Erectile dysfunction (ED) following human radical prostatectomy (RP) is a well-documented event that may involve a neuropraxia-induced apoptosis leading to corporal fibrosis. We developed a canine model to assess anti-apoptotic protein, phosphorylated AKT$^{473}$ (AKT/PKB$^{473}$) and the pro-apoptotic protein, phosphorylated-Bad$^{(p\text{Ser}136)}$, as markers of apoptosis in the corporal cavernosal tissue. Seven adult male canines ($n = 7$) underwent RP. At three RP time points (0.5 hr pre-RP, 0.5 hr post-RP, and 1 hr post-RP), whole blood was extracted from corporal cavernosal tissue and systemic brachial veins of each animal. To avoid rapid commencement of apoptosis, samples were stored on ice ($\leq 30$ sec.) and centrifuged at 14,000Xg for 15 minutes. Equal amounts of protein were resolved on 4-20% gradient SDS-PAGE gels and probed with rabbit antibodies against phosphorylated-AKT/PKB$^{(p\text{Ser}473)}$, phospho-Bad$^{(p\text{Ser}136)}$, and endothelial nitric oxide synthase (eNOS). Results were quantified by the Gel-Pro©™ program and expressed as mean integrated optical densitometry units (IOD). In four of the six samples, phospho-AKT$^{(p\text{Ser}473)}$ levels in the corpora cavernosal plasma increased from the pre-RP to 0.5-hour post-RP, returning to baseline at 1-hour post-RP. AKT levels remained unchanged in the peripheral plasma. Phospho-Bad$^{(p\text{Ser}136)}$ levels were obtained in three of the six samples and were elevated at 1-hour post-RP. eNOS levels reflected the activity of AKT and were elevated only at the 0.5-hour post-RP. Our early results in a canine model demonstrate that radical prostatectomy induces corporal cavernosal apoptosis with early up-regulation of phospho-AKT and eNOS and subsequent elevation of the pro-apoptotic Bad$^{(p\text{Ser}136)}$ protein. These protein markers reflect ongoing apoptosis in the corporal cavernosal tissue and can quantify the degree of neuropraxia following radical prostatectomy.