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JOURNAL ARTICLE

The effect of cardiac arrest on the blood-testis barrier to albumin, tumor necrosis factor-alpha, pituitary adenylate cyclase activating polypeptide, sucrose, and verapamil in the mouse

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Impotence commonly occurs after events such as acute myocardial infarction, coronary bypass, head trauma, and cerebral bleeding, including subarachnoid hemorrhage. We hypothesize that the hypoxia accompanying these events could damage the blood-testis barrier (BTB) and so cause testicular dysfunction, a possible cause of impotence. We examined the effect of cardiac arrest in mice on testis weight and various aspects of BTB function. Testis weight was decreased by about 24% 12 hours after cardiac arrest but had recovered fully by day 7. The testis/serum ratio for albumin was increased 12 hours after arrest, showing a disruption in the vascular BTB with recovery by 24 hours. The testis/serum ratio for sucrose was not consistently elevated, showing that the Sertoli cell BTB remained intact. The testis/serum ratio for verapamil was increased on day 3 of cardiac arrest, suggesting impaired function of the BTB's p-glycoprotein efflux transporter. Transporters for pituitary adenylate cyclase activating polypeptide and tumor necrosis factor-alpha were not affected by cardiac arrest. These results show that cardiac arrest affects testis weight and some aspects of BTB function. Such changes might have long-term effects on testicular function.