Previously we have shown that central sensitization, induced with an intradermal injection of capsaicin, undermines the inherent plasticity of the spinal cord. In our studies, plasticity is assessed with a simple instrumental (response-outcome) task. Male rats are transected at the second thoracic vertebra and are tested 24-48 h later. During testing, subjects receive shock to one hindleg (outcome) when the leg is extended (response). They quickly learn to maintain the leg in a flexed position, minimizing net shock exposure. Rats that have been injected with capsaicin (1 or 3%) in the hindpaw fail to learn, even when they are tested on the leg contralateral to the injection. Based on similar studies with uncontrollable electrical stimulation, we hypothesized that the loss of plasticity induced with central sensitization may be reversed by naltrexone combined with instrumental training. To test this, spinalized rats were given a subcutaneous injection of capsaicin (0 or 1%) in one hindpaw. Six hrs later they were given naltrexone (7 µg in 1 µl, i.t.) or saline (1 µl, i.t.) combined with instrumental training of the injected hindlimb. Then 24 hrs later, the capacity for instrumental learning was assessed on the contralateral hindlimb. Subjects treated with capsaicin and then saline failed to learn at both 6 and 30 hrs, supporting previous findings. Treatment with naltrexone and instrumental training, however, restored plasticity in the cord. Subjects given naltrexone and behavioral training learned the instrumental response at 6 and 30 hrs post injection. These results suggest that a clinically relevant stimulus (peripheral inflammation) can induce a behavioral deficit in spinalized animals, and could affect recovery of function after a spinal injury. Instrumental training combined with naltrexone (i.t.) is a potential behavioral therapy for restoring plasticity in the sensitized spinal system.