
"It has, in fact, been my object in preparing these lectures, to sit, so to speak, on the hedge between general medicine and ophthalmology, or—to put it in another way—to convert what might be regarded as a no-man's land between these two departments into an everyman's land." "I have no wish to make my remarks encyclopaedic, and would prefer to be eclectic, picking out from the mass of accumulated data of medical ophthalmology, those features which I think are of interest."

These two sentences from different parts of Williamson-Noble's three Lettsomian Lectures exactly express what he has actually done, and in the doing of it he has put the Reactions of the Eye to General Disease into a smaller and sharper focus than usually is the case. Such, at any rate, is likely to be the opinion of most ophthalmologists. If the reader will look up in the dictionary the word "abstract" he will find that it has a number of shades of meaning. For example, it may mean a précis, or, on the other hand, a theft. The reviewer is distracted as to which of these meanings to adopt and must endeavour to combine them, since a précis pure and simple would occupy too much space.

The author first deals with measles which is associated not only with conjunctivitis and corneal ulcers but with non-paralytic convergent squint. "The toxin of measles seems in a fair number of cases to have some peculiar selective action on the fusion centre in the brain, and to bring about concomitant convergent squint, apart entirely from anything in the way of encephalitis." Dealing with the reactions of the iris and ciliary body "which must be considered together," he explains the circulation of the aqueous and the various disturbances which occur in it, makes special reference to the gelatinisation of the fluid, particularly in gonococcal iritis, and to localisation of inflammation to a small area of the ciliary body in syphilitic infection, this leading to gummatous degeneration of the ciliary body and sclera; thus a benign condition (episcleritis) may be simulated when in reality degeneration may be progressing to perforation of the eyeball.

The type of iritis produced by focal infection "usually bears a close resemblance to that caused by the gonococcus. It is subject to relapses, may remain unilateral for a long time, and usually results in the formation of narrow adhesions between the iris and lens, in distinction to the syphilitic type of iritis which is commonly
bilateral and causes broad adhesions.” ... “An interesting feature in many of these cases is paralysis of the sympathetic on the affected side. ... It is a nice point, and one which has not yet been decided, whether the sympathetic paralysis is the primary cause of the cyclitis. If there are such things as trophic fibres, and if their course to the eye is via the sympathetic nerves, then interference with these nerves will reduce the resistance of the iris and render it more liable to bacterial invasion.” The possibility of endocrine factors taking a share in the causation of chronic iritis and cyclitis is illustrated by a case history. Still dealing with focal infection, what the author has to say about anaphylaxis and elective localisation must be read in the original, but the following may be noted. “No definite opinion seems to have been expressed yet as to whether interstitial keratitis following a blow or injury to the eye can be regarded as attributable to this or not.”

A very large part of what the author has to say about the reactions of the iris and ciliary body to syphilis is occupied by his discussion of the Argyll Robertson pupil. Only the opening statement can be given here. “In the first place, what exactly is the Argyll Robertson pupil? It seems to consist essentially in the absence or a marked diminution in the reaction of the pupil to light with preservation of its reaction to accommodation-convergence. There may or may not be associated miosis, and the consensual reflex to light on illuminating the fellow eye may or may not be present. The lesion is to be regarded as the consequence of destruction, whether by toxins, new growth or trauma, of a certain part of the pupillo-reflex arc. It would therefore follow that, although the Argyll Robertson pupil is in a high percentage of cases due to neurosyphilis, it is not necessarily pathognomonic of this disease.” Discussing the cerebral connections, the author says, “The special ability of syphilis to produce the lesions is to be attributed to the peculiar affection of the syphilitic toxin for the terminal arborisations of afferent nerves, the toxin spreading from the aqueduct into the subependymal tissues. The localisation of the lesion to this part of the brain may also serve to explain the miosis which is so frequently present, since it is probable that fibres carrying inhibitory impulses from the cortex to the sphincter iridis pass through the periaqueductal tissue, and so are affected by the toxin diffusing into this tissue.” (The author indicates that this is only one theory of its localisation.)

The author commences the second lecture by consideration of the lens, reviewing the latest work on its changes in diabetes, and emphasising the necessity, in estimating the utility of the eyes, of remembering to investigate the refraction. Calcium deficiency in connection with the cataract associated with tetany is discussed. Tuberculosis occupies several columns of the Lancet. No précis of these could do them justice, but it is worth noting in particular the
author's view on tuberculin treatment. "... There is no question in my mind that a number of cases of obstinate infection of the uveal tract and of the cornea do better with tuberculin than with any other form of treatment." Four columns are devoted to high blood pressure. The changes in the retinal vessels are reviewed, with diagrams and references to recent literature. As the author says, "an enormous literature has grown up recently around this subject." Friedenwald's classification of vascular anomalies is given. One or two sentences may be transcribed from what is said on high blood pressure. "The condition of the small vessels passing to the macula may be of great importance in cases of arteriolar sclerosis. In fact, in some cases of vascular disease of the most serious type, these may be the only vessels showing any visible change. (A mydriatic is probably required, and for this purpose cocaine is recommended). The changes observed in these vessels are an alteration in their course, whereby they become corkscrew-like, and an increase in the brightness of their reflex, which renders them like silver wire." Again, "if sclerosis should persist in the retinal arteries over a period of years, the retina itself may begin to show changes... Histologically, the change consists in the formation of hyaline nodules in the outer molecular layer of the retina. These nodules are visible with the ophthalmoscope as bright white dots, seldom larger in diameter than one of the main veins. They are usually grouped at the posterior pole of the eye, and may occasionally form a star or a fan-shaped figure round the macula." The discussion of the causes of the appearances in albuminuric retinitis does not lend itself to the making of a précis, or even to the "theft" of isolated parts of it. With regard to diabetic retinitis the author says, inter alia "The haemorrhages are for the most part small and circular, owing to their situation in the deeper layers of the retina. There are no cotton wool patches, and the white areas present have a soapy appearance with clean-cut edges. Sometimes they are arranged in a circle round the macula. Finally, there is no retinal oedema to speak of, and there is no star figure." Dealing with the causation of diabetic retinitis here is the opinion given. "It appears, therefore, that arterial sclerosis and renal disease are concomitant, possibly contributory, but not causative of this form of retinitis... The essential lesion in the disease (i.e., the retinitis) has seemed to me to be the recurrence of the deep-seated retinal haemorrhages."

The third lecture hardly lends itself to any form of précis writing. It concerns the retinal changes in anaemia, and the reactions of the optic nerve to disease in other parts of the body and this includes retrobulbar neuritis, papilloedema and optic atrophy. The first named subject (anaemia) must be read word for word in the original. From the others a few sentences may be quoted, notably regarding
retrobulbar neuritis in its relation to disseminated sclerosis. "Its aetiology is still rather a problem. . . . At one time the majority of these cases were attributed to direct extension from sinus disease, but this does not appear to be so. In a series of 76 cases of retrobulbar neuritis, E. D. D. Davis found only 5 in which it was attributable to manifest nasal sinus suppuration, while 14 of the 46 in which a cause could be discovered were due to disseminated sclerosis and 9 to syphilis. Disseminated sclerosis thus seems to be the commonest single cause of retrobulbar neuritis. It is probably commoner than is indicated by Davis's figures, since it may not be until years after the optic nerve involvement that other signs become manifest which would warrant a diagnosis. So that, of the 30 cases in which no cause could be found, it is more than likely that a goodly proportion would turn out to be disseminated sclerosis." Next, one or two references to papilloedema and to optic atrophy. It is pointed out that the pathology of papilloedema is still a subject of discussion and that in some quarters it does not seem to be understood. "The whole thing can be expressed very shortly (diagram given). The subdural space of the optic nerve is normally in connection with the subdural space of the brain through the optic foramen. If, therefore, there is an increase in the general pressure of the cerebro-spinal fluid, such increase will be transmitted to the subdural space of the nerve unless it is distorted in some way at the optic foramen so as to block the communication. The increase of pressure is not, however, transmitted to the interior of the eye. The effect, so far as the optic disc is concerned, is therefore similar to that on, say, the hand when a Bier's bandage is put round the arm, and oedema must obviously occur. . . . Whether the oedema is due principally to blockage of the lymphatics or of the vein does not seem to matter very much." Speaking generally, papilloedema has no localising value. Reference is made to Leslie Paton's findings in this regard.

The author agrees with Leslie Paton that it is futile to attempt to divide optic atrophies into only two classes, and gives some examples.

Lastly, the reviewer may refer to two pitfalls to which the author draws attention in estimating optic atrophy. The first, well known to every ophthalmologist, is the varying physiological tint of the disc. The second refers to the colour as seen by different forms of illumination, and especially the apparent pallor which may be noticed when using an electric ophthalmoscope "particularly if equipped with a new lamp and battery. My own practice in a doubtful case is not to express any opinion until I have had the opportunity of examining the disc through a dilated pupil with a reflecting ophthalmoscope and a yellowish source of light such as is provided by a carbon filament lamp. Even then I like to know
something about the fields and the corrected visual acuity before saying very much."

These scrappy references and quotations require that the reviewer should apologise to the author, though, in the former's favour it may be said that, since nearly thirty columns of closely reasoned matter in the Lancet are involved, a book notice would be more suitable and distinctly more easy to write. And will not a book notice ultimately be required?

**Ernest Thomson.**

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**BOOK NOTICES**


This report carries a step further the reports of similar but not identically constituted committees published by the Medical Research Council in 1922 and 1923; but that step is a very important one. The earlier committees had arrived at the conclusion that the factor of primary importance in the prevention of Miners’ Nystagmus was the supply of adequate illumination, particularly for workers at the coal face. Measures based upon the recognition of this factor have, however, had no success in diminishing the amount which is being paid by coalowners in compensating sufferers from the disorder.

The present report confirms the previous findings as to the importance of adequate lighting if the physical jerking of the eyes is to be avoided, but points out that this feature may never have been noticed, or may have subsided, in many cases in which severe psychological symptoms of the disease exist, while on the other hand nystagmic jerking of the eyes is quite frequently present in miners still engaged in their work and in whom the incapacitating psychoneurotic symptoms may never develop.

The conclusion is reached, and it is a very important advance, that in Miner’s Nystagmus we have to deal with a psychoneurosis allied to disabilities of this class in other industries, and to shell-shock. Accordingly, that the affected miner needs to be encouraged to do such work as he is capable of performing, and not allowed, or as at present even practically compelled to remain idle.

That, for patient or observer to concentrate on the cure of the physical manifestation of eye jerking is unjustified, and impedes recovery.

The report ends with the words: "The Committee are strongly of opinion that the practical treatment of the disease from an administrative point of view, should consist in the elimination of a hopeless