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Title: The unmasking phenomenon in chronically crushed cutaneous afferents: plasticity of pad pathways

Location: Hall A-C

Presentation Time: Saturday, Nov 15, 2008, 2:00 PM - 3:00 PM

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Abstract: During the facilitation (unmasking) of the sural (SU)-evoked intraspinal field potentials (IFPs) induced by the acute section of the saphenous (Saph) or the superficial peroneal (SP) nerves, there is a concurrent change in the tonic PAD of SU terminals that depends on which nerve is first sectioned. That is, cutting the SP nerve, increases the SU tonic PAD while sectioning the Saph nerve reduces the tonic PAD (Abs Soc Neurosci 77.13HH20, 2007). We now investigated the extent to which the unmasking and the changes in tonic PAD of SU terminals induced by the acute section of the SP nerve are modified after a chronic SU nerve crush (CSNC). In 3 cats, 2 weeks after CSNC, SP conditioning pulses (2 xT) produced strong phasic PAD of the SU intraspinal terminals and inhibited the IFPs recorded within the dorsal horn (1.4-1.6 mm depth) in the L6-L7 segments generated by SU stimulation (1.2-2xT). After the acute SP nerve section the SU IFPs were only slightly increased (to 108±7 %), in contrast with the effects observed in non-chronic preparations (159±42% $p<0.001$ Mann Whitney), suggesting reduced unmasking. SP acute nerve section still increased the SU antidromic responses produced by intraspinal microstimulation at the same site (127±35 %), to about the same extent as in the non-chronic preparations (139±13 % $p>0.05$), suggesting increased tonic PAD. In contrast, by 3 weeks after CSNC (n=2), the acute SP nerve section now reduced the SU IFPs to 70±36% and the SU antidromic responses to 86±32%. That is, the tonic PAD was reduced and there was no more unmasking of the SU responses. Yet, as in 2 weeks after CSNC, SP conditioning stimulation either before or after the SP section still produced a clear PAD and still inhibited the

SU IFPs. By 4 weeks after CSNC (n=1), the acute SP nerve section still reduced the SU IFPs (58±21%) but now increased the SU antidromic responses (177±89%). Although we have not yet examined the complete time course of the effects produced by CSNC, the present set of observations already indicates that 2 weeks after SCNC, acute section of the SP nerve fails to unmask the otherwise "normal" responses of dorsal horn neurons to stimulation of low threshold SU afferents. There is in addition a transient reversal in the effects of SP section on the tonic PAD, even though conditioning stimuli still produce a strong phasic PAD and inhibit the SU IFPs. It is suggested that separate pathways and/or mechanisms mediate the tonic and phasic PAD of the SU terminals and the unmasking of the SU responses.

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