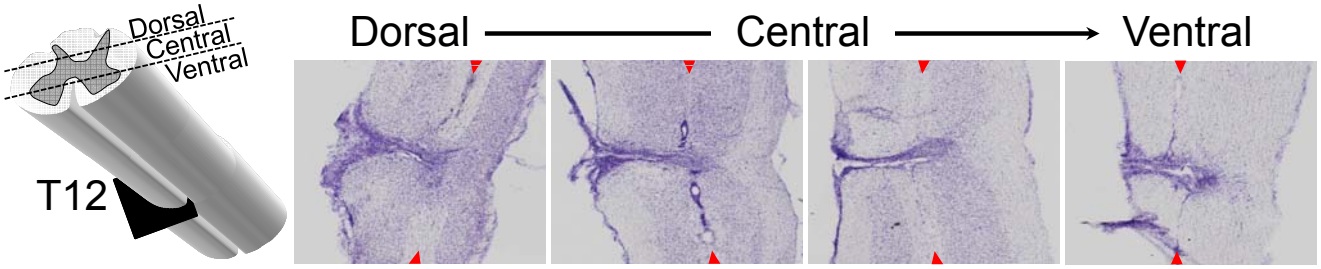

Figure S1. Histological evaluation of lateral hemisection. Schematic representation of hemisection at T12. Dashed lines indicate horizontal planes of tissue sections evaluated. Images show representative examples of histological evaluation of cresyl violet stained horizontal sections of spinal cords with scarring that encompasses over 50% of the side-to-side dimensions of the spinal cord through its entire dorsal and ventral extent, in a manner compatible with complete lateral hemisection. Arrowheads indicate midline.

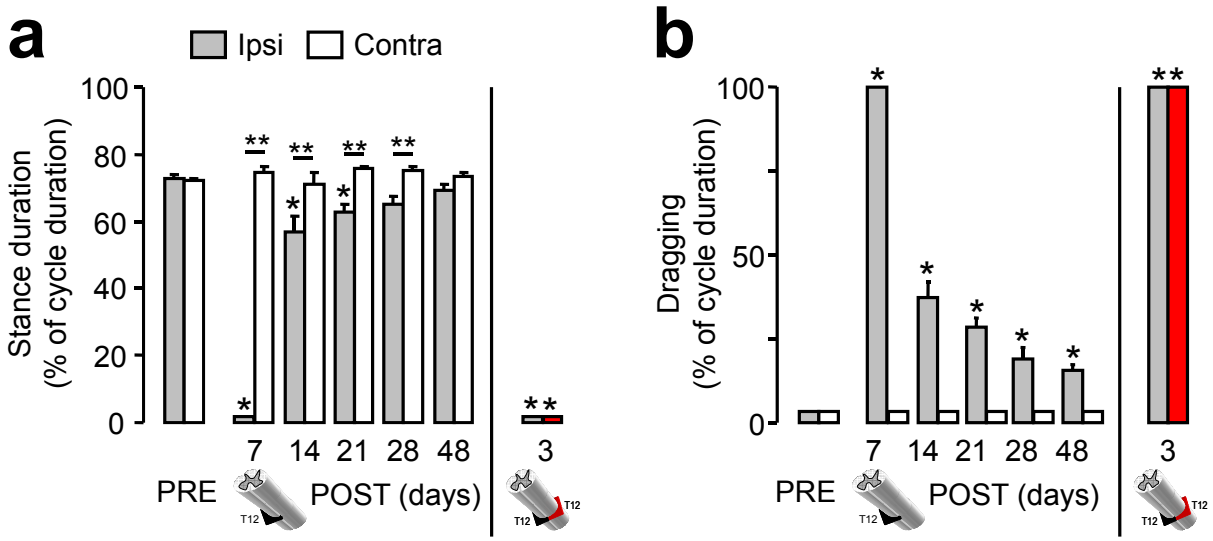

Figure S2. Recovery of supraspinal control of stepping after a lateral hemisection at T12. (a) Bar graphs of average (n = 8 mice, 10 steps per animal) normalized stance duration pre and 7- 48 days post left T12 hemisection, and 3 days after a delayed (ten weeks) right T12 hemisection. **(b)** Bar graphs of paw dragging expressed as percent of cycle duration (n = 8 mice). Error bars, S.E.M. *, **: statistically significant difference between pre- and post-lesion values, and between ipsilateral and contralateral hindlimbs, respectively.

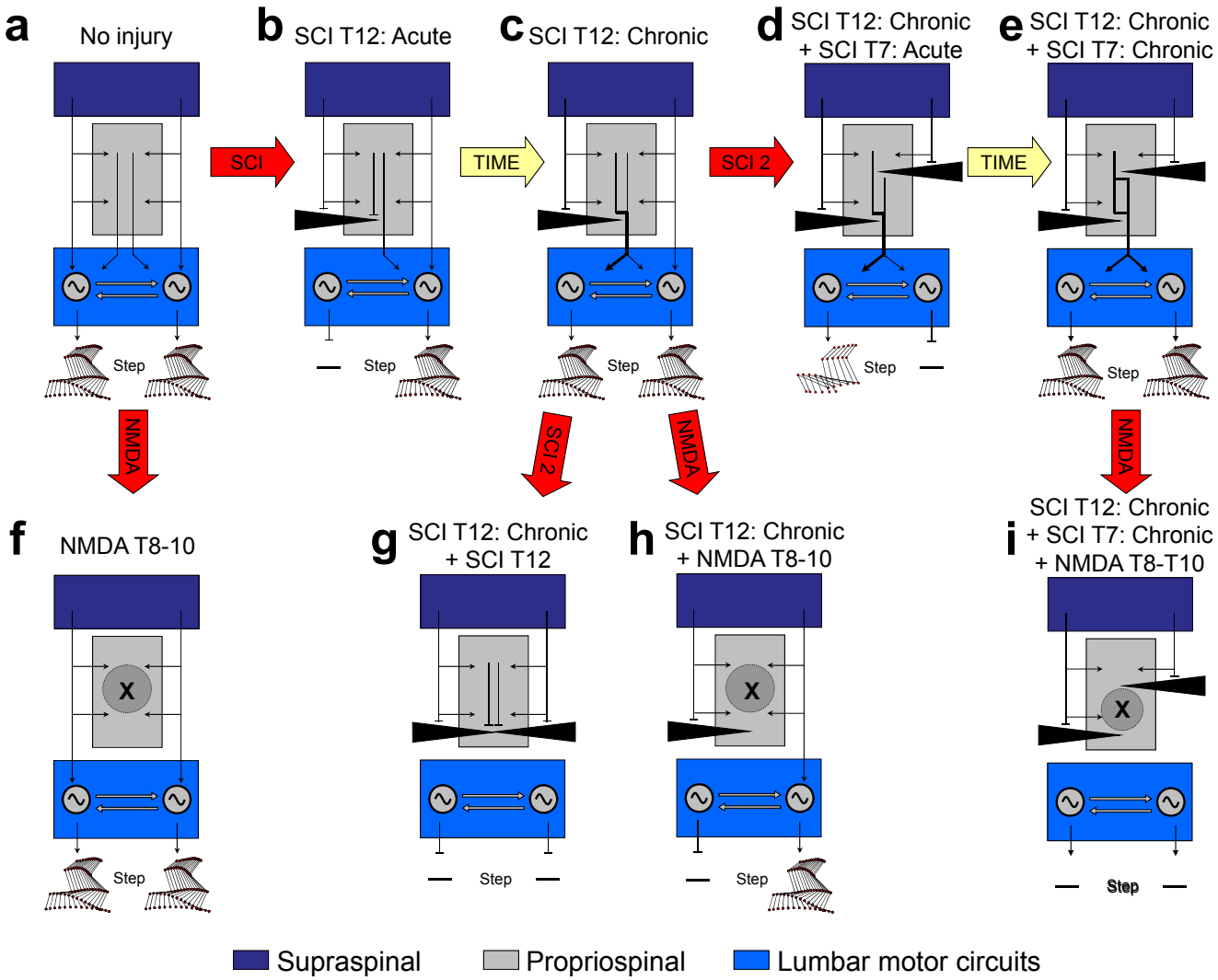

Figure S3. Schematic drawings summarizing the effects of the different combinations of lateral hemisections and NMDA lesions on the direct and indirect supraspinal control of stepping. **(a)** Control of stepping by direct supraspinal connections to lumbar motor centers in uninjured mice. **(b)** Acute loss of stepping ipsilateral to a T12 lateral hemisection that interrupts supraspinal connections on that side. **(c)** Chronic recovery of stepping after lateral T12 lateral hemisection in spite of loss of supraspinal connections. **(d)** Acute loss of ipsilateral stepping and partial loss of recovered contralateral stepping after a delayed lateral hemisection at T7. **(e)** Chronic recovery of bilateral stepping after T12 and delayed T7 lateral hemisections mediated by indirect propriospinal relay connections in spite of the loss of direct supraspinal connections to the lumbar motor centers. **(f)** Preserved supraspinal control of stepping after NMDA-mediated ablation of intrinsic spinal cord neurons at T8-T10 in otherwise uninjured mice. **(g)** Loss of recovered stepping after delayed contralateral lateral hemisection at T12. **(h)** Loss of recovered stepping on the side of the T12 lateral hemisection but not on the uninjured side after ablation of intrinsic spinal cord neurons at T8-T10 **(i)** Loss of recovered stepping on both sides after T12 and delayed T7 lateral hemisection after ablation of intrinsic spinal cord neurons at T8-T10.

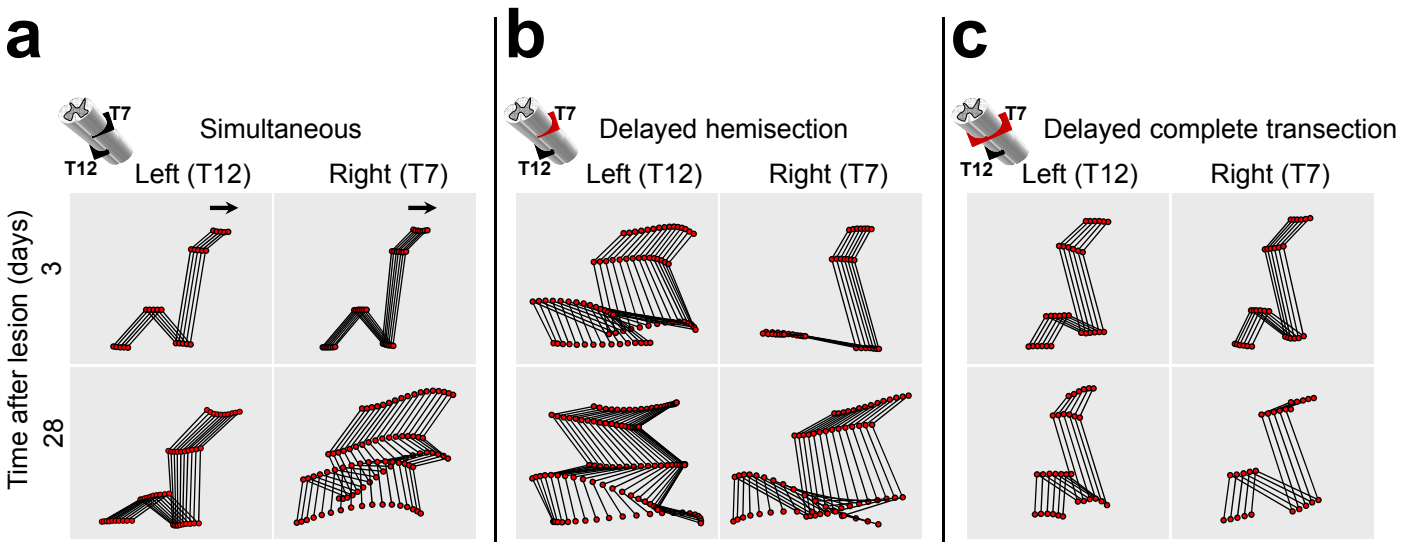

Figure S4. Recovery of supraspinal control of stepping after delayed but not simultaneous left-T12 and right-T7 hemisections or after delayed complete transection at T7. (a-c) Stick diagrams of left and right hindlimb movements during swing (or dragging) at 3 and 28 days after either simultaneous left-T12 and right-T7 hemisections (a), delayed right-T7 hemisection 10 weeks after left-T12 hemisection (b), or delayed complete-T7 transection 10 weeks after left-T12 hemisection (c).

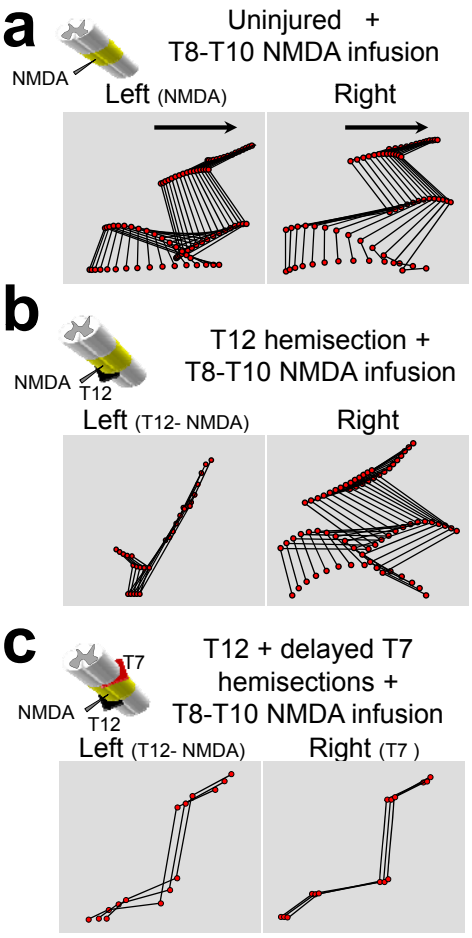

Figure S5. Excitotoxic ablation of T8-T10 neurons abolishes the recovered control of stepping after a T12 hemisection and after a T12 hemisection followed by a delayed T7 hemisection. (a-c) Stick diagrams show left and right hindlimb movements during the swing phase at 2-5 days after NMDA injection in uninjured mice (a), and in mice that had recovered from a left T12 hemisection (b) or from left T12 and delayed right T7 hemisections (c).