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Neutrophils and the blood-brain barrier dysfunction after trauma

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Despite the fact that neutrophils are essential for the protection from invading pathogens, hyperactive neutrophils may elicit detrimental cerebral damage after severe trauma. The neutrophil interactions with the neurovascular unit entail endothelial dysfunction involving endothelial leakage, formation of edema, coagulation abnormalities, disturbed hemodynamics, tissue infiltration etc. These elements of the "whole body inflammation," designated systemic inflammatory response syndrome (SIRS) in conjunction with intracerebral proinflammatory activities, are important triggers of post-traumatic cerebral damage and mortality according to the "second hit" concept. From the immunologic point of view, the brain is an immune privileged site, known to resist autodestructive inflammatory activity much more efficiently than other organs because of the highly efficient diverse functions of the blood-brain barrier (BBB). However, both the underlying strategy of the BBB to maintain cerebral protecting functions against the post-traumatic neutrophil-mediated "second hit" and how activated neutrophils may overcome the BBB are currently unknown. Therefore, this review summarizes the current understanding of the "second hit," the BBB physiology, and its role in the maintenance of cerebral immune privilege, and discusses recent findings that may explain the pathophysiologic neutrophil-BBB interactions occurring after severe trauma, thus offering novel therapeutic options to protect from post-traumatic brain damage.