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Multimodal, live imaging of peri-infarct depolarisation (PID) during global forebrain ischemia in the rat cerebral cortex

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PID and associated changes in cerebral blood flow (CBF) spontaneously occur in the cortex during cerebral ischemia and may contribute to the extension of brain infarcts. We have set out to characterize PID-related changes in membrane potential and hemodynamic variables in rat cerebral cortex. In anesthetized, male Sprague-Dawley rats (n=13) a closed cranial window was mounted over the parietal bone and loaded with voltage sensitive (VS) dye. The left femoral artery and vein were cannulated for the monitoring of mean arterial pressure (MAP) and for blood withdrawal. Global forebrain ischemia was induced by bilateral common carotid artery occlusion combined with subsequent hypovolemic hypotension (MAP<40 mmHg). Using selected illuminations we acquired synchronous changes in membrane potential (VS dye method); cerebral blood volume with green (540–550 nm); hemoglobin saturation with red (620–640 nm) illumination, and CBF by laser speckle contrast imaging. PID occurred at a MAP value of 41.2±3.7 mmHg and at a CBF value of 43.4±4.9%. In most cases the depolarization was not followed by the recovery of membrane potential, and these events were associated with CBF reduction (19±3.6%). In conclusion, PID appears to generate at the lower limit of the autoregulatory range of CBF, originates at a focus with high vulnerability to ischemia, and propagates similar to that known for cortical spreading depression. PID associated CBF responses most often display inverse neurovascular coupling.

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