Abstract: Damage to the primary visual cortex (V1) or its immediate afferents results in a loss of conscious vision in corresponding parts of the visual field in each eye – in essence, causing a “cortically-induced blindness”. While blinding diseases are a significant scourge in our aging society, when it comes to cortical blindness, most of the sparse rehabilitation effort leans heavily towards substitution or compensation for the vision lost, rather than towards vision restoration. One contributing factor has been the mistaken belief that the adult visual system lacks the kind of plasticity inherent in other cortical [i.e., motor] systems, which underlies their ability to recover function after damage. Research in our laboratory over the last 14 years has critically examined this problem first using an animal model of visual cortical damage, then using affected humans. In both cases, we found that gaze-contingent visual training can restore direction and orientation discrimination of moving, static, simple and complex visual targets at trained, blind field locations, even in chronic stroke patients. Localized, discrimination training also generated large swaths of improvement in visual detection sensitivity measured by Humphrey perimetry, inside the original blind field border. The amount of improvement attained did not depend on patient demographics (age, time post-lesion, etc.), but instead, was directly proportional to the amount of training performed. However, both Humphrey perimetry and psychophysical measurements showed that recovered vision was not completely normal, with poorer sensitivity for luminance contrast. Visual psychophysics also demonstrated residual deficits in fine difference discriminations for both motion and orientation, which modeling revealed to be caused by abnormally high levels of internal processing noise. Current research in our laboratory is focused on better understanding the nature and sources of this noise, and the neural mechanisms that underlie training-induced recovery. Together with our collaborators, we have also started manipulating training parameters and using adjuvant therapies (both pharmacological and electrical) in an attempt to speed up the rate and magnitude of recovery and overcome residual visual deficits at trained, blind field locations.