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SPINAL CORD UNIT ACTIVITY: BEHAVIORALLY RELATED EXCITABILITY CHANGES IN THE AWAKE CAT. L.S. Sorkin*[1], T.J. Morrow[1,3], and K.L. Casey[1,2,3] (Spon: B.R. Trefz), Depts. of [1]Physiology, [2]Neurology, and [3]V.A. Med. Ctr., Univ. of Michigan, Ann Arbor, Mi. , U.S.A.

Aim of Investigation: When a cat is eating, its behavioral responses to a noxious stimulus occur at a significantly lower probability than when it is not eating (Casey,K.L., et al Neurosci. Abs. 1977). This study attempts to determine whether this difference is reflected in the excitability of spinal cord cells.

Methods: Cats were implanted with a chronic microelectrode device (Morrow, T.J., Brain Res. Bull. 5: 91-93) in the lower lumbar vertebral column. Natural and electrical stimuli applied to the skin were used to characterize units during alternate periods of eating and noneating.

Results: Twenty five units with well defined receptive fields were recorded. Seven of these were presynaptic, since they responded to each suprathreshold driving stimulus between 1 and 100 Hz. and had no latency changes as the frequency increased. Eight were postsynaptic and 10 were not classified. Ten additional units which could not be electrically driven were associated with joint movement or position (4), discrete, voluntary movements (4) or tendon stretch (2).

In two cases each, evoked slow potentials and postsynaptic activity in the dorsal cord was markedly attenuated or eliminated during eating, and resumed with cessation of feeding. In no instance was such modification associated with presynaptic units.

Conclusion: These results are consistent with the hypothesis that reduced behavioral responses to somatic stimuli may in part be attributed to reduced excitability of dorsal spinal neurons.

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A LIGHT AND ELECTRON MICROSCOPICAL ANALYSIS OF THE MORPHOLOGY AND SYNAPTIC CONNECTIONS OF ULTRAFINE PRIMARY AXONS WHICH TERMINATE IN LAMINA I OF THE SPINAL DORSAL HORN. S. Gobel, W.M. Falls* and E. Humphrey*. Neurobiology and Anesthesiology Branch, NIDR, NIH, Bethesda, MD 20205, USA

The aim of this investigation is to understand how nociceptive inputs might reach lamina I neurons. This study describes primary axons in lamina I whose endings are much finer (0.5 μm or less in diameter) than those previously reported either in the dorsal horn or in the ventral horn.

Crystalline horseradish peroxidase (HRP) was applied to the cut central ends of cervical and lumbar dorsal roots in adult cats. Following survival times ranging from 8-18 hrs, the cats were perfused with an aldehyde fixative and the terminal arbors of HRP-filled primary axons were examined in lamina I in parasagittal and horizontal planes.

Ultrafine primary axons arise from fine parent branches ($\sim$ 0.3 μm) and give rise to numerous, scalloped endings which sit in the center of small glomeruli. Within these glomeruli, they synapse on dendrites containing synaptic vesicles (type 2 dendrites) as well as on dendrites without synaptic vesicles (type 1 dendrites). Type 2 dendrites form dendrodendritic synapses on type 1 dendrites and dendroaxonic synapses on the ultrafine primary endings. Small axonal endings form axoaxonic synapses on the ultrafine endings and axodendritic synapses on type I dendrites.

The ultrafine primary endings in lamina I are thought to originate from primary neurons with either unmyelinated (C) or extremely fine myelinated axons. Inputs transmitted by ultrafine primary axons which are thought to respond to noxious stimuli are subject to presynaptic modulation from at least two sources, i.e., from synaptic vesicle containing dendrites and from small axons within lamina I.