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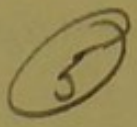

REPEATED PAROXYSMAL FAILURE OF SIGHT, IN CONNECTION WITH HEART-DISEASE.*

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In the cases, by no means uncommon, in which patients come under care for temporary failure of sight, in a large proportion the symptoms are due either to megrim, or to a temporary and varying failure of accommodation, or to some less common local conditions. But a few cases remain in which we can invoke neither central nervous disturbance, as in megrim, nor any transient nervous or muscular failure of accommodation, and of which temporary arrest of blood-supply to the retina seems the only feasible explanation.

These patients who, so far as I have yet seen, are always the subjects of valvular heart-disease, are subject repeatedly to loss of sight for a short time. The attacks vary considerably in severity, but they all agree in general features; the failure comes on suddenly, lasts a short time (from a few minutes to an hour or two), affects only one eye, and passes off almost as quickly as it came on. Not only is the affection asymmetrical, but the same eye suffers repeatedly, whilst its fellow may escape altogether. In certain of the cases, an attack at length occurs which, instead of passing off as usual, leads to permanent blindness. Such cases, although rare, are well known and have attracted the attention of many observers (especially Knapp, Moos, Loring, Mauthner), and various explanations have been offered.

The fact that the cases occur in connection with heart-disease seems to point to some circulatory disturbance as the cause of the symptoms. The difficulty is to explain a disturbance or arrest of blood-supply which, whilst it happens repeatedly, is, as the symptoms show, not only very localised, but always affects precisely the same part. We cannot suppose that the same artery, whether in the eye or the optic centre, is repeatedly plugged by embolism; nor can the symptoms be accounted for by a general failure of the cerebral circulation, nor by fainting; for the patients, though often suffering much from headache and giddiness, never actually faint during the attacks, nor does either posture or exertion seem related in any way to their onset. We seem to be driven by exclusion to the hypothesis of some permanent local disease of the ophthalmic artery, or its branch to the retina. Thus, whilst we can readily suppose that disease of the arterial coats, narrowing the blood-channel to the retina, might cause no symptoms, so long as the general circulation was healthy; we may note that the local conditions mentioned below make it likely that if, with the ophthalmic artery narrowed by disease, the intracranial circulation were also liable to disturbance from heart-disease, the stream of blood to the retina might now and then be suddenly stagnated for a short time.

The natural elasticity of the coats of the eye offers an especial obstacle to the entrance of blood; the arterial twig which supplies the retina is

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* Read at a meeting of the South London District of the Metropolitan Counties Branch.

one of the most insignificant branches of the ophthalmic artery, and its current, therefore, relatively weak, and lastly, the current in the ophthalmic artery itself must be a weak one; for this artery is given off at a right, if not an obtuse, angle from the tortuous part of the internal carotid. A narrowing of the lumen, which would have but little effect in a vessel carrying a strong stream, might hinder or stop the flow of a feeble stream, either by increasing the friction or serving as the nucleus for a thrombus—a result which, though capable of recovery and repetition, might at any time become permanent. Such lasting blindness at length occurred, *e. g.*, in a case reported by Loring, after very numerous temporary attacks, which were spread irregularly over a period of five-and-twenty years.

The hypothesis of local arterial disease certainly, however, makes it difficult to account for the tendency to symmetry which is noticed in many of the cases, the second eye often becoming subject to the same symptoms after the first has suffered for some time, or has become blind. The difficulty, however, is not greater than in chorea where, as Dr. Dickinson has shown, there is a strong tendency to symmetry in the distribution of the arterial lesions. In one of my own patients, chorea had occurred many years before.

I know of no *post mortem* examination of the ophthalmic artery in such a case, and of only one record of ophthalmoscopic examination during one of the attacks. In this case, Mauthner* found that the retinal arteries were empty during the blindness; but that when, a few minutes later, the sight had returned, the arteries had resumed their proper size—changes, it may be noted, in perfect accordance with such an explanation as I have offered. In many instances, these cases remain "functional" ones throughout our observation of them; and I should not have ventured to ask your attention to the subject now, had I not had the opportunity of watching two patients in whom the onset of permanent blindness of one eye, from vascular blocking, drew special attention to the nature of the temporary attacks in the other. In the first case, one eye having itself been subject to the transient seizures, ultimately went blind with the ophthalmoscopic signs of arterial plugging; after which event, the second eye became the victim of similar transient attacks.

Case I.—*Transient Attacks of Failure of Right Eye, followed by Permanent Blindness, with Ophthalmoscopic signs of Arterial Blocking: subsequently, many temporary attacks of greater or less Blindness in Left, with contraction of Visual field: Mitral Stenosis: Chorea ten years ago.*—Jane S., aged 26, came to the South London Ophthalmic Hospital on February 9th, 1878, for defect of the right eye. On the 4th, she had gone to bed seeing as well as usual, and, on waking on the 5th, noticed something amiss, and on trial found the right eye was nearly blind. It did not alter between that time and her admission, when she could only see shadows at six inches. The pupil scarcely acted alone, but acted well with its fellow. The ophthalmoscope showed the appearances commonly taken as characteristic of embolism: dense white haze of the central region of the retina and of the disc; the branches of the arteria centralis reduced to about half their proper size, quickly disappearing at a distance from the disc, and not pulsating at all, even on the firmest pressure, though some branches on the disc were slowly emptied by this means; the veins much diminished in size on the disc, some being even smaller than corresponding arteries, diminishing little, or in some cases not at all, towards the periphery, but never getting larger. An island of retina, from the disc towards, but not quite up to, the yellow spot was clear, and on inspection proved to

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* *American Journal of Medical Science*, July 1874, p. 273, from Stricker's *Med. Jahrbücher*, 1873.

be supplied by two little arteries, which gave evidence of being branches of the ciliary system, and not of the arteria centralis.* These two arteries pulsated easily on light pressure, and were, therefore, pervious; the corresponding part of the disc was much less hazy than the rest, but far from healthy.

February 16th. Pulsation was now easily obtained in the central trunk, and all its main divisions at the centre of the disc; but it did not extend up to the border of the disc. The retinal haze was less dense; the vessels, even the finer ones, which were hidden by it last week, were now visible, and thrown up by the haze behind them. Vision was unchanged.

March 2nd. Vision was unchanged. The retina and disc were nearly clear, but the disc was pale all over. All the vessels (including the two cilio-retinal arteries) were much shrunken, but the arteries pulsated well on pressure. There was a small hæmorrhage near the yellow spot (revealed by disappearance of the haze).

The ophthalmoscopic appearances have remained the same, excepting that the disc has become perfectly clear. In particular, it is to be noted that at the last examination (January 16th, 1879), the arteries all pulsated easily. She had bare perception of light with the eye.

She was a well-nourished woman, accustomed to live with her parents and earn her living by needlework; of very nervous temperament, easily blushing, her lips trembling, and one side of the face often twitching a little when spoken to. She had for some time been liable to headaches and coldness of the extremities. She had never had rheumatic fever, nor any rheumatism; but, at the age of sixteen, she was for six weeks an in-patient of King's College Hospital with "chorea", chiefly of the left side, which, she says, was attributed to a fright, though she denied knowledge of such a cause. Her mother is "very hysterical", and subject to "a sort of faint" when upset. Dr. Allen Sturge, who kindly examined the patient's heart on the day of her admission, found a well-marked mitral systolic *bruit*.

The interest of this case, for our present purpose, turns upon the symptoms in the other eye and on her headaches and general condition. It will be remembered that she said, on first admission, that she was subject to headaches and cold feet. But these headaches became much more troublesome after the occurrence of the arterial blockage in the right eye; and she had a number of troublesome symptoms, including attacks of temporary dimness or loss of sight, in the sound (left) eye. Thus, on March 2nd, 1878, "much headache" is noted.

March 23rd. She complained of "everything dancing up and down" before the left (good) eye; vision, 2 Jäger barely.

April 29th. Aching and burning in the left eye; vision, $\frac{20}{60}$, with $-\frac{1}{60} = \frac{20}{30}$.

May 22nd. Yesterday, she had an attack of dimness of the left eye, lasting five minutes. She now read 4 Jäger badly; and $\frac{24}{60}$; $\frac{1}{60} = \frac{24}{20}$.

June 7th. She was admitted into St. Thomas's Hospital to rest the eye completely in subdued light.

June 11th. She had had about six attacks of dimness in the left eye within the last three weeks. They lasted a few minutes; everything became dim, but she was never in total darkness. She never had an attack of dimness without headache being present at the time, though she often had the headache without the dimness. She did not think that use of the eye brought on the headaches. She was liable to palpitation and to feelings of faintness, but did not faint. She did not feel

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* See *Ophthalmic Hospital Reports*, vol. ix, part II.

faint when the dimness came on. She had lately been losing flesh. Catamenia were scanty and too frequent. The bowels were generally constipated. She now said that, before the right eye went blind, she used to have attacks of dimness in it like those she now has in the left eye. Ophthalmoscopic examination of the left eye showed the disc decidedly red, but quite clear; all the retinal vessels tortuous and somewhat distended. Refraction was myopic, about $\frac{1}{40}$.

June 16th. She had another attack, lasting three quarters of an hour, "like a thin gauze"; her head was very bad at the time.

June 20th (afternoon). Vision had been dim all the afternoon, but she had no headache; it was now = 4 or 6 Jäger at nine or ten inches, and $\frac{20}{60}$, with $-\frac{1}{48} = \frac{20}{30}$ badly. Sight seemed to vary. She complained of a sort of double sight, "like the reflection of the last thing I look at", probably persistence of retinal images. Ophthalmoscopic examination gave negative results.

July 3rd. She was ordered to use plain smoked glasses. They gave relief for a time. She was at the seaside (Southend) for a month in August, and returned much better. She tried bathing, but could not get warm after it. Whilst there, she had two dim attacks; one came on while sitting on the beach on a bright day; in a moment everything was in a mist and she could see neither ships nor people; it lasted "two or three minutes". Her head had been bad for some days before and continued so some time after.

Between September and Christmas, she had only one attack, but it was unusually long, and she "was afraid the sight would not come back". It came on quite suddenly, while she was in the street, and she had to ask someone to lead her home; it appears not to have been total blindness, but, as usual, "everything in a mist". It lasted "about ten minutes". She felt faint but did not faint away. Headache had preceded it, and continued afterwards. She thus described the manner in which the sight returned. "When it begins to come back, it is like looking at a light a long way off in a dark place", and "the light seems gradually to get larger till it all comes back."*

She had no idea what brought on the dimness. She never had an attack whilst lying down. Neither weariness nor emotion seemed related to the attacks.

Dec. 27th. She had no attack since the last noted, but complained of two black spots constantly dancing before the eye. Ophthalmoscopic examination of the left eye showed doubtful congestion of the disc and of the retinal veins.

Jan. 16th, 1879. Lately she had "continual throbbing" in the left eye, without pain. Vision, 1 Jaeger badly at six inches, and $\frac{20}{60}$. Ophthalmoscopic examination was much as before; the retinal vessels seemed very numerous and seemed more tortuous than usual, but not more than at the first examination, and the meaning of the fact was doubtful. Firmer pressure was needed to produce pulsation in the arteries than usual. Pulse 102, full and soft. Dr. Greenfield examined the heart and reported mitral stenosis.

In August 1878, I took the visual field, with Förster's perimeter, and found considerable contraction, both for white and red. The left eye, besides being liable to the attacks described, has throughout been "weak", so that she has done little or no needlework during the past year. The asthenopia has been of the kind so common in young nervous adults, and no doubt has been increased by the low degree of myopia which is present.

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* This was almost exactly the description given by a patient with temporary occlusion of the arteria centralis, the plug being in a short time pushed on into one of the branches.—*Lancet*, Oct. 2nd, 1875.

At the present time (February), she has gained flesh and is suffering less from headaches, under a rather long course of iron and aloes, with assafœtida.

CASE II. *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.*—Alfred F., aged 28, married, a butcher's assistant, pale, spare, nervous manner, with very little facial hair, easily perspiring, and subject to twitching of the face when spoken to, was under Mr. Liebreich's care for defect of the left eye, which had been discovered accidentally early in 1878.

I saw him in June in the following condition. With the left eye, he could only see shadows; the pupil was of same size as the right, and acted independently, but dilated very quickly to atropine. There was diffuse haze of the disc and retina for a long distance around, including the yellow spot region; the disc was rather pale. The retinal arteries were diminished in size and pulsated spontaneously on the disc though not to emptiness; the slightest pressure causes full pulsation. There was abundant pigmentation of the lower and outer part of fundus in a somewhat sector-like pattern by spots, rings, and stellate collections, chiefly in the retina; some brownish patches were seen. I have no doubt the whole of the pigment-changes were the result of hæmorrhage, very probably of retinal hæmorrhage. The appearances are still nearly the same, and the disc has not become white.

These appearances, though suggestive, under the circumstances, of some cause of sudden arrest of circulation, were not such as are left behind in the typical cases of embolism, where the disc after some months or weeks becomes very pale and quite clear.

The interest of the case is chiefly in the other eye, which, since the failure of the left, has been subject to attacks of temporary loss of sight.

The patient had never had either rheumatic fever or chorea, and did not remember ever being laid up. There was no history of syphilis. Dr. Greenfield was kind enough to make a very careful examination of the heart in August, and reported "very marked aortic obstruction and regurgitation; great dilatation of the left ventricle; typical Corrigan's pulse". He had been deaf of the left ear for eight or nine years. He was not liable to epistaxis. He never had any fits. He was formerly subject to sick headaches but never had dimness of sight with them.

On the evening of May 29th, whilst serving a customer, his right eye suddenly failed, so that he could not finish his sale; and though he could see the gaslights, he was so blind that he had to be led home. In two hours, the sight returned perfectly, and next day he went to work as usual. The only symptom attending the loss of sight was "beating in the temples".

June 1st, 5 P.M.—He had a similar attack, but not so severe in degree, and lasting only a few minutes.

June 3rd, 9 P.M.—He had another attack, followed by pain in both eyes and disturbed sleep.

June 7th. Another attack lasted an hour. He had used some blisters to the temples and considered they had relieved the "thumping" in the head.

July 3rd and 4th. He had a severe attack of pain in the head. He had had no eye attack for the last three weeks.

August 1st. Dimness again came on in the right eye, and lasted an hour. The headache had been very bad for a week or so before and continued for many days after.

From the end of August to the end of October, he attended Dr. Greenfield, and took belladonna and sal volatile with great benefit to the headaches and beating in the temples. In December, he got a cough, and has since then had more palpitation and shortness of breath.

Dec. 23rd. While returning home by train in the evening, the sight of the right eye again went away. He described it as "like a cloud passing quickly over the sight", and thought it reached its acme in "about a minute"; he said it was *not absolutely sudden.* Having to change trains, a friend led him to a seat, and he waited till the sight returned. This occurred in about half an hour, the sight returning *quite quickly*, so that "it came back all at once in a minute".

This, he said, was the only attack since August 1st. The dimness was not attended by either headache or giddiness. It is particularly noted that faintness was never a concomitant symptom of the failure of sight.

The sight of the right eye was normal, whenever tested, allowance being made for a low degree of myopic astigmatism. The ophthalmoscopic appearances of the disc were consistent with health, although the retinal arteries were if anything too large, and pulsated on the slightest pressure (not quite spontaneously, as in the left eye); there were some small filamentous opacities seen in the vitreous body, near the disc. Though central sight was normal, the field of vision was smaller in all directions than in health, and the visual field for red was markedly contracted, conditions which might probably be accounted for by a deficient general supply of blood to the retina.

He was afterwards for a short time in the hospital under Dr. Stone, but getting worse refused to remain. He had an attack of partial dimness, complained much of accommodative asthenopia, and the pupils were larger than in health. There was a small diffused ecchymosis on the sclerotic of the right eye.

Whatever may be the precise explanation of such cases as the above, the association of the eye-symptoms with serious heart-disease makes it a matter of some clinical interest to distinguish between them and some of the more trivial affections.

From cases of dimness due to variations of accommodation the heart cases are as a rule, easy to diagnose. This accommodative failure generally occurs from overuse of eyes which are optically defective (usually hypermetropic) and need spectacles, and it is seldom asymmetrical. But it must be admitted that in the accommodative cases, and in some few others, *e.g.*, some cases of threatened glaucoma, the distinction cannot always be drawn so easily as would be expected on theoretical grounds. But the cases of megrim blindness are, I think, those in which the distinction is of the greatest importance and at the same time most difficult to draw.

It is well known that certain of the subjects of megrim are liable to a very peculiar affection of sight, in which a part of the field of vision becomes obscured by a flickering or waving cloud, the edges of which in many persons are sharply defined, serrated, and brilliantly coloured.

With an intelligent patient, there could be no difficulty in distinguishing between the *momentary* obscuration of the *whole* visual field of *one* eye which characterises the cardiac cases, and the *gradual though rapid* flickering or quivering cloud, beginning at one spot and spreading in the course of minutes over something like half the field of vision, affecting both eyes, and distinguishable even when the eyes are shut, which marks the amblyopia of megrim. But many who are clearly the subjects of megrim amblyopia do not give so definite an account as the above, often no doubt because they do not observe well. In such cases, the differential diagnosis may be difficult,

particularly as a liability to severe headaches and to coldness of the extremities is equally common in the cardiac as the megrim patients.* But even here it may be noted that, when megrim patients are liable to both amblyopia and headache, the latter usually follows the eye-symptoms and that vomiting is a common climax to the atttack ; whilst in the heart cases vomiting is not usual, and the headache generally precedes the blindness, and continues after it has passed off. No very useful distinction can be drawn between the two groups in respect to *age*, although, according to Allbutt,† some of the best marked cases of ocular megrim are seen rather late in life, whilst the heart patients are often young. I think it open to question whether some cases of permanent, supervening on repeated transient, blindness in connection with headaches, and which have been attributed to megrim, have not really belonged to the cardiac group.‡

In trying to account for the symptoms in my two cases, I had entirely forgotten that Dr. Loring, in 1874,§ had offered the same explanation as the one which had occurred to me and that he had indeed developed the hypothesis at some length.‖ It must be added that Dr. Knapp, more than ten years ago,¶ recorded some cases of his own, and referred to one by Moos, which evidently belonged to the same group as these I I have read, but, in which the occurrence of repeated embolisms of some of the *choroidal* arteries was thought to furnish the best explanation, the nutrition of the corresponding part of the retina suffering quickly in consequence.

It needs, indeed, but slight reflection to see that the embolic and thrombotic processes, which have been proved to occur in the ophthalmic vessels are able to give very different results according to the position, size, and permanence of the obstruction, and also according to the precise relations to one another, and mode of branching of the central retinal vein and artery as they run in the stem of the optic nerve ; and, in suggesting that disease of the coats of the ophthalmic or of the central retinal artery may, when it occurs in conjunction with aortic or mitral disease, sometimes cause transient stagnation of the retinal circulation, I would claim for it no more than its due share as one factor in the important clinical family of blindness from vascular blockage.

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* Some difficulty may arise in distinguishing the heart cases from the " epileptiform defects of sight", described by Dr. Hughlings Jackson. The latter, however, whether consisting in subjective sensations of colour or in loss of sight, appear to be symmetrical, and this will serve to distinguish them even if there be no other epileptic symptoms.

† *Brit. and For. Med. Chir.-Rev.*, April 1874.

‡ See, *e. g.*, a case published by Mr. Hutchinson in connection with xanthelasma and sick headaches. There is no note of the heart ; but the man had had rheumatic fever several times, and extreme diminution of the retinal arteries, with white atrophy of the disc, are noted in the eye which had become permanently blind.—*Ophthalmic Hospital Reports*, viii, p. 56.

§ *American Journal of Medical Science*, April 1874.

‖ " May not the trouble", says Loring, " have been caused by some morbid local process producing a stasis of blood at the spot", *i.e.*, in some part of the central retinal vessels (p. 319). Again (p. 323), " With aortic obstruction in a feeble patient might it not occur that for a moment or two the action of the heart should be so reduced that it could not force the blood....to so distant an outpost of the general circulation as that of the retina". " ....there may have been some anatomical condition which would make it, in this case, more difficult for the heart to force the blood into the left eye than the right, and hence the recurring blindness always taking place in the left and never in the right".

¶ Graefe's *Archiv für Ophthalmologie*, Band xiv, Heft 1, p. 237.