AN ADDRESS ON OPHTHALMOLOGY IN ITS RELATION TO GENERAL MEDICINE.

*The Annual Oration delivered before the Medical Society of London.**

By J. HUGHLINGS JACKSON, M.D., F.R.C.P.,

Physician to the London Hospital, and to the Hospital for the Epileptic and Paralysed; etc.

I CANNOT find appropriate words to express how much I feel the honour of being asked to give the annual Oration before the Medical Society of London. I think, after all, the best return I can make is not to waste your time in a fruitless endeavour, but to begin my real task at once.

The subject I have taken up for this address is one which interests me more deeply than any other whatever. It is on the bearing of a very special department of medicine on general medicine. I wish to show how we have profited and may profit by what ophthalmic surgeons have done in their special field of work. I think it the luckiest thing in my medical life, that I began the scientific study of my profession at an ophthalmic hospital.

As scientific medical research goes on, there is greater specialisation of investigation, just as, in the development of society, there is that continually increasing specialisation, called division of labour. This being so, all the more need is there that there should be greater integration, just as along with division of labour there is need for co-operation of labourers. I do not think there is any class of men in our profession to whom the term "specialist", in its bad sense, can be less justly applied than to ophthalmic surgeons; for it is plain enough that their department potentially includes the whole range of medicine and surgery. The ophthalmic surgeon operates for cataract; he treats palsies of ocular muscles and studies anomalies of refraction; he notes local tissue-changes, which are parts of some wide pathological condition. No class of men in the profession have done more for the methodical investigation of disease than ophthalmic surgeons. There is no harm in studying a special subject; the harm is in doing any kind of work with a narrow aim and with a narrow mind. Differentiation of medical investigation has led to results of inestimable value. If, in the smallest degree, I succeed in furthering the integration which should quickly follow this necessary differentiation, I shall feel that my address will be of some little value.

The direct utilitarian value of ophthalmological knowledge needs no comment. But the value of such knowledge for general medicine, although recognised, is not even yet thoroughly appreciated. Physicians have not fully applied the facts and principles which ophthalmic surgeons have discovered for them. It is not too much to say that, without an extensive knowledge of ophthalmology, a methodical investigation of diseases of the nervous system is not merely difficult, but impossible. It is plain at first glance. The nerve of the most special of all the special senses goes to the eye, and the end of it can be seen by the ophthalmoscope. The muscles of the eyeball are supplied by three nerves, each coming from a different part of the encephalon; the cornea is supplied by the fifth nerve; the orbicularis palpebrarum is supplied by the portio dura. There are, then, nine cranial nerves, and six of them supply the eyeball and ocular apparatus. By the ophthalmoscope, not only can we see a nerve-ending, but also an artery, a vein, and a nerve-expansion. We can see the effects of an embolism; we can watch arterial pulsation in disease of the aortic valves; we can see tubercle in the choroid, and diagnose syphilis and Bright's disease. Further, the commonest ophthalmoscopic appearances occurring in physicians' practice point to the general nature of intracranial disease; optic neuritis supplies, not decisive, but yet the best evidence there is to be had for the diagnosis of organic (gross) brain-disease.

I do not think that a student can learn what he requires to know in ophthalmology as preparatory to a study of diseases of the nervous system, except under the direction of an ophthalmic surgeon. For example, an ordinary knowledge of paralyses of the muscles of the eye is not sufficient; there are many things about them which can only be properly taught by a skilled ophthalmic surgeon. There are some things which it may at first glance seem needless for a physician to know, such as anomalies of refraction; but I shall show that

great blunders are made from ignorance of this department of ophthalmology.

But this is not all. Ophthalmic surgeons investigate their cases very methodically and very precisely. And thus, for the sake of scientific discipline, a study of ophthalmology is most important, even if we could make the assumption that the student would never be consulted for any sort of affection of the eyes, either alone or along with other symptoms. I do not know of any kind of work better fitted for correcting loose habits of observation and careless thinking than a study of palsies of ocular motor nerves. If these disorders of movement were studied carefully before the study of such more complex disorders as locomotor ataxy and ataxy of articulation was undertaken, we should hear less of metaphysical explanations, more about movements and less about a faculty of co-ordination, and we should, I think, hear less of "loss of volition" and "loss of memory" as explanations of physical defects. I wonder what an ophthalmic surgeon would say if, as an explanation of a patient's inability to look in this or that direction, he were told that the patient had lost the memory of that ocular movement, or that paralysis of the external rectus was due to loss of volition, or that confused sight was owing to disorder of co-ordination. Would he not say that these explanations, seeming to explain everything comfortably, really explain nothing whatever? Yet just such "explanations" are given of some symptoms of nervous disease: for example, in some cases of aphasia in locomotor ataxy and disease of the cerebellum.

Being a physician, I am desirous of saying as little as possible on the purely ophthalmological side of the matters to be considered. I wish to point out the wide value of certain ophthalmological facts, and especially their bearing on the methodical investigation of nervous diseases. Sometimes particular facts from ophthalmology are used merely as illustrations, merely to introduce some general medical question which ophthalmological evidence helps to elucidate. The subject even thus narrowed, however, is still so very extensive, that I can only speak fragmentarily and very briefly.

Before I do this, let me mention one case, and it is a type of many other medical cases, which shows that varied ophthalmological knowledge is necessary to the physician. A patient had epileptiform seizures, beginning in his left great toe; he had paralysis of the right third nerve; there was slight double optic neuritis; there was old choroidal disease in the left fundus; there was also an old nebula on the left cornea, and some astigmatism. We found *post mortem* syphilitic tumour of the right cerebral hemisphere and a syphilitic neuroma of the right third nerve. Without ophthalmic knowledge, we might have had a very confused notion of the relation of the disease of the cerebral hemisphere to the patient's defect of sight.

Before entering into the consideration of particular topics, I must remark that Dr. John Ogle was the first physician in this country to use the ophthalmoscope in medical cases. There is another physician who has done work of inestimable value in medical ophthalmology. Dr. Clifford Allbutt's work, *The Ophthalmoscope in Nervous and Renal Diseases*, has done more than any other work towards what I would call the integration of ophthalmology with general medicine.

I can say very little of affections of the superficial parts of the eyes. The most important to physicians is interstitial keratitis. This affection is, Mr. Hutchinson has shown, a certain proof of congenital syphilis. It usually goes along with a dental malformation, which, as he has shown too, is evidence equally certain. This important double discovery could scarcely have been made except at an ophthalmic hospital, since the cases are rare and are only met with in numbers at a large ophthalmic hospital. Here is a great debt we owe to one whom I would call an ophthalmic surgeon, were he not distinguished by having done good work in other departments of surgery. Having in the corneal and dental peculiarities a certain warrant for the diagnosis of syphilis, we can study the doings of congenital syphilis in other organs. In investigations of diseases of the nervous system, I have profited very largely in the diagnosis and therapeutics of non-ocular diseases from these tests of inherited syphilitic taint.

Passing over numerous other important organic affections, I will speak of anomalies of refraction. It is exceedingly important that the physician should study anomalies of refraction and their indirect effects. I do not mean that it is necessary for him to have that familiar knowledge which the ophthalmic surgeon must have, but, for general medical purposes, he should be able to diagnose them. It is not necessary that the physician should be able to cut for stone, but it is needful that he should be able to diagnose or to suspect vesical calculus, or he will subject his patient to a long course of inefficient treatment. A knowledge of the indirect effects of anomalies of refraction would, doubtless, save us from serious misinterpretations of symptoms. Mr. Brudenell Carter records the case of a patient who came complaining of "peri-

* This report contains many paragraphs omitted in the delivery of the address.

odical confusion of vision". He worked at an office commanding a view of a large clock on the other side of a court-yard; he "could see the hands of the clock when they were approximately vertical, but lost them when they were approximately horizontal". Such a case would be inexplicable without ophthalmological knowledge. Mr. Carter diagnosed astigmatism and found it.

We should learn how to estimate degrees of hypermetropia by the ophthalmoscope; because, when a patient is too ill to permit testing the state of his refraction in the ordinary way, the ophthalmoscope may be the only means of deciding. I could relate a striking case in which the discovery of a high degree of hypermetropia by the ophthalmoscope in the hands of Mr. Couper was of inestimable value in unravelling a complex case in which there were partial aphasia, defect of sight, and right-sided convulsion, due to cerebral tumour.

We have learned from an ophthalmic surgeon that the common kind of strabismus is not really of nervous origin, as, before Donders's researches, it was universally supposed to be. It is indirectly owing to congenital shortness of the eyeball, to hypermetropia. This is an enormous gain both for diagnosis and for therapeutics. It is now so well known, that we are scarcely conscious of the serious blunders from which Donders has saved us. Strabismus indirectly owing to hypermetropia being taken to be of nervous origin, has, no doubt, often introduced sad confusion into the report of a case of brain-disease, and led to erroneous prognosis and misdirected treatment.

In some cases of abnormal refraction, there is a simulation of brain-disease, or, at any rate, patients who only want appropriate glasses are occasionally treated by physicians for brain-disease. I have been speaking of hypermetropia, but the remark applies to other abnormalities of refraction.

Mr. Brudenell Carter has reported a remarkable case of myopia simulating brain-disease in the eighth volume of the Clinical Society's *Transactions*. This report should be carefully studied by physicians. The patient, for supposed brain-trouble, took a voyage to Australia, but was no better for it. He was told that "he must abandon the idea of carrying on the family business, or of taking any active part in life". This patient was immediately thoroughly and permanently cured by the adaptation of appropriate glasses. The possibility of an anomaly of refraction or any eye-defect being at the bottom of that patient's troubles seems not to have occurred either to himself or to his doctors. He did not consult Mr. Carter for any defect of sight, but because he had heard that ophthalmic surgeons had an instrument useful in the investigation of disease of the brain. I could relate several cases of hypermetropia in which the diagnosis of brain-disease had been made by physicians and refuted by ophthalmic surgeons. Here is one. A medical student, twenty-one years of age, had been obliged to give up his work because reading brought on attacks of vomiting and frontal headache. The vision of each eye for near and distant objects seemed good to ordinary examination, and there were no ophthalmoscopic changes, except some dilatation of retinal veins. To cut a long story short, after travelling about two years, doing nothing towards his career, Mr. Tweedy, in May last, fitted him with a pair of spectacles, remedying hypermetropia and astigmatism. The patient has been well ever since; has returned to his medical career, and has graduated at the University of London. Without some knowledge of anomalies of refraction, the physician would never suspect the real nature of such a case as that of this medical student. Mr. Carter writes, in his paper referred to, that his advice was received by the patient and his father with polite incredulity, although fortunately it was acted on. I find that patients whose symptoms appear to them to depend entirely on something wrong in the head or in the liver receive with something more than incredulity the statement that they require glasses, and I fail now and then to make them consult an ophthalmic surgeon. Yet it is very ludicrous to give "nervous" and other remedies for a state of things which requires only a scientifically adapted mechanical aid.

There is another thing in connection with hypermetropia which deserves the attention of physicians. Mr. Couper has found that in some cases of hypermetropia the optic discs, in consequence of over-use of the eyes, become reddened and even swollen. The redness and swelling may, he tells me, be so marked that this comparatively unimportant condition, remediable by spectacles, is occasionally mistaken for and treated as slight neuritis depending on cerebral disease. The true explanation of these cases was given by Mr. Couper in a paper read before the Hunterian Society in 1870. In hypermetropia, the strain on accommodation is great; and thus there is not only congestion of the disc, but oedema also. Mr. Couper says that the button-like prominence thus given to the disc is easily recognised in the indirect image by the exaggerated parallax when the object-lens is moved. Its amount may be approximately expressed by the lens answering to the difference in the hypermetropia for the disc and the adjoining retina

respectively, as ascertained by the ophthalmoscope. Mr. Couper has found the oedema and swelling most marked in young subjects; the redness most marked in older persons who have never used adequate spectacles, and especially in those who have also taxed their eyes severely. In the former, perfect visual acuteness is usually obtained either immediately or in a few weeks by the use of proper glasses. In the latter, this perfect result is not always attained. The prolonged oedema and loss of vascular tonus seems to impair the acuteness of the retina. Occasionally the redness is so intense as to conceal the choroidal boundary of the disc in great part. Besides its very obvious direct interest, this fact has an important indirect bearing. The congestion and oedema from overwork of the hypermetropic eye seem to me to throw light on cases in which functional abuse leads to paralysis—on cases of writers' cramp, for example. In such cases, there is not a single fact as to their morbid anatomy; hence all our so-called pathological knowledge of them can only be hypothetical. In ophthalmic practice, is a case where we can see the effects of overwork in producing a morbid state, not, it is true, of a nerve-centre, but of a nerve-periphery. The facts stated seem to me important also as bearing on one way in which congestion of the brain results. As overwork of the eye causes congestion and oedema of the optic disc, so overwork of the brain causes congestion and even possibly oedema of the brain. I believe it produces arterial fatigue, and thus loss of arterial tonus. We cannot afford to overlook even indirect evidence bearing on so obscure a subject as cerebral congestion. Congestion of the brain, although frequently spoken of, is very difficult to prove even *post mortem*. Wilks and Moxon say that "*post mortem* fulness of the vessels of the brain and its membranes is of no pathological value whatever as showing brain-disease, as asphyxia produces it". They do not deny the existence of congestion of the brain as a fatal disease. It interests me very much to find that the conclusion that overwork of brain produces cerebral congestion by arterial fatigue has been reached by Niemeyer.

The nature of hypermetropia is a matter of great interest to physicians in one very important direction. The eyeball is incompletely developed; it is small; all its axes are short, especially the visual axis. More significant, there is a slighter optic nerve and a less retinal expansion; the nervous system of the eyeball is less developed. There is, if I may use such an expression, an exaggeration of the normal astigmatism. When the abnormal refraction is corrected by glasses, there remains usually a diminished acuteness of retinal vision—further evidence that the nervous system of the eyeball is less developed. It is noteworthy that objects appear to be smaller even when proper convex glasses are used. Hence the hypermetropic eyeball is an imperfect or ill-developed one. Moreover, hypermetropia, according to Donders and others, often goes along with an imperfect development, or, at any rate, a peculiar appearance, of the face; and, if one eye be hypermetropic, the face on that side alone is abnormal.

It seems to me that we have here a good illustration of what I believe to be the nature of the inheritance in one class of cases of insanity. No one inherits "asthenopia"; he inherits hypermetropia. I believe, similarly, that insanity is not transmitted in the sense of disease of the brain. In those who inherit a tendency to insanity, a congenitally imperfect brain has been transmitted; in those who inherit a tendency to asthenopia, a congenitally imperfect eye. There is not transmitted an insane neurosis, nor a tendency to any kind of disease of the higher divisions of the nervous system. There is transmitted a healthy brain, but one of which the highest nervous arrangements are few. Such a person is easily rendered insane by any kind of ill health, or by mental overwork or worry—by causes which would not affect those whose highest nervous arrangements are well developed. Holding this opinion, which is neither practically nor theoretically equivalent to the current doctrine, I do not believe that insanity is, as is commonly assumed, interchangeable in inheritance with such neuroses as epilepsy, chorea, neuralgia, etc.; any more than I believe that "asthenopia" is interchangeable in inheritance with other forms of "amaurosis". That epileptics often become insane is admitted, but only because epilepsy damages the brain, as drinking and many other things do. The imperfection of the inherited brain is, I speculate, that the highest cerebral centres are imperfectly developed the lower being, in comparison, over-developed. In other words "the controlling centres" are little developed. To become insane is to have defect in the controlling or inhibitory centres; and this permits over-activity of the lower centres. A person with such a brain easily becomes excited by alcohol, easily delirious during an acute illness and is more liable than other persons to have insanity after an acute illness. The reason is that, having fewer controlling processes, he can bear little interference with them. He begins his mental life with deficit, just as "the hypermetropic individual begins his accommodation with a deficit" (Donders). There are various degrees of hyper-

tropia; so, no doubt, to carry the illustration further, there are various degrees of congenital ill-development of the highest cerebral centres. Hypermetropia from ordinary use of the eyes becomes manifest at different ages, according to its degree (roughly speaking, one-eighth at eight years of age, one-twentieth at twenty years of age, and so on); so it is possible that insanity becomes manifest at different ages, even from ordinary wear and tear of a civilised life, according to the degree of ill development of the highest cerebral centres.

I should extend the principle to all organs. The inheritance of phthisis is, I submit, an inheritance of small lungs—of lungs easily overworked.

Leaving the consideration of anomalies of refraction, I pass to a very important disease, in which loss of accommodation is a symptom. I suppose it is to ophthalmologists that we are indebted for our knowledge of what so-called diphtherial "amaurosis" really is. It is one of the most singular forms of palsy. Diphtherial palsy altogether seems unlike any other. I believe that diphtherial amaurosis gives us a clue to its nature. Diphtherial amaurosis is that part of diphtherial paralysis which is most easily studied. The state of things can be studied with absolute precision, and therefore I suggest that, in a scientific investigation of the nature of that very complex thing diphtherial paralysis, we should begin here. Whilst urging on physicians the ophthalmological study of diphtherial paralysis, I would particularly urge on ophthalmic surgeons a careful study of the researches of Greenhow into this very singular disease.

In diphtherial amaurosis, the power of accommodation of the eyes is diminished or lost, and thus the patient (supposing him to be emmetropic) cannot read or see small objects, but can see in the distance. Obviously, the term amaurosis is improper; the deep parts of the eyes are normal. So far as I know, paralysis of the ciliary muscles on the two sides is only found in a high degree after diphtheria. After any debilitating illness in a hypermetropic person, we may have a condition which is symptomatically a minor degree of it: the patient's hypermetropia, heretofore latent, becomes manifest.

The peculiarity of diphtherial amaurosis is, that the paralysis is of parts supplied through a ganglion of the sympathetic chain. There is not paralysis nor paresis of the third nerve, but of parts of it which are supplied through the lenticular ganglion. In other regions, we see the same thing. The defective articulation is owing to paralysis of muscles of the palate supplied through Meckel's and the otic ganglia. The very slow pulse which, as Jenner and Greenhow have pointed out, is found in some cases, may be explained by affection of the cervical ganglia of the sympathetic. I grant fully that I cannot show that a similar state of things applies to the rest of diphtherial paralysis. Indeed, we could not reasonably suppose that the pathological process in diphtherial paralysis has any special malice against sympathetic ganglia as such; we can only suppose that cells of certain histochemical constitution easily give way, and, at the most, that those of these which first give way are most largely represented in the higher ganglia of the sympathetic.

There is one other direction of inquiry. In the case of hearing, there is evidently an accommodative apparatus analogous to that in the eye. Omitting the muscles of the pinna, which in man are rudimentary, there are, inside the tympanum, the tensor tympani and the stapedius muscles; we have to take into account also the tensor palati, because it has to do, if not with what I call the accommodation of the ear, at any rate with the functions of hearing. The otic ganglion, Troeltsch says, is of the same importance to the ear as the lenticular ganglion is to the eye. It is, then, I think, legitimate to inquire if we do not find in diphtherial palsy occasionally affection of hearing analogous to the defect of sight. One could only expect slight defect, because the muscular apparatus of the ear has not so much to do with hearing as the ciliary muscle has with sight. Besides, if the hypothesis be correct, that the otic ganglion in such a case is affected, there would be no palsy of the stapedius muscle; and, more than all, the tensor tympani and the tensor palati would not be entirely paralysed, as they receive a supply directly from the internal pterygoid branch of the fifth nerve, as well as from the otic ganglion. Moreover, the tensor tympani is the accommodator for high sounds; and thus, on the hypothesis, we should only expect loss of power to recognise high sounds. So far, then, from expecting to find "deafness" in diphtherial paralysis, I should expect only slight interference with the power of appreciating high-pitched sounds. I have met with but one case of slight affection of hearing in diphtherial paralysis. A medical man said it was not enough to impair his hearing for ordinary purposes, but, to use his expression, "enough to render music unintelligible". It was just the kind and degree of defect one would expect to find. Slight defects of hearing are not likely to be noted by unskilled persons. It is a remarkable

fact that double hearing has never been noted except in musicians (Roosa), although, of course, it may occur in anybody. It would seem to me that the investigation of the states of hearing in diphtherial paralysis could only be possible in an intelligent and also educated patient, and when the investigator is an aural surgeon. For there is the obvious difficulty, as Greenhow insists, that, if there be deafness, it may be owing to some affection of the Eustachian tube, a relic of the active diphtheritic process. In cases of Menière's disease, and in other ear-affections, the aural surgeon notes the patient's power of perceiving particular notes; and I see no reason why the state of hearing in cases of diphtherial paralysis should not be equally carefully noted.

Before leaving this subject, I would mention that the clearest account of the peculiar difficulty of articulation which occurs in paralysis of the palate is given by Donders in an appendix to his exposition of diphtheritic paralysis of the ciliary muscles; it is not an uncommon thing for paresis of the lips to be suspected when the palate is solely affected.

[To be continued.]

THE LUMLEIAN LECTURES ON THE MUSCULAR ARTERIOLES: THEIR STRUCTURE AND FUNCTION IN HEALTH AND IN CERTAIN MORBID STATES.

Delivered at the Royal College of Physicians of London.

By GEORGE JOHNSON, M.D., F.R.S.,

Fellow of the Royal College of Physicians; Professor of Clinical Medicine and Senior Physician to King's College Hospital; etc.

LECTURE III.—*Concluded.*

Renal Asthma: Symptoms and Proximate Cause.—The Dry and Inactive Skin of Bright's Disease: its Proximate Cause.—Is Diuresis a Result of High Arterial Tension?—Is High Arterial Tension a Cause of Albuminuria?—Uræmic Convulsions and other Nervous Symptoms: their Relation to Epilepsy: their Proximate Cause: Principles of Treatment.—Two Forms of Impaired Vision in Bright's Disease: the Proximate Cause of each.—Cholera Collapse and Pulmonary Embolism compared.—Cholera Collapse and Spasmodic Asthma compared.—Conclusion.

THERE is good reason to believe that some of the more formidable nervous symptoms which result from uræmia—in particular uræmic convulsions and a form of transient amaurosis—are directly due to cerebral anæmia consequent on sudden extreme contraction of the muscular arterioles.

There can, of course, be no question that uræmic convulsions are of an epileptic character. A large amount of evidence points to the conclusion that both the loss of consciousness and the convulsions of epilepsy are the results of sudden and extreme anæmia of the brain. In man, and in most, if not in all, warm-blooded animals, a rapid and very copious hæmorrhage usually causes convulsions. Kussmaul and Tenner state (*On the Nature and Origin of Epileptiform Convulsions caused by Profuse Bleeding*, New Sydenham Society, 1859) that in numerous cases of dogs, cats, and rabbits, they observed, without a single exception, violent and general convulsions preceding death by loss of blood. In order to produce this result, the hæmorrhage must be rapid. If it occur slowly, so that the vital powers are gradually exhausted, death then occurs with swooning, drowsiness, and delirium without convulsions.

The same experimenters found that an interruption of the supply of blood to the head of a rabbit, by ligature or compression of the arteries of the neck, produces epileptic convulsions as surely as hæmorrhage does. In about one hundred rabbits they ligatured or compressed the carotids and subclavians, from which, be it remembered, the vertebral arteries proceed; and in every case, except that of one very old lean and feeble animal, convulsions occurred.

In order to excite convulsions, they found it necessary to close all the four arteries which supply the brain. If but one carotid or one vertebral artery remained pervious, the animal was enfeebled and more or less paralysed, but not convulsed. And again, if, during the height of the convulsion, the ligature were removed from one carotid, the convulsions generally ceased immediately, and there was a sudden change from the most frightful spasm to complete relaxation of the muscles. The description of the convulsions thus artificially produced with, as it