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Anatomical distribution and response patterns of reticular neurons active in relation to acoustic startle

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A population of reticulospinal neurons with short latency response to startle-inducing stimuli was identified in the nucleus reticularis pontis caudalis (NRPC) and nucleus gigantocellularis (NRGC) of the medial pontomedullary reticular formation. The threshold and magnitude of response to auditory stimuli was correlated in these cells and in the muscles mediating startle. Startle-related neurons were significantly more likely to have high conduction velocity spinal projections than adjacent cells not related to startle. Startle-related cells were not 'dedicated' to startle, but were active in relation to spontaneous movements. Both the unit response of the startle-related cells and the startle response recorded in muscles were suppressed by the prior presentation of a weak prepulse. Thus, prepulse inhibition of startle occurs at, or prior to, the medial pontomedullary reticular formation. We conclude that these reticulospinal cells convey the output of the brainstem system modulating and triggering startle.

The acoustic startle reflex (ASR) in mammals, which is elicited by sudden, intense auditory stimuli, involves a series of rapid, phasic contractions of the skeletal muscles throughout the body[9,27]. Because of its simplicity, the startle response and its inhibitory control can be readily investigated in intact humans and animals. Startle and its modification has been used as a model system for the study of habituation[5,19], sensitization[19], and the effects of drugs[3,4,6] and toxins[24,53] on behavior.

Evidence from several sources has implicated the brainstem reticular formation, especially the NRPC, in startle instigation. Lesions of NRPC reduced or abolished ASR[2,7,8,13,20,22,29], while electrical stimulation of this area produced startle-like responses[7,8]. However, the response properties and distribution of neurons that might be mediating the reflex are unknown. In the present paper, we report finding in the behaving animal a population of cells whose location, projection, response latencies and response magnitudes are consistent with their mediation of the startle response.

The amplitude of the startle response can be greatly reduced by prior presentation of another, usually weaker prestimulus (P) (either auditory, visual, or tactile) at an appropriate lead interval, a robust phenomenon which has been termed 'prepulse inhibition'[23]. This inhibition has been hypothesized to occur either in the brainstem or in the spinal cord[7,28,30]. We find that neural responses to startle-inducing stimuli were suppressed by prestimuli, which indicates that prepulse inhibition occurs at, or prior to, the medial pontomedullary reticular formation in the startle circuit. A preliminary report of some of these findings has been made[45].

Twenty-eight adult cats were implanted bilaterally with chronic mechanical microdrives, containing bundles of microwires (32 μm diameter) aimed at the

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medial pontomedullary reticular formation and/or midbrain (0.5–2.5 mm lateral to midline), and with fixed electrodes for the recording of electroencephalogram (EEG), electrooculogram (EOG), lateral geniculate (LGN) spikes, and neck electromyogram (EMG)[41]. (EEG, EOG, and LGN waves were utilized for the identification of animals' sleep-waking states.) EMG responses were measured in the splenius, biventer cervicis, and complexus neck muscles. For purpose of quantification, EMG amplitudes were averaged and integrated over 50–100 ms following stimulus onset after full-wave rectification.

Broad-band noise stimuli, having a 20 ms duration and a 1 ms rise/fall time, were gated through a Grason-Stadler 829E electronic switch, amplified, then delivered through two Mitsubishi SG-69TB 3-way midrange loudspeakers. The two speakers were located on diagonally opposite ends of the top of the recording chamber (58 × 56 × 86 cm internal dimensions) about 50 cm from the animal's head. The ambient noise level inside the chamber was 55 dB SPL (re. 20 μN/cm^2, A Scale). Prestimuli with an intensity between 70 and 100 dB were generated by amplifying a 0.2 ms pulse and feeding the resulting signal to a Soundolier C803 loudspeaker situated between the other two speakers. Calibration of stimulus intensity was carried out with a Bruel & Kjaer Type 2209 Impulse Precision Sound Level Meter. The standard stimulation sequence was 50–200 trials of startle-inducing stimuli with or without prestimuli. Inter-stimulus interval (ISI) was varied between 40 and 60 ms, and the inter-trial interval between 10 and 40 s. The order of stimulus presentation was counterbalanced between sessions to avoid systematic changes in response due to habituation, a shift in posture or order effects. All stimuli were delivered in quiet waking state, as determined by polygraphic and visual observation. A MINC (LSI-11) computer averaged 8 channels of muscle/unit response. A second MINC computer was employed simultaneously to generate spike train statistics and peristimulus time histograms.

Auditory response was examined by presenting clicks or white noise bursts between 70 and 120 dB. Threshold, discharge pattern, and latency of responsive units were then determined. To measure a unit's auditory threshold, stimulus intensity was incremented or decremented in 10 dB steps, and 50 trials were run at each intensity level. Unit threshold was defined as the lowest intensity in which discharges were significantly ($P < 0.05$) increased from the spontaneous activity level, following the statistical method of Syka and Radil-Weiss[46]. A battery of tests was employed to identify each unit's behavioral correlate[39,40,42,43]. The locations of recorded units were determined as previously described[41].

Spinal projections were determined with chronically implanted bipolar stimulating electrodes in the ventral funiculus at the second cervical (C_2) and/or the first lumbar (L_1) spinal cord levels. Two electrodes were implanted 3.7 mm ventral to the dorsal surface and 0.5 mm lateral to the exit of dorsal roots, one on each side, and one midline electrode was placed 4.0 mm ventral to the dorsal surface. Spinal stimuli were 0.2 ms pulses up to 1.0 mA in intensity. Antidromic responses were identified according to the general criteria: fixed latency (<0.1 ms variation), high frequency following (400–800 Hz), and collision with orthodromic spikes. Latencies for antidromic responses were measured with stimulus intensities at 1.5 times the excitation threshold. Axonal conduction velocities were calculated from the latency differential between L_1 and C_2 antidromic responses. Because not every RS neuron projected down to the lumbar cord, for purpose of statistical comparison between auditory-responsive and non-responsive cells, response latencies to C_2 stimulation were used.

We found that 26.5% (105 out of 396) of medial reticular neurons were excited by startle-inducing acoustic stimuli. Fig. 1 shows the oscilloscope tracings (Fig. 1A) and the averaged response (Fig. 1B) of two pontine startle-related units and that of the biventer muscles recorded simultaneously. The threshold for neck EMG startle was between 90 and 100 dB in well-habituated, waking cats. Consistent with a previous report[47], EMG responses were usually biphasic or triphasic, with an initial excitation followed by suppression and/or a late excitation. The actual pattern of the EMG response was, however, dependent on the particular muscle group recorded, the posture, and the arousal state of the animal during stimulation. The response latency was usually shorter and the amplitude larger in the alert state as compared to that in sleep states, and in active muscles as compared to that of the relaxed muscles. The latency

for neck EMG startle response in the alert cat was between 6 and 12 ms, and the duration was between 5 and 15 ms at 110 dB. The pattern of unit discharge to the startle-inducing stimulus was similar to that of the neck muscle EMG response (Fig. 1B). The response thresholds for medial pontomedullary cells were in

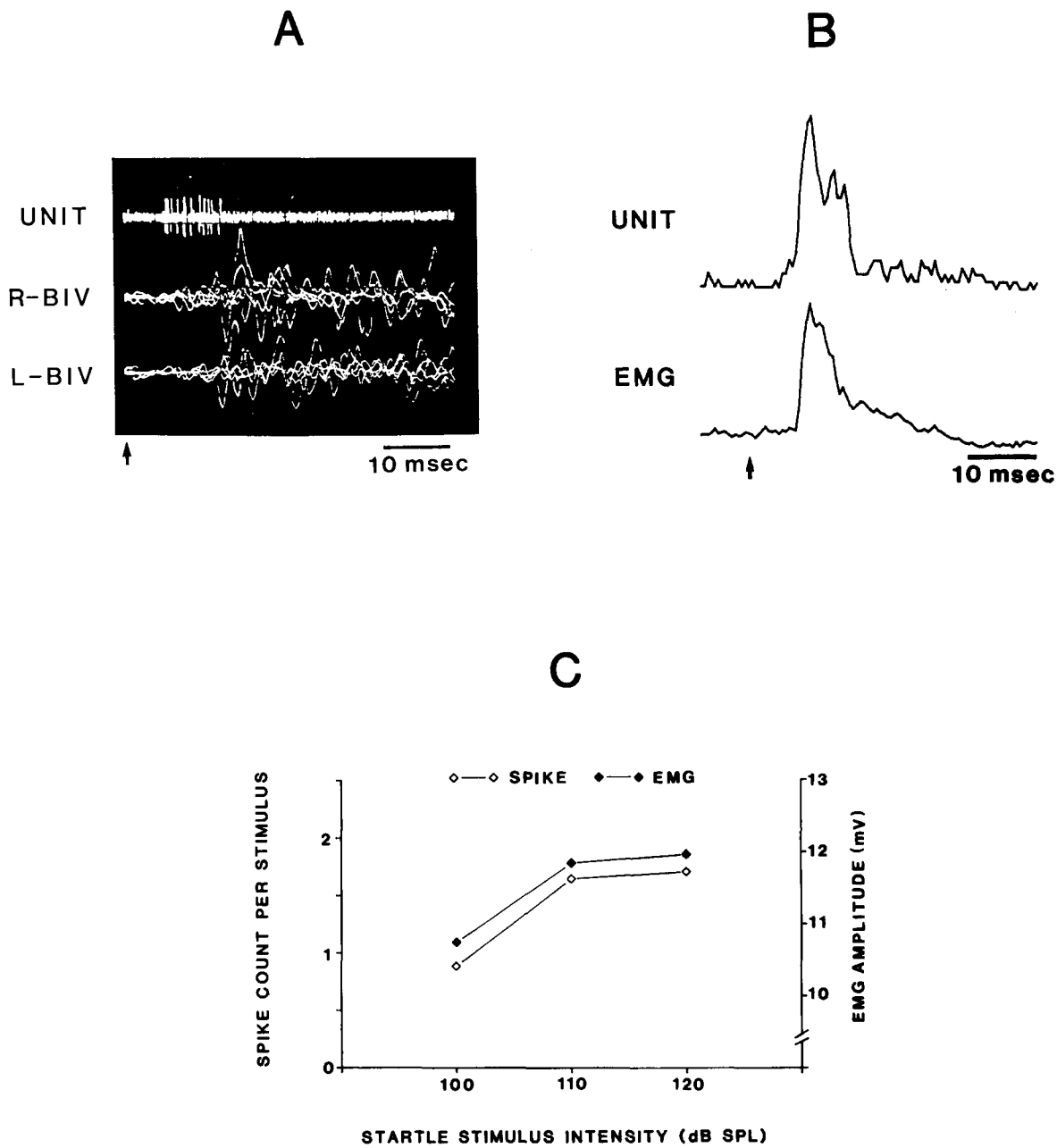

Fig. 1. A comparison of unit and neck EMG responses to startle-inducing stimuli. A: oscilloscope tracings of unit and EMG (left and right biventer) responses to a 120 dB click stimulus superimposed over 6 sweeps. This RS unit in left NRPC discharged during spontaneous ipsilateral and contralateral head movements. It had an estimated conduction velocity of 95 m/s. B: unit and EMG (left complexus) responses to a 120 dB, 30 ms tone pip (1.5 kHz) averaged over 200 trials while the animal's neck was voluntarily flexed to the ipsilateral (left) side of the unit. This RS unit in left NRPC discharged to ipsilateral active and passive head movements. The estimated conduction velocity was 135 m/s. C: parallel increase in unit discharge and integrated EMG amplitude as stimulus intensity increases from 100 to 120 dB. EMG and unit data were averaged over 50 trials. EMG values were integrated over 50 ms following stimulus onset. Cell discharge was taken as the average number of spikes within 30 ms of stimulus onset.

the same range as that for the neck muscle startle response; most cells did not respond to weak or slow-onset stimuli including human voice and other laboratory noises, and only responded to loud hand-claps and startle-inducing stimuli over 90 dB. Of the 12 units for which parametric threshold determinations were made, 10 had thresholds greater than 90 dB (range from 90 to 105 dB), while the other 2 had thresholds below 80 dB. Startle-related cells were tested for response to stimuli between 90 and 120 dB. All of these cells showed a strong correlation between stimulus and unit response magnitude as did the motor response to startle. This relationship is illustrated in Fig. 1C, which shows a parallel increase in unit discharge and EMG amplitude as the intensity of the eliciting stimulus increased from 100 to 120 dB.

Cells with short latency response to startle-inducing stimuli were distributed over a large portion of the medial pontomedullary brainstem, ranging from NRPC to NRGC, between P4 and P11. Cells were located between 0.8 and 2.2 mm lateral to the midline. Fig. 2 shows the anatomical distribution of these units and their corresponding response latencies. The response latencies for these units, which ranged from 3 to 52 ms, averaged 12.1 ms. Seventy-one percent (75 of 105, average latency 8.2 ms) responded to startle-inducing stimuli with a latency less than 19 ms, which is well within the duration of the startle response.

The majority of startle-related cells discharged in relation to spontaneous movements. We were able to identify the behavioral correlates of 97 of the 105 responsive cells recorded. Sixty-six percent (64 of 97) of these cells were related to axial movements involving head, neck or other parts of the spinal column. Other startle-related units discharged during multiple facial movements (n = 8), visual and somatosensory stimulation (n = 12), limb movements (n = 6), multiple head and neck movements (n = 6), or jaw

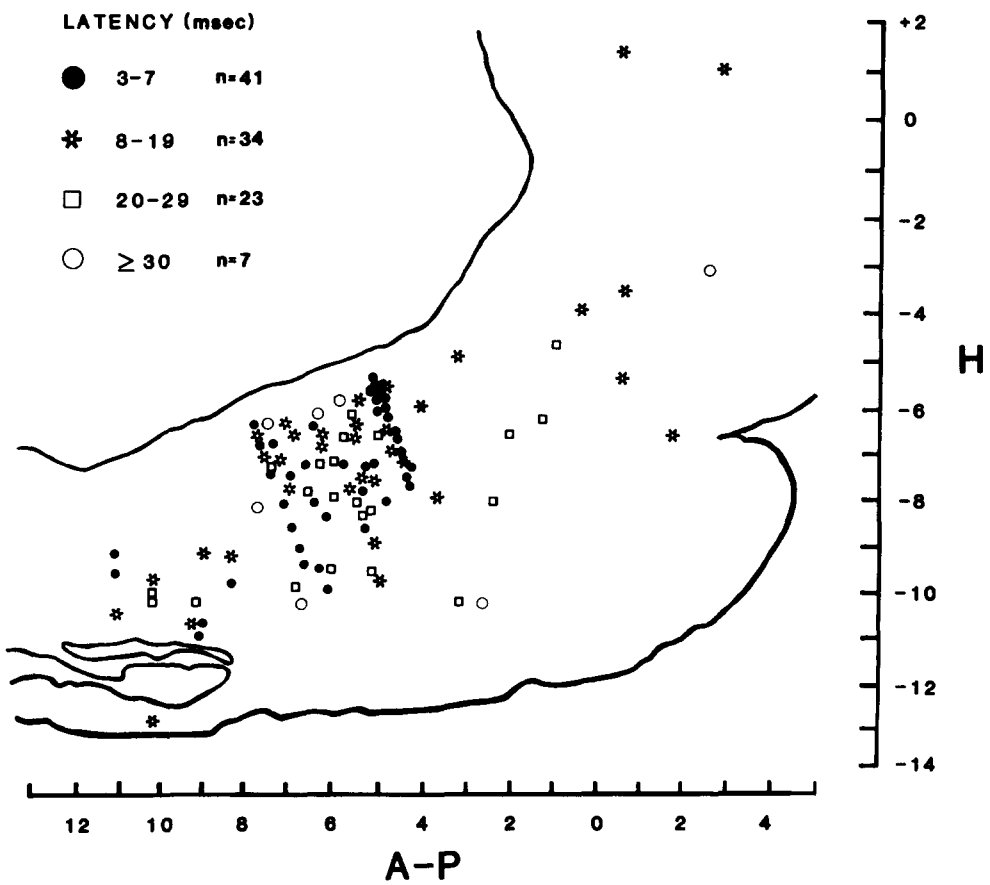

Fig. 2. A schematic drawing of the brainstem reticular formation showing the distribution of auditory responsive units and their corresponding response latencies.

closing ($n = 1$)[42,43]. None of the cells related to single facial movements, eye movement, or tongue movement, responded to startle-inducing stimuli.

When the animal's body was curved to one side, the side with greater EMG tone had a larger magnitude and shorter latency response to startle stimuli. The response of tonically active axial movement-related cells to startle-inducing stimuli also depended

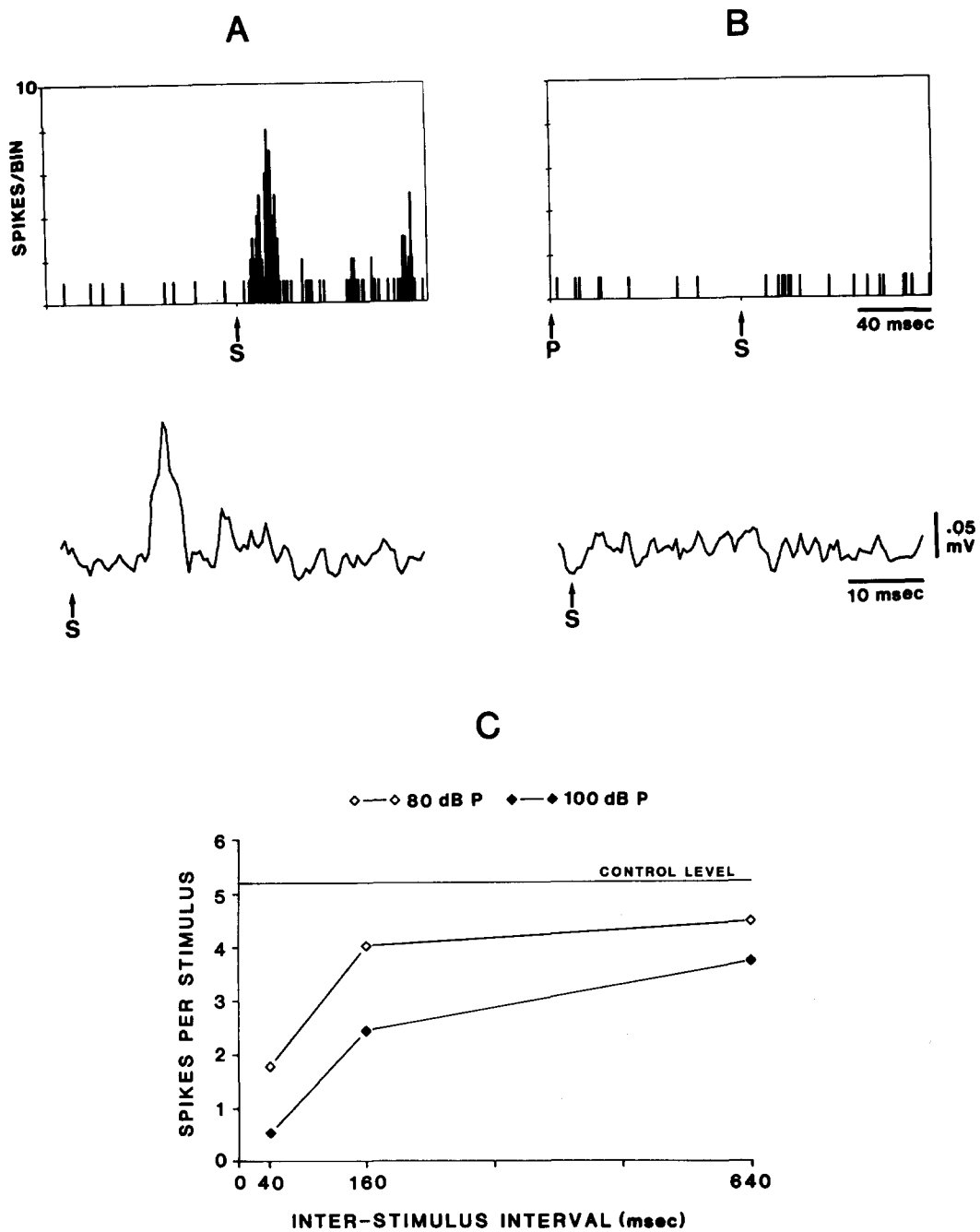

Fig. 3. An example of neck EMG startle response (lower graphs) and the discharge of a pontine unit (upper graphs) with (B) and without (A) the prestimulus. The prestimulus was 80 dB, and the eliciting stimulus 110 dB. Fifty trials were run in each condition. C: unit discharge to a 110 dB startle-inducing stimulus as a function of the intensity of the prestimulus and the interval between the prestimulus and the eliciting stimulus. Fifty trials were run in each condition.

on the posture of the animal during stimulation. Startle-induced response in 4 tonically active cells which showed maximal sustained firing to ipsilateral axial movement was examined. Unit response to both auditory and orthodromic spinal stimulation was reduced by 40–60% in a contralateral posture. These cells may mediate the asymmetry of EMG startle response as a function of posture.

Ninety-seven cells, among which 41 were auditory responsive, were tested for their spinal projections. As a group, auditory responsive neurons were significantly more likely than non-responsive neurons to have spinal projections: 57% (24 of 41) of the responsive neurons were identified as RS, while only 23% (13 of 56) of the non-responsive neurons were identified as RS ($P < 0.01$, χ^2-test). The percentage of RS cells responded to startle-eliciting stimuli in the present study (65%, 24 of 37) is comparable with that reported in decerebrate cats (70%)[36]. The estimated average conduction velocity for auditory responsive RS neurons was 117.3 ± 7.3 m/s (range from 76 to 153 m/s). Non-responsive RS neurons had significantly slower conduction velocities (87.4 m/s) than responsive neurons ($P < 0.05$, Mann-Whitney *U*-test).

The neural mechanisms for prepulse inhibition are unclear. If prepulse inhibition is occurring exclusively in the spinal cord, one would expect that the response of brainstem startle-related neurons would be unaffected by prestimuli. On the other hand, if prepulse inhibition is occurring before the spinal cord, one would expect a reduced response to be seen in the activity of brainstem output neurons. Eleven auditory responsive units were tested for prepulse inhibition. All showed dramatic discharge reduction ranging from 30% to 90% of baseline levels with an 80 dB prepulse presented 100 ms prior to the 110 dB startle-inducing stimulus. Fig. 3 illustrates the reduction of a unit's discharge (89% reduction) and EMG amplitude (87% reduction) by a prestimulus (Fig. 3B) as compared to that of the control trials without the prestimulus (Fig. 3A). This cell did not respond to the 80 dB prepulse and yet its response to the subsequent, more intense, startle-inducing stimulus was greatly suppressed. Cells which discharged to prepulse stimulations also showed discharge suppression by the prestimulus. Similar to the whole-body startle[23], prestimulus suppression of the unit discharge was a function of both the intensity of the prestimulus and the interval between the prestimulus and the eliciting stimulus. Fig. 3C shows an example of the effect of prepulse intensity and ISI on the discharge of startle-related neurons. We did not find a reduced prepulse effect in those cells in which response to startle-inducing stimuli was reduced in a less-favorable posture. This is consistent with the hypothesis that the inhibition and the elicitation of startle are independent processes[23].

Previous lesion and stimulation data had implicated NRPC in startle elicitation. The present study identifies neural elements in this region whose activity is correlated with startle and its modulation by prepulse. Together these data strongly support the identification of RS neurons in the medial pons and medulla as the brainstem output neurons mediating startle. Startle-related cells represent a subpopulation of cells in the medial pontomedullary region. In contrast with adjacent non-responsive cells, they are significantly more likely to have spinal projections. As a group, they have a very high conduction velocity, averaging 117 m/s, which is at the high end of the conduction velocity values reported for RS cells[10,25,31,32,34,44,48–50]. We find that these cells are not 'dedicated' to startle, but are normally active during specific classes of movement in waking, and also during the motor activation of REM sleep[39,40,42,43]. This relationship could not be seen in previous studies in decerebrate, paralyzed and/or anesthetized animals[11,12,14,21,26,36]. The present results also indicate that many previous acute studies of reticular cells emphasizing auditory and 'polysensory' response may actually have been observing startle response[17,18,37,38]. We find that, in the intact, unanesthetized animal, reticular cell response to auditory stimuli with an intensity of less than 90 dB is quite rare, while response to startle-inducing stimuli is relatively common. It is possible that some of these auditory responsive cells in the present study are also involved in startle response elicited by other modalities, e.g. tactile. The distribution of these cells is similar to that reported for cells responded to somatosensory stimulation[12].

Since reticulospinal neurons are known to have monosynaptic connections with motoneurons[15,16,33,35], they represent a major brainstem output pathway for triggering the startle reflex. The rostrocaudal distribution of RS cells with short latency re-

sponse to startle would allow the recruitment of startle response in several muscle groups[33]. We hypothesize that reticulospinal cells with short response latencies initiate the startle reflex, while cells with longer response latencies participate in the long latency components of the startle reflex.

The suppression of reticular response to startle-inducing stimuli by prestimuli, in parallel to the suppression of neck EMG response, provides the first direct evidence that this suppression occurs at or before pontomedullary RS neurons. The present results and studies of the inferior colliculus[52] are consistent with modulation of startle by the prestimulus early in the sensory processing of the startle-inducing stimulus. These results, however, do not rule out the possibility that additional prepulse modulation also occurs at the level of spinal cord[1,7,51].

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