Conclusions

Neoantergan conferred no apparent benefit upon 25 specially selected and carefully observed asthmatic patients.

Neoantergan was markedly successful in reducing the severity of the symptoms of 15 cases of hay-fever.

When working with asthmatic patients an erroneous impression of the efficacy of a new preparation is likely to be formed unless a very careful scheme of trial is planned, preferably using a control preparation.

It is with pleasure that I acknowledge the help received from Dr. W. N. Mann during this investigation, and I am grateful to Dr. W. R. Thrower, of Messrs. May and Baker, for kindly supplying the anthisan. This work is an abstract from part of a Cambridge M.D. thesis, and for permission to publish it I am indebted to Sir Lionel Whitby, Regius Professor of Physic, Cambridge University.

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CHIASMA LESION DUE TO FALL IN EPILEPTIC FIT

BY

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Damage to the optic nerve due to a fall in an epileptic fit is surprisingly rare, although optic-nerve lesions may follow slight head injuries. Chiasmal damage occurring after epileptic seizures has not been previously recorded in the literature. The following case, which ended in optic atrophy and blindness in one eye, is of special interest because of its unusual course and visual-field changes.

Case Report

The patient, a single man aged 32, suffered from epilepsy, probably of traumatic origin. Generalized fits followed a severe head injury sustained six years before. These recurred at intervals of one to two months, with sudden falls and no aura. At the end of November, 1944, he fell on his forehead during a fit. There was a swelling over the right eye due to extravasation of blood. He was confused for the rest of the day—as usual after his seizures. Next day, when mentally clear, he complained of blindness in his right eye. Examination revealed a complete loss of vision in this eye, and a marked temporal-field defect of the left eye with normal visual acuity. This was evident in subsequent examinations. Simple and stereoscopic x-ray examination of the skull immediately afterwards did not show any fracture.

About a fortnight after the accident there was a distinct difference in the two disks, the right being paler. After a month there was an obvious atrophy of the right disk, with paleness, narrowing of the large retinal arteries, and reduction of the small vessels. This condition was still unchanged when a complete examination of fundus, visual field, and acuity was carried out in May, 1945, six months after the accident. Since further developments were not anticipated no regular examinations were made. On July 17, 1945, however, the patient reported that a few days previously he had begun to see again with the right eye and that vision had gradually improved. Immediate examination showed a central scotoma in his right

eye with full peripheral visual fields. The visual acuity for finger-counting was 3 ft. (0.91 metre). An examination two days later showed that the central scotoma had disappeared and that the acuity had risen to 6/9. The visual field in the left eye still showed some temporal defect, but to a less degree than in the first period after the accident.

On Aug. 2 he reported that the sight of the right eye had again become worse, and attributed this to an injury inflicted by a patient, who he alleged threw lumps of soil into his face. He also stated that a few days previously he had had to bend his head while helping with weeding in the garden. The visual field two days later showed a central scotoma and a concentric constriction of the peripheral field; the visual acuity had fallen to 1/60. Subsequent examinations showed that the scotoma gradually increased in size, the peripheral field became more and more restricted, leaving eventually a pericentral annular island of vision, and within a week the patient was again blind in the right eye. The visual field of the left eye showed no definite temporal defect during this phase. The direct pupillary reaction to light in the right eye was from the beginning of the observation sluggish and poor, decreasing in the first week after the accident until it became fairly stable after the atrophy of the optic nerve had been established. No definite change was noted during the short spell of recovery. Up to January, 1948, no further change has occurred; the right eye remains blind, with atrophy of the disk; the left eye shows full visual field and normal acuity. The direct light reaction, though very poor, is still obtainable.

Discussion

Complete blindness and optic atrophy of the right eye after a fall in an epileptic fit, and a temporal-field defect of the left eye, pointed to a lesion in the chiasmal region affecting the fibres of the right optic nerve and the nasal retinal fibres from the left eye. This type of injury has been repeatedly discussed in the literature. The skull fracture which often accompanied such lesions suggested a direct traumatic damage to the nerve, and specimens have been shown in which the nerve in this section had actually been torn. Early writers were mainly concerned with the possible types of skull injury which might produce this nerve damage. Various mechanisms have been suggested (Liebrecht, 1912; Coppez, 1929; Rand, 1937), but more recent experience has made it clear that bone injury and anatomical nerve lesions are not the only cause of the traumatic chiasma syndrome, and that vessel damage alone may have the same effect and produce the initial symptoms (Traquair, 1933, 1942; Dandy, 1932; Rodger, 1943; Hughes, 1943). Rodger differentiates bleeding into the sheaths and subvagal and intraneural haemorrhages.

There can be little doubt that in our patient there was a vascular lesion; the remission and subsequent relapse cannot be explained in any other way. The fact that central vision was the last to return when the recovery took place, and that a central scotoma was the first symptom when the relapse occurred, seems to indicate an intraneural damage. The onset immediately after the injury is in favour of traumatic rupture of the vessel wall, with consequent intraneural haemorrhage (Rodger, 1943). This would also account for the relapse. After a slight injury the vessel, which had been closed, started to leak again, causing a gradually increasing extravasation—a mechanism well recognized in meningeal and brain-vessel damage, when a minor incident may cause renewed extravasation long after the initial trauma.

With regard to the progress of our case the changes during the first few weeks after injury were as anticipated. It is the general experience that a visual-field defect and loss of visual acuity occur immediately after the injury, while atrophy of the optic nerve usually appears within four weeks. In seven cases of optic-nerve lesions Rodger (1943) noted an average interval of 17 days, the shortest

interval being four days. In our case blindness set in immediately after the accident, and atrophy was distinct after a fortnight.

Concerning the prognosis of these cases there has been general agreement (Traquair) that if no signs of improvement in visual acuity occur during the first four weeks no further change can be expected. But in our patient there was a rapid and almost complete recovery as late as five months after the injury. This is difficult to explain; it is contrary to all experience that recovery should have started after so long a time. One might think of a hysterical fixation of an original organic blindness, but nothing was observed in the general behaviour of the patient to support this, and there were no psychogenic elements. There were, on the other hand, optic atrophy and a transitory scotoma—findings which do not permit a functional explanation.

It is likely that the primary site of the lesion was in or near the anterior chiasmal angle, affecting mainly the right optic nerve but also damaging the crossed fibres of the left. It is interesting to note that the temporal-field defect of the left eye gradually diminished and had entirely disappeared after the relapse. We will therefore have to assume that on this occasion the chiasma had been spared, the lesion being restricted to the right optic nerve.

Summary

A case is reported in which blindness and optic atrophy in the right eye, with a temporal-field defect in the left eye, followed a fall in an epileptic fit. X-ray examinations of the skull were negative. There was no fracture.

Not less than five months after the injury normal visual acuity with full visual fields in the left eye were re-established; during this period of recovery central vision was the last to return to normal. The temporal-field defect in the left eye was at the same time greatly reduced.

Shortly afterwards, following a trivial blow on the face, his sight and visual-field condition deteriorated, a gradually extending central scotoma appeared, and visual acuity fell rapidly until the right eye became blind again. There was no temporal-field defect in the left eye at this time. Atrophy of the right disk remained unchanged during the whole period. There was no further change one year later.

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Dr. Esmond R. Long, director of medical research and therapy, National Tuberculosis Association, has been appointed editor-in-chief of the *American Review of Tuberculosis*, official journal of the N.T.A.'s medical section, the American Trudeau Society, to succeed the late Dr. Max Pinner. A new position of managing editor has been created, and Dr. Walsh McDermott, associate professor of medicine, Cornell University Medical School, New York, has been appointed to the post. Dr. Long, who is also director of the Henry Phipps Institute, Philadelphia, has been director of the N.T.A.'s medical research since January, 1947. He is a past president of the N.T.A. and is a consultant on tuberculosis to the United States Army, the Veterans Administration, and the U.S. Public Health Service. A graduate of the College of Physicians and Surgeons, Columbia University, Dr. McDermott is associate attending physician of New York Hospital and was associate editor of the seventh and eighth editions of the Cecil-Loeb *Textbook of Medicine*. He is a member of the American Trudeau Society's Committee on Medical Research and Therapy and of the Antibiotics Study Section and the Tuberculosis Study Section, National Institute of Health, U.S. Public Health Service.

JUVENILE GENERAL PARALYSIS

A CASE WITH A SPONTANEOUS BUT SHORT-LIVED REMISSION

BY

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Although a considerable number of cases of juvenile general paralysis have been reported in the literature the disease is relatively rare and its incidence in cases of congenital syphilis is stated by Stewart (1933) to be about 1%. Schmidt-Kraepelin (1920) gives the figure as 1.7%, but White and Veeder (1922) and Smith (1933) are in agreement that the incidence is less than 1%. Most authorities agree that the sex distribution is approximately equal, although there is a slight tendency towards a preponderance of males; thus out of 145 cases reported by Mott (1899), Watson (1903), Schlicht (1915), Schmidt-Kraepelin (1920), Klauder and Solomon (1933), and Menninger (1930), 83 were males and 72 females. According to Menninger (1936) there are two types of onset, the first being insidious, when the child is apparently subnormal from birth, and the second in which an acute breakdown occurs after several years of seemingly normal development. Menninger in an analysis of 542 cases gives the age of onset as 3 to 5 years in 9.4%, 6 to 10 years in 24%, 11 to 15 years in 36.2%, 16 to 20 years in 26.8%, and 21 to 25 years in 3.6%. In the majority of cases, therefore, the illness begins between the ages of 10 and 15. Menninger states that in a series of 653 cases psychic trauma was regarded as the precipitating factor in 2.7% and that convulsions occurred in 17% before the diagnosis was established.

Case Report

A boy, aged 11 years 3 months, was admitted to this hospital in November, 1946, as he was not responding to out-patient treatment at a child-guidance clinic in another area.

Family History.—The father, a Naval petty officer aged 38, was stated to be fit and well. He has had little to do with the boy's upbringing owing to his service overseas. When interviewed in December, 1946, he admitted that he had had primary syphilis in 1929 but had received a full course of treatment over two years. His Wassermann reaction was: blood +, C.S.F. negative. He was having treatment at a V.D. clinic. The mother, aged 36, is fit and well. She is a kindly woman, but ineffective in handling the family and lacking in understanding. Her Wassermann reaction was: blood +, C.S.F. negative. She has had no treatment. **Siblings:** There are three sisters, aged 16, 7, and 4. All are bright and alert, with negative serology.

Personal History

The boy weighed 8 lb. 12 oz. (3.98 kg.) at birth, walked at 12 months, and began talking at 15 months. Clean habits were established early, and he appears to have been a perfectly normal child until a few days before his fifth birthday, when his home was destroyed by enemy action. He was uninjured, but was said to have screamed persistently for several hours after the incident. He was apparently terrified during the next three days, and there were many outbursts of screaming during this time. On the fourth day he had an epileptiform attack during which he exhibited loss of consciousness and left-sided rigidity followed by generalized clonic movements. In the succeeding nine months he had frank epileptic major attacks with incontinence and tongue-biting at the rate of about one a week. These persisted despite anticonvulsant drugs prescribed by his doctor. During this time he progressively became more dull and was excessively timid and anxious. There was, in fact, a complete personality change, and whereas he had formerly been a bright, cheerful, alert, self-possessed child, he now became very quiet and reserved, and was made fun