Central nervous system disorders produce the undesired, approximately rhythmic movement of body parts known as pathological tremor. Typical treatments are medications and deep brain stimulation surgery, both of which include risks, side effects, and varying efficacy. Since the pathophysiology of tremor is not well understood, empirical investigation drives tremor treatment development. This dissertation explores tremor from a mechanical systems perspective to work towards theory-driven treatment design. Unlike typical treatments, the mechanisms for mechanical tremor suppression are well understood: applying joint torques that oppose tremor-producing muscular torques will reduce tremor irrespective of central nervous system pathophysiology. However, typical engineering actuators are rigid and bulky, preventing clinical implementations. This dissertation explores dielectric elastomers as tremor suppression actuators to improve clinical implementation potential of mechanical tremor suppression. The primary drawback of dielectric elastomers is their relatively low actuation levels compared to typical actuators. This research develops a tremor-active approach to reduce actuation requirements: actuators only actuate to oppose tremor, while the human motor system must overcome the passive actuator dynamics. Simulations with recorded tremor datasets demonstrate excellent and robust tremor suppression performance. Benchtop experiments validate the control approach on a scaled system. In addition to tremor suppression, this research investigates a delay-based model for parkinsonian tremor. Besides tremor, Parkinson's disease generally inhibits movement, with typical symptoms including rigidity, bradykinesia, and increased reaction times. One possible mechanism that could cause movement inhibition and tremor is excessive central nervous system inhibition producing unaccounted feedback delays that cause instability. This dissertation develops an optimal control model of human motor control with an unaccounted delay between the state estimator and controller that represents the increased inhibition projected from the basal ganglia to the thalamus. Simulations that incorporate tremor resetting and random variation in control saturation produce simulated tremor with similar characteristics as recorded tremor.