The increase in volume of intracranial contents will determine the rise of intracranial pressure. Figure 3.2 shows the intracranial pressure-volume relationship. Initially, a small increase in the volume of the intracranial contents causes no rise in pressure; a small amount of CSF can move into the spinal subarachnoid space, which is very slightly distensible. However, the skull being a relatively closed container, a critical volume is soon reached when a small rise in intracranial pressure occurs. Benign Cerebral Glioma, Volume II synthesizes the considerable amount of information on the classic and evolving tools in the clinical treatment of these neoplasms. The text also presents practical guidelines for contemporary clinical management with the framework of currently available knowledge. Volume II discusses the clinical aspects of benign gliomas, including Benign Cerebral Glioma, Volume II synthesizes the considerable amount of information on the classic and evolving tools in the clinical treatment of these neoplasms. The text also presents practical guidelines for contemporary clinical management with the framework of currently available knowledge.

Benign meningiomas used for the study did not have perifocal edema; therefore histologically unchanged tissue surrounding the meningioma was studied as the perifocal zone. We considered this tissue to be intact or relatively intact. [4] proposed to use PAI-1 and uPA as prognostic markers of meningiomas. Malignant tumors and metastases: glioblastoma and breast cancer metastasis in the brain. Metabolic processes involving urokinase were more active in the tissues of both primary and secondary malignant tumors of the cerebral hemispheres than those involving thrombokinase (see Table). Both uPA-AG and uPA-act were significantly elevated in both malignant tumors; however uPA-AH/uPA-act ratio was significantly increased only in primary tumors and their perifocal zone.