Dorsal Spinocerebellar Tract Neurons Are Not Subjected to Postsynaptic Inhibition During Carbachol-Induced Motor Inhibition

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Xi, Ming-Chu, Jack Yamuy, Rong-Huan Liu, Francisco R. Morales, and Michael H. Chase. Dorsal spinocerebellar tract (DSCT) neurons in Clarke’s column are not subjected to postsynaptic inhibition during carbachol-induced motor inhibition. J. Neurophysiol. 78: 137–144, 1997. Dorsal spinocerebellar tract (DSCT) neurons in Clarke’s column are not subjected to postsynaptic inhibition during carbachol-induced motor inhibition. The mean amplitude of both monosynaptic excitatory postsynaptic potentials (IPSPs) and disynaptic IPSPs evoked in DSCT neurons following stimulation of group I muscle afferents after the injection of carbachol was similar to that evoked before the injection of carbachol. There were no significant changes in the mean input resistance and membrane time constant of DSCT neurons during carbachol-induced motor inhibition. We conclude that, in contrast to lumbar motoneurons, DSCT neurons in Clarke’s column are not postsynaptically inhibited during carbachol-induced motor inhibition. The present findings also indicate that transmission through the DSCT is not modulated by postsynaptic inhibition at the level of DSCT neurons during carbachol-induced motor inhibition.

INTRODUCTION
Despite the large number of electrophysiological studies of dorsal spinocerebellar tract (DSCT) neurons (see reviews by Mann 1973; Walmsley 1991), only a few reports have discussed the possibility that the activity of these neurons could be modulated during the behavioral states of sleep and wakefulness. A study by Carli et al. (1967) indicated that sciatic-nerve-evoked cerebellar responses are suppressed during the rapid-eye-movement periods of active sleep. In their recent studies, Soja et al. (1995, 1996) reported that the spontaneous spike activity of most DSCT neurons decreases during active sleep when compared with wakefulness or quiet sleep. Because extracellular recording techniques were employed in these studies, these authors were not able to determine whether the decrease of activity of DSCT neurons during active sleep was due to disfacilitation or postsynaptic inhibition.

Electrophysiological studies have indicated that single spinal cord inhibitory Ib interneurons mediate both the postsynaptic inhibition of DSCT neurons and the nonreciprocal inhibition of lumbar motoneurons (Brink et al. 1983a,b; Hongo et al. 1983a,b; Rudomin et al. 1987). Pharmacological study indicates that these Ib interneurons are glycinergic (Rudomin et al. 1990), and there is substantial evidence that indicates that lumbar motoneurons are subjected to a process of glycinerically mediated postsynaptic inhibition during naturally occurring active sleep or carbachol-induced motor inhibition (Chase et al. 1989; Soja et al. 1991; Yamuy et al. 1994). Therefore we reasoned that if DSCT neurons were found to be postsynaptically inhibited after the injection of carbachol, this would be strong evidence indicating that these Ib interneurons are responsible for the postsynaptic inhibition of lumbar motoneurons during carbachol-induced motor inhibition. In the present study, intracellular recording techniques were utilized to examine the activity and electrophysiological properties of DSCT neurons in Clarke’s column in the cat anesthetized with α-chloralose before and during the state of active-sleep-like motor inhibition induced by the injection of carbachol into the nucleus pontis oralis (NPO). Our findings indicate that DSCT neurons were not postsynaptically inhibited during carbachol-induced motor inhibition. We therefore conclude that the population of Ib interneurons that innervate both DSCT neurons and motoneurons does not discharge after the injection of carbachol, which suggests that they are not responsible for the postsynaptic inhibition of motoneurons that occurs during carbachol-induced motor inhibition. An additional conclusion that can be drawn from this study is that sensory transmission within the DSCT is not influenced by postsynaptic inhibition during carbachol-induced motor inhibition. Preliminary results of this study have been reported previously (Xi et al. 1995).

METHODS

Surgical procedures
Experiments were performed on six adult cats weighing 3.0–4.5 kg. Surgical procedures, which were carried out under halothane anesthesia, have been described in detail in the preceding paper (Xi et al. 1997). Briefly, the following left hindlimb nerves were excised at their distal ends and positioned on silver stimulating electrodes: hamstring, triceps surae, and tibial distal to the triceps surae branches. The sciatic nerve, before its division in the popliteal fossa, was also placed on a stimulating electrode. In addition, the left quadriceps nerve was placed in a bipolar cuff stimulating electrode. The spinal cord was exposed, by laminectomy, from segment...
L₅ to S₂ and from segment T₁₂ to T₁₃. The right L₇ dorsal and ventral roots were cut distally to monitor the activity of motoneurons. Spinal and leg pools were constructed with skin flaps and filled with warm mineral oil (37°C). An occipital craniotomy was performed to expose the cerebellum. After completion of all surgical procedures, α-chloralose (60 mg/kg iv) was gradually substituted for halothane over a period of 5 min. The chloralose solution was filtered to produce a more stable anesthetized preparation (Kohlmeier et al. 1996). Supplementary doses of α-chloralose (20 mg/kg iv) were administered throughout the remainder of the experiment, as necessary, to maintain anesthesia. The level of anesthesia was ensured by checking that the pupils were constricted, and that blood pressure and heart rate were stable and did not alter in response to a paw pinch. During the recording session the cat was immobilized with gallamine triethiodide (Flaxedil, 1.0 mg/kg) and artificially ventilated. Cardiovascular and respiratory parameters were maintained within physiological limits (see Xi et al. 1997). All experimental procedures were conducted in accord with the Guide for the Care and Use of Laboratory Animals (7th edition, National Academy Press, Washington, DC 1996).

The data presented in the accompanying paper (Xi et al. 1997) and previous studies from our laboratory (Kohlmeier et al. 1996; López-Rodríguez et al. 1995) have shown that the brain stem–spinal cord inhibitory system that mediates atonia during active sleep can be activated in the α-chloralose anesthetized preparation following the injection of carbachol into the NPO. We therefore utilized this preparation to explore the origin of the premotor neurons that are responsible for the postsynaptic inhibition of lumbar motoneurons during active sleep.

**Recordings and stimulation**

The experimental paradigm and principal synaptic connections of Ib interneurons are shown in Fig. 1A. The recording sessions commenced 2 h after the cessation of halothane administration to ensure the systemic clearance of halothane (Cowles et al. 1968; Yanagida et al. 1975). Intracellular recordings were obtained from antidromically identified DSCT neurons in the left side of spinal cord segments L₃ and L₄ (adjacent to the midline and ~2 mm beneath the surface of the cord). Simultaneously, the cord dorsal potential was recorded with the use of a silver ball electrode that was placed on the surface of the rostral portion of L₇. Intracellular recordings were also obtained from identified lumbar motoneurons. Electrodes used for intracellular recording were glass micropipettes filled with 2 M potassium citrate (tip resistances 20–40 MΩ). The electrodes were connected to a high-input impedance preamplifier (Axoclamp 2A). High-gain (×100) DC and low-gain (×10) DC intracellular activity as well as AC records of cord dorsal potentials and ventral root activity were displayed on an oscilloscope and stored on a video cassette recorder by means of a PCM recording adapter (Vetter, Model 4000).

Peripheral nerves were stimulated with bipolar silver electrodes with the use of pulses 0.15 ms in duration and intensities <2 times threshold for the most sensitive fibers to excite predominantly group Ia and Ib afferents (Asif and Edgley 1992; Hongo et al. 1983a,b; Matthews 1972). A tungsten needle stimulation electrode was inserted into the ipsilateral anterior lobe of the cerebellum and a silver ball stimulating electrode was placed on the dorsolateral surface of the cord at the level of T₁₂-T₁₃. These electrodes were used to stimulate axons of DSCT neurons for their identification by antidromic activation. The Ia monosynaptic reflex, which was used to monitor carbachol-induced motor inhibition, was evoked by stimulation of the right L₇ dorsal root at an intensity just suprathreshold for group I afferents (Morales et al. 1987b; Pereda et al. 1990).

**FIG. 1.** A: diagram of experimental paradigm and of relevant synaptic connections of Ib interneurons. Dorsal spinocerebellar tract (DSCT) neurons in Clarke’s column were penetrated with microelectrodes (1) and identified by antidromic stimulation of the ipsilateral dorsolateral funiculus (2) and the anterior lobe of the cerebellum (3); the peripheral nerves were stimulated with bipolar stimulating electrodes (4) to excite group I afferents. Thick lines: neuronal pathway of inhibition of both DSCT neurons and lumbar motoneurons from the common Ib interneurons. Ib IN, Ib inhibitory interneuron; MN, motoneuron; VR, ventral root. B: identification of a DSCT neuron by antidromic activation and synaptic potentials evoked by stimulation of group I muscle afferents. Antidromic action potentials were evoked following stimulation of the ipsilateral lateral funiculus at the level of T₁₂ (1) or the anterior lobe of the cerebellum (2). Monosynaptic excitatory postsynaptic potentials (EPSPs) were evoked by stimulation of tibial (1.7 times threshold; 3) and sciatic nerves (1.7 times threshold; 4). A spike in B3 is truncated. Note that the monosynaptic EPSP in B4 is reduced by the following disinhibitory postsynaptic potential (IPSP). Disinaptic IPSPs were evoked by stimuli applied to the hamstring (1.7 times threshold; 5) and triceps surae nerves (1.8 times threshold; 6). Top traces: intracellular records from DSCT neurons. Bottom traces: cord dorsum records from L₇ segment of spinal cord. Each trace is an average of 10 trials.

**Data collection and analysis**

The intracellular data reported in this study were obtained from identified DSCT neurons with action potential amplitudes >65 mV. The data were digitized off-line and analyzed with a microcomputer (Macintosh Ici) with the use of specially designed software. The segmental latencies of evoked synaptic potentials were measured from the incoming volleys in the cord dorsal potential to the onset of potentials. The mean spontaneous discharge rate was calculated with the use of samples of spike activity that lasted for a minimum of 5 min with a stable resting membrane potential.
Resting membrane potential values were determined by measuring the difference between the DC potential recorded immediately before and after withdrawing the microelectrode from the cell. antidromically evoked action potentials were utilized to measure spike amplitude. Input resistance was determined by the “direct” method with the use of computer-averaged voltage responses (100 trials) to the injection of low-intensity (1–3 nA) current pulses 50–150 ms in duration, as described in the preceding paper (Xi et al. 1997). Because most DSCT neurons fired spontaneously in this preparation, only hyperpolarizing current pulses were used. Each voltage response to a single current pulse was displayed on a computer monitor before the average of all samples; the trial was discarded if the voltage response was overridden by spontaneous spikes. Determination of the membrane time constant was based on an analysis of the decay phase of the averaged cell membrane voltage change produced by current pulses injected through the microelectrode. This procedure involved successively “peeling” exponential terms with the longest time constant from semilog plots of V or dV/dt versus t (Morales et al. 1987b; Zengel et al. 1985). Means ± SE of measurements were used to express values of the experimental data. The statistical level of significance of the difference between sample means was evaluated with the use of the paired or unpaired one-tailed Student’s t-test (P < 0.05). A one-tailed analysis was used because the present study was undertaken to determine whether DSCT neurons were postsynaptically inhibited during carbachol-induced motor inhibition.

**Carbachol administration**

Carbachol (1 µg in 0.25 µl of saline) was injected into the NPO as described in detail in the accompanying paper (Xi et al. 1997). The induction of motor inhibition, which was monitored by recording the amplitude of the Ia monosynaptic reflex from the right L1 ventral root (Morales et al. 1987b; Pereda et al. 1990; Xi et al. 1997), was deemed to be effective on the basis of a significant reduction in the mean amplitude of the reflex following the administration of carbachol (in the present study there was a mean reduction of 48.5%; number of injections = 6; P < 0.001). The site of carbachol injection was confirmed anatomically by the histological procedures that have been described in the previous paper (Xi et al. 1997).

**RESULTS**

Intracellular recordings were obtained from 40 DSCT neurons. These cells were antidromically activated by stimulation of the ipsilateral lateral funiculus with a mean antidromic latency of 0.9 ± 0.2 (SE) ms (range: 0.6–2.0 ms, n = 40), and of the anterior lobe of the cerebellum with a mean antidromic latency of 3.3 ± 0.3 ms (range: 2.2–5.0 ms, n = 40; Fig. 1, B1 and B2). The mean conduction velocity of these DSCT neurons was 84.8 ± 0.9 m/s, which was calculated on the basis of postfixation conduction distances from the stimulating site in the cerebellum to the recording site in the spinal cord. This value is consistent with data of previous studies (Burke and Rudomin 1977; Lundberg 1964; Mann 1973). These neurons were further characterized by the presence of monosynaptic excitatory postsynaptic potentials (EPSPs) and/or disynaptic inhibitory postsynaptic potentials (IPSPs) that were evoked by stimulation of group I afferents from hindlimb muscle nerves. Examples of these synaptic potentials are presented in Fig. 1, B3–B6. Stimuli of strengths sufficient to activate both group Ia and Ib afferents elicited short-latency EPSPs and/or IPSPs (≥1.8 times threshold). The mean latency of the EPSPs was 1.0 ± 0.1 ms (range: 0.5–1.5 ms, n = 69). On the basis of this short latency, these potentials can be considered as monosynaptic (Eccles et al. 1961; Hongo et al. 1983a). The mean latency of the IPSPs was 2.1 ± 0.1 ms (range: 1.4–3.1 ms, n = 65). This latency, which was ~1 ms longer than that of the monosynaptic EPSPs, was within the latency range of disynaptic IPSPs that has been reported by previous investigators (Hongo et al. 1983a,b; see review by Mann 1973).

**Spontaneous activity of DSCT neurons**

Twenty-one DSCT neurons were recorded intracellularly during control conditions (before the injection of carbachol), 13 were recorded during carbachol-induced motor inhibition, and 6 were recorded both before and during carbachol-induced motor inhibition.

High-gain membrane potential recordings were examined to determine whether large-amplitude spontaneous IPSPs, similar to those that bombard motoneurons during active sleep and carbachol-induced motor inhibition (Chase et al. 1989; Lópex-Rodríguez et al. 1995; Morales and Chase 1982; Morales et al. 1987a,b; Xi et al. 1997), could also be recorded from DSCT neurons after the injection of carbachol. Intracellular records from the same

**FIG. 2.** Intracellular records from a representative DSCT neuron before (A, resting membrane potential: −60.5 mV) and 15 min after injection of carbachol (B, resting membrane potential: −61.0 mV), which was effective in producing motor inhibition. Expanded traces illustrate high-gain records of spontaneous membrane potentials. Spikes are truncated. Note that the pattern of membrane potential activity was not changed during carbachol-induced motor inhibition, i.e., the membrane potential of the neuron was characterized by the presence of large-amplitude (>1 mV) spontaneous EPSPs. B: averaged Ia monosynaptic reflex elicited by dorsal root stimulation (I) during control conditions. D: reflex 15 min after injection of carbachol in same cat. Amplitude of Ia monosynaptic reflex was reduced in B by 43%. Each trace is an average of 20 trials.
DSCT neuron before and during carbachol-induced motor inhibition are shown in Fig. 2. A and C. Before the injection of carbachol, the membrane potential of this and other DSCT neurons was characterized by the presence of spontaneous depolarizing potentials. Some of these depolarizing potentials reached firing threshold and initiated action potentials. During carbachol-induced motor inhibition there was no change in the general behavior of spontaneous synaptic activity. In particular, we did not detect IPSPs in any of the DSCT neurons (19 neurons), which is in clear contrast to the observation that discrete IPSPs bombard lumbar motoneurons during active sleep and during carbachol-induced motor inhibition.

During control conditions, 22 of 27 DSCT neurons (81%) exhibited spontaneous spike activity. The mean discharge rate was 15.6 ± 2.8 spikes/s (range: 1.1–42.5 spikes/s, 22 neurons). An example of the pattern of spontaneous cellular discharges is illustrated in Fig. 3A. After the administration of carbachol, 15 of 19 DSCT neurons (79%) also exhibited spontaneous spike activity (Fig. 3B). The mean discharge rate was 11.0 ± 2.4 spikes/s (range: 2.2–40.3 spikes/s, 15 neurons). These values were not significantly different.

Five of six DSCT neurons recorded before and after the injection of carbachol discharged spontaneously. The discharge rate before and after the injection of carbachol was then compared in each of these DSCT neurons. The mean discharge rate of two DSCT neurons decreased significantly after the injection of carbachol (before the injection of carbachol: 18.9 ± 2.0 and 25.8 ± 1.3 spikes/s; after the injection of carbachol: 10.1 ± 2.2 and 19.5 ± 2.2 spikes/s, respectively; P < 0.05). An example of spontaneous synaptic activity of one DSCT neuron is presented in Fig. 3C and D. The discharge rate of the remaining three DSCT neurons was not significantly reduced after the injection of carbachol (before the injection of carbachol: 23.1 ± 2.6, 9.3 ± 0.6, and 3.7 ± 1.1 spikes/s; after the injection of carbachol: 23.1 ± 1.8, 8.2 ± 1.0, and 3.0 ± 0.8 spikes/s, respectively).

**Evoked synaptic potentials in DSCT neurons**

The amplitudes and latencies of synaptic potentials evoked by stimulation of group I muscle afferents were examined before the injection of carbachol and compared with those after the injection of carbachol. Typical records of the disynaptic IPSPs in the same DSCT neuron before and during carbachol-induced motor inhibition are shown in Fig. 4, A and B. These IPSPs were evoked by stimulation of the hamstring nerve. The IPSP evoked after the injection of carbachol was almost identical to that evoked during control conditions. There was no significant change in the mean amplitude of the disynaptic IPSPs of DSCT neurons (before the injection of carbachol: 3.0 ± 0.3 mV, range: 1.1–6.3 mV, n = 32; after the injection of carbachol: 3.3 ± 0.4 mV, range: 1.3–5.6 mV, n = 23), nor was there any significant change in the mean latency of disynaptic IPSPs recorded before and after the injection of carbachol (before the injection of carbachol: 2.1 ± 0.2 ms, range: 1.4–3.1 ms, n = 37; after the injection of carbachol: 2.2 ± 0.1 ms, range: 1.5–3.1 ms, n = 28).
Input resistance was examined in DSCT neurons recorded before and/or during carbachol-induced motor inhibition. Figure 5, A and B, show sample records showing the voltage responses to the injection of 1-nA hyperpolarizing current pulses into the same DSCT neuron recorded both before and during carbachol-induced motor inhibition. The amplitude of the voltage response of this neuron to a 1-nA current pulse increased from 4.9 to 5.4 mV. The mean input resistance of DSCT neurons was 6.2 ± 0.4 MΩ (range: 4.0–11.3 MΩ, 19 neurons) during control conditions; it was 7.4 ± 1.1 MΩ (range: 3.1–13.5 MΩ, 11 neurons) during carbachol-induced motor inhibition (Fig. 5C). However, the difference was not statistically signifi-

Electrophysiological properties of DSCT neurons

The mean resting membrane potential for DSCT neurons that were recorded during control conditions was −61.8 ± 1.0 mV (range: −55.0 to −70.0 mV, 22 neurons), whereas the corresponding value for DSCT neurons during carbachol-induced motor inhibition was −63.1 ± 1.1 mV (range: −56.0 to −72.5 mV, 18 neurons). These values were not significantly different. In six DSCT neurons that were recorded before and during carbachol-induced motor inhibition, there was also no significant change in the mean resting membrane potential following the injection of carbachol (−61.9 ± 1.8 mV, range: −56.0 to −68.5 mV vs. −63.9 ± 1.7 mV, range: −56.0 to −69.0 mV, before and after the injection of carbachol, respectively).
cant. There was also no significant change in the mean input resistance following the injection of carbachol for DSCT neurons that were recorded before and after the injection of carbachol (5.6 ± 0.8 MΩ; range: 2.9–7.4 MΩ vs. 7.0 ± 1.1 MΩ; range: 3.1–10.2 MΩ, 5 neurons, before and after the injection of carbachol, respectively).

The membrane time constant was also measured in DSCT neurons before and/or during carbachol-induced motor inhibition. There was no statistically significant change in the mean time constant (before the injection of carbachol: 11.3 ± 1.0 ms, 11 neurons; after the injection of carbachol: 12.0 ± 1.2 ms, 8 neurons).

**DISCUSSION**

The present study indicates that the spontaneous and stimulus-evoked synaptic activity of DSCT neurons during carbachol-induced motor inhibition did not change compared with synaptic activity observed during control conditions. These findings contrast with the striking hyperpolarization in motoneuron membrane potential and the appearance of motoneuron-directed IPSPs that occur during active sleep and carbachol-induced atonia.

Large-amplitude spontaneous IPSPs in high-gain recordings from DSCT neurons were not observed and the resting membrane potential of DSCT neurons was not significantly hyperpolarized during carbachol-induced motor inhibition. In addition, the mean amplitude of both the monosynaptic group I EPSPs and disynaptic IPSPs evoked in DSCT neurons after the injection of carbachol was similar to that recorded before the injection of carbachol. Furthermore, the mean input resistance and time constant of DSCT neurons after the injection of carbachol was not significantly different from that before the injection of carbachol. During naturally occurring active sleep or carbachol-induced motor inhibition, there is a significant decrease in input resistance and membrane time constant of α-motoneurons (Morales and Chase 1981; Morales et al. 1987b; Soja et al. 1991; Xi et al. 1997). The decrease in input resistance is the consequence of postsynaptic inhibitory processes (Carlen and Durand 1981; Carlen et al. 1980; Llinas and Terzuolo 1964; Smith et al. 1967). On the basis of the above data, we conclude that DSCT neurons are not postsynaptically inhibited during motor inhibition induced by the pontine administration of carbachol. Because we believe that the injection of carbachol in α-chloralose-anesthetized cats activates the neural system that is involved in the generation of motor atonia during active sleep (Chase and Morales 1990), we were clearly present in these motoneurons. Therefore the data indicate that an active-sleep-like state, vis-à-vis motoneuron inhibition, was induced following the injection of carbachol.

With the use of extracellularly recording techniques, Soja et al. (1996) reported that the spontaneous spike activity of most DSCT neurons (84%) is reduced during active sleep in the chronic intact unanesthetized cat. They further classified the pattern of the decrease in spike activity of DSCT neurons into two types, tonic suppression that is sustained throughout the entire episode of active sleep (type I) and phasic suppression only during active sleep (type II). The authors proposed that the suppression of spike activity of DSCT neurons during active sleep may reflect two processes: disfacilitation and/or postsynaptic inhibition. In the present study we did not observe any evidence of the postsynaptic inhibition of DSCT neurons during carbachol-induced motor inhibition. Therefore the suppression of spike activity of DSCT neurons during active sleep may be due to processes that occur during the rapid-eye-movement phase of active sleep that are not present in the chloralose-anesthetized preparation, or it may be due to disfacilitation. In theory, the disfacilitation of DSCT neurons during active sleep could arise as a result of the cessation of Ia afferent fiber discharge (Kubota et al. 1967), or a decrease in the activity of other spinal cord neurons (Pompeiano et al. 1967), and/or the active-sleep-related withdrawal of descending excitatory influences.

If Ib interneurons, which inhibit both DSCT neurons and lumbar motoneurons, are the inhibitory interneurons responsible for motor inhibition during active sleep or carbachol-induced motor suppression, they should exhibit certain characteristics. For example, large-amplitude spontaneous IPSPs should be present in intracellular recordings from DSCT neurons after the administration of carbachol. In addition, the amplitude of group I afferent-evoked disynaptic IPSPs should be greater after the injection of carbachol because the same group I afferent inputs would be expected to activate a larger number of Ib interneurons. Finally, the latency of disynaptic IPSPs evoked in DSCT neurons should also be reduced because the increase in activity of Ib interneurons would be expected to shorten the latency to spike generation in these interneurons.

From the results of the present study, we conclude that the inhibitory Ib interneurons that make synaptic contact on DSCT neurons are not activated following carbachol administration because 1) no discrete IPSPs were observed in any DSCT neuron after the injection of carbachol at the same time the active-sleep-like specific IPSPs are clearly present in lumbar motoneurons (López-Rodríguez et al. 1995; Xi et al. 1997; present study) and 2) the mean amplitudes and latencies of disynaptic IPSPs evoked by stimulation of group Ib muscle afferents in DSCT neurons before the injection of carbachol were practically identical to those present after the injection of carbachol. Therefore we suggest that the Ib interneurons that inhibit both DSCT neurons and lumbar motoneurons are not likely to be the inhibitory interneurons responsible for carbachol-induced motor inhibition.

Our previous studies have shown that during active sleep or carbachol-induced motor inhibition, following electrical stimulation of the nucleus reticularis gigantocellularis in the
medullary reticular formation, long-latency IPSPs are elicited in lumbar motoneurons (López-Rodriguez et al. 1995; Pereda et al. 1990; Soja et al. 1987; Yamuy et al. 1994). These and other electrophysiological studies (Jankowska et al. 1968; Peterson et al. 1979; Takakusaki et al. 1989, 1994) also indicate that certain projections from the medullary brain stem to lumbar motoneurons are not monosynaptic, but rather involve one or more interneurons. It has also been shown that during active sleep and carbachol-induced motor inhibition the postsynaptic inhibitory potentials that impinge on lumbar motoneurons are mediated by glycine (Chase et al. 1989; Soja et al. 1991; Yamuy et al. 1994). Premotor inhibitory interneurons that use glycine as a neurotransmitter and that are activated during active sleep and carbachol-induced motor inhibition can therefore be considered as candidate neurons for mediating the postsynaptic inhibition of lumbar motoneurons. At the present time, only Renshaw cells (Morales et al. 1988) and, on the basis of the present data, Ib inhibitory interneurons that have axon collateral projections to Clarke’s column, can be excluded as candidate inhibitory interneurons.

There is, however, anatomic evidence to indicate the existence of a direct inhibitory descending pathway from the brain stem to spinal motoneurons (Holstege and Bongers 1991). This finding was based on a study in which ~15% of the terminals of direct descending fibers from the ventromedial lower brain stem to lumbar motoneurons in the rat were found to contain glycine, which indicates the existence of monosynaptic glycinergic inhibitory projections from the lower brain stem to spinal motoneurons. It was suggested that this descending pathway may be responsible for the postsynaptic inhibition of spinal motoneurons during active sleep without the use of spinal cord segmental inhibitory interneurons (Holstege and Bongers 1991).

The DSCT is one of the major ascending sensory pathways that convey sensory information from muscle spindles, Golgi tendon organs, joints, and cutaneous receptors in the lower extremity to the cerebellum (see review by Mann 1973). Via these same afferents, information also reaches the cerebral cortex through axon collaterals of DSCT neurons that project to the dorsal medullary relay nuclei (Asif and Edgley 1992; Johansson and Silfvenius 1977; Landgren and Silfvenius 1971). After the administration of carbachol, most DSCT neurons still exhibited spontaneous spike activity, which suggests that sensory information can be relayed by DSCT neurons during carbachol-induced motor inhibition.

The following conclusions can be derived from the present results: 1) DSCT neurons are not postsynaptically inhibited during carbachol-induced motor inhibition at a time when active-sleep-like specific IPSPs are present in lumbar motoneurons. 2) Ib interneurons that inhibit both DSCT neurons and lumbar motoneurons are not likely to be the inhibitory interneurons responsible for the postsynaptic inhibition of motoneurons during carbachol-induced motor inhibition, and 3) transmission through the DSCT is not modulated by postsynaptic inhibition at the level of DSCT neurons during carbachol-induced motor inhibition. However, these conclusions need to be confirmed in the intact unanesthetized preparation.

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