Vibrotactile Coding Capacities of Spinocervical Tract Neurons in the Cat

V. Sahai, D. A. Mahns, N. M. Perkins, L. Robinson, and M. J. Rowe
Department of Physiology and Pharmacology, School of Medical Sciences, The University of New South Wales, Sydney, Australia

Submitted 11 May 2005; accepted in final form 23 November 2005

Sahai, V., D. A. Mahns, N. M. Perkins, L. Robinson, and M. J. Rowe. Vibrotactile coding capacities of spinocervical tract neurons in the cat. J Neurophysiol 95: 1465–1477, 2006. First published November 30, 2005; doi:10.1152/jn.00484.2005. The response characteristics and tactile coding capacities of individual dorsal horn neurons, in particular, those of the spinocervical tract (SCT), have been examined in the anesthetized cat. Twenty one of 38 neurons studied were confirmed SCT neurons based on antidromic activation procedures. All had tactile receptive fields on the hairy skin of the hindlimb. Most (29/38) could also be activated transsynaptically by electrical stimulation of the cervical dorsal columns, suggesting that a common set of tactile primary afferent fibers may provide the input for both the dorsal column-lemniscal pathway and for parallel ascending pathways, such as the SCT. All but 3 of the 38 neurons studied displayed a pure dynamic sensitivity to controlled tactile stimuli but were unable to sustain their responsiveness throughout 1 s trains of vibration at vibration frequencies exceeding 5–10 Hz. Stimulus-response relations revealed a very limited capacity of individual SCT neurons to signal, in a graded way, the intensity parameter of the vibrotactile stimulus. Furthermore, because of their inability to respond on a cycle-by-cycle pattern at vibration frequencies >5–10 Hz, these neurons were unable to provide any useful signal of vibration frequency beyond the very narrow bandwidth of ∼5–10 Hz. Similar limitations were observed in the responsiveness of these neurons to repetitive forms of antidromic and transynaptic inputs generated by electrical stimulation of the spinal cord. In summary, the observed limitations on the vibrotactile bandwidth of SCT neurons and on the precision and fidelity of their temporal signaling, suggest that SCT neurons could serve as little more than coarse event detectors in tactile sensibility, in contrast to DCN neurons the bandwidth of vibrotactile responsiveness of which may extend beyond 400 Hz and is therefore broader by ∼40–50 times than that of SCT neurons.

INTRODUCTION

Tactile afferent nerve fibers from the limbs project to the spinal cord and establish connections with central neurons that contribute to one or more ascending somatosensory pathways that project in parallel to thalamocortical levels of the brain. Perhaps the principal tactile ascending pathway is the dorsal column-lemniscal pathway (e.g., Douglas et al. 1978; Mountcastle 1974) formed by a direct projection of afferent fibers up the spinal dorsal columns to target neurons of the dorsal column nuclei (DCN), whereas others arise in the spinal dorsal horn, for example, as the spinocervical-lemniscal pathway or spinothalamic pathway (Willis andCogghell 1978).

In the case of the dorsal column-lemniscal pathway, the individual DCN neurons display a striking capacity to code reliably for the various parameters of tactile stimuli applied either to the glabrous skin (Connor et al. 1984; Douglas et al. 1978; Ferrington and Rowe 1982; Ferrington et al. 1987a) or to hairy regions of skin (Sahai et al. 2006; Zachariah et al. 2001). The dorsal column nuclei (DCN) neurons display sensitively graded stimulus-response relations as a function of changes in the intensity of both vibrotactile stimuli and static forms of skin indentation, and, in addition, retain a tightly phase-locked pattern of response to vibrotactile stimuli over a broad bandwidth of vibration frequencies extending up to ∼400 Hz (Connor et al. 1984; Douglas et al. 1978; Ferrington and Rowe 1982; Sahai et al. 2006; Zachariah et al. 2001). The high security of synaptic transmission between tactile afferent fibers and their DCN target neurons (Coleman et al. 2003a,b; Ferrington et al. 1987a,b; Gynther et al. 1995; Rowe 2002a,b; Vickery et al. 1994; Zachariah et al. 2001) enables the DCN neurons to retain, in their rates and patterns of impulse activity, a reliable signal of the intensity and periodicity parameters of vibrotactile perturbations encountered in either the glabrous or hairy skin.

Within the parallel spinocervical tract (SCT) pathway there is also reported to be secure transmission between tactile afferent fibers and the postsynaptic neurons of the dorsal horn (Brown et al. 1987a–c; Hongo and Koike 1975), although it appears, at least in the cat, that for this SCT system, and for the spinothalamic system, the tactile inputs may be confined to the hairy skin, with little representation of glabrous skin-associated tactile afferent fiber classes (Brown 1982; Willis et al. 1975). Furthermore, in the cat at least, there is an absence of slowly adapting SCT responses to static forms of tactile stimuli (Brown and Franz 1969) and, in both cat and the macaque, limited vibrotactile responsiveness, although in the macaque most observations were made at the next level of the SCT system, in the lateral cervical nucleus (Downie et al. 1988). However, there may be significant species differences in the characteristics and functions of the SCT system as the primate SCT system is known to receive glabrous as well as hairy skin input (Downie et al. 1988), and has a much slower central conduction speed than does the SCT system of the cat (Anderson 1962; Downie et al. 1988; Mark and Steiner 1958).

In the present study in the cat, we have investigated quantitatively the response characteristics and tactile coding capacities of individual SCT neurons to permit precise comparison with their DCN counterparts (Connor et al. 1984; Douglas et al. 1978; Ferrington et al. 1987a–d; Sahai et al. 2006; Zachariah et al. 2001), in particular, for the coding of information about the frequency and intensive components of vibrotactile stimuli applied to the hairy skin of the limbs.
METHODS

Animal preparation

Experiments were performed on 18 adult cats (2–4 kg in weight) anesthetized, in 10 cases, by intra-peritoneal injection of α-chloralose (70 mg/kg), with subsequent intravenous doses of 7 mg/kg as required, and, for five cats, by intraperitoneal injection of sodium pentobarbitone (35 mg/kg) with later intravenous infusion of 1–2 mg · kg\(^{-1}\) · h\(^{-1}\). In a further three cats, anesthesia was induced with a combination of alfaxonol and alphalodone acetate (Saffan, 18 mg/kg im), a short acting anesthetic, and maintained with gaseous anesthesia (1–3% halothane in a 80%:20% mixture of \(\text{N}_2\):\(\text{O}_2\)) until a mid-collicular decerebration and transection of the spinal cord at C1 were performed. These three different circumstances for general anesthesia were used to determine whether any obvious differences in SCT responsiveness emerged that might warrant more systematic study to elucidate factors responsible for the response behavior of SCT neurons. However, no such differences were apparent.

The cranium was stabilized in an \(~45^\circ\) ventro-flexed position with bars inserted into the external auditory canal and an angled mouth piece support. The lumbar spinal cord was supported by metal clasps attached to the L\(_1\) and L\(_2\) lumbar spinal processes before exposing lumbar (L\(_3\)–L\(_6\)) and cervical (C\(_1\)–C\(_3\)), regions of the cord that were protected under warm paraffin oil. The animal was artificially ventilated at a rate and volume that maintained end-tidal CO\(_2\) in the range of 3–4.5%. Additional stability of the cord was achieved on some occasions by the intermittent use of short-acting (20–30 min) doses of muscle relaxant (gallamine triethiodide, 20 mg), and where necessary by performing a pneumothorax, and/or by covering the lumbar cord with an agar gel (4% wt/vol).

Recording and stimulation procedures

Extracellular recordings were obtained using tungsten microelectrodes (impedance, 2–6 M\(\Omega\)) from the L\(_1\)–L\(_6\) dorsal horn at medio-lateral and dorsoventral locations consistent with Rexed’s (1952) lamina III–V, that is, 900–2,100 µm from the cord dorsum and ~500–900 µm lateral to the midline. This was the case for 46/50 neurons, while the remaining 4 units were isolated in the dorsolateral tracts of the spinal cord in the pathway taken by ascending SCT axons, namely, 100–500 µm lateral to the spinal cord entry zone of the primary afferent fibers (Taub 1964). On isolation of a single neuron, the depth from the cord surface was noted, and the excitatory receptive field mapped using calibrated von Frey hairs. Precise and reproducible mechanical stimuli were delivered, from a mechanical stimulator fitted with circular probes (1–6 mm diam) that were applied to the point of maximum sensitivity within the receptive field of the neuron. To accurately position the stimulator on the skin surface, the hindlimb was shaved and the distal limb supported, with ventral surface uppermost, by molded plasticine.

To study the response characteristics of dynamically sensitive neurons, 1-s trains of sinusoidal vibratory stimuli were delivered normal to the skin surface at frequencies of 5–300 Hz at amplitudes \(\equiv300\) µm. These were usually superimposed on a 1.5- to 2-s background step indentation of 400 µm amplitude and were repeated at intervals of no less than 10 s to allow for recovery of skin position between successive stimuli.

Antidromic identification of spinocervical tract neurons

Dorsal horn neurons were examined for their SCT identity by antidromic activation by means of bipolar stimulation (100 µs pulses, \(\leq10\) mA) from the ipsilateral dorsolateral funiculus (DLF) at the C\(_3\) level, which corresponds to the caudal region of the lateral cervical nucleus in which SCT axons terminate. However, to verify the SCT identity of each neuron according to criteria established in earlier studies (Brown 1981, 1982; Brown and Franz 1969; Taub and Bishop 1965), it was also necessary to employ a second set of bipolar stimulating electrodes, positioned at the C\(_1\) level of the DLF, to confirm that the neuron could not be activated antidromically from beyond the rostral extent of the lateral cervical nucleus. Antidromic activation from C\(_3\) was confirmed with the collision technique (Darwin-Smith et al. 1963; Mackie et al. 1998) in which an orthodromic spike, generated in the SCT neuron by skin stimulation, was able to extinguish the putative antidromic spike for a period, called the interaction time, corresponding to twice the antidromic response latency plus the absolute refractory period for the axon (estimated to be \(~0.5\) ms). A third set of stimulating electrodes was placed more medially on the cord surface at the level of the DCN entry zone to determine whether the SCT neurons were activated trans-synaptically via axon collaterals of primary tactile afferent fibers that provided direct input to the DCN. This was done to determine whether neurons of both parallel ascending pathways, the SCT and the dorsal column-laminal system, received their input from a common subset of tactile afferent fibers.

Analysis of vibrotactile responsiveness in SCT neurons

Procedures for the quantification of the responsiveness of SCT neurons were similar to those described in the associated paper for the study of DCN neurons (Sahai et al. 2006). In brief, the recorded SCT impulse activity was displayed on an oscilloscope and fed to a discriminator from which standardized output pulses were relayed to a counter unit and a laboratory computer for the construction of impulse traces and response histograms from which quantitative measures of phase-locking, based on vector strength values (Sahai et al. 2006), were derived for the vibrotactile responses of SCT neurons.

Testing for GABA-mediated inhibitory constraints on SCT responsiveness

In five experiments, intravenous bicuculline methiodide (2.5 mg/kg) was administered to assess whether GABA-mediated inhibitory processes may limit the responsiveness of SCT neurons to vibrotactile stimulation. In each case, the response of the SCT neuron to precise and reproducible stimuli was tested first, under control conditions, then in the 2- to 20-min period after bicuculline injection, and again later, after recovery from the effects of bicuculline.

RESULTS

Thirty-four tactile-sensitive neurons were isolated for study within the L\(_1\)–L\(_6\) spinal dorsal horn at depths of 900–2,000 µm, consistent with a lamina III–V location. A further four units were recorded lateral to the dorsal horn in the DLF (Taub 1964). All 38 displayed an exquisite sensitivity to brushing of the shaved skin surface and were activated by movement of the cut hairs within their follicles or by puffs of air. The majority of this sample (21/38 neurons) were identified as SCT neurons by means of antidromic activation (Taub and Bishop 1965) from the ipsilateral C\(_3\) level of the spinal cord (see METHODS and Fig. 5A). Conduction velocities for the DLF-projecting axons of the SCT neurons, derived from the antidromic conduction latencies (3–9 ms) and measured conduction distance from the lumbar cord to C\(_3\), were in the range 34–100 m/s (56.5 ± 3.6, mean ± SE), in agreement with earlier calculated values (Brown 1981, 1982; Taub and Bishop 1965).

Receptive fields and adaptation characteristics for dorsal horn neurons

No obvious distinction was apparent between SCT neurons and the remaining unidentified subset of dorsal horn neurons in
receptive fields or functional characteristics. Most neurons isolated (27/38) had well-defined, punctate receptive fields on the hairy skin distal to the knee, often near the margins of the glabrous footpad regions. Others (11/38) had larger fields on the skin of the thigh. Greatest sensitivity and responsiveness, apparent in the bursts of spikes generated by low intensity (≤50 mg wt) von Frey hair stimulation, were found often in a central region of the receptive field, or near the margin for those fields abutting the glabrous skin. We should emphasize that we sought to record many times within the region of the lumbar dorsal horn where, on grounds of topographic representation, one might expect to find representation of the hindlimb glabrous footpad skin. We did this in the belief that others in past studies may have missed this representation in the cat. However, we were unable to find this glabrous representation and our results, in this respect, therefore corroborate the many studies by Alan Brown’s group on SCT neurons of the lumbar cord in the cat (e.g., Brown 1981, 1982; Brown and Franz 1969).

All but 3 neurons in the sample of 38 displayed a pure dynamic sensitivity as reflected in their transient response to the onset and offset components of a controlled 1.5- to 2-s step indentation of the skin delivered at the point of maximum sensitivity within the receptive field. The remaining three showed evidence of some maintained response during the step (see Fig. 7), which could justify their functional designation as slowly adapting tactile-sensitive neurons (see Discussion) (see also Tapper et al. 1983).

Vibrotactile responsiveness of SCT neurons

The sensitivity and responsiveness of SCT neurons to controlled vibrotactile stimulation were routinely tested by applying 1-s trains of vibration at a range of frequencies. The individual SCT neuron the responses of which are illustrated in Fig. 1 was typical of all 21 SCT neurons examined in this way. All responded to the first cycle of the vibration train, usually with a burst of two to six impulses at high frequency. However, in striking contrast to the behavior of DCN neurons that respond throughout the vibration train with high levels of impulse activity (Connor et al. 1984; Douglas et al. 1978; Ferrington and Rowe 1982; Ferrington et al. 1987a–c; Sahai et al. 2006; Zachariah et al. 2001), the SCT neurons could not sustain their response throughout the 1-s vibration train at vibration frequencies >5–10 Hz. In Fig. 1, the SCT neuron responded on each cycle at 5 Hz at an amplitude of 300 μm but responded on only two cycles at the lower amplitude of 100 μm. At 10 Hz, the cycle-by-cycle response could be sustained for only six cycles of vibration and then only at the higher amplitude of 300 μm. At 20 and 30 Hz, in Fig. 1, C and D, the neuron rarely responded beyond the first one to two cycles of the vibration train as was also found when the SCT neurons were tested at vibration frequencies as high as 200–300 Hz.

When stimulus-response relations were constructed by plotting the magnitude of response, in imp/s, against vibration amplitude for a range of vibration frequencies, these quantified relations revealed a very limited capacity of the SCT neurons to signal in a graded way the intensity parameter of the vibrotactile stimuli. In Fig. 2, the relations are plotted for ≤21 different SCT neurons at the four vibration frequencies of 5, 10, 20, and 50 Hz. At the lowest frequencies (5 and 10 Hz, Fig. 2, A and B), there is a coarse grading of the response output over the amplitude range of 100–300 μm, but up to a maximum response level of only ~15–20 imp/s in most neurons. At the higher vibration frequencies of 20 and 50 Hz (Fig. 2, C and D), the grading of output for individual neurons is even coarser and therefore less well able to provide a systematic signal of the intensity change in the vibrotactile stimulus. Indeed, as most of the response at these frequencies was confined to the very onset of the vibration train (Fig. 1), any grading in the stimulus-response relation is largely or entirely accounted for by the different number of impulses in this onset burst and is related to the magnitude of the abrupt, nonsinusoidal onset of this first cycle rather than reflecting, even coarsely, the amplitude of the sustained, sinusoidal vibration train.
Phaselocking and temporal patterning in the vibrotactile responses of SCT neurons

Although the neural coding for the intensive component of vibrotactile stimuli appears to be based on an impulse rate code (see Discussion), the signaling of the frequency parameter may be dependent on phaselocking and temporal patterning of the impulse activity in the primary afferent fibers and their central target neurons (Ferrington and Rowe 1980a,b; Mountcastle et al. 1969; Sahai et al. 2006; Talbot et al. 1968). However, it is crucial, if the impulse pattern is to encode this parameter that the level of response, whether in the individual neuron, or in an ensemble of activated neurons, is high enough to reflect the inherent periodicity of the vibration. The PSTHs in Fig. 3, left, constructed from five consecutive responses at 5, 10, 20, and 30 Hz, for a representative SCT neuron, confirm the inability of these neurons to sustain vibrotactile-induced response beyond 5–10 Hz. However, at these very low frequencies, the impulse activity is phaselocked to the vibration waveform, thus providing a potential signal of vibration frequency in the temporal pattern of activity within the individual neuron, but only for vibration frequencies of ≤10 Hz. Although the CHs in Fig. 3 show relatively tight phaselocking of the impulse activity, with all, or almost all, impulse activity concentrated within less than half the vibration cycle period at all four vibration frequencies, it is clear from the associated PSTHs that, at 20 and 30 Hz, essentially all impulse occurrences are associated with the first cycle of vibration and therefore can provide no useful signal of the vibratory character of the stimulus. Furthermore, this is confirmed in the TIH distributions in Fig. 3, right, that show that almost all interspike intervals at 20 and 30 Hz are concentrated in the first address of the histogram and therefore reflect the short intervals within the burst of spikes discharged on the first cycle of the vibration train. There is virtually no representation of interspike intervals near 33 ms (for the 30-Hz vibration) or near 50 ms for the 20-Hz vibration. However, at 5 and 10 Hz, there is some representation of interspike intervals near 200 ms (for 5 Hz) and near 100 ms (for the 10-Hz vibration) although, even at these frequencies, the most prominent interspike intervals are very short, reflecting the intervals within the burst of impulses occurring principally on the first cycle of vibration (Figs. 1 and 3).

Limitations on the vibrotactile responsiveness of SCT neurons and other dorsal horn neurons

The narrow bandwidth (~5–10 Hz) of vibrotactile responsiveness in SCT neurons could not be attributed to either an anesthetic-induced depression or to a tonic descending inhibitory influence operating on dorsal horn responsiveness as the same response behavior was encountered in SCT neurons sampled in spinal preparations in which the effects of the short-acting, induction anesthetic agent had passed by the time of the electrophysiological recording. These SCT neurons therefore displayed similar frequency-dependent attenuation of their transsynaptic and antidromic responses as was observed in the SCT neurons studied in the anesthetized preparations. It was also found that the three SCT neurons with slowly adapting responses to static skin displacement, which may therefore have received a contribution from SAI or SAIi tactile afferents arising in hairy skin, displayed the same narrow bandwidth of vibrotactile responsiveness as the purely dynamically sensitive SCT neurons. This is apparent for one of these SA neurons in Fig. 4, where A shows a sporadically maintained response to a static step indendation at an amplitude of 400 μm, although at the two higher intensities (800 and 1,200 μm), the response appears to be suppressed for some time after the initial burst response to the step onset. In B, the maintained response to the step (at 400 μm) is again apparent in the absence of vibration (0 Hz) and, when vibration was superimposed on the step, there was some response on most cycles at the low frequencies of 5 and 10 Hz but little at 50 Hz. As the same limited vibrotactile responsiveness was also found in other dorsal horn neurons, which were not confirmed SCT neurons, it appears that the observed response behavior must reflect the fundamental transmission characteristics for dorsal horn synapses, whether for SCT neurons, or for other classes of dorsal horn neurons.
FIG. 3. Response histograms displaying the capacity of a representative SCT neuron for phase-locked, temporally patterned responses to vibrotactile stimuli. In each of the 3 columns, the histograms were constructed from 5 consecutive responses to 1-s trains of vibration at 5, 10, 20, and 30 Hz. In the left-hand column, the peristimulus time histograms (PSTHs) show the response profile throughout and beyond the vibration stimulus, revealing the inability of the neuron to sustain a vibration-induced response at frequencies >5–10 Hz. Middle: the cycle histograms (CHs) have an analysis time corresponding to the vibration cycle period and display the probability of impulse occurrences throughout the cycle period. Right: time interval histograms (TIHs) display the distribution of interspike intervals during the response to the 1-s vibration trains. The arrow below the abscissa within each TIH indicates the fundamental period for the vibratory stimulus.

FIG. 4. Slowly adapting responsiveness in an SCT neuron to static skin displacement. A: traces show responses obtained to a 2-s static skin displacement at the indicated amplitudes. B: response traces for this neuron when a 1-s vibration train, at the indicated frequencies, was superimposed on a 2-s, 400-μm step indentation.
Limitations on SCT responsiveness after antidromic or transynaptic stimulation within the spinal cord

Similar limitations on the responsiveness of SCT neurons were apparent after either antidromic stimulation at C3 or transynaptic activation by means of electrical stimulation at the DCN entry zone of the cervical cord. First, in Fig. 5, the traces in D show responses of an SCT neuron after antidromic stimulation at 5 and 10 Hz, but the antidromic response could not follow stimulation rates at ≥20 Hz, except for the first three or four stimuli (at 20, 100, and 300 Hz). The time of occurrence of the stimulus at the C3 level of the cord is indicated by the regular black marker above each trace. However, SCT units recorded from their axons in the DLC were able to follow at a fixed latency, and with metronome regularity, antidromic stimulation over sustained segments at rates as high as 300 Hz (Fig. 5B). Presumably this differential antidromic following capacity reflects the occurrence of inhibitory processes, instigated by the C3-induced antidromic or transynaptic inputs to the dorsal horn, which operate on the soma of the SCT neuron, limiting its capacity to follow repetitive stimuli.

Most of the tactile-sensitive dorsal horn neurons sampled (29/38) could also be activated transynaptically by electrical stimulation of the cervical cord at the DCN entry zone as could several other neurons for which cutaneous receptive fields could not be found, either because their peripheral input appeared to come from muscle sources, or we failed to identify any peripheral source of input, even though the dorsal horn neurons activated in this way were presumably driven by an antidromically generated volley that traveled down primary afferent axons of the dorsal columns before traversing collateral axonal branches that enter the dorsal horn. All units activated transynaptically in this way, whether recorded in the dorsal horn or DLC, were limited in their capacity to follow repetitive stimulation at rates of <5–10 Hz (Fig. 6). Clearly, therefore there are profound limitations on the responsiveness of SCT or other dorsal horn neurons that are apparent whether the neuron is activated from the periphery, for example, by means of vibrotactile inputs, or by central activation following antidromic or trans-synaptic stimulation.

Mechanisms contributing to the limited vibrotactile responsiveness of SCT neurons

To explore mechanisms that might account for the limited responsiveness in SCT or other dorsal horn neurons, we investigated the hypothesis that potent inhibitory actions are generated within the dorsal horn, soon after the arrival of excitatory inputs, in this case, associated with vibrotactile inputs from the periphery. This was examined by the administration (intravenous) of bicuculline, a blocking agent for the inhibitory neurotransmitter gamma aminobutyric acid (GABA). Its actions were tested on five dorsal horn neurons, three of which were identified SCT neurons, in circumstances in which responsiveness could be quantified prior to and after bicuculline administration. In four, there was some enhancement of responsiveness in association with the bicuculline administration, but in the fifth no effect. In Fig. 7A, the top trace shows the response of an identified SCT neuron to a ramp indentation of the skin, in the control (pre-bicuculline) condition in which a variable number of spikes (usually 1–4) is discharged, at somewhat

![FIG. 5. Impulse traces recorded from confirmed SCT neurons in the dorsolateral funiculus (DLF in A and B) and the dorsal horn (DH in C and D). The impulse traces of A show the extinction of an antidromic spike (●) when an orthodromic spike (□) falls within the interaction period (bottom). Impulse traces shown in B show the capacity of the ascending SCT axon to propagate antidromic spikes at rates ≤300 imp/s, whereas those in D show the limited capacity of the SCT postsynaptic element within the dorsal horn to sustain antidromic invasion beyond 20 Hz. Impulse traces shown in A and C are 50 ms in duration, and 1,000 ms in B and D except for the lowermost traces (300 Hz) where the time scale is expanded 10-fold (100 ms in duration). Marker pulses above the traces in B and D indicate the time of occurrence of stimulation at the C3 level.](http://jn.physiology.org/pdf_images/jn_095_03_1470_0034.png)
variable latency. The filled squares in Fig. 7B plot the mean (± SE) spike count in this response for five repetitions of the ramp at 2-min intervals for 20 min prior to bicuculline injection. After bicuculline administration, there was an increase in the spike number in the response to the ramp (A, middle), an effect the time course of which is plotted in the diamond symbols in Fig. 7B. This enhancement appears to peak ~5 min after bicuculline administration but was significant (ANOVA, P < 0.05) for the 20 min over which the responses were monitored. In the period 50–70 min after bicuculline, the response had returned to pre-bicuculline levels as reflected in both the lowest impulse traces in Fig. 7A and the 50- to 70-min segment plotted in Fig. 7B.
The bicuculline-induced enhancement was also apparent in vibrotactile responsiveness as shown in the impulse traces and stimulus-response relations of Fig. 8. In Fig. 8, A–C, response traces show a higher level of response to each of the three amplitudes of the 5-Hz vibration train with bicuculline (B) than before (A) or after recovery (>45 min) from the bicuculline administration (C). Quantification of the effects on vibrotactile responsiveness of this SCT neuron at four vibration frequencies (5–50 Hz) is shown in the stimulus-response relations of Fig. 8. In Fig. 8, A–C, response traces show a higher level of response to each of the three amplitudes of the 5-Hz vibration train with bicuculline (B) than before (A) or after recovery (>45 min) from the bicuculline administration (C). Quantification of the effects on vibrotactile responsiveness of this SCT neuron at four vibration frequencies (5–50 Hz) is shown in the stimulus-response relations of Fig. 8. For all four relations, responsiveness was significantly elevated (ANOVA, \( P < 0.05 \)) by bicuculline, to impulse rates approximately double those of control values. This was also the case for comparison of responsiveness at each given frequency and amplitude, with the exception of the 10 Hz (100 \( \mu \)m) value (paired \( t \)-test with Bonferroni’s correction).

As the bicuculline was administered systemically, we cannot be certain whether the bicuculline-induced enhancement of SCT responsiveness reflects a blockade of GABA-inhibitory mechanisms within the dorsal horn or a more general effect. To resolve this issue unequivocally would require localized iontophoretic application to the neuron under study. However, as the bicuculline-induced elevation in SCT responsiveness was observed in a spinoally transected preparation as well as in the pharmacologically anesthetized animals, it appears that the bicuculline effect on SCT responsiveness is unlikely to have been of supraspinal origin. Furthermore, as we observed no consistent blood pressure changes associated with the bicuculline effects on the dorsal horn neurons it appears that nonsepspecific vascular effects are also not responsible for the observed effects.

The capacity of the SCT neurons to respond in a phase-locked, patterned way to the vibration stimulus appeared unchanged, for the most part, with bicuculline administration (Fig. 9) as there was little evidence for systematic changes in measures of phaselocking accompanying the modest elevation in response level. This was demonstrated, for example, in Fig. 9, by constructing cycle histograms (CHs) the analysis times (abscissa) of which correspond with the cycle period of the particular vibration stimulus. The CHs use a pulse associated with the onset of each vibration cycle as a stimulus marker and show the time of occurrence of impulses during successive cycles of the vibration train. In Fig. 9, for each of the four vibration frequencies, the pre- and post-bicuculline distributions were superimposed as \( \square \) and \( \blacksquare \), respectively. Quantitative measures of the tightness of phaselocking based on the vector strength, calculated for each CH distribution and expressed as

**FIG. 8.** Enhancement of vibrotactile responsiveness accompanying bicuculline administration. A–C: impulse traces showing responses of an SCT neuron to a 5-Hz vibrotactile stimulus at 3 amplitudes (100, 200, and 300 \( \mu \)m), 1st in the control circumstance (A), 2nd, in the period from 2 min after intravenous injection of bicuculline (2.5 mg/kg; B) and 3rd, after a delay of >45 min after bicuculline injection (C). D–F: stimulus-response relations constructed from responses of the SCT neuron to 5-, 10-, 20-, and 50-Hz vibration at 3 amplitudes (100, 200, and 300 \( \mu \)m) 1st, in the control circumstance (D), 2nd, in the period from 2 min after intravenous injection of bicuculline (2.5 mg/kg; E) and 3rd, after a >45-min delay after the bicuculline injection (F).
the resultant ($R$) (Coleman et al. 2003a,b; Sahai et al. 2006), revealed similar $R$ values at 5 Hz (0.79 and 0.70). At the higher vibration frequencies (10–50 Hz), there was little response in the pre-bicuculine period with most impulse occurrences scattered across the distribution, reflected in the low values of $R$ (0.09–0.26). However, after bicuculine, at 10 Hz, the neuron became responsive with phaselocked activity ($R = 0.58$), whereas at 20 and 50 Hz, the activity increase was minor and dispersed across the cycle period with $R$ values of $\leq 0.2$.

**DISCUSSION**

**Distributed processing of tactile inputs to the CNS**

The observed activation of dorsal horn neurons by dorsal column stimulation near the DCN entry zone was most probably mediated by antidiromic volleys generated in tactile afferent fibers the axons of which bifurcate on entry to the spinal cord and supply a direct axonal projection to the dorsal columns and input to neurons of the dorsal horn. As the great majority (>75%) of the sampled SCT and other dorsal horn neurons could be activated in this way, it appears that common tactile inputs to the central neurons system may be utilized for processing at dual or multiple sites in a series of parallel distributed central systems.

**Regional and functional specialization in the tactile representation within the SCT system**

Despite the apparent common source of tactile input to different parallel ascending pathways, including the dorsal column-lemniscal and SCT systems, there are clearly some regional differences in the cutaneous sources of input to these two systems. Furthermore, there appear to be differences in the functional classes of tactile afferents represented within the two systems. First, in terms of regional differences, our analysis is in agreement with earlier studies in the cat by Brown’s and Willis’ groups in finding little or no evidence of tactile input from glabrous skin regions to neurons of the SCT (Brown 1975; Brown and Franz 1969; Brown et al. 1986) or spinotactato- 

**vibration-induced responses of an SCT neuron.** The cycle histogram distributions in A–D were constructed from responses to 5-, 10-, 20-, and 50-Hz vibration stimuli and, in each case, consist of 2 superimposed CHs. □, constructed as controls prior to bicuculine injection; ■ (with the enhanced response level), constructed in the period from 2 min after bicuculine injection. The quantitative measures of phase-locking based on the vector strength or Resultant ($R$) for the control (pre-bicuculine) CHs and for the CHs constructed after bicuculine injection are indicated for each paired distribution. Each CH contains 50 addresses and was constructed from 5 consecutive responses.

**FIG. 9. Effect of bicuculine injection on phase-locking of vibration-induced responses of an SCT neuron.** The cycle histogram distributions in A–D were constructed from responses to 5-, 10-, 20-, and 50-Hz vibration stimuli and, in each case, consist of 2 superimposed CHs. □, constructed as controls prior to bicuculine injection; ■ (with the enhanced response level), constructed in the period from 2 min after bicuculine injection. The quantitative measures of phase-locking based on the vector strength or Resultant ($R$) for the control (pre-bicuculine) CHs and for the CHs constructed after bicuculine injection are indicated for each paired distribution. Each CH contains 50 addresses and was constructed from 5 consecutive responses.

**representation in the SCT system, at least at the lumbar level of the spinal cord in the cat, is in stark contrast to its prominence in both the cuneate and gracile divisions of dorsal column nuclei (Bystrzyzka et al. 1977; Connor et al. 1984; Douglas et al. 1978; Dykes et al. 1982; Ferrington and Rowe 1982; Gordon and Jukes 1964; Murray et al. 1997; Perl et al. 1962; Zhang and Rowe 1997) and therefore implies a quite marked regional segregation and demarcation in the distribution of tactile processing among central ascending tactile pathways. In particular, the absence of glabrous skin input to the cat SCT system means that the three principal classes of tactile receptors and afferent fiber classes arising in this region of skin in the hindlimb are denied access to this ascending system.

Some further exclusion of input to the SCT system may apply also for certain classes of tactile afferent fibers associated with the hairy skin as SA inputs from this region are absent or sparsely represented (Brown and Franz 1969; present study) in contrast to their prominence within the DCN (Douglas et al. 1978).

**Representation within the SCT system of dynamically sensitive inputs from hairy skin**

Within the cuneate and gracile divisions of the DC-lemniscal pathway, dynamically sensitive tactile neurons can be divided into two functional classes according to their differential sensitivity to vibrotactile stimuli (e.g., Connor et al. 1984; Douglas et al. 1978; Dykes et al. 1982; Ferrington and Rowe 1982;
Murray et al. 1997). This dichotomy applies for inputs from both glabrous and hairy skin and, in the case of the latter (Sahai et al. 2006), consists of one class of neurons most sensitive to low-frequency (≤50 Hz) vibration the input of which comes selectively or predominantly from HFA fibers, and a second class, sensitive to a broader bandwidth of vibration (≥300 Hz) that was driven principally by PC sources (Sahai et al. 2006). In the present study, the vibrotactile-sensitive SCT neurons were each tested over a broad range of vibration frequencies, ≈200–300 Hz; however, all were limited in their responsiveness to the lowest vibration frequencies, ≤5–10 Hz, with no persuasive evidence for any dichotomy of the type observed for DCN neurons. This behavior of the SCT and other dorsal horn neurons studied is consistent with earlier reports that PC inputs are not represented in the responses of either SCT (Brown 1981; Downie et al. 1988) or STT neurons (Willis et al. 1975) of the dorsal horn. However, an alternative explanation (Tapper et al. 1983) for the absence of PC and indeed SA responses in dorsal horn neurons could be that the PC and SA inputs exist but are synaptically curtailed beyond a transient onset response of the SCT neurons.

Bandwidth of vibrotactile responsiveness in SCT neurons: comparison with DCN neurons

As none of the SCT or other dorsal horn neurons was able to sustain its response to the 1-s vibration trains much beyond the first cycle at vibration frequencies >10 Hz, the SCT system is effectively limited in its bandwidth of vibrotactile responsiveness to no more than 5–10 Hz. These observations support the view that the limited vibrotactile responsiveness of lateral cervical nucleus (LCN) neurons in the macaque monkey (Downie et al. 1988) can be attributed to synaptic transmission characteristics at the prior dorsal horn relay of the SCT system as was suggested by preliminary observations on a small sample of SCT neurons in the Downie et al. study (1988). The behavior of SCT neurons represents a dramatic difference in functional properties from those of neurons in the parallel, dorsal column-lemniscal pathway, the vibrotactile bandwidth of which may extend beyond 400 Hz and is therefore effectively ≥40 times broader than that of SCT neurons (Connor et al. 1984; Douglas et al. 1978; Ferrington et al. 1987a–c; Ferrington and Rowe 1982; Sahai et al. 2006; Zachariah et al. 2001). Indeed, some DCN neurons retain significant responsiveness over the whole range of human vibrotactile sensitivity, up to ~1,000 Hz (Connor et al. 1984); this is consistent with them having a crucial role as part of the neural substrate for this aspect of sensory performance (Douglas et al. 1978; Rowe 1990a,b).

Coding for the intensive parameter of vibrotactile stimuli by SCT neurons: comparison with DCN neurons

On account of their narrow bandwidth of vibrotactile responsiveness, the SCT neurons are clearly very limited in their capacity to signal reliably, information about the intensive or pitch parameters of vibrotactile events.

In the case of both primary tactile afferent fibers and DCN neurons, the coding for the intensity of vibrotactile stimuli appears to be based on the total impulse traffic in the population of responding neurons with two aspects of neural responsiveness contributing to the grading or scaling of this signal as a function of intensity changes (Douglas et al. 1978; Johnson 1974). First, the individual fibers or DCN neurons tend to display a sensitive grading of response (in imp/s) over a range of intensities, and second, as intensity increases, there is a progressive recruitment of additional receptors, afferent fibers, and, in turn, central neurons as the stronger stimuli spread further in the skin. However, the present results reveal that for individual SCT neurons, the response levels are very low and stimulus-response relations display only a very coarse and poorly graded signal of vibration intensity, suggesting that any scaling of this intensive parameter in the SCT system, in terms of impulse rate, would be limited almost entirely to the contribution based on progressive recruitment across the population of receptors, afferent fibers, and central SCT neurons. Signaling of intensive changes in vibrotactile disturbances is therefore likely to be vastly poorer over the SCT system than in the DC-lemniscal pathway.

Limitations on responsiveness and vibrotactile frequency coding in SCT neurons: comparison with tactile afferent fibers and DCN neurons

The failure of SCT neurons to maintain high levels of responsiveness throughout the 1-s segments of vibrotactile stimulus trains cannot be attributed to limitations in the response behavior of the HFA fibers. These input fibers are known to respond well to trains of vibration at frequencies up to ~100 Hz (Merzenich and Harrington 1969; Zachariah et al. 2001). In addition, the DCN target neurons of the HFA fibers respond sensitively over a similar frequency bandwidth (Sahai et al. 2006). Furthermore, the DCN target neurons of the HFA fibers are able to respond with very high security of transmission even when the input is derived from a single HFA fiber or single fibers of other tactile afferent classes including the HFA, PC, SA1, and SAII classes (Ferrington et al. 1987a,b; Gynter et al. 1995; Rowe 2002a,b; Vickery et al. 1994; Zachariah et al. 2001). In addition, the DCN responses to vibrotactile stimuli can retain great precision in the temporal patterning of their spike activity in a way that reliably reflects and signals the periodicity of the vibrational stimulus.

In stark contrast to this behavior of neurons in the DC-lemniscal pathway, the response levels in the SCT neurons are far too low, even in the presence of bicuculline-mediated antagonism of GABA inhibition to allow any equivalent capacity for coding the frequency or “pitch” parameter of vibrotactile events in an impulse pattern code as this temporal patterning of SCT activity was never maintained throughout the vibration stimulus at frequencies above ~10 Hz. Whether additional dorsal horn inhibitory processes generated by the afferent input and dependent on mediators other than GABA, for example, glycine or adenosine triphosphate (De Koninck and Henry 1992; Salter and Henry 1987), might also contribute to the limitations on SCT responsiveness is unclear.

The dramatic difference in functional properties between the SCT and DCN neurons is somewhat surprising as many earlier reports had identified a high security of transmission between HFA fibers and SCT neurons, based, for example, on the observation that single HFA fibers can generate complex mono- and polysynaptic EPSPs leading to spike output from the SCT neuron (for example, Brown et al. 1987a–c; Hongo 2001). In addition, the DCN responses to vibrotactile stimuli can retain great precision in the temporal patterning of their spike activity in a way that reliably reflects and signals the periodicity of the vibrational stimulus.
stimulus has occurred, though it is no longer possible to specify system lesions is the capacity to recognize that a mechanical vibration frequencies, the phase-locking of responses to vibrotactile stimuli in SCT neurons would be considerably poorer than in DCN neurons, and therefore the capacity of the HFA fiber-SCT neuron linkage to convey information about the frequency parameter of vibrotactile disturbances in a temporal pattern code would be inevitably poorer than that of the HFA fiber-cuneate neuron linkages. This was confirmed in the present study, even at low vibration frequencies of 5 and 10 Hz where the quantitative measures of phase-locking are much poorer than those for DCN neurons (Sahai et al. 2006).

Role of the SCT system in tactile sensory signaling

The present study has confirmed the preponderance of phasic responsiveness in SCT neurons that was recognized in earlier studies (e.g., Brown and Franz 1969), characteristics that should equip these neurons to detect and signal the novel or changing features of tactile stimuli. However, the current analysis of SCT responsiveness for controlled vibrotactile events reveals a very limited capacity for signaling discriminative information about the bandwidth, intensity or frequency parameters of these stimuli. Furthermore, the limitations on the precision and fidelity of temporal signaling in SCT neurons, in comparison with DCN neurons, suggests that the SCT system could serve as little more than a coarse event detector in the tactile domain, a role that is further constrained in a regional sense, as its processing seems confined largely to inputs from the hairy skin. The present observations and conclusions, for example, the demonstrated inability of SCT neurons (and probably other classes of dorsal horn neurons) to signal vibrotactile information beyond a bandwidth of 5–10 Hz, are consistent with the finding that primates with DC lesions were unable to discriminate between trains of tap stimuli presented to the skin at frequencies exceeding 10 Hz (Makou et al. 1996; Vierck 1998). The present electrophysiological evidence for the role of the SCT as a coarse event detector provides corroborative support for the earlier behavioral observations that led to Mountcastle’s summary (1974) that “What remains in the mechanoreceptive sphere after large fiber or dorsal system lesions is the capacity to recognize that a mechanical stimulus has occurred, though it is no longer possible to specify its location, intensity or shape”. One might now add that the discrimination of the precise temporal features of mechanical stimuli, whether in simple vibrotactile events or more complex tasks of spatiotemporal tactile discrimination, will also be lost in association with DC lesions of the cord.

Evolutionary and comparative roles of SCT and DC spinal tactile systems

The limited capacities of SCT neurons in these aspects of tactile signaling raise the question of why this ascending system should operate in parallel with the far more discriminative DC system. From an evolutionary perspective, one finds no clear delineation into separate DC and SCT spinal pathways in the evolutionarily early chordates, such as amphioxus. However, both systems appear to be present in amphibians, reptiles and birds, although in some cases their components, for example, the cuneate or gracile divisions of DCN, or the LCN, as the projection target of the SCT system, are said to be present as rather primitive homologues rather than equivalent structures to their mammalian counterparts (Ebbesson 1967; Necker 1989; Norton 1969).

As these evolutionary comparisons are based almost entirely on anatomical assessments, it is unclear whether the dynamic signaling capacities of the SCT and DC-lemniscal systems have remained consistently different across generic and species borders in the way we have found with the cat. Perhaps the existence of both systems represents, at least in the tactile signaling capacities of the SCT system, a form of redundancy in the evolution of sensory systems. Alternatively, subtle differences in projection targets of each system at higher levels of the nervous system may confer some differential and unique role on the two systems.

ACKNOWLEDGMENTS

We acknowledge the technical assistance of C. Riordan and D. Sarno.

GRANTS

This work was supported by the National Health and Medical Research Council of Australia and the Australian Research Council.

REFERENCES


