Supporting Information

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SI Materials and Methods

We searched our data for evidence that fluctuations in tremor amplitude resulted from changes in the efficiency with which spinal interneuron circuits cancelled descending oscillatory drive from M1. First, for each recording session made with the spinal chamber, we calculated the spectral power in the finger acceleration recording during the ramp phase of each finger flexion trial. Trials were sorted by the total power in the 6 to 13 Hz range, and separated according to whether they were above or below the median level. Fig. S1*A* shows the mean finger acceleration power spectrum for high and low tremor trials so categorized. The average power in the 6 to 13 Hz range was 0.0096 $(m/s^2)^2$ and 0.0033 $(m/s^2)^2$ for high and low tremor trials, respectively (Fig. S1*J*). Tremor power was 2.9 times larger in the high tremor trials; this difference was significantly greater than when we segregated the trials at random (Monte Carlo test with 100 repetitions, two-tailed estimated *P* value shown in Fig. S1*J*).

As well as recordings from the spinal electrodes, we also had available to us signals from chronically implanted microwire electrodes in the hand representation of the left M1. These used insulated 50-μm-diameter stainless steel wires, which had been inserted into the cortex transdurally at the end of recordings from M1 just before sealing the chamber (see ref. 1 for further methodological details). Fig. S1*B* shows the power spectrum of these M1 LFP recordings, separated into high and low tremor trials. Power in the 6- to 13-Hz band was significantly higher during high tremor trials (Fig. S1*K*). By contrast, power in the central tremor band in the spinal LFP recordings was unaltered for high and low tremor trials (Fig. S1 *C* and *L*).

The coherence between the M1 and SC LFP recordings and finger acceleration is shown in Fig. S1 *D* and *E* separately for high and low tremor trials. In both cases, coherence was significantly higher in the 6- to 13-Hz band during the high tremor trials (Fig. S1 *M* and *N*).

The phase of LFP-acceleration coherence is shown separately for low and high tremor trials in Fig. S1 *F* and *G*. Fig. S1 *H* and *I* show the circular histograms calculated using the mean phase between 6 and 13 Hz at each recording site for M1 (black) and SC (red) using low (Fig S1*H*) and high tremor trials (Fig. S1*I*). The phase difference between M1 and SC mean coherence phase was approximately π radians, and this phase shift did not alter significantly between the low and high tremor trials (Z test, $P > 0.05$).

These results are consistent with the idea that spinal circuits cancel oscillatory inputs in the frequency range of central tremor, and that changes in tremor amplitude result in part from a limitation of this cancellation system. This interpretation is illustrated schematically in Fig. S2. We suggest that on low tremor trials, oscillations from M1 converge with antiphase oscillations from SC, resulting in substantial (but not complete) cancellation. The residual signal sums with oscillations from other sources (e.g., mechanical resonance and oscillations caused by the stretch reflex feedback loop) to yield the observed tremor. On high tremor trials, oscillations from M1 are increased in amplitude. This increase is not matched by corresponding changes in the amplitude of SC oscillations. Although SC activity remains in antiphase to M1 activity, cancellation is thus not as effective, resulting in a greater amplitude of tremor. On these trials, a greater fraction of the observed tremor results from uncanceled central oscillations. Coherence measures the fraction of one signal that is correlated with another. Consequently, the coherence between observed tremor and both M1 and SC recordings is increased in the high tremor trials.

We can only speculate as to why the SC circuits seem unable to follow the increases in the amplitude of oscillatory input from M1 during high tremor trials. However, for a system implemented using neurons with a fixed maximum firing rate, and with fixed amplitude synaptic inputs, it is not unreasonable that oscillations should not be able to increase above a certain limit.

1. Witham CL, Wang M, Baker SN (2007) Cells in somatosensory areas show synchrony with beta oscillations in monkey motor cortex. *Eur J Neurosci* 26:2677–2686.

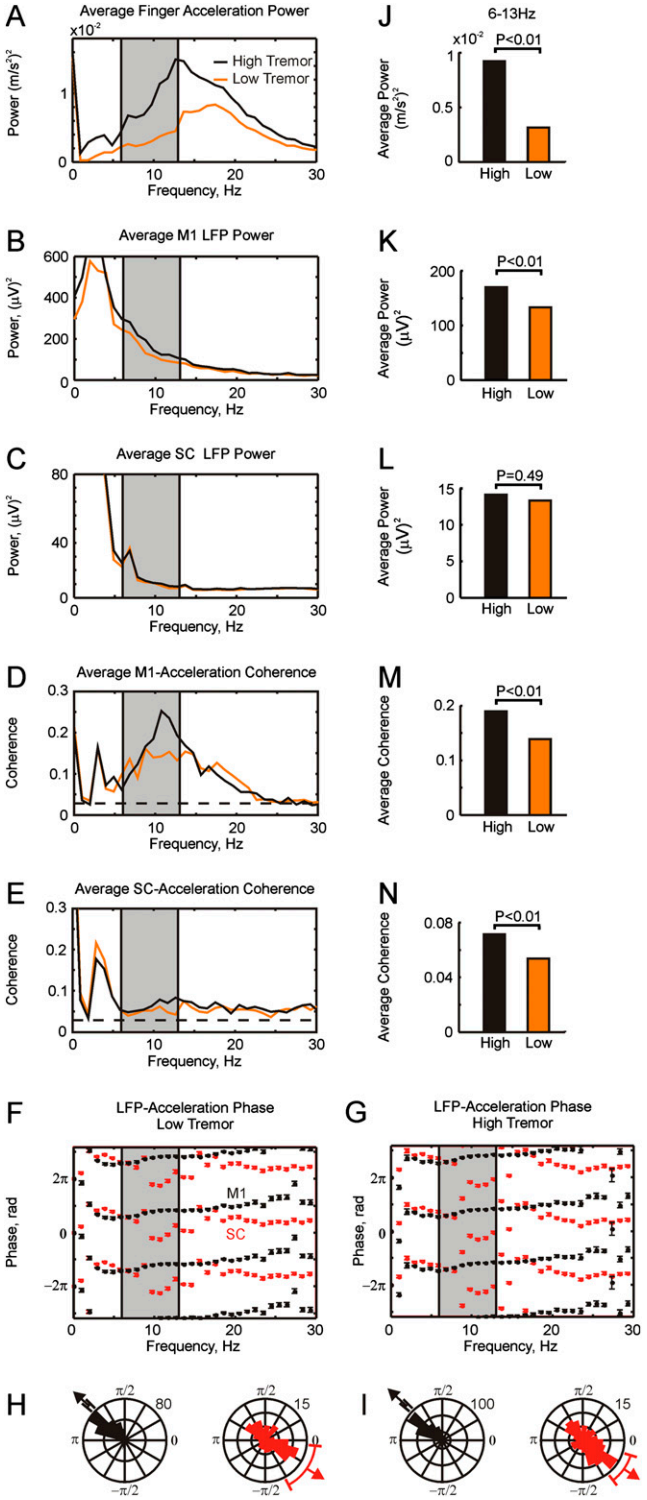

Fig. S1. Spectral measures during SC recordings, separated by high (black lines) and low (orange lines) tremor trials, during the ramp movement of flexion trials: (*A*) average finger acceleration power spectrum; (*B*) average power spectrum of LFP recorded from microwire electrodes in left M1 hand representation; (*C*) average power spectrum of SC LFP; (*D*) average coherence between M1 LFP and finger acceleration; and (*E*) average coherence between SC LFP and finger acceleration. (*F* and *G*) Phase of coherence between LFP and finger acceleration for low (*F*) and high (*G*) tremor trials respectively. (*H* and *I*) Rose plots of the phase for each recording site averaged over the 6- to 13-Hz range. Colors mark data from M1 (black) and SC (red). (*J–N*) Mean values over the 6- to 13-Hz range of the corresponding spectral measures shown alongside in *A–E*. Significance of the differences between high and low tremor trials has been assessed by using a Monte Carlo method.

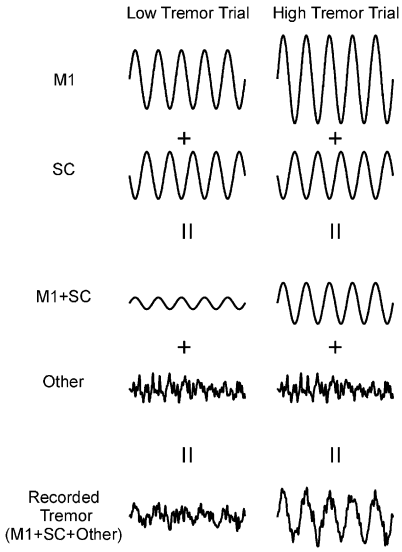

Fig. S2. Schematic illustration of how incomplete cancellation of oscillations in the descending drive from M1 by SC interneuron circuits could lead to changes in tremor amplitude and the coherence between M1 or SC and tremor recordings.

Table S1. The percentage of LFPs recorded from each monkey in each area

Area	Monkey R	Monkey D
DCN	54%	46%
PMRF	58%	42%
SC	63%	37%
M1	59%	41%