

cephaly, and idiocy as the late results of birth trauma, is mentioned in support of these claims, the partial correctness of which had to be conceded by Wohlwill, who is an adherent of the dysontogenetic dogma.

These observations have reduced greatly the value of a powerful argument advanced heretofore in favor of Cohnheim's theory, that is, the high incidence of medullo-glioblastomas among children, which was attributed to blastomatous transformations of congenital glioblastic anlagen. Beneke claimed in contradistinction that regenerative glioses, representing sequelae of a cerebral trauma sustained during birth, are the basis for this phenomenon. The evidence thus far presented indicates that embryonic developmental anomalies are the blastogenic anlagen for a relatively limited number of cerebral neoplasms, but have no proven or probable relation to the great majority of these tumors. Some circumstantial evidence even suggests that focal glial proliferations, resulting from birth trauma, may occasionally assume such a role.

Before the conception of a possible transition of reactive, regenerative, and reparatory glial proliferations into neoplastic formations is given serious consideration in connection with traumatic episodes, a detailed survey of the ultimate outcome of cerebral glial reactions of various origin is essential. Meyer and Cook reported the presence of a diffuse gliosis of the white matter of the brain in mental defectives. These investigators stressed the absence of any developmental errors and of immature or blastomatoid characteristics of the proliferating glial cells, such as observed in the glioses associated with tuberous sclerosis and von Recklinghausen's disease. The glial proliferations in mental defectives were attributed by Meyer and Cook to abnormalities in the vascular pattern of the brain, causing a locally deficient oxygenation of the white matter, which in turn resulted in the glial proliferation. Marked and especially perivascular glial reactions are observed in connection with certain infections of the brain (epidemic encephalitis) and with numerous chemical poisonings (mercury, lead, and manganese). No data are available as to the occurrence of neoplastic sequelae from such conditions.

It is known that in multiple sclerosis, a disease of the central nervous system characterized by multicentric glial foci in the spinal cord and by gliosis of the brain, multiple, small, cerebral gliomas may occur (Müller). Syringomyelia (spinal gliosis with multiple cavitation of the spinal cord and medulla oblongata) is accompanied by the occurrence of small, tumor-like glial nodules within the gliotic areas located in the grey matter, and occasionally multiple small gliomas in the brain exhibiting a diffuse gliosis (Müller). Since the etiology of multiple sclerosis and syringomyelia is highly controversial (for multiple sclerosis an infectious origin and chronic lead poisoning have been favored recently, and for syringomyelia surgical trauma sustained at birth has been accused), the value of this evidence is more of a general and circumstantial character, but favoring the existence of interrelations between reactive glioses and gliomatoses of the brain.