changes the author holds that we must take for epilepsy the view that we take for many other disorders, namely, that pathological anatomy can distinguish certain peculiarities which, even though not constant, yet taken together when present are probably more or less characteristic and of value for making a probable diagnosis. Just as in general paralysis certain areas show a predisposition to certain types of change, and an exacerbation of the disease runs parallel with certain further cerebral changes, so he thinks that epilepsy is probably a disease process—possibly of more than one type not yet distinguished—which tends to affect certain areas in a way not wholly unlike many other processes, but with some predominant characteristics, and associated with certain other abnormalities frequent though not peculiar to the disorder. Thus he regards sclerosis of the cornu ammonis and cell degeneration with glia proliferation of the Purkinjë cells of the cerebellum as among the most constant changes, though not invariable, and believes that when these occur together with certain developmental defects attributed to an innate inferiority, akin to stigmata of degeneration, and also with Chaslin's "Randsclerosis" (sclerosis of the superficial layers), the whole picture gives as great a probability for the diagnosis of epilepsy as do similar degenerative changes for general paralysis when associated with other typical paralytic pathological changes.

He emphasizes that the value of pathological anatomy has been in the past, and is likely to be in the future, the gradual narrowing of the field by the exclusion of other conditions which were previously regarded as epilepsy, from, for instance, gross cerebral lesions, to the entity of the late form of amaurotic idiocy recently established by the author. It yet remains to discover whether, when all other entities have been separated out, there will remain any unified group to be regarded as genuine or idiopathic epilepsy.

Part IV: Genealogical [*Genealogisches*]. *Rüdin, E.*

The many difficulties of this form of research upon epilepsy are pointed out, beginning with the uncertainty of diagnosis and thus of the phenotype which is the starting-point. Much work of a rather unsystematic kind has been done which gives as a whole an impression that epilepsy and a great number of other psychopathic conditions are genetically connected. Very little has yet been completed which conforms to the strict requirements of modern methods. As far as it goes, it seems to establish with some degree of certainty the fact that idiopathic epilepsy does rest on some inherited basis, and that this basis is definitely recessive and not dominant. Far more must be done before we can know what other factors are part of this inherited factor, and whether they are monomeric or merely associated and separable. The author stresses the importance of remembering that it may be some clinically quite different manifestation which forms part of the entity (*e.g.*, cataract and myotonia atrophica), and that here clinical and geneological workers can help one another. Research into inheritance

may disclose some associated condition having in common with epilepsy some physical disorder which may give the clue to the nature and causation of both.

As far as any surmise can be made from the material available, it would seem that certain forms of congenital weakmindedness are the most frequent associates of epilepsy. The relation to alcoholism is definitely not proven; there is no convincing evidence that alcoholism in the parents, apart from the general psychopathic tendency which leads to the alcoholism, has any effect in producing epileptic descendants. For true dipsomania there is some evidence of a relation both to epilepsy and to manic-depressive insanity. Between epilepsy and other psychoses no constant relationship has been shown.

The author pleads for intensive research on approved lines, studying the descendants of known epileptics, with and without collateral taint, and of apparently normal parents of epileptic children and their collaterals. He mentions that his figures probably err on the side of being too low, as he neglects those children that have died with infantile convulsions, some at least of whom would probably have been true epileptics if they had lived. He hopes that geneological research may help towards clearing up the question of the relation of idiopathic epilepsy to convulsions of other origin, and the connection between inherited disposition and external precipitating causes, and may also lead to some classification of the possibly different hereditary groups which present a similar clinical picture, and are so far grouped together as idiopathic epilepsy.

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(1) *The Sequelæ of Encephalitis Lethargica.* (*Brain, vol. xlvii, part* 1, 1924.) *Duncan, A. G.*

(2) *Memorandum on Encephalitis Lethargica.* (*Ministry of Health*, 1924.)

(3) *General Considerations about Encephalitis* [*Allgemeine Betrachtungen ü. d. Enzephalitis*]. (*Schweitz. Archiv für Neurol. und Psychiatrie, vol. x*, 3, 1922.) *v. Monakow.*

(4) *A Case of Encephalitis with Cortical Localization* [*Un cas d'encéphalite epidemique aigüe à localisation corticale.*] (*Bull. et mem. Soc. med. des Hôpitaux de Paris*, 45, *Pt.* 3, 1921.) *Delater and Rouquier.*

(5) *Behaviour Changes Supervening upon Encephalitis in Children.* (*Lancet, vol. cciii, October* 28, 1922.) *Auden, G. A.*

The recent recrudescence of epidemic encephalitis is of especial interest to psychiatrists on account of the mental symptoms which may occur during the acute stage or develop at a later period. Dr. Duncan followed up a series of 136 cases for two to five years, and was able to trace 83 to a recent date. In 78 of these mental sequelæ appeared to have become chronic. The mental symptoms took the form of defective memory for recent and remote events, inability to fix the attention, abnormal drowsiness and tendency to narcoleptic attacks, sometimes alternating with nocturnal