

Changes of cerebral blood flow during the secondary expansion of a cortical contusion assessed by ¹⁴C-iodoantipyrine autoradiography in mice using a non-invasive protocol.

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Abstract

Although changes of cerebral blood flow (CBF) in and around traumatic contusions are well documented, the role of CBF for the delayed death of neuronal cells in the traumatic penumbra ultimately resulting in secondary contusion expansion remains unclear. The aim of the current study was therefore to investigate the relationship between changes of CBF and progressive peri-contusional cell death following traumatic brain injury (TBI). CBF and contusion size were measured in C57Bl6 mice under continuous on-line monitoring of (ETp)CO₂ before, and at 15 min and 24 h following controlled cortical impact by ¹⁴C-iodoantipyrine autoradiography (IAP-AR; n = 5-6 per group) and by Nissl staining, respectively. Contused and ischemic (CBF < 10%) tissue volumes were calculated and compared over time. Cortical CBF in not injured mice varied between 69 and 93 mL/100mg/min depending on the anatomical location. Fifteen minutes after trauma, CBF decreased in the whole brain by approximately 50% (39 +/- 18 mL/100mg/min; p < 0.05), except in contused tissue where it fell by more than 90% (3 +/- 2 mL/100mg/min; p < 0.001). Within 24 h after TBI, CBF recovered to normal values in all brain areas except the contusion where it remained reduced by more than 90% (p < 0.001). Contusion volume expanded from 24.9 to 35.5 mm³ (p < 0.01) from 15 min to 24 h after trauma (+43%), whereas the area of severe ischemia (CBF < 10%) showed only a minimal (+13%) and not significant increase (22.3 to 25.1 mm³). The current data therefore suggest that the delayed secondary expansion of a cortical contusion following traumatic brain injury may not be caused by a reduction of CBF alone.

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