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Previous work showed that in the anesthetized cat, acute section of the saphenous (Saph) nerve increased the amplitude of the L3-L7 cord dorsum potentials (CDPs) and the L6 intraspinal field potentials (IFPs) evoked by stimulation of low threshold sural (SU) afferents (unmasking phenomenon). At the same time, the tonic PAD of SU terminals was reduced, leading to increased synaptic efficacy of SU intraspinal terminals. In contrast, sectioning the superficial peroneal (SP) nerve increased the SU-evoked CDPs and IFPs and also the SU tonic PAD. This suggested that in this case the facilitation of the SU-evoked responses was not entirely presynaptic (see Abs Soc Neurosci 146.10/I6, 2006). We now report that the effects on the SU tonic PAD produced by Saph and SP acute nerve section depend on the “state” of the spinal neuronal circuitry at the moment of nerve damage. Changes in tonic PAD were inferred from changes in the SU nerve antidromic responses produced by L6 dorsal horn microstimulation. As reported before, sectioning the SP nerve first, increased the SU tonic PAD (n=5). However, sectioning in the same preparation the Saph nerve after the SP nerve was cut now increased the SU tonic PAD (n=3). In other experiments sectioning the Saph nerve first (n=3), reduced the tonic PAD. In one of these experiments sectioning the SP nerve after cutting the Saph nerve also reduced the SU tonic PAD. These observations suggest that the previous history of the spinal neurons mediating the tonic PAD determines their response to nerve damage and that this response is modified by the damage itself. The resetting of the tonic PAD after peripheral nerve section could be involved in the development of secondary hyperalgesia. Experiments in course are aimed to determine if the reversal of the tonic PAD is also observed by anesthetizing instead of cutting the Saph or SP nerves and if it involves changes in spinal circuitry only, or if it is mediated by supraspinal structures.

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