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MYOPIA

THE RESULT OF CONSTITUTIONAL DISEASE.

17.

BY

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MYOPIA THE RESULT OF CONSTITUTIONAL DISEASE.

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Thirty years ago, Donders wrote: "The cure of myopia belongs to the *pia vota*. The more our knowledge of the basis of this anomaly has been established, the more certainly does any expectation in that direction appear to be destroyed, even with respect to the future." Now Donders was a great man, and we have very much for which to thank him. But when he wrote this, I believe he did a great injury to ophthalmic science and imposed a severe check on its development, from which it has not yet recovered. Since then, numbers of ophthalmic surgeons have written on the causation and treatment of myopia, and have copied and recopied what he taught, continuing to state that myopia is incurable and unpreventable, and that our race is doomed to become more and more myopic as its civilization progresses. Such being the present state of our knowledge on the subject, I make no apology for bringing forward this paper, for it points to a cause of myopia, a constitutional cause, a cause capable of being treated; and insists that myopia should therefore be regarded as a preventable, if not a curable, disease.

In a former paper* I have expressed the opinion that both hypermetropia and myopia are very often asso-

* Some Points of Relationship of the Eye to the Cardio-Vascular System. A paper read before the Harveian Society.—Vide *Lancet*, May 2nd and 9th, 1891.

ciated with certain constitutional conditions, mainly showing themselves in the cardio-vascular system, and constituting what, for want of a more accurate term, I called a "myopic and hypermetropic circulation," and I described what I considered the differences between them, namely, differences in pulse and cardiac signs. I also claimed as evidence of constitutional difference between the myope and hypermetrope the comparative freedom of the former from optic neuritis—a difference which subsequent observations have tended to confirm.

Since reading that paper, I have been engaged in more closely studying the subject, especially with regard to myopia and its associated constitutional conditions.

I now propose to give some of my conclusions and some cases illustrative of them, and, in so doing, I shall invert the usual order, and give my theories first, so that my cases may be considered from my own point of view.

I wish to call attention to the frequent association of myopia with vascular disease. To what extent these two conditions are dependent on each other has yet to be determined—that is, whether there is one disease affecting both the vascular and ocular structures and causing changes in shape and condition in both; or whether the vascular disease so affects the nutrition and tension of the eye as to cause its dilatation.

Now the study of the causes of myopia has generally been the study of its secondary causes, while the primary causes have been neglected. Myopia has been compared to rickets, to which it holds a close resemblance, and the reasonable study of it should be based on the same plan. The mechanical causes of the various deformities produced in the bones in rickets are of great interest, and the rules which regulate the form the bones take are well understood. But the most thorough knowledge of all the deformities and the mechanical causes of their production can help us but little in its treatment. Even if we go a step further, and say the

cause of rickets is an undue yielding of the bones, owing to their softened condition, we are still no nearer the true cause, and therefore no nearer its true treatment, and, until its *constitutional* cause is understood, we can merely correct or alleviate its deformities by splints, etc.

Now just as in rickets the deformities called first for attention, so it has been in myopia. The eye has been given its splints and crutches, in the shape of correcting glasses, and this has been done so well that the wearers are rather proud of them, and consider them as a sign of higher intellect, and a necessary product of advanced education. As in rickets the mechanical causes have been thoroughly studied, so in myopia the mechanical cause of the shape the eye takes is very well understood; but the reason why the sclerotic should dilate, expand, and yield, is still unknown. In fact, we at present know nothing of the true *causes* of myopia; we only know some of its effects; and it would be just as reasonable to say that the causes of rickets are standing, walking, sitting, or any of the positions which result in the deformities, as to say that myopia is caused by extra use of the eyes, by the action of the ocular muscles, the height of the orbit, or any other of the many "causes" to which it is assigned.

A myopic eye has been held to be a *healthy* eye with too long an axis, and within certain limits this may be so. But the fact that an eye obtains good vision when aided by glasses is no proof that it has not been damaged by disease. Myopia may in this connection be compared with heart disease, specially with dilated heart, which, like myopia, may be caused by disease and strain, acting alone or together. These factors, either separately or conjointly, may be the cause of heart or eye dilatation, and to a certain point the dilatation may take place without any *disturbance of function*. But this does not alter the fact that it is a diseased condition. In my present paper, I do not claim to have found the primary cause of myopia, but

I think I shall have got one step nearer it if I can show that the change in the eye is not an isolated one, affecting the eye only, but is part of a general constitutional change.

The acute or rapid production of myopia is, I think, not sufficiently recognised.

If inquiry is made of patients as to when the "short-sightedness" began, they will often give a most definite history of the time of its onset or increase. Of course it is easy to deny this history, and insist upon the proof of good vision previously; but this method of proceeding is hardly fair. If a patient can give a history of onset of "short-sight" so comparatively sudden that he fixes it as having occurred in the course of a few weeks, he may, I think, be reasonably believed. The constitutional causation of the *increase* of myopia is recognised to a limited extent. Nettleship says, "Myopia seldom increases after the age of 25, unless under special circumstances. General enfeeblement of health, as after severe illness or prolonged suckling, seriously increases the risk of its progress, even after middle life." [Nettleship, "Diseases of the Eye," p. 280.] But it is only when the proof is so positive that there is no possibility of denying it, that the acute increase of myopia is allowed, and histories of the acute onset of myopia are seldom sought for, and rarely believed. In fact, the trouble and ingenuity that is usually taken to explain it by, or attribute it to, any cause except the most obvious is truly wonderful. But surely it does seem most reasonable that, if constitutional causes are recognised as an important factor in the *increase* of myopia, they should also be an important factor in its original causation.

Cases of acute myopia are, of course, comparatively rare. Myopia is generally a chronic condition, of slow onset, and slow progress; so slow that the patient only recognises its progress by the occasional necessity of changing his glasses. My argument is, that if acute

constitutional disturbance can produce acute myopia, chronic constitutional disturbance may result in simple myopia; and it is the evidence of this constitutional condition that I shall endeavour to produce.

The condition is one that might reasonably be termed "vascular rickets," as it appears to depend on a yielding of the vascular and ocular walls. It may be defined as a disease chiefly of city life, the result of faulty nutrition, developing during the period of youth and growth, and producing deformities of the eye and cardio-vascular system. It frequently appears first at one of the nutritional changes of life, such as second dentition, puberty, commencement of menstruation, or the period just before full growth.

I offer as evidence of the vascular disturbance associated with myopia the following conditions:—

1. Spontaneous hæmorrhages (epistaxis; menorrhagia; retinal hæmorrhages).

2. Capillary congestion.

3. Cardio-vascular disease (high arterial tension; cardiac hypertrophy; valvular disease).

The tendency to hæmorrhage is recognised in myopia as a *local* condition; but what I wish to insist on is, that there is a *general* tendency to hæmorrhage, not limited to the eye, but occurring as spontaneous hæmorrhages in other parts of the body. These hæmorrhages in some instances precede the occurrence of myopia, and they are valuable symptoms of a faulty state of the vascular system. They are in themselves probably beneficial, affording relief to the vascular system, and the myopia would appear, in some cases, to come on when this relief ceases. In this connection, the treatment of myopia by bleeding (heurtelouping) is interesting, as being apparently an unconscious imitation of nature's own method.

Of these spontaneous hæmorrhages, epistaxis I consider a common accompaniment of myopia, which it often appears to precede. In some cases, it has been

a most marked symptom, seriously interfering with the patient's business.

Menorrhagia, often a recognised symptom of cardio-vascular disease, I consider one of the associated symptoms in myopia, sometimes taking the place of epistaxis in the male, and produced by much the same causes. The catamenia are either too frequent, too copious, or both. Delay in the establishment of the menstrual periods appears sometimes to cause rapid increase of myopia.

The tendency to retinal hæmorrhage in myopes is well known.

I consider as evidence of capillary congestion the full red lips and rather high-coloured cheeks frequently observable chiefly in young myopes. They are also very rarely anæmic.

Proceeding to the cardio-vascular condition of myopes, the pulse is readily felt. The pulse-rate, except in rapid progressive cases, is rather slow—that is, it is often slower than might be expected from the general appearance and behaviour of the patient, but it is within normal limits. It is a large, full pulse. The arterial walls feel thin. The tension is full, and often distinctly high. The pulse is recurrent, not only at the wrist, but also in other arteries, *e.g.*, the temporal.

The heart shows signs of enlargement of very variable degrees, and this is probably due to both dilatation and hypertrophy.

There is generally some alteration in the rhythm and accentuation of the sounds. An accentuation of the second aortic sound is perhaps the most frequent.

Well-marked valvular disease is also not infrequent.

Now the vascular disturbance I have described above is, I believe, nothing else than a form of high arterial (or pulse) tension, or the effects of it.

I am aware that I use the term high arterial tension in a wider sense than that generally accepted. Pulse tension like ocular tension, is, to a certain extent, a matter for each,

individual to decide what is normal or physiological, and what is pathological. No distinct line can be fixed between the two. The extremes are well marked, and no one can fail to recognise them. The recognition of slight increase in pulse tension is largely an individual matter; and in forming the diagnosis it is of importance not to be guided by the pulse alone, but to take into consideration the condition of the heart, and other symptoms.

I do not wish to lead any one to suppose that, in the majority of cases of myopia the changes in the pulse, etc., are anything very obvious, or that they can be made out in every case. But I believe that any one who will take the trouble to observe a few cases thoroughly will very soon be convinced of their existence.

There are many admirable descriptions of the condition of high pulse tension as it occurs in adults, but I know of none as it occurs in the young; yet I believe it to be a condition by no means rare. But as it gives rise to a different class of symptoms in the young and the adult, it has not been recognised as the same condition. The difference in the characters of the pulse is, I think, explained by the different ages at which the increased tension occurs. Thus in renal cases, or cases of high pulse tension occurring in late life, we have an increased pressure in vessels which have already lost much of their elasticity, and have become firm, fixed, and undilatable. But high pulse tension occurring in a young subject, when the vascular walls are soft and elastic, must produce results far different. The arteries, however great the pressure, still have a reserve of elasticity, and hence can never become hard, as in the renal pulse. Again, in the young, a constant or frequent strain on the arterial walls will gradually cause them to yield and become stretched, and hence the large thin-walled arteries of the myope, due to the stretched fibrous coat.

I consider the recurrent pulse is another result of long-continued high arterial tension, and it is but

reasonable to expect that continued high arterial tension should not only cause the larger arteries to dilate, but also the smaller ones, thus giving rise to a more free anastamosis between the vessels. This free anastamosis is probably to a large degree compensatory. The possessor of a habitual high pulse tension becomes accustomed to it and tolerates it, whereas a subject of low tension acquiring high tension later in life, when the arterial wall has become thickened and fixed, suffers from its constitutional effects. Myopes seldom develop high arterial tension in its typical form as met with in renal disease.

That the capillaries are not generally dilated is evidenced by the existence of high arterial tension. But I think that it may be the dilatation of the capillaries in acute progressive myopia which causes the marked difference in pulse in those cases from the pulse in ordinary myopia, the pulse in the former being rapid, soft, and compressible.

Now it is, I think, in no way an unreasonable theory that a chronic vascular disorder should give rise to myopia, *i.e.*, to an enlargement of the eye-ball.

In fact, an increase of blood-pressure as a cause of myopia has been recognised, taught, and accepted by most writers on the subject, myopia having generally been attributed to a softening due to congestion caused by prolonged stooping, pressure on the chest, etc. All this I freely admit. But while the local and temporary increase of blood-pressure has been recognised and treated, the general constitutional increase has been left unrecognised and untreated ; and I believe that it is this constitutional high pulse tension which alone causes the local increase of pressure to be injurious. It is the *constant* high pulse tension which does the harm, and interferes with the nutrition. The vascular system is very well adapted to withstand occasional increase of blood-pressure ; but it is with the blood-vessels and eye,

as with other structures, that *constant* pressure causes atrophy and thinning.

If the vascular supply to the upper limbs be interfered with, whether by heart disease, aneurism, or phthisis, the finger-tips are apt to become clubbed. The eye is also an extremity, in that it has a terminal circulation, and if the general circulation is affected, it is perfectly reasonable to expect that the nutrition of the eye will be affected, and that over-growth may take place.

A great deal has been written on the prevention of myopia in school children by school hygiene, of which the principal factors are

(1) The prevention of stooping ;

(2) The avoidance of eye-strain by good light and good print.

Very much has been done in this way, but its result on the prevention of the increase of myopia has been very disappointing, for the evidence goes to show that the myopia continues to increase in spite of it, and that, as the higher classes are reached, so a larger percentage of myopes is found.

Now my contention is, that the myopia continues to progress because its causes are not recognised, and therefore not removed.

I hold, then, that myopia is generally evidence of a constitutional disease, either past or existing. Of a past disease it may be the only remaining evidence, the other effects having been recovered from, just as some alteration in the shape of the bones may be the only remaining evidence of rickets.

The cause of the myopia may have been active for a few years only, during which time its effects have been evident on other structures besides the eye ; but the cause passing away, they have recovered, whereas the fibrous sclerotic, once damaged and stretched, does not recover. The eye, when once myopic, apparently remains so. This, I think, is the reason why, in some

myopes, the pulse and heart signs are indistinguishable from the normal ; the cardio-vascular system may have completely recovered, its walls being muscular, and therefore able to recover, if the stretching does not persist, and has not been of too long duration. In most myopes, however, I think I can detect evidences of past damage on the cardio-vascular system. The constitutional symtoms I have described as being associated with myopia, I do not consider as being by any means limited to it. And from my observations of the constitutional state in eye-patients I have been led to include with the myopes, cases of high astigmatism, whether myopic or hypermetropic ; some cases of hypermetropia with bad vision, and cases of lamellar cataract.

My attention having thus been directed to the subject of astigmatism, I have been endeavouring to find out the views of authors on the subject of its production and cause. Whether it is liable to increase, and if so in what direction ? Is it congenital, or acquired ? And are young children astigmatic any more than they are myopic ?

But in English text-books I can find no mention of the subject. Some of the American observers seem, however, to have taken up the subject, and to regard astigmatism as allied to myopia, in that they recognise its tendency to change. Dr. S. D. Risley speaks of "eyes passing while under observation from hypermetropic to myopic refraction by the turnstile of astigmatism." He also notes retino-choroidal changes in these cases, but considers that the astigmatism is the cause of the eye-ball distension. (Oph. Review, vol. vi. p. 276).

Astigmatism is, of course, elaborately classified for the optician's benefit ; but from a medical or surgical point of view, it is absolutely unclassified.

With regard to astigmatics, I regret that I have not so closely observed their constitutional condition as I

have in the uncomplicated cases of hypermetropia and myopia ; that is to say, I have not made notes of them, so that I can only speak from my general impressions. But, speaking thus, I would say that all astigmatics of high degree, whether hypermetropic or myopic, should be classified constitutionally with the myopes, as cases of *yielding eyes*—eyes which have yielded from a constitutional cause ; and the mere fact of their requiring a convex or concave lens should not determine the class to which they are to be assigned.

Cases of hypermetropia with bad vision and of lamellar cataract are probably both due to constitutional causes, which may account for their similarity to cases of myopia. On the other hand, some myopes are constitutionally indistinguishable from emmetropes. Some of these are —as I have explained—due simply to the constitution having entirely recovered from its injury, while the eye is left defective. But there are others to which I do not think this explanation will apply.

I have endeavoured to show that what I consider to be a common cause of myopia is high pulse tension, or, rather, that a common cause can give rise to vascular and eye changes. I would, therefore, ask you to consider how far my theory is borne out by the conditions under which we find myopia occurring. I think it is generally accepted that myopia is commoner amongst the sedentary, the well-to-do, and Germans. It is also generally accepted that high arterial tension, when not due to actual disease, is often produced by a full meat diet, rich living, and deficient exercise. Add to this, taking of beer or very little fluid, and you have the most favourable conditions for producing high arterial tension, which is caused by the contraction of the small arterioles, due to the presence in the blood of uric acid and other waste products in excess. This excess is due partly to excess of formation, partly to a gradual accumulation arising from a deficiency in

excretion of renal, biliary, intestinal, and cutaneous products.

Now all these conditions we have in the modern city school-child, well-fed (or over-fed, for the amount of exercise taken), with hurried meals and insufficient time for digestion, followed by a hurried walk, and with deficient excretion arising from deficient exercise. In the German, we find very similar conditions, and a variation of the same applies to the young ladies of the upper classes, who also put an extra strain on their nutrition and hearts by tight-lacing.

This, then, I offer as an explanation of one of the causes of myopia. Granted a faulty nutrition, affecting ocular and vascular structures, and an increased arterial and therefore ocular tension, the mechanical causes usually given amply account for the direction in which the eye yields.

Certain diseases are specially prone to cause the onset or progress of myopia; and while they are not diseases usually classified together, they have certain points in common: they all profoundly affect the general nutrition, and most, if not all, the vascular system.

In rheumatism, which I do not mean to limit to rheumatic fever, we certainly have a vascular disease and a nutritional disease, notably affecting fibrous tissues, and hence probably the yielding of the fibrous sclerotic.

That primary syphilis should be followed by myopia is, perhaps, only what might be expected, when it affects the eye itself, producing retino-choroidal and vitreous changes, and it is only in these cases that I have obtained evidence of it. Its tendency to cause vascular and nutritional disease is well known.

Mr. Phillips has noticed a connection also between myopia and inherited syphilis.

In one or two young alcoholic subjects (pot-boys) I have also found high myopia.

Some of my cases point to typhoid fever as the cause of the myopia. It certainly affects nutrition, though, as

regards its action on the vascular system, I cannot speak definitely.

In two cases of acute myopia, I have found phthisis associated. This may be only accidental, and I certainly do not consider the myope as a typically phthisical subject.

In pregnancy, an accepted cause of increase of myopia, we undoubtedly have a nutritional disturbance of the most general kind, and almost every structure is liable to change, and in addition we have a vascular change, namely, high arterial tension. So far, however, I have not met with any case of myopia with pregnancy as its primary cause.

I think I shall be stating the result of general experience when I say that choroiditis is commonly associated with myopia—that is, taking all forms of choroiditis, in the majority of cases the refraction is myopic; often only slightly so; but still, generally myopic.

I have already stated that I consider primary syphilis, when accompanied with eye-symptoms, as being sometimes a cause of myopia; and when the disease takes the form of choroiditis, I consider it equally a cause of the myopia which follows.

Choroiditis is, in most cases, accepted as evidence of constitutional disease, whether that disease be syphilis, congenital or acquired; rheumatism; gout; defects of menstruation; or tuberculosis. "Choroidal changes, like those of the retina, are for the most part the result of special diseases." (Gower's Med. Opthal., p. 119.)

"Inflammatory and degenerative changes often occur, some of them entirely local, as in myopia; others symptomatic of constitutional or of generalised disease, such as syphilis and tuberculosis." (Nettleship, "Diseases of the Eye," ch. xii. p. 178.)

And yet this generally admitted symptom of constitutional disease, when it occurs in conjunction with myopia, is said to be "entirely local," and it is only when it occurs in a more than usually violent form, as

in some cases of acute progressive myopia, that it is admitted to have a constitutional cause, and is allowed a name—posterior sclero-choroiditis.

The divisions between the various kinds of myopia are purely artificial, and only matters of degree. There is, of course, a vast difference between the extremes of myopia; but there is no hard and fast line to be drawn between the various kinds. Therefore it is reasonable to look for a common cause for all kinds of myopia, and if disease and constitutional disturbance are found as a cause in high myopia, it is not unreasonable that a departure from health, whether sufficient to be termed a "disease" or not, be sought for in the lesser degrees of myopia.

Choroiditis may, in some cases, progress independently of the increase in myopia. Donders says: "The atrophic crescent is not absent in higher degrees of myopia, and when in moderate degrees the atrophy may be in youth still wanting, it is developed, *even without increase of the myopia*, at a more advanced time of life." (Donders' "Accommodation and Refraction of the Eye," p. 448.)

The occasional occurrence of crescents in hypermetropes, and in cases of lamellar cataract without myopia would seem to indicate that the myopic crescent is a form of choroiditis, and not the result of the myopia.

Why, when in addition to choroiditis and vitreous opacities there is dilatation of the eye, it should be held that the dilatation is the cause of the other two, it would be hard to say, especially as the dilatation is equally likely to be the result of the faulty nutrition, which has produced both the choroiditis and vitreous opacities.

Admittedly, choroiditis may progress a long way before it gives rise to any ophthalmoscopic signs. "Congestion of the choroid is not commonly recognisable by the ophthalmoscope. That active congestion does occur is certain; and it would seem that myopic eyes are especially liable to it." (Nettleship, "Diseases of the Eye," ch. xii.)

Some amount of choroidal disturbance is discoverable

in almost all myopic eyes; and I contend that it is a more warrantable assumption that myopia is caused by choroiditis, than that choroiditis is caused by myopia; I believe that the cause of both will be found in the cardio-vascular changes which I have endeavoured to describe.

In conclusion, then, I claim that there is sufficient evidence for considering myopia to be the result of constitutional disease; first, on what I consider the reasonable deductions from the generally accepted teaching of the cause and increase of myopia; secondly, on my own observations on the constitutional condition of myopic patients. The generally accepted teaching and my own observations may be briefly compared as follows:—

1. Myopia a local inherited tendency.	1. Myopia an inherited tendency, associated with inherited constitutional disease.
2. Increase of myopia caused by local vascular congestion and local increase of blood-pressure.	2. Commencement and increase of myopia caused by general and local vascular congestion and general and local increase of blood-pressure.
3. Increase of myopia caused by some constitutional diseases.	3. Commencement and increase of myopia caused by some constitutional diseases.
4. Myopia accompanied by a tendency to local hæmorrhages (retinal).	4. Myopia accompanied by a general tendency to hæmorrhage (epistaxis, menorrhagia, &c).
5. Myopia a product of artificial and civilised life.	5. Cardio-vascular disease also a product of artificial and civilised life.
6. The myopic eye shows evidence of degeneration in its tendency to change, its increase in size, and its liability to choroiditis, vitreous opacities, and detachment of retina.	6. The cardio-vascular system shows evidence of degeneration in high pulse tension, vascular dilatation, tortuous arteries, and heart disease.

In giving the following cases, I do not wish it to be supposed that I have based my theories on them alone.

For some time past, I have made a practice of making a more or less thorough physical examination of all refraction cases coming under my observation. Of some of these I have the notes, and the cases that I give are taken from among them.

My sincere thanks are due to Mr. Couper and Mr. Lawford for kindly affording me the opportunity of carrying on my observations. Also to Mr. Gunn for his permission to refer to one of his cases.

Case I.

Onset of myopia at 36 years of age, following rheumatic fever, preceded by habitual epistaxis and menorrhagia; cardio-vascular disease; progressive myopia.

July 4, 1891.—M. J. W. (female), age 43.

Previous History.—Healthy, except for severe epistaxis, with which she had been troubled until fifteen years ago. It was so severe that she always had to be prepared for it at night, when it would wake her up almost choked. Rheumatic fever nine years ago; ill for two or three months. About seven years ago, sudden severe pains in left eye; sight not affected at the time; sight gradually got misty. Four years ago, attended at Westminster Ophthalmic Hospital, when she was ordered 2D. sph. Catamenia very painful; copious; every three weeks. Have ceased now; ceased suddenly.

Family History.—F. slight rheumatism. One sister short-sighted.

Physical Examination.—Arteries full and tortuous; no recurrence.

Heart, apex displaced outwards in the nipple line. Impulse forcible; heaving.

V. R.— 5.50 = $\frac{6}{12}$
L.— 11.0 = $\frac{6}{12}$

Case II.

Myopia; rheumatism; rheumatic fever.

Sept., 1891.—B. S. B., age 33. Builders' foreman. Comes complaining of pains and stiffness of limbs.

Previous History.—Chorea as a boy; got quite well.

18 months ago rheumatism 5-6 weeks.
13 „ „ pleurisy and rheumatic fever.
3-4 „ „ influenza.
3 weeks „ quinzy throat.

Physical Examination.—Pulse 80. Arteries large, pulse tension raised, but not very high. The examination of the chest did not reveal anything except an accentuated second sound.

The pulse, however, was what I should have described as a "myopic pulse," and, on examination, I found that he had high myopic astigmatism.

V. R. $<\frac{6}{60}$ Ophthalmoscopic Estimation—10 and —14.
L. $<\frac{6}{60}$ —4 and — 9.

He had never worn glasses.

In this case the rheumatic tendency appears strongly; for not only is there history of rheumatic fever, but the rheumatic tendency is to be noticed in the chorea, tonsilitis, and rheumatic attacks.

Case III.

Myopia; rheumatism; epistaxis; cardio-vascular changes.

I. V. (male), age 17, clerk, has noticed short sight about two years. Has had "rheumatics," keeping him in bed several times. Last attack three years ago. First attack nine years ago, when joints were swollen. Is subject to epistaxis, though not so much as formerly.

Pulse 80, soft and recurrent.

Heart, apex 4th and 5th interspace about in nipple line.

Impulse forcible, slightly heaving.

Accentuated 2nd sound to right of sternum.

Loud 1st sound. No murmur (?).

V. R. $<\frac{6}{60} - 3{\cdot}50 = \frac{6}{6}$.
L. $<\frac{6}{60} - 3{\cdot}50 = \frac{6}{6}$.

Ophthalmoscopic Examination.—Rather unhealthy fundus, disturbance of pigment, halo round macula.

Note.—Mr. William Bull published three cases of myopia associated with rheumatic fever in the *Hospital Gazette* (Nov. 22nd, 1890), under the title, "What is the connection between mitral stenosis and myopia?"

Case IV.

Acute myopia; cardio-vascular symptoms; menorrhagia.

Sept., 1891.—E. E., age 21, dressmaker.

History.—In March, 1891, noticed that she was rapidly getting short-sighted. One month ago, "could not use her eyes"; one week ago, gave up using them entirely.

Family History.—Two brothers, short sight; no rheumatism.

General Symptoms.—"Weak on the chest," shortness of breath on exercise; menstruation regular, very excessive, weak and faint at the periods, not much pain.

Physical Examination.—Pulse, 100 to 90. High pulse tension. Full tense arteries.

R. —4 cyl.
L. —4 cyl. —3 sph.

Case V.

Acute onset of myopia; epistaxis.

July 11, 1891.—A. W., age 20, sailor.

Previous History.—Had very good sight as a schoolboy, 13 years old. His eyes were examined at school by a professional examiner, and given a first-class certificate. One and a-half years ago, while at sea, became short-sighted in the course of a few months. Spontaneous nose-bleeding three or four months ago. Has grown three inches in two years.

No family history of myopia.

Note.—This case is of interest partly on account of the definite history of the acute onset of myopia. The fact of its occurring at sea is interesting, as the conditions of life on board ship are exactly those liable to produce myopia, by causing high pulse tension.

I have come across other myopic sailors, though in the case of sailors the element of stooping and looking at near things is conspicuous by its absence.

Case VI.

Myopia; cardio vascular symptoms.

June 9, 1891.—A. W. (female), age 17.

Is attending at Queen Square Hospital under Dr. Tooth, who sent her to Moorfields.

A rather tall, thin girl—*i.e.*, body and limbs thin; cheeks fat; lips red; weak, delicate-looking.

Physical Examination.—Pulse 128, small, and extinguished only with considerable pressure; a recurrent pulse, both at wrist and temporal artery, which is rather tortuous. Heart, apex diffused; impulse felt in fourth, fifth and sixth interspaces, in and outside nipple line; excited action; no distinct murmur; first sound not quite clear at apex.

Family History.—Father died of consumption.

$$\text{V.}\quad \begin{cases} \text{R. } \frac{3}{60} \dfrac{-1{\cdot}50 \text{ cyl. horiz.}}{-8 \text{ sph.}} = \frac{6}{12} \\ \text{L. } \frac{2}{60} \dfrac{-1{\cdot}50 \text{ cyl. horiz.}}{-6{\cdot}50 \text{ sph.}} = \frac{6}{18} \end{cases}$$

Nov. 10, 1891.—Pulse 140; temporal arteries markedly tortuous; fundus examined; the vessels on leaving the optic discs turned towards the nasal side of fundus.

Case VII.

Myopia and epistaxis.

Oct. 3, 1891.—E. T., age 16, turner.

Near-sighted all his life. Brothers and sisters also short-sighted. Always subject to nose bleeding, so much so that his nose bleeds every time he washes his face, and interferes with his work. Pulse 60, no high pulse tension; heart sounds normal, no impulse felt; hands rather blue and cold.

$$\text{V.}\quad \begin{cases} \text{R. } \frac{6}{60} \dfrac{-3 \text{ sph.}}{-1 \text{ cyl.}} = \frac{6}{6} \\ \text{L. } \frac{6}{60} \dfrac{-3{\cdot}5 \text{ sph.}}{-1 \text{ cyl.}} = \frac{6}{6} \end{cases}$$

Case VIII.

Myopia and epistaxis.

E. S. (female), age 14.

She was a very large child at birth; has always been healthy, except that she has always been liable to epistaxis since a baby; first menstruated six months ago; "short-sight" first noticed when 11 years old; refraction highly myopic.

Case IX.

High myopia; epistaxis; hæmorrhage in macula region.

1891.—E. R., age 27, engineer.

History.—General health fairly good; suffered from nose-bleeding very much between 16 and 20 years of age, not since; no rheumatism.

Family History.—Father and mother "not short-sighted." One brother and one sister, about the same as himself.

Physical Examination.—Thin, poorly-covered chest; heart

sounds regular, no murmur; pulse 84, fairly full, normal; high myopia right and left, about — 18 D; sight failing in right eye; hæmorrhage in macular region.

Case X.

Myopia and cardiac symptoms.

I. S. (male), age 13.

Heart examined. Apex beat outside nipple line, heaving impulse; first sound accentuated, and much louder than second sound, even in aortic area.

V. R. $\frac{-2 \text{ D. sph.}}{-3 \text{ D. cyl.}} = \frac{6}{18}$

V. L. $\frac{-5 \text{ sph.}}{-2{\cdot}5 \text{ cyl.}} = \frac{6}{18}$

Case XI.

Acute myopia preceded by epistaxis; phthisis; unusual form of myopia.

Oct. 17, 1891.—S. T. (female), age 24, box-maker.

History.—Six months ago, her sight failed suddenly—*i.e.*, in the course of a week or two—so that she was unable to see "to cross the road without screwing up her eyes"; previously she had seen well. Up to 12 months ago, suffered frequently from nose-bleeding, occurring two or three times a week; this had lasted for some years (two or three) previously.

Physical Examination.—Heart, nothing distinctive; pulse full, soft, recurrent; phthisis well marked (left apex).

V. R. $\frac{6}{60}$ Under Atropine Oct. 21. $< \frac{6}{60}$ —3 sph. $= \frac{6}{12}$

V. L. $\frac{6}{24}$ Under Atropine Oct. 21. $< \frac{6}{60}$ —4 sph. $= \frac{6}{18}$

Oct. 21. *Ophthalmoscopic Examination.*—There is a marked difference in refraction between the macula and disc.

	R.	L.
Mac.	—3 D.	—3 D.
Disc.	—8 D.	—7 D.

The O.D.'s appear tilted towards the nasal side, and the vessels turn in that direction.

Case XII.

High myopia; "weakness of chest."

Oct. 14, 1891.—L. Y. (female), age 23.

Much troubled with chronic cough and shortness of breath.

Chest Examination.—Heart, apex fourth interspace just inside nipple line; slight thrill at apex; no lung mischief.

High myopia.

R. —16 sph. = $\frac{6}{60}$
L. —16 sph. = $\frac{6}{36}$

Case XIII.

Myopia; cardio-vascular symptoms; epistaxis.

J. C. (male), age 25.

Always "short-sighted," occasional nose-bleeding.

Chest Examination.—Heart, apex beat fourth and fifth interspaces in nipple line; heaving impulse; heart-sounds of a booming character; pulse 124.

One sister said to be "short-sighted."

R. $\frac{-2 \text{ cyl.}}{-5 \text{ sph.}} = \frac{6}{9}$
L. $\frac{-3 \text{ cyl.}}{-4 \text{ sph.}} = \frac{6}{9}$

Ophthalmoscopic Examination.—Retinal vessels turned towards nasal side of fundus.

Case XIV.

Myopia and heart disease.

M. P., nurse, age 36; looks very much older.

Complains of "short sight"; has always had it, and the glasses last ordered do not suit; general health has never been good. In 1884 she had "rheumatic or typhoid fever"—the diagnosis was uncertain; subsequently she had "nephritis."

Physical Examination.—Temporal arteries prominent and tortuous; heart's impulse heaving and diffuse, outside nipple line; loud systolic murmur heard all over cardiac area, especially at apex.

V. R. $\frac{6}{60}$ $\frac{-7 \text{ sph.}}{-4 \text{ cyl.}} = \frac{6}{12}$
L. $\frac{6}{60}$ $\frac{-3 \text{ sph.}}{-.75 \text{ cyl.}} = \frac{6}{24}$

Note.—In some cases, the occurrence of myopia may precede the definite signs of constitutional disease; but I do not consider that this is in any way opposed to the theory that myopia is of constitutional origin, for my contention is not so much that the cardio-

vascular disease produces the myopia as that both cardio-vascular and myopic symptoms are the result of a common cause. Hence I consider it of equal importance to carefully note the diseases which follow myopia as those which precede it.

Case XV.

High myopia and heart disease in a child.

E. F., age 8.

Family History.—Father "short-sighted" and "rheumatic."

Physical Examination.—Pulse 80, soft, recurrent; heart, apex displaced outside nipple line; impulse heaving; loud musical murmur to right of sternum.

V. R. —17 D. = $\frac{6}{60}$
L. —17 D. = $\frac{6}{60}$

Moderate crescents, choroids thin and pigment rather patchy.

Case XVI.

Myopia and Syphilis.

H. K. (male), age 23.

In 1885, was under Mr. Hulke's care, and is noted as a case of central choroiditis in both eyes; he was treated with iodide of potassium; his vision is noted as R. $\frac{6}{12}$ and L. $\frac{6}{9}$, and his refraction said to be hypermetropic.

Dec. 2nd, 1891.—

V. R. $\frac{6}{18}$ —·75 cyl. = $\frac{6}{12}$
L. $\frac{6}{60}$ —3 sph. = $\frac{6}{18}$

Note.—This case shows the production of myopia as the result of choroiditis, probably syphilitic. The vision obtained in 1885 without the aid of glasses was the same as that obtained in 1891 with the aid of a — cylinder in one eye and a — spherical in the other.

Case XVII.

Myopia and epilepsy.

A married woman, age 31 (but looking 10 years older).

Does not think that she has ever seen well; the sight has got worse lately. Four years ago, "typhoid fever," after which she had her first fit; she has frequent fits now, and during the fits she passes both urine and fæces, and bites her tongue. Had rheumatic fever 10 months ago, and has had rheumatism ever since.

Physical Examination.—Revealed but little, except an unusual relationship between the pulse and respiration, the former being 88 per minute, the latter 30 per minute.

$$V. \quad \left.\begin{array}{ll} R. & -14 \text{ sph.} \\ L. & -14 \text{ sph.} \\ & -2 \text{ cyl.} \end{array}\right\} = \frac{6}{24}$$

Note.—I give this case, not because there is any marked cardio-vascular change (except in so far as the epileptic fits may be considered as evidence), but because it corresponds with some points in other cases, in which attacks of a more or less epileptic character occur in myopes.

In this case, I consider that the constitutional disturbance which caused the myopia has now progressed still further and has probably some connection with the production of the fits.

With this case it is interesting to compare two cases published by Mr. Nettleship (B.M.J., 1879).

CASE 1.—"Repeated paroxysmal failure of sight in connection with heart disease."

Refraction, myopia, $\frac{1}{40}$.

CASE 2.—Blindness of left eye with changes, the result of retinal hæmorrhage; thrombosis; afterwards, repeated attacks of transient blindness of right eye, and contraction of visual field with small opacities in vitreous body; spontaneous arterial pulsation; severe aortic disease.

Low degree of myopic astigmatism.

Also a case in Mr. Gunn's clinic of a woman about 35, who was myopic in a high degree, and had frequent attacks of transitory blindness. She had, in addition, well marked heart disease.

CASE XVIII.

Myopia and chronic bronchitis in a child.

J. L. (male), age 7.

Three years ago, attended under Mr. Hulke; he then had myopia —12 D.; his myopia has now increased to —15 D.; he is rather fat, and looks much older than his age; he shows no definite cardio-vascular disturbance, and is now in fair health; previously however, he suffered for some years from chronic bronchitis.