Fig. S2. Ischemic depolarization and subsequent histopathology

SHR (n=5) were surgically prepared for DC potential recording at a series of mediolateral electrode positions 2 mm caudal to bregma, and were then subjected to tandem MCA/CCA occlusion, as detailed in the manuscript (Study 1, part 2). No depolarization occurred at 1 mm lateral positions that were devoid of pathology 24 h after occlusion (open circles). Conversely, maximal depolarization was evident within 15 min at 5 mm lateral sites (and at 6 mm, not shown), that later exhibited consistent infarction (closed circles). At intermediate positions the kinetics of ischemic depolarization was also highly predictive of tissue fate. Persistent depolarization invariably was associated with infarction regardless of recording site, whether evident immediately or occurring with a delay of 1 or 2 h (e.g., at 4 mm lateral). Transient depolarization at 1 h with subsequent repolarization characterized a 3 mm lateral site exhibiting selective neuron loss at the infarct margin (closed square). A single electrode at 3 mm lateral, located just within the eventual infarct margin, failed to exhibit detectable depolarization during this acute recording interval and must have depolarized later. Since the depolarization magnitude declined progressively under these recording conditions it could no longer provide a reliable index of local physiological status at time points later than 2 h.