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*From* "THE PRACTITIONER" *for December*, 1904.

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# RETINAL EMBOLISM, HOMONYMOUS HEMIANOPSIA, AND DOUBLE OPTIC NEURITIS, IN CASES OF ANÆMIA.

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THE objects of this communication are to record certain cases of anæmia, each one of which was complicated by evidences of a lesion in one or other part of the visual apparatus; to submit that in two of these cases certainly, and probably also in a third, the lesion responsible for this special complication was a thrombosis; and to urge that such a record adds force to the suggestion that the unusual condition—a double optic neuritis—present in the fourth case may reasonably be ascribed to the same cause. All the patients were women presenting distinctive evidences of anæmia in a more or less pronounced degree, though in none of them could it be said that the anæmia was extreme. In each, too, the anæmia was of a "simple" order, that is, it was unaccompanied by any qualifying event or circumstance indicating a more precise or specific diagnosis. And yet again, the record of each case exhibits a considerable improvement in the general condition of the patient under the influence of appropriate dietetic and hygienic measures associated with the administration of iron. The particular clinical feature, however, which marks these cases with a common interest is the presence in each of distinctive signs of the existence of some unusual disturbance in the course of the visual path. In two of them there was evidence of retinal embolism, in the third the patient was the subject of homonymous hemianopsia, whilst the fourth showed a very considerable degree of double optic neuritis. It must of course be admitted that these several conditions appear, on the surface at least, to differ widely from one another, certainly in the site, and perhaps also in the nature of the lesions responsible for them. But they all occurred, it is necessary to note, in cases of otherwise uncomplicated anæmia

and apart from any appreciable abnormal condition capable of explaining them except the anæmia. Hence it must be presumed that the complications already stated, namely, retinal embolism, homonymous hemianopsia, and double optic neuritis, were all direct or indirect consequences of the defective quality of the blood. The only alternative suggestion is that of chance or coincidence, and this, unlikely on *a priori* grounds, is further weakened when the cumulative testimony of the cases is given due weight, and when the case-records are studied in detail. It therefore seems necessary to accept the proposition that anæmia may, in some way or other, cause in different cases such effects as retinal embolism, homonymous hemianopsia, and double optic neuritis. The possibility that it may lead to these various events by distinct and different pathological processes must of course be allowed, though the imagination does not readily furnish the required suggestions. On the other hand, it may be held that thrombosis—an occurrence not improbable in the circumstances—will cover them all. Applying this to the individual cases it is certain, in the first place, that such an incident as embolism of the central artery of the retina demands a preliminary thrombus-formation either in the left heart or in the arterial system on the proximal side of that artery, and even if it be argued that the plugging of the artery was thrombotic and not embolic, thrombosis remains the essential factor in the result. It is beyond question that anæmia in the form of chlorosis includes among its possibilities the occurrence of thrombosis. Indeed, according to Professor Welch[1] no less than six distinct hypotheses have been framed to explain the relationship between the two. In the great majority of the recorded cases the thrombus-formation has taken place in the cerebral sinuses or other venous channels. But instances of primary arterial thrombosis, though rare, are not unknown.[2] Therefore it is in principle no novel or revolutionary suggestion to submit that in two separate cases of anæmia (Cases 1 and 2) the development of retinal embolism is a particular exhibition of a tendency which is recognised as accompanying generally the anæmic state, in a degree more or less pronounced. Unless it can be supposed that in the cases in question cardiac valve disease existed and was overlooked, there is, indeed, no opportunity to escape from the conclusion

that the occurrence of retinal embolism in each of these two patients means a preceding arterial thrombosis.

The position is similar, though perhaps attained by a less inevitable and obvious series of events, in reference to the patient the subject of homonymous hemianopsia (Case 3). Of sudden onset and existing for thirteen years without material modification there can be no hesitation in ascribing this symptom to a lesion, vascular in origin and non-progressive in character, in the left visual path posterior to the optic commissure, affecting, in all probability, either the optic radiations or the grey matter of the occipital lobe. There is certainly in the record of the case nothing to support a diagnosis of hæmorrhage, and whether thrombus-formation *in situ*, or embolism, be adopted, it serves equally the argument that thrombosis is the essential basis of the event. The history of the case, it will be admitted, affords the strongest possible presumption that at the date of the onset of the hemianopsia the patient was suffering from chlorosis, and the later record practically puts out of court any possible suggestion of tumour or other advancing lesion. Hence in this instance, also, it can be claimed that an unusual complication in a case of anæmia, namely, homonymous hemianopsia, finds its most probable explanation in a process of thrombosis. Thus the two cases of retinal embolism (Cases 1 and 2) and the instance of homonymous hemianopsia (Case 3), all occurring in anæmic women, are found not only to share certain clinical features in common but also to rest on an identical underlying pathological process, namely, either thrombus-formation *in situ*, or embolism following thrombosis in some part of the circulatory apparatus on the hither side of the lesion immediately responsible for the visual defect.

There remains the case (Case 4) of double optic neuritis. The argument that when this condition occurs in the course of chlorosis it is dependent on a thrombosis of one or more of the intracranial veins or sinuses has been stated in detail in previous communications,[3] and in the case now recorded it cannot be said that there is any specific point which adds weight to that argument. But in view of the fact that the records here submitted multiply the symptomatic disturbances in anæmic women which must find in thrombosis their immediate explanation,

it may be claimed that they do in some measure strengthen the suggestion that the optic neuritis that occasionally complicates chlorosis (*e.g.* in Case 4) has in all probability a like interpretation. No doubt in the cases of retinal embolism (Cases 1 and 2) the thrombus-formation took place within arterial channels, and possibly the same may be true in the case of homonymous hemianopsia (Case 3). But this does not compel a similar restriction in the instance of optic neuritis (Case 4). Once recognise the tendency to intravascular blood-clotting, and the incident must be admitted as a possibility in all orders of the blood-vessels. That optic neuritis in chlorosis is due to arterial thrombosis is highly improbable, if only for the fact that when this condition occurs in syphilitic or other disease of the cerebral arteries optic neuritis does not accompany it. Indeed this fact has been urged against the conclusion that the optic neuritis of chlorosis is a result of sinus-thrombosis. It may be questioned whether this is a valid argument. The effects which follow obstruction of an artery are not identical with those caused by obstruction of the venous return, and in view of the obscurity which veils the exact relationship between intracranial disease and the development of optic neuritis it is hardly safe to conclude that thrombus-formation in the intra-cranial veins or sinuses is incapable of producing optic neuritis because a similar change within an artery does not produce this result. And there remains the undoubted fact that now and then a patient with chlorosis dies somewhat suddenly with acute evidences of intracranial disturbance, including optic neuritis, and a post-mortem examination reveals no appreciable lesion other than sinus thrombosis.

Another argument advanced against the suggestion of sinus-thrombosis as the cause of double optic neuritis in chlorosis is that the optic neuritis may, and with relative frequency does, occur apart from other evidence of intracranial disease. Whilst this is true in part it does not cover all the facts. Thus such symptoms as unusually severe headache, vomiting, and diplopia due to an ocular paralysis, have been observed in association with optic neuritis occurring in the course of chlorosis. There is, indeed, in connection with chlorosis a graduated series of cases from those in which optic neuritis is the sole unusual event to those in which an acute development of such cerebral

symptoms as delirium, coma, paralysis, and optic neuritis terminates in death, and the discovery of sinus-thrombosis on post-mortem examination. And, to quote again from Professor Welch's article, "not very infrequently after death in one or more of the intracranial sinuses thrombi are found which had occasioned no recognisable symptoms during life." There is, therefore, a double answer to the objection that sinus-thrombosis cannot be the cause of optic neuritis in chlorosis because there occurs with the neuritis no other evidences of cerebral disturbance. In the first place this objection as a statement of fact is not wholly accurate, and secondly sinus-thrombosis, as the above quotation testifies, may exist and yet may fail to produce any such evidences. It is therefore conceivable that it may cause optic neuritis as its sole and unaccompanied clinical consequence. But it may be said that the fact stated by Professor Welch proves too much, so far as the present argument is concerned. If sinus-thrombosis may be discovered after death in persons who during life did not show optic neuritis, this, it may be held, is a demonstration of the incapacity of sinus-thrombosis to cause optic neuritis. But intracranial tumours are exactly in the same position. Some of these are, and some are not, accompanied by optic neuritis, and occasionally such a tumour is discovered after death though during life no suspicions of its presence existed. Now the absence of optic neuritis from some cases of brain-tumour will not be advanced against the doctrine that intracranial tumours may, and frequently do, cause optic neuritis. Equally the absence of optic neuritis from some cases of sinus-thrombosis does not prove that this condition can never number optic neuritis among its consequences. In short, the non-occurrence of optic neuritis in arterial thrombosis, its absence from some cases of sinus-thrombosis, and its development in chlorosis apart from other cerebral symptoms, neither separately nor together render untenable the position that the optic neuritis which is seen in some cases of chlorosis is in all probability due to the occurrence of thrombosis within some of the intracranial veins or sinuses. The case (Case 4) reported with this article is, as already stated, merely one further example of optic neuritis in chlorosis without any feature specially assisting the solution of the clinical problem presented by such a combination. It

finds an obvious bond with the other three cases in that in all of them the only discoverable condition competent to explain the distinctive events which marked their course was the qualitative defect of the blood. Thus in this defect an explanation of these events, in some sense or other, must be found. That this explanation in the cases of retinal embolism and of homonymous hemianopsia is afforded by the suggestion of thrombosis can hardly be open to question. And in view of this conclusion it may be urged that the same pathological process offers itself as a not unlikely interpretation of the case in which double optic neuritis is the peculiar feature. It is certainly noteworthy that the unusual events occurring in four cases of anæmia appear to find in thrombosis —an admitted possibility in the circumstances—an adequate and inclusive interpretation. The presentation of a rival suggestion having an equally comprehensive claim is not a task that may be easily accomplished.

## ABSTRACTS OF CASE-RECORDS.

*Case I.*—L. R., æt. 21, maidservant. Fourteen days before examination at the Central London Ophthalmic Hospital complained of headache and discomfort in her left eye, which, on closing her right eye, she found to be blind. Ophthalmoscopic appearances as of comparatively recent embolism of the central artery; left pupil no direct but free consensual light-response; V. = no P.L. Decided pallor of mucous membranes and loud venous hum at the root of the neck on each side; cardiac impulse and left border of dulness in nipple-line, sounds pure. Physical examination otherwise negative. Some complaint of breathlessness on exertion and menses deficient in amount.

Personal history of occasional "pains in arms," but no definitely rheumatic incident, and family history also free from evidences of rheumatism.

After a month's treatment the general condition of the patient had much improved, but the defective vision of the left eye remained without change.

(I am indebted to Mr. Brittin Archer for the opportunity of seeing this patient.)

*Case II.*—A. G., æt. 42, hospital matron, unmarried. Loss

of sight in the right eye observed in the morning seven days before examination and after a severe attack of vomiting during the preceding night. Right fundus shows the appearances seen in retinal embolism; pupil-response to light not sustained; counts figures at four feet; the perimeter detects a limited area of vision above and to the outer side of the fixation point, otherwise the field is obliterated. General signs of anæmia, including venous hum over the right jugular vein and a systolic murmur over the base of the heart. No other evidence of disease to physical examination.

The personal history is one of good health except for (1) an illness attributed to overwork five years ago, (2) excessive menstruation and anæmia two years later; no suggestion of rheumatism at any time. The family history included evidences of diabetes and perhaps also of tubercle, but no indication of rheumatism.

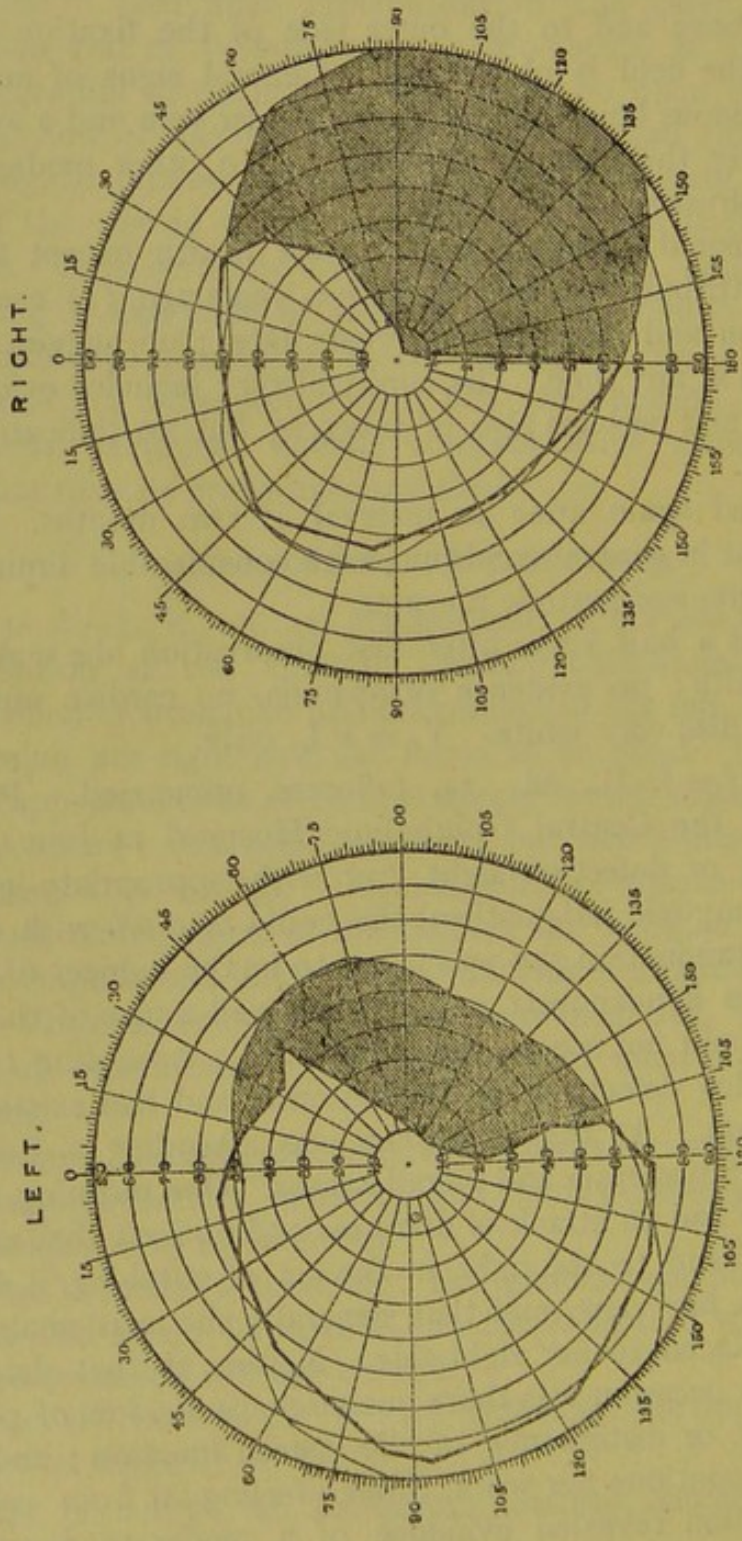
Examined again after an interval of two months. Great improvement in general condition, with considerable diminution of venous hum and systolic murmur.

Four and a half years after first observation she was seen in good health; no evidence of anæmia, no cardiac murmur. Right optic disc very white. V. = P.L. only.

*Case III.*—J. B., æt. 34, tailoress, unmarried. Patient attended at the Central Ophthalmic Hospital in June, 1904, complaining of defective sight, but with appropriate glasses (compound myopic astigmatism) she could read 6/6 with either eye. On examination she was found to be the subject of right homonymous hemianopsia, and she was well aware of the fact that she "could not see to the right side." According to her statement this defect was of sudden onset and had existed for thirteen years. At that time she was attending a London hospital for "dyspepsia and bloodlessness." One morning, when at work, she had an attack of dizziness and noticed that as she carried her hand to the right, in the act of stitching, it disappeared from her view, and that generally she was unable to see things placed to her right side. Neither at that date nor on any other occasion was there unconsciousness, loss of power in the limbs, or disturbance of the speech function; and she was able to continue her work, though feeling far from well.

Examination revealed evidence of a moderate degree of

anæmia, but there were no signs of disease in the thoracic or abdominal viscera, or in the nervous system (except as above). The light-reaction of the pupils was normal, as was also the appearance of each fundus oculi.




*Case III.—Right Homonymous Hemianopsia in a woman of 34 years. Onset sudden, when suffering from "dyspepsia and bloodlessness" at 19 years of age.*

Personal history free from all illness except as already recorded; no form of rheumatic disease, and no suggestion of syphilis.

There can be no doubt about the accuracy of the facts as far as the hemianopsia is concerned, as the patient has in her possession the visual charts which were taken shortly after the onset of the right-sided blindness.

(I have to thank Mr. W. Ilbert Hancock for the opportunity of observing this case.)

*Case IV.*—E. H., æt. 21, maidservant. In March, 1904, suffered from severe frontal headache and consulted Dr. Fraser, of Harpenden, who, recognising optic neuritis, sent patient to hospital. On admission decided evidences of anæmia with double optic neuritis (swelling in right 4 dioptres, in left 5 dioptres); no other signs of disease to physical examination, and vision to usual tests little if at all prejudiced. Left hospital at end of June in good general condition with fundus oculi on each side almost normal in appearance.

## REFERENCES.

[1] *System of Medicine* (Clifford Allbutt), Vol. VI., p. 202.
[2] *Ibid.*, p. 200.
[3] *Glasgow Hospital Reports*, Vol. II., p. 119; *British Medical Journal*, February 8, 1902; and *Lancet*, April 30, 1904.
[4] *Loc. cit.*, p. 202.