as Janus-faced, looking one way towards death and the other towards life.[10] Survival depends on the relationship between the self-destructive and the life-preserving tendencies. If the former are entirely absent we are not dealing with a suicidal attempt.

Steroid Glaucoma

In 1954 J. François[1] pointed out that cortisone drops applied to the eye for the treatment of conjunctivitis increased the intraocular pressure, reduced visual function, and caused optic atrophy. H. Goldmann[2] brought further cases to our notice in 1962. Unfortunately the problem is still with us.

At a recent meeting of the Ophthalmological Society of the United Kingdom S. J. H. Miller[3] described six cases of corticosteroid-induced glaucoma, and five of these patients had severe loss of visual field which was permanent. The primary condition for which each patient was treated was episcleritis, atopic eczema, allergic conjunctivitis, pinguecula, vernal catarrh, and sympathetic ophthalmitis. None had a family history of glaucoma so far as was known. All of them complained of intermittent haloes or blurred vision and had considerably raised intraocular pressure. They responded dramatically to cessation of therapy in that the intraocular pressure returned to normal limits with one exception. None of them showed continuing deterioration of the field of vision after cessation of treatment, and indeed one or two showed an improvement; the severity of the field loss was proportional to the total quantity of steroid given.

In recent years the effects of topical corticosteroids on the fluid dynamics of the eye have received careful study, chiefly by B. Becker[4 5] and M. F. Armaly.[6-9] The topical corticosteroids which have been reported to increase the pressure are cortisone itself, hydrocortisone, betamethasone, and dexamethasone in ascending order of potency. The hypertensive response over a period of four weeks is always greater in eyes with open-angle glaucoma. The rise is not accompanied by signs of congestion and resembles that seen clinically in glaucoma simplex. It has also been found that an individual with apparently normal eyes who has a parent with open-angle glaucoma reacts to topical corticosteroids by a rise in pressure greater than that of his contemporary who has no family history, and the rise is comparable in size to that found in a patient with frank glaucoma.

Becker[5] proposed a genetic hypothesis to explain the difference in response to topical corticosteroids found in a normal eye and in one with open-angle glaucoma. Glaucoma simplex is considered to be a heritable trait and to represent the homozygous recessive state with a frequency of 4% in the general population. Theoretically the heterozygous state should have a frequency of 32% and the homozygous dominant 64%. The size of the steroid effect on intraocular pressure divides a series of normal people into two groups: group I with the less response consists of individuals who do not bear the recessive gene, and group II with the greater response consists of individuals having the recessive gene either in the heterozygous or in the homozygous state.

Armaly[9] has carried this one stage further. Applying 0.1% dexamethasone 21-phosphate three times daily to the right eye in 80 volunteer subjects, he has defined three significantly different groups by the size of the rise in pressure. Group I showed a low level of response with an average rise of 1.6 mm. and formed 66.2% of the sample. Group IIa showed an intermediate level of response with an average rise of 10 mm. Hg; this formed 28.8% of the sample. Group IIb showed a rise of pressure of 16 mm. Hg or greater, and this group formed 5% of the sample. These percentages are very close to the theoretically derived figures based on Becker's genetic theory.

G. Paterson[10] has reported a study on a series of siblings and children of patients known to have glaucoma simplex. The results were analysed so as to compare Armaly's figures from a sample of the general population with those taken from a group of people who were known to carry the gene of glaucoma. Theoretically none of these latter patients should be in group I, but should show either an intermediate rise indicating the heterozygous state or a large rise indicating the preclinical stage of glaucoma, the disease being age-dependent. In fact Paterson's figures showed that 33% of the siblings and 33% of the children actually fitted into group I.

There are several possible explanations for this divergence from expectation. The total dosage of steroids employed by the two workers was not the same. It may be that the penetrance of the gene is incomplete in some cases or that the heritable trait is not the disease of glaucoma itself but rather the pressure response to corticosteroids. Further investigation is required to resolve this paradox.

Meanwhile the clinical lesson to be learnt from this work is the danger of topical corticosteroids, particularly beta- and dexamethasone, when prescribed for a period longer than a week unless ophthalmic supervision is close. The possibility, however, of using corticosteroid eye drops to detect genetic liability to glaucoma may prove to be of practical value.

Electroencephalography in Childhood

The sudden occurrence of a local lesion in the brain is usually accompanied by a prompt change in the electrical activity of the neighbouring regions. This can be recorded by the electroencephalograph (E.E.G.), though the relation between the lesion of the brain and the concomitant electrical changes is not fully understood. Frequently, however, the E.E.G. record changes continually until the condition has either resolved or become static.[1]

The E.E.G. findings in childhood differ from those in adults, both in normal subjects and in patients with diseases of the brain. For example, the progression of brain tumours in children is considerably different from that of adults, both clinically and electroencephalographically. Similarly, acute lesions of the brain, such as abscesses, various forms of encephalitis, or vascular disorders, also have different features according to the age of the patient. In children the E.E.G. changes are often disproportionately greater than those seen in apparently similar conditions in adults.

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[8] —— ibid., 1964, **71**, 636.
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