted the twin pulses to occur in the period 20–90 ms before an orienting response. When the food target was moved through the receptive field of the neuron under study, the level of excitability was increased (Fig. 12*C*). Both the first and second SD spikes occurred earlier than in the control state. Because the first SD spike occurred earlier, there was a longer interval before the second antidromic action potential arrived, and hence the SD membrane was not in a refractory state. For this test, the second SD spike was present in 94% of the trials (Fig. 12*F*, column labeled Orientation R). The probability of this distribution deviating from the control distribution because of chance alone was <1/1,000 [$\chi^2(1,n = 33) = 90.2$, $P < 0.001$]. All 13 oTR(S)Ns tested with this technique were at a significantly higher level of excitability immediately before orienting movements to targets within their visual receptive fields. In Fig. 12*E* there is shown the superimposition of the traces shown in Fig. 12, *A–C*. Note the systematic changes in the latency of the first SD spike as the behavioral conditions were varied.

The food target was also moved ipsilaterally, away from the cell's visual receptive field (Fig. 12*D*). The second SD spike was not present in any of the 44 trials attempted in this condition (Fig. 12*F*, column labeled Orientation L). This was significantly different from the control distribution [$\chi^2(1,n = 44) = 8.8$, $P < 0.005$]. In the tests just described, the level of arousal was presumed to be the same, regardless of whether the animal oriented right or left, yet the level of excitability of the neuron was very dependent on the direction of orientation.

The results in this section lead us to conclude that, during attentive fixation, oTR(S)Ns located in both SCs are at a reduced level of excitability and that it is only in the SC involved in generating an orienting movement that excitation is increased. However, from the above experiment we were not able to determine whether the reduction in excitability was the result of inhibition, disfacilitation, or a combination of these mechanisms.

DISCUSSION

Identification of TRNs and TRSNs

We have described the comprehensive procedures used to electrophysiologically identify TR(S)Ns in alert cats. Grantyn and Grantyn (1982) injected horseradish peroxidase into tectal neurons antidromically activated from the contralateral PDB and upper cervical spinal cord of the anesthetized cat and reported that TRSNs arose from a single class of large, multipolar neurons. Later, Moschovakis and Karabelas (1985) identified two morphologically distinct cell types that projected an axon into the contralateral PDB of the cat. One population, which they called *X cells*, had large somata, large-diameter axons, and no recurrent collaterals back into the SC. This population of neurons represented most of the cells projecting into the PDB and is probably identical to the population of TRSNs described by Grantyn and Grantyn (1982). Moschovakis and Karabelas (1985) also described a second population, which they called *T cells*. These neurons had smaller somata, smaller-diameter axons, a projection to the contralateral SC, and a recurrent collateral back into the ipsilateral SC. Only 3 out of their 16 T cells projected into the PDB.

Most of the neurons that we identified from either the PDB or C_1 had short antidromic latencies, accounting for the bell-shaped portion of the latency histograms in Fig. 3. They appeared to represent a somewhat uniform population of large cells because *1*) they usually could be recorded over a large range of vertical electrode travel through the SC (e.g., up to 300 μm); *2*) when the recording electrode was

positioned close to the cell, the extracellularly recorded spike was very large in amplitude; and *3*) their short antidromic latencies implied that they had large-diameter axons. This is further evidence that these neurons presumably represented the X cells described by Moschovakis and Karabelas (1985) and the TRSNs described by Grantyn and Grantyn (1982).

Neurons that we identified with antidromic latencies >0.7 ms from the PDB (32% of population) or >0.9 ms from C_1 (33% of population) accounted for the skewed portion of the latency histograms shown in Fig. 3. These neurons are presumed to be small because *1*) they were recorded over a narrow range of vertical electrode travel through the SC, *2*) the amplitude of their extracellularly recorded spikes was always small, and *3*) it was assumed that they had small-diameter axons because of their long antidromic latencies. It is tempting to assign these neurons to the T-cell category, but their greater proportion in our hands, compared with the Moschovakis-Karabelas study, suggests that the neurons we identified with long antidromic latencies may have been both T cells and small X cells.

Experimental evaluation of neuronal excitability in the alert behaving animal

The latency of an antidromic response can be used to estimate the direction and approximate magnitude of changes in soma membrane potential, without having recourse to intracellular recording (for review see Lipski 1981). This approach is therefore particularly well suited to evaluate neuronal excitability in alert behaving animals. A particularly useful measure is the time difference between the IS and SD spikes (IS-SD delay). Factors affecting this delay are discussed at length by Lipski (1981). According to this author, the IS-SD delay, which increases when the soma is hyperpolarized, can be significant in some classes of neurons, achieving values as high as 1 ms and ranging from 10 to 100 μs/mV change in membrane potential, depending on the initial value of the delay.

Our "twin-pulse antidromic" technique makes use of the dependence of the IS-SD delay on soma excitability but does away with the need to precisely measure latency. In our approach, the only observation required is the presence or absence of the second SD spike. Thus even casual visual inspection of an oscilloscope trace during an experiment can give insight into the relationship between a neuron's excitability and the animal's behavioral state.

Sensory responses of oTR(S)Ns

CONVERGENCE OF MULTIMODAL SENSORY INFORMATION ONTO ORIENTATION TR(S)Ns. The deeper layers of the SC represent a neural substrate for the convergence of multimodal sensory information (Gordon 1973; Meredith and Stein 1985, 1986b; Stein and Arigbede 1972b; Stein et al. 1976; Wickelgren 1971). Multisensory convergence onto TR(S)Ns has been demonstrated by Meredith and Stein (Meredith et al. 1987; Meredith and Stein 1985, 1986a,b), who found that many of these cells responded best to stimulation in one modality and that stimuli in different modalities, when presented simultaneously, generated complex interactions that could enhance or depress the neural responses. These authors suggested that the neuronal discharge patterns, although not reflecting the precise stimulus features, represented an integration of available sensory information relevant for controlling orienting behavior.

In agreement with Meredith and Stein, our observations suggest that visual, auditory, and somatosensory inputs disperse locally in the TR(S)N layer in a quasirandom manner such that their combined effect varies from cell to cell. Consider these examples. *1*) There were considerable differences between neurons with regard to their visual responsiveness. A few oTR(S)Ns did not yield even a single action potential in response to the most provocative form of visual stimulation. Yet, using our "twin-pulse antidromic" technique, we found these "silent" cells to be depolarized by the visual input. *2*) Other oTR(S)Ns responded much better to sensory stimulation in a nonvisual modality. For example, the visual and auditory responses of *cell M8* were weaker than its somatosensory responses. *3*) We were fortunate enough on three occasions to record simultaneously, on two adjacent microwires, the activity of two oTR(S)Ns with overlapping visual receptive fields. The maximum firing frequency of the response to a given sensory stimulus varied considerably between proximal neurons. Yet, the entire ensemble of active oTR(S)Ns were modulated similarly by the animal's behavioral state.

The visual, auditory, and somatosensory "maps" in the deeper layers of the anesthesized cat's SC are in spatial register (Gordon 1973; Stein et al. 1976; Wickelgren 1971). In agreement with this organization, we have found that, in alert cats and for oTR(S)Ns with multimodal sensory responses, the boundaries of visual, auditory, and somatosensory receptive fields occupied similar regions of space when the cat looked straight ahead. It has been suggested that the deeper layers of the primate SC are organized into motor error coordinates and that the spatial location of receptive fields of acoustically sensitive neurons are modulated by eye position (Jay and Sparks 1987a,b). Put another way, the auditory signal was transformed from head-centered coordinates, the initial frame of reference for localizing an auditory stimulus, into eye motor error coordinates required for the foveation of the stimulus.

Although we tried, we were not able to demonstrate a shift in the location of oTR(S)N auditory or somatosensory receptive fields as gaze, head, and body positions were varied in either the alert or lightly anesthetized cat. Several factors contributed to rendering such results inconclusive: *1*) oTR(S)Ns have large receptive fields with imprecise borders; *2*) oTR(S)N sensory responses show considerable trial-to-trial variability; and *3*) movement of the visual axis far enough to one side or another, to different positions relative to the body or head axes (to study possible shifts in somatosensory or auditory receptive fields, respectively), required use of a target that the alert animal tracked and therefore attended to, a behavioral condition that we have shown profoundly influenced the responsiveness and excitability of oTR(S)Ns.

FIXATION SUPPRESSES oTR(S)N SENSORY RESPONSIVENESS. When the cat fixated a food target, it was much harder to excite an oTR(S)N using either visual, auditory, or so-

matosensory stimuli, provided the visual receptive field of the neuron under study was off the area centralis. Presentation of spatially disparate sensory stimuli has been shown to reduce the sensory responsiveness of collicular neurons in anesthetized and paralyzed cats (Meredith and Stein 1986b; Rizzolatti et al. 1974). These interactive effects between two sensory stimuli may also be linked to the evidence for intracollicular inhibitory connections (Douglas and Vetter 1986), whereby activation of one collicular locus leads to inhibition of neurons at other loci. However, interaction between spatially disparate sensory signals alone cannot account for all of the dramatic attenuation of sensory responses that occurred during fixation of a food target. When behaviorally nonsignificant stimuli (e.g., open hole in the screen, empty spoon, block of wood) were fixated, or when the cat fixated spontaneously about the laboratory, the sensory responses of oTR(S)Ns were not significantly modified. The attenuation in oTR(S)N sensory responsiveness was related to the behavioral significance of the target that was being fixated. During the course of an experiment, the hungry animal was continuously being reinforced with food and therefore, maintained almost constant fixation on the food target when it was present. Because oTR(S)Ns project directly onto the eye and head premotor circuits, their reduced sensory responsiveness during fixation was presumably responsible for decreasing the probability that a peripheral stimulus might evoke an orienting response.

The dramatic decrease in oTR(S)N responsivity during attentive fixation could be the result of a reduction in afferent sensory drive onto the cells, a constant afferent input impinging on cells with lower levels of excitability, or a combination of these phenomena. If the fixation-induced attenuation of oTR(S)N sensory responses also resulted from a decrease in afferent input, then fixation signals would have to interact with sensory signals in each modality independently, before activation of oTR(S)Ns. The deeper layers of the SC receive some direct sensory projections (e.g., retinotectal: Beckstead and Frankfurter 1983; Berson and McIlwain 1982; spinotectal: Wiberg and Blomquist 1984). Indirect evidence suggests that these pathways are not influenced by the behavioral significance of the stimulus to which they respond. Sensory responses recorded in primary visual (Wurtz and Mohler 1976b) and somatosensory (Hyvarinen et al. 1980) cortices of the monkey are not enhanced when the animal attends to the stimulus. When attention is directed elsewhere, there is no attenuation of visual responses in monkey primary visual cortex (Moran and Desimone 1985). Furthermore, several studies have found that visual responses of neurons in the superficial layers of the cat SC are more stereotyped than responses in the deeper laminae (Dreher and Hoffmann 1973; Sprague et al. 1968; Stein and Arigbede 1972a,b; Sterling and Wickelgren 1969), implying that changes in excitability levels of deeper layer neurons may be an important factor in shaping their sensory responses.

ORIENTATION ENHANCES oTR(S)N SENSORY RESPONSIVENESS. Presaccadic enhancement of visual responses was first described by Goldberg and Wurtz (1972b), who demonstrated that about one-half of the visually responsive neurons in the superficial layers of the monkey SC had responses that could be enhanced when a saccade was made to the stimulus. Two forms of enhancement were described an early component, which appeared as a more intensive initial visual response, and a late component, which manifested itself as a prolongation of the visual response. The type of enhancement was dependent on the time of the eye movement relative to the onset of the stimulus target (Wurtz and Mohler 1976a,b). We observed both early and late forms of enhancement on cat oTR(S)Ns. The early component, consisting of a marked increase in the initial phasic response to the visual target, was observed when the cat oriented to the target within 200 ms of its onset. This movement-related enhancement of oTR(S)N visual responses was similar to that previously reported by Grantyn and Berthoz (1985), who also recorded from oTR(S)Ns in alert cats. When the animal delayed its orienting response beyond 200 ms, a prolonged train of spikes continued until the onset of movement. In the third paper of this series (Munoz et al. 1991), we will show how the enhanced phasic responses influence the trajectory of coordinated eye-head orienting gaze shifts. In the next paper (Munoz and Guitton 1991), we will consider the form of enhancement that manifests itself as a sustained discharge with an intensity related to the vector between current gaze position and a target of interest (i.e., gaze position error). This discharge maintains activity at a specific collicular site in the eventual probability that the site will produce the phasic discharge that generates the subsequent movement.

VISUAL RESPONSES. Despite the behaviorally induced variability in sensory responses, it was possible to obtain some detailed information on the characteristics of oTR(S)N visual responses. These could be studied when the animal passively fixated without there being present any soliciting sensory stimulus, such as food. This condition was typically met when the cat kept its visual axis immobile and fixated a large opaque blank screen. In these conditions oTR(S)Ns could be activated by a wide variety of visual stimuli, of which either novel or behaviorally significant stimuli elicited the strongest responses. Precise boundaries of visual receptive fields were difficult to delineate, but they appeared very large, particularly when compared with neurons in the superficial collicular laminae (McIlwain 1975) and with neurons in layer 5 of the striate cortex, which project to the SC (Palmer and Rosenquist 1974). oTR(S)Ns did not appear to code specific features of a visual stimulus, such as size, shape, and orientation. The lack of specificity in oTR(S)N visual responses in alert behaving cats provides further evidence supporting the classic notion that the deeper laminae of the SC are primarily concerned with localizing sensory stimuli that are relevant for the control of orienting behavior rather than with the more elaborate analysis of stimulus features.

oTR(S)Ns were very sensitive to the direction of stimulus motion. All neurons had a strong directional preference for stimuli moving away from the vertical meridian, through their receptive fields, and into the periphery of the contralateral visual field. A similar directional preference has been shown in several studies of unidentified neurons located in the deeper laminae of the cat SC (Gordon 1973; Grantyn and Berthoz 1985; Hoffmann and Cynader 1976; Sprague et al. 1968; Stein and Arigbede 1972a; Syka et al.

1979; Wickelgren 1971). Within the context of orientation control, it is interesting to speculate on the significance of directional preference in these neurons. When a stimulus is moving away from the vertical meridian, toward the periphery, an orienting movement is required to ensure that the stimulus remains on the area centralis. By comparison, when a stimulus is moving from the periphery toward the center of the visual field, an orienting movement may not be required because, depending on stimulus velocity, the stimulus may reach the visual axis within the reaction time.

Overview of factors modulating oTR(S)N excitability

Figure 13 schematically illustrates the main points of this paper. oTR(S)Ns receive multimodal sensory inputs (Meredith and Stein 1985, 1986b) and in turn project on the brain stem and spinal cord premotor centers implicated in gaze control (Grantyn and Berthoz 1985; Grantyn and Grantyn 1982). We have demonstrated that a fixation signal impinges on, and reduces the excitability of, oTR(S)Ns (i.e., cells located off of the area centralis representation) during the behavioral act of attentive fixation, thereby decreasing the probability that a sensory stimulus will evoke an orienting movement. This fixation signal is presumed, but not proven, to be inhibitory.

A further modulatory input onto oTR(S)Ns is shown in Fig. 13 as a cognitive weighting capability, which is necessary if the animal is to attach significance either to a spatial location void of all sensory cues or to one sensory cue rather than another. This anticipatory input enhances the sensory responsiveness of oTR(S)Ns, thereby increasing the probability of a stimulus evoking an orienting movement. We do not know whether this cognitive input impinges directly on oTR(S)Ns or before activation of oTR(S)Ns, is added to visual signals coding the relevant target. A mechanism that explains how intracollicular and cognitive mechanisms affect neuronal excitability will be proposed in the subsequent two papers (Munoz and Guitton 1991; Munoz et al. 1991).

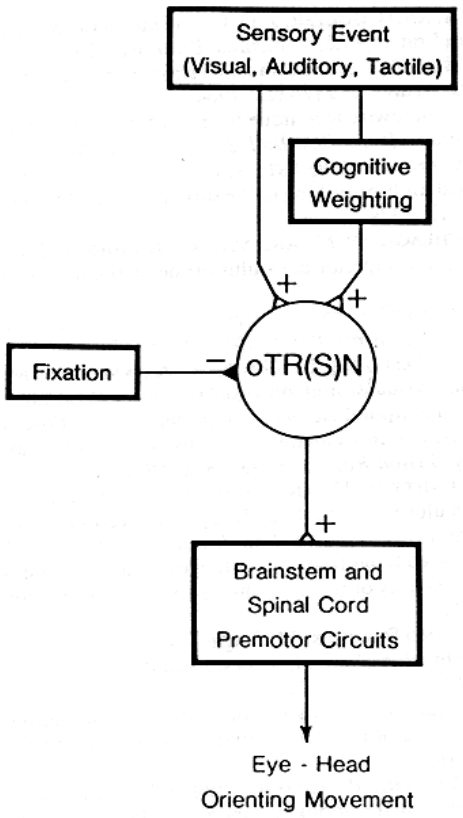

FIG. 13. Schematic overview of main results presented in this paper and illustrating how sensory inputs onto oTR(S)Ns can be modulated to produce desired behavioral responses to sensory stimuli. An important proposal is that the act of attentive fixation directly inhibits oTR(S)Ns.

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