

one and that additional factors must be active to bring about this result.

It is therefore essential for the establishment of definite causal relations between a trauma and a cerebral neoplasm in the individual case to obtain anatomical evidence demonstrating the close local connections between the traumatic residues and the cerebral tumor. The following residues are of significance in this respect: scars in the skin of the head and changes in the bony skull and meninges (healed fracture, depressions, bony splinters, thickenings of periost, meningeal thickening, meningeal tears, organized meningeal hemorrhage, arachnoid cyst, and meningo-cerebral adhesions) above or opposite to the site of the tumor; fibro-gliar scars with surrounding reactive cerebral gliosis, old blood pigment, cerebral cysts (former liquefaction necroses or hematomas), and foreign bodies (bullets and shell splinters) in close proximity, in direct contact with, or surrounded by the glioma. The intracerebral lesions may exist on the side previously affected directly by the trauma or may be found on the opposite side of the brain in the direction of the traumatic extension, being the sequelae of the counter-coup (Adler; and Naville).

In the evaluation of these lesions proper consideration must be given to the fact that the significance of some of them regarding a traumatic origin is greatly limited, as identical changes may occur within or adjacent to the tumor as the result of the neoplastic growth. Spontaneous necroses and hemorrhages, often followed by cystic formation, are found rather commonly in gliomas, especially in the cellular types, and less often in the more mature fibrillar varieties [in 80 per cent (Karitzky)] (Doerr; and Schley). A perigliomatous increase of glia cells is a frequent normal feature caused by a direct compression of the cerebral tissue surrounding the neoplasm and being then only an apparent one (Karitzky), or representing a reactive gliosis (Singer), which may even involve parts of the brain remote from the neoplasm, and being the result of secondary circulatory disturbances or a response to the production of toxic substances from degenerating and necrotic parts of the blastoma (Agostini). Karitzky even doubted that the presence of scar tissue in a glioma was always indicative of a traumatic residue, as he observed, during the study of 133 cerebral tumors, in 2 cases scar tissue within the neoplastic tissue. As these scars occurred in close relation to necroses, Karitzky argued that these scars represented the cicatricial end results of primary degenerations of the neoplastic tissue. A growing blastoma may obliterate in the course of its progress many traumatic residues originally present in the brain.

A careful and thorough anatomical and histological study of the brain and its membranes and a cautious and discriminating interpretation of the observations made is essential in every case of alleged traumatic cerebral neoplasia, as clearly demonstrated by the considerations noted above. It is necessary for a reliable evaluation of a case to obtain the following additional data: a) The histological type of the tumor must be ascertained, as the proliferative activity of immature tumors (medulloblastomas-glioblastomas) is much greater than