This report concerns the pathological findings in the brain of a white woman, 74 years old at death.

Thirteen years prior to death, the patient began to have severe intermittent bilateral frontotemporal headaches, failing vision, and bitemporal hemianopsia. Roentgenograms of the skull showed an enlarged sella turcica without other bony changes. A diagnosis of pituitary adenoma was made. Permission for surgical operation and biopsy was refused, and the patient was treated with roentgen-ray therapy for a total dose of 3200 r. This resulted in slight improvement in the headaches, but vision continued to fail and the patient became blind.

About 8 months prior to death, decreased psychomotor activity, confusion and ptosis of the right eyelid became apparent in addition to the blindness. This progressed to the time of admission 8 days before death, when the patient was noted to be disoriented. The right pupil was not reactive to light, and the right eye would not cross the midline to the right. Deep tendon reflexes were slightly more active on the right side, with a positive Babinski's sign. Strength of muscles was normal, and sensory changes were not demonstrated. The spinal fluid was clear, under decreased pressure, contained no cells, and the quantity of fluid obtained was inadequate for chemical studies. There was increasing coma, and episodes of shock and cyanosis occurred during the last week of her life. Just prior to death roentgenogram of the chest revealed a pulmonary lesion compatible with a diagnosis of carcinoma.

Autopsy. Metastatic carcinoma was present in the lungs, liver, lymph nodes and bone marrow. The primary site was not determined, but an origin in the gall bladder was considered likely.

The major finding in the brain was a firm, pink-gray, partially lobulated tumor, 5.5X4X3 cm. in size, adherent to the orbital plate of the frontal and sphenoidal bones, extending downward into the sella turcica with compression of the pituitary gland against the hypophysial fossa, and extending upwards into the base of the brain for a distance of about 3 cm., compressing the olfactory nerves, the optic chiasm, the right third cranial nerve and infiltrating into the hypothalamic structures and left basal ganglia (Fig. 1). On section, the tumor was somewhat variegated in appearance and consistency with areas that were hard and white, and areas that were firm and gray.

Microscopically, two types of tissue were apparent, one an adenocarcinoma, the other a meningioma, largely meningothelial, partly fibroblastic in character (Fig. 2). These tissues were intimately intermingled in areas, while elsewhere large masses of one tumor tissue was present to the exclusion of the other. In such circumstance, the hard white tissue proved to be carcinoma, the firm gray, meningioma. It is estimated that the meningiomatous tissue comprised approximately 45 per cent of the total. The tumor pressing upon the pituitary gland was found to be exclusively meningiomatous, while the tumor infiltrating into the basal ganglia was exclusively carcinoma. A few isolated carcinoma cells were present in the subarachnoid space adjacent to the tumor, and within a small old cystic infarct in the right caudate nucleus, but no carcinomatous tissue was found in distant portions of the brain.

* This study was supported in part by Grant C8044 of the National Cancer Institute, and by a special traineeship BT-463 from the National Institute of Neurological Diseases and Blindness, U. S. Public Health Service.
Fig. 2. Photomicrograph of tumor, showing two clusters of carcinoma cells (upper left, lower right) within meningioma. Hematoxylin and eosin, X186.

**COMMENT**

The metastatic carcinoma was present within the meningioma and within the adjacent neural tissues. It is possible that the carcinoma had metastasized into the neural tissues adjacent to the meningioma, with subsequent infiltration into the latter, or conversely, had metastasized into the meningioma with subsequent infiltration into the brain. We know of no way to determine which of these two possibilities occurred in this instance. It is of interest that of the 6 reported instances of metastasis of carcinoma to primary intracranial tumor known to us, the latter proved to be a meningioma in 5, the sixth being a nerve-sheath tumor. This may serve to make more likely the possibility that we too are dealing with a true metastasis to a meningioma. The slow growth of such tumors within the brain may be assumed to be one factor in such metastasis. We are reluctant to engage in speculation on the biological significance of localization of metastasis to the meningioma, since we cannot exclude the first alternative mentioned above, a “collision tumor”, which would render the presence of the carcinomatous tissue in the meningioma a spatial accident.

From a clinical standpoint, the degree to which a suprasellar meningioma may mimic a pituitary adenoma, hardly needs comment. The accentuation of effects of the tumor caused by superimposition of carcinomatous tissue, is likely to be a very rare event.

**SUMMARY**

A case of suprasellar meningioma within which a carcinoma had metastasized or infiltrated from adjacent tissue, is documented.

**REFERENCES**