

## The Impact of Shock on Reactivity to a Tactile Stimulus

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We have previously shown that exposure to either three brief (0.75 s) or three long (25 s) tail shocks can induce a strong hypoalgesia in rats as measured by tail withdrawal from radiant heat, and that these two examples of hypoalgesia are mediated by different neural systems. The present experiments evaluate whether exposure to either brief or long shock disrupts reactivity to a tactile stimulus applied to the tail. We found that subjects exposed to brief shock are hyperreactive to the tactile stimulus. This effect was observed on all of our response measures: latency to exhibit a tail movement, magnitude of tail movements, latency to vocalize, and vocalization magnitude. Similarly, exposure to long shock increased reactivity to tactile stimulation. However, this effect was only evident on three of the four measures: latency to vocalize, vocalization magnitude, and magnitude of tail movements. Implications of the results are discussed. © 1990 Academic Press, Inc.

Prior work has shown that exposure to a variety of aversive stimuli (e.g., shock, cold water, or defeat) can induce a strong decrease in pain reactivity, or "hypoalgesia" (Akil, Madden, Patrick, & Barchas, 1976; Bodnar, Kelly, Brutus, & Glusman, 1980; Miczek, Thompson, & Shuster, 1982). Depending on the characteristics of the "stressor," this hypoalgesia has been shown to be mediated by antinociceptive systems that are both anatomically and neurochemically distinct (for reviews see Maier, 1989; Terman, Shavit, Lewis, Cannon, & Liebeskind, 1984; Watkins & Mayer, 1982). For example, we have shown that different neural systems mediate the hypoalgesia observed after three 1.0 mA shocks depending upon whether the shocks are brief (0.75 s) or long (25 s) (Grau & Meagher, 1987; Grau, Illich, Chen, & Meagher, 1990; Meagher & Grau, 1987; Meagher, Grau, & King, 1989, 1990).

We (Grau, 1987a) and others (Terman *et al.*, 1984; Watkins & Mayer,

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