Result.—There was no significant difference between the two groups, and no substantial increases were noted in the C group.

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AGE INCIDENCE AND PROGNOSIS OF EPILEPSY

BY

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In the course of some 20 years' experience of epilepsy in hospital and private practice I have been impressed by the number of cases of epilepsy of unknown origin beginning in middle and later life. While all writers recognize that idiopathic epilepsy may arise at any age, an onset in later life is generally thought to be rare. Organic disease of the brain which may cause epilepsy is commoner in later life, and therefore epilepsy in such patients is more likely to be a symptom of progressive intracranial disease than in children or adolescents. A statement such as the following, taken from the latest edition of Osler's *Medicine* (Christian, 1942), is typical: "It is well always to be suspicious of 'epilepsy' beginning in adult life, for in a majority of such cases the disease is not epilepsy" (italics mine).

Some statements, however, are much more dogmatic. For example, Penfield and Erickson (1941), in their recent book, *Epilepsy*, which is a very fine study of the surgical aspects of the problem, lay great stress on the age of onset of fits as a factor in diagnosis, though they are careful to state that idiopathic epilepsy may occur at any age. They say:

"In cases of onset of seizures between 35 and 55, the most frequent cause is cerebral neoplasm" (italics mine). "After 55 years the various cerebral lesions produced by abnormality of the cerebral blood vessels become more frequent, though the strong probability of neoplasm must still be remembered." And lastly: "Idiopathic epilepsy comes on in youth, not in early infancy, and not in middle age or old age."

Age of Onset

As such statements did not correspond with my experience, I instituted, with the aid of Flight Lieut. H. G. Miller, formerly medical registrar, and Dr. Frank Robertson, first assistant, an inquiry into the age of onset in my cases, and, secondly, into the cause of attacks in the later age groups.

TABLE I.—Age of Onset of Epilepsy: Present Investigation

Age (Years)	Private	Hospital	Total
0-10	50 (22%)	83 (22%)	133 (22%)
11-20	63 (28%)	122 (33%)	185 (31%)
21-30	49 (21%)	82 (22%)	131 (22%)
31-40	28 (12%)	44 (12%)	72 (12%)
Over 40	41 (17%)	40 (11%)	81 (13%)
Total	231	371	602

The cases number 602. The private cases cover a period of 18 years, hospital cases 12 years; hospital out-patient records before this time were removed for salvage without my know-

ledge. No cases seen during the past two years have been included, as insufficient time has elapsed to judge of their outcome. A study of the figures will reveal that there is a general correspondence between the private and hospital groups, and that of the whole series 25% showed the first evidences of epilepsy after the age of 30 and 13% after the age of 40. The group of 81 patients with onset of attacks over the age of 40 were chosen for special follow-up.

Table II shows a comparison with previous records of the age of onset of epilepsy. While all the records show the

TABLE II.—Age of Onset of Epilepsy: Comparison with Previous Records

Age (Years)	Gowers (1,450 Cases)	Turner (1,000 Cases)	Nattrass (602 Cases)
0-10	29%	31.3%	22%
11-20	46%	46.7%	31%
21-30	15.7%	12.2%	22%
31-40	6%	5.8%	12%
Over 40	3.3%	4.0%	13%

highest incidence in the second decade, the figures of these eminent observers (Gowers, 1901; Turner, 1907) indicate only 9 to 10% of patients in whom the first fits occurred after the age of 30, as compared with 25% in my series.

Results of a Follow-up

Table III summarizes the results of a follow-up of the 81 patients who developed attacks diagnosed as epilepsy with onset after the age of 40, and in whom at the time of initial examination no cause could be found for the attacks.

TABLE III.—81 Cases diagnosed Epilepsy of Unknown Origin with the First Attack after the Age of 40

Untraced	24
Deaths	16
Tumour proved 5, probable 2	7
G.P.I.	1
Cerebral arteriosclerosis	1
Heart failure	2
Air raid	1
Pancreatic tumour, ? hypoglycaemia	1
Unknown	3
Living and traced	41

The number of untraced patients (24) is perhaps not surprising considering the years involved in the inquiry. The 41 patients living and traced were nearly all re-examined; of the few who could not attend adequate information was obtained. The present condition of these 41 patients is indicated in Table IV.

TABLE IV.—Present Condition of 41 Traced Patients of the Series in Table III

3 patients have had only 1 fit:	
11 years ago, at age 53	
7	55
6	43
1 patient had 2 fits at age 47; now aged 54	
6 others have had no fits for the past 9 years (2 cases), 8 years (1 case), 5 years (1 case), 2 years (2 cases)	
The remainder continue to have fits at very variable intervals	

In some cases the frequency of fits remains unchanged, but on the whole there seems a tendency to diminution in the frequency and severity of attacks with the passing years. None of these patients shows any symptom or sign of intracranial tumour. Their general condition on the whole is good; most of them are living almost normal lives and are at work. For example, one patient is the manager of a large branch of an insurance company though he is the subject of major seizures and has very severe myoclonic twitching from time to time between attacks. Another patient had 7 major nocturnal attacks between the ages of 67 and 77, when they ceased. He is now aged 83, and is exceptionally well.

With regard to evidence of cerebral arteriosclerosis, a moderate degree of hypertension is found in a proportion of cases (Table V).

TABLE V.—Incidence of Hypertension (41 Cases re-examined)

B.P. in one patient	= 210/120
B.P. " " "	= 190/130
B.P. " " "	= 190/100
B.P. " " "	= 195/95
B.P. " " "	= 180/100 (Attacks ceased since 1935)

Hypertension seems to us to occur in no greater proportion in these patients than in non-epileptic subjects of the same age, and the height of the blood pressure has not been found to

bear any relation to the frequency of fits. Blood pressure in these patients has shown little variation since the onset of their fits. Other evidences of cerebral arteriosclerosis—for example, strokes and mental deterioration—are notably absent. Only one case showed marked mental changes when he first came under observation; this condition improved with the response of the fits to treatment, and the man has been back at work for some years.

Jacksonian Epilepsy

The question arises whether Jacksonian epilepsy is especially suggestive of tumour. Among the total of 81 patients with onset over the age of 40, 7 are known to have had Jacksonian attacks (Table VI). Of these, 6 are dead, the cause being shown.

TABLE VI.—Significance of Jacksonian Epilepsy among 81 Patients

Number known to have Jacksonian attacks	7
Died	6
Cerebral arteriosclerosis	2
G.P.I.	1
Heart failure	2
Unknown cause	1

The mortality rate is probably exceptional, but the point which emerges is that Jacksonian attacks, while revealing the site of the disturbance, give no indication of the nature of the lesion.

Time of Epileptic Attack

A last point of clinical significance is the time of epileptic attacks. Among the 41 surviving patients of the last group the time of attack was: nocturnal only, 13; diurnal only, 16; irregular, 12. The purely nocturnal incidence of attacks has two important advantages: first, such attacks, occurring in bed, are unassociated with danger of injury, and the patient is able to lead an ordinary life by day; secondly, there is a better chance of controlling nocturnal attacks by maximum doses of an anti-epileptic remedy given at night.

Summary and Conclusions

The age of onset of epilepsy is considered in a series of 602 cases.

81 patients in whom epileptic attacks began after the age of 40 were followed up, and the results of the inquiry are recorded.

Making full allowance for errors in diagnosis, it is clear that the majority of these patients who began to have fits after the age of 40 could not be the subjects of cerebral tumour, severe cerebral arteriosclerosis, G.P.I., or other progressive disease. Most of them continue to have epileptic attacks from time to time without other evidences of disease.

These observations provide no contribution towards the aetiology of idiopathic epilepsy, but they suggest some conclusions which may be of practical value: (1) According to my experience idiopathic epilepsy is by no means uncommon in persons of mature years. (2) Though fits may be the first symptom of intracranial tumour it is rare for them to precede all other symptoms and signs by months or years. (3) In the later years of life epilepsy has remarkably little effect on intellectual powers and general health.

I am interested in this matter from the point of view of teaching. I would suggest that students should be advised always to regard epilepsy as a symptom and to search for the cause both in the nervous system and elsewhere; and, secondly, to try to discover the site of the disturbance by careful analysis of the aura or earliest stage of the fit; but that these rules should apply to epilepsy at any age, and we should not stress unduly the incidence of progressive disease as a cause of epilepsy in the later years of life.

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THE SCOPE OF PREVENTION IN OPHTHALMOLOGY

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The recent publication by Marshall and Seiler* of their statistical analysis of 3,219 persons certified blind during 1929–35 raises the question of the preventive function of ophthalmology. There is at the present time a total of 74,000 blind persons in England and Wales whose maintenance costs the State £4,500,000 annually, while practically nothing is spent on planned prevention of blindness by improved facilities for treatment, by education of the public, or by research.

Marshall and Seiler's figures bring out certain noteworthy points. First, the seven most important causes of blindness are, in order of occurrence:

Cataract	16.77%	of the total examined
Myopia	16.34%	" "
Vitreous disease	13.95%	" "
Uveitis	10.72%	" "
Glaucoma	8.69%	" "
Congenital anomalies and abiotrophies	7.45%	" "
Injuries	6.42%	" "

Secondly, although at first sight the first four items would suggest that the greatest amount of blindness occurs in the older age groups and does not therefore carry so great an economic importance, yet actually only half the cases became blind after 50, and in two-thirds of these the underlying cause had started before that age. In view of the longer expectation of life now than formerly, and the increasing average age of the population in general, as well as of workers in responsible positions, it is obvious that most of the causes of blindness begin to operate early enough to constitute a risk of economic importance. Thirdly, the relatively low percentage of blindness from injury appears surprising until we realize two points—namely, that the position of injury in the list of causes varies between 10.3 and 5.8%, according to whether the first eye, the second eye, or both, are involved; also, most severe industrial eye injuries lead to loss of one eye rather than both, only those of great severity such as explosions and head injuries appearing to any extent in statistics of blindness, yet the time lost from injuries short of this order is of great economic importance.

Although, because of the certification of blind persons and the payment of pensions to the blind, statistics are readily available, what we lack is a precise knowledge of the amount of working-time lost and of inefficiency and disability caused by eye injuries and diseases which do not end in blindness but which nevertheless should be, if possible, reduced in number. Even if the condition does end in blindness there is a latent period of illness and steadily increasing disability during which the patient is unable to work. This is brought out by Marshall and Seiler's tables, which show the average times between the onset of the cause of blindness and the actual certification. In some cases it is considerable; for example, in all accidents the difference between the mean age of onset and the mean age of blindness is 3.11 years; in glaucoma it is 2.94, in cataract 5.1, and in inflammatory conditions it is higher still (e.g., trachoma 25.58, and uveitis 12.4), while it is highest in myopia (36.24 years). Quite apart from these figures exists the mass of time lost from accidents which recover and from accidents leading to the loss of one eye only, which never come to certification. As pointed out before, these must be borne in mind as explaining the, at first, surprisingly low figures for blindness as a result of injury, and it is to be emphasized that in any discussion on prevention the non-blinding conditions must be taken into account.

When we consider the list of conditions involved from the point of view of possible reduction of eye disease it becomes obvious that advances could occur mainly in three directions—

* Marshall, John, and Seiler, H. E. (1942). *Brit. J. Ophthalm.*, 26, 337, 385, 434.

H. Weyler and C. C. Dustin (*New Engl. J. Med.*, 1942, 227, 785) record their observations on a woman aged 24 who presented paroxysmal auricular tachycardia throughout the latter part of four pregnancies over a period of five years.