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A CASE OF HOMONYMOUS, LEFT-SIDED INFERIOR TETARTANOPSIA FOLLOWING TOXIC DOSE OF SALICYLATE OF SODA.

By J. H. CLAIBORNE, M.D.,
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A large, short-necked gentleman of 57 years of age consulted me in July, 1904, on account of some ill-defined obscuration of vision, which had not been relieved by glasses. His business was matching and comparing cotton samples and he experienced an annoyance in his work which he found some difficulty in describing. His vision for far and near, in both eyes, had been tested by a surgeon, and found to be normal under correction. He had always been a free liver, and two months before I saw him had been on a moderate champagne spree, — a thing in which he was accustomed to indulge periodically.

The spree which immediately antedated the present trouble lasted about a week. Toward the end of it he took a bath, which was followed by some indefinite pains in the top of his head, arm, and leg. They were thought to be rheumatic by his physician,

who gave him salicylate of soda. The dose was large enough to produce physiological, characteristic symptoms of tinnitus; he took as much as eighty grains in twenty-four hours, according to the statement of his medical attendant. Twenty-four hours after commencing the treatment, vision became very misty, and a dark spot appeared toward the left. The salicylate was reduced to one-half the amount, but no clearing of vision resulted. It was then entirely discontinued. Within a week or ten days the vision had cleared gradually, until it was apparently normal, except for the indefinite blur already referred to.

The patient was sent me in consultation by the oculist whom he consulted, and this gentleman wrote me that at the time he first saw him there were normal fields of vision.

He had a compound hyperopic error and presbyopia. His vision with the right eye was 20/20, and the twenty-foot line was read easily and accurately. In the left eye, however, the vision was barely 20/20, and the line was read with apparent uncertainty.

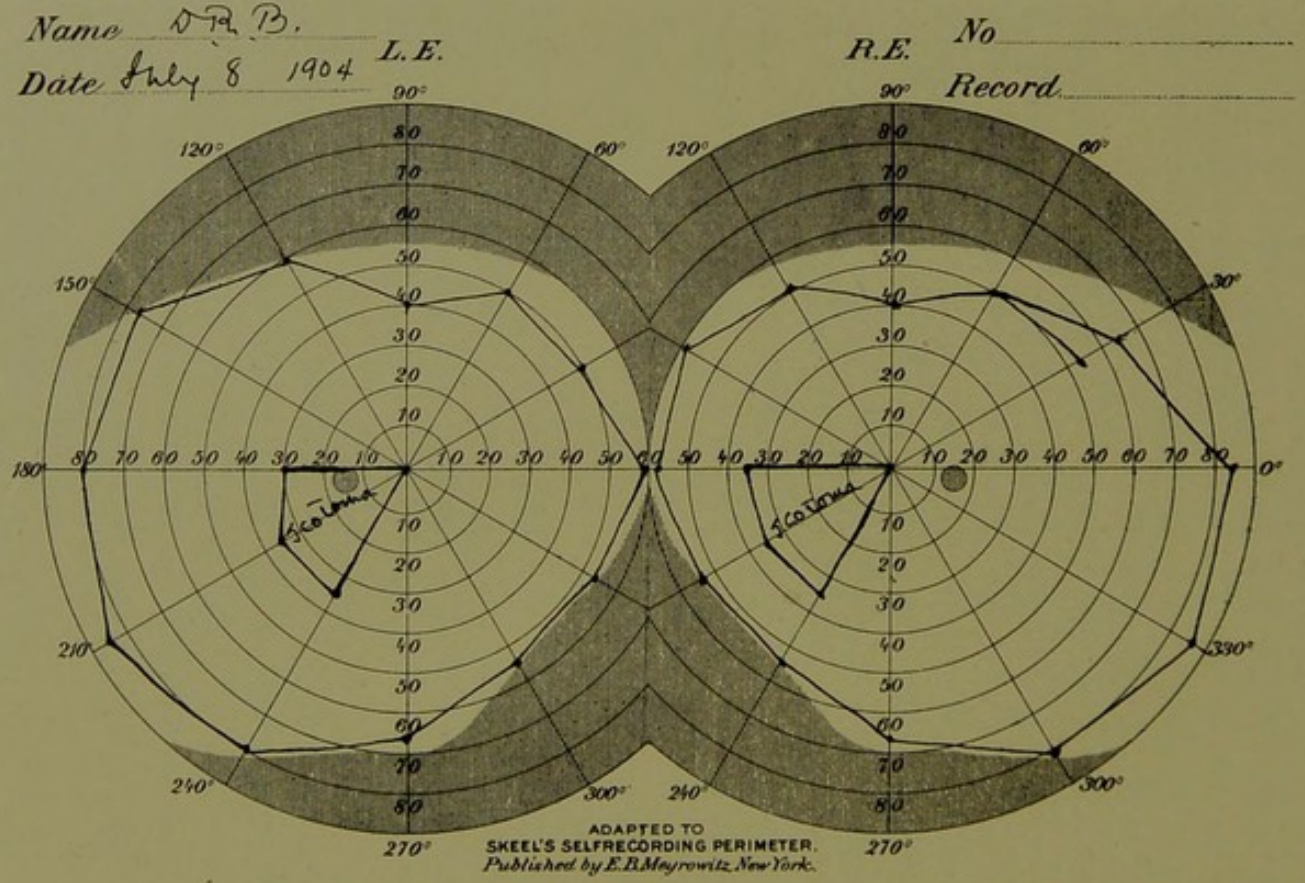
On cross-questioning him closely, I obtained the statement that when the blur came on, it was much denser to the left, that it persisted in that region after the right side had cleared, and that during the general obscuration the darker spot referred to appeared on the left side.

He denied any specific history, and his urine subjected to morphological and chemical analysis yielded practically normal results. At the time I saw him, the status was as follows:

R. eye=20/20 m+75 D. ⌒ +.25 Dc. ax. 180°.
L. eye=20/20?w.+75 D. ⌒ +.25 Dc. ax. 180°.
+2.75 D. s. ⌒ cyl. as above, for reading.

In each eye the retinal veins were a trifle large, arteries normal, disc normal; the rest of the fundus normal. No external signs; orthophoria, central color perception normal; pupillary reaction normal; left grip weak; left knee-jerk exaggerated; no Rhomboid; no disparity in sensation.

My colleague, who referred this patient to me, wrote that when he saw him just after the illness there was a possible pallor of the discs.



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The fields of vision in the two eyes constitute the interest in this case.

It will be observed that the extent of the field for form is about normal, but downward and to the left in each eye, almost mathematically symmetrical, is a scotoma which is absolute, and which represents a triangle. In the right eye the three points marked on the base were at 35° and the sides ran clean and straight to the apex at the center. In the left eye, almost the same thing occurred, only the point in the horizontal plane marking the limit of the scotoma was at 30° instead of 35°. I frequently tested this to see if I was in error, but repeated trials convinced me it was correct. It will be observed, too, that there was vision from the base of the triangle out to the periphery.

This scotoma is consistent with the fact that the obscuration of vision was mainly toward the left, and is probably the dark spot originally observed by the patient. Its position will also account for the fact that the final central vision in the left eye was not so good as in the right.

The fields for red and green are almost, if not quite, as interesting as that for form.

Let us look first at the field for red. In the right eye it is distinctly erratic and irregular. It will be observed that downward and to the left there is wanting a quadrant of 90°. In the left eye the field is still more contracted and erratic, but the temporal limit is naturally greater than the nasal in the right eye. There is here also a quadrantal deficit of 90°, but there is in the blind area along the sector nearest to the vertical, an islet of about 20°, in which red was distinctly recognized. Very careful trial a number of times proved this fact, and likewise the fact that it existed nowhere else in the red-blind area.

The field for green is quite as interesting. Let us look first at the right eye. It will be observed that the green-blind area invades the right half of the field below to the extent of 30°; also the upper half to the extent of 30°, so that this defect is not, strictly speaking, quadrantal.

In the left eye the green-blind area invades the right field,

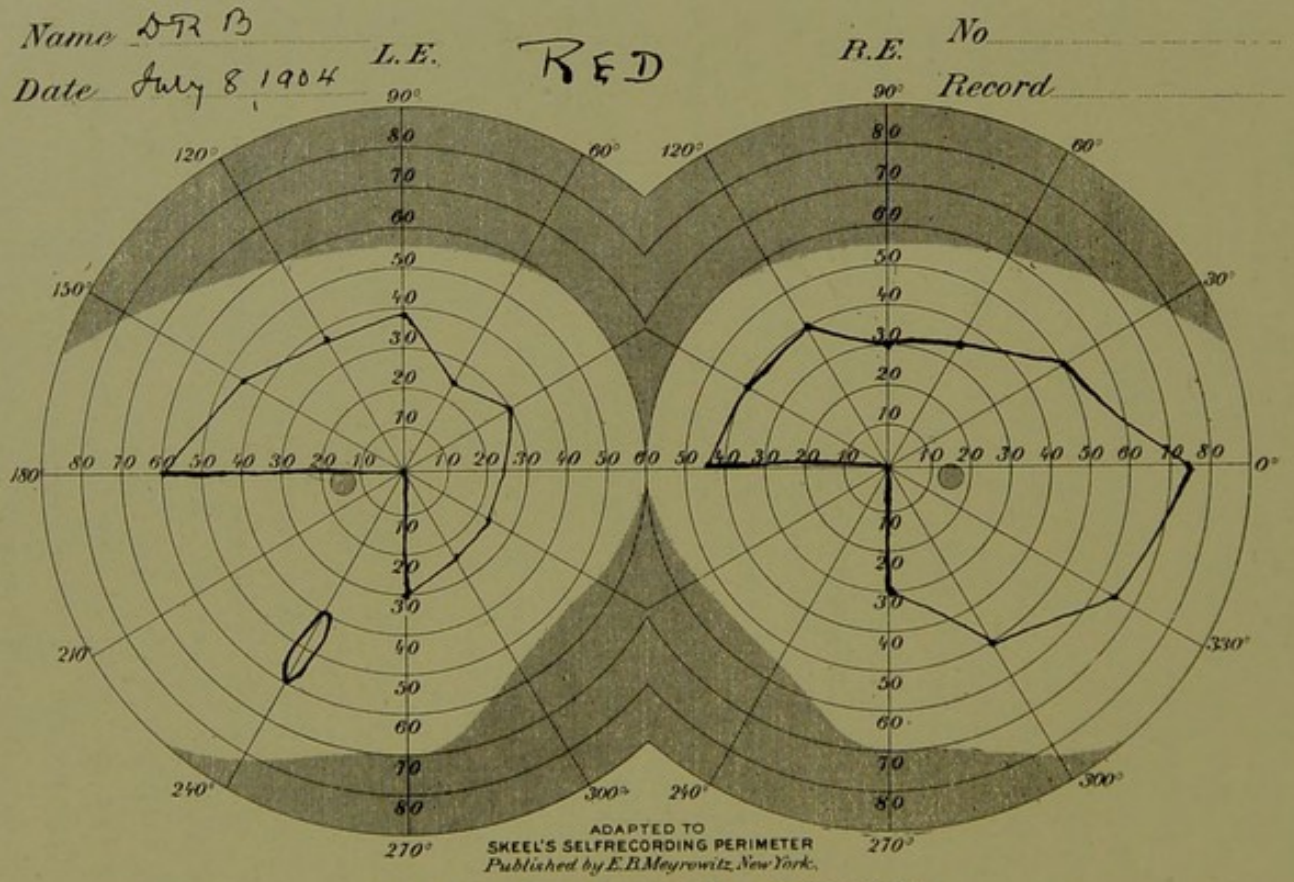
30° below, but remains at horizontal limit to the left. As in the field for red there was found along the same sector an islet of vision for green. It differed, however, very slightly in length. As has been observed, the central perception for red and green seemed normal. It can be readily understood how this might be in the right eye, but it would appear reasonable that it should be slightly modified in the left eye, even as the central vision for form was, and indeed in all probability it was, but in the nature of things was not sufficiently striking for the patient to make the comparison.

After a comparatively patient and thorough search of the literature of the visual field, I have not been able to find anything like these fields, either for form or colors.

I am acquainted with the fugacious hemianopsic field for colors and also with the fugacious hemianopsic field for form, but I have never before seen a symmetrical form scotoma in a quadrantal scotoma for colors, in which the form scotoma was smaller than the color scotoma. The islets of green and red in the color-blind areas are interesting, particularly in their symmetrical character. I have seen islets for form in blind areas, but never an islet for colors in a color-blind area. It is interesting also to observe the invasion of the right field below by both the red and green-blind area and of the upper and left field by the green-blind area. And yet, it is not unreasonable, at least, in the case of green, since the green sense is less acute than the red, and the field naturally smaller. Similar islets of vision for form are at times found in the blind areas in quinine amaurosis.

I believe this is the first observation that has been made on the color field following salicylate of soda intoxication. The similarity of intoxication by the salicylate to that induced by quinine has been often noted. I, myself, have reported a case in which eighteen grains of quinine produced a transient but typical amaurosis.

As to the contributory intoxication from champagne, it was obvious that the patient was not a regular drinker, but that he occasionally got on "sprees," always from champagne, but never



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drank heavily during them. He was always capable of attending to business during these "sprees," and they never degenerated into a debauch. They were accustomed to last about a week.

I think it can be safely said that while the champagne was a contributory cause, the symptoms were those of salicylate poisoning, and the resultant fields may be cited as the result of it.

De Schweinitz, in "'Toxic Amblyopsias," 1896, cites all the cases extant at that time of visual disturbances due to salicylic acid and salicylate of soda. He cites the case of Gatti, published in 1880, of a young girl of 16 years, who took eight grammes in ten hours. At the last dose she fell asleep and awoke entirely blind. The optic discs were normal, but the retinal veins were well filled. These conditions persisted after the restoration of vision, which took place in twenty-four hours after the blindness began.

Earlier than this, Riess, 1875, made the observation that five-gramme doses of salicylate caused tinnitus and disturbances of vision. Knapp, in 1881, in describing quinine blindness, remarked that he had seen three cases presenting precisely analogous symptoms, due to large doses of salicylate of soda.

De Schweinitz has made experiments on dogs with salicylate of soda. In the first experiment he gave a dog sixty grains of soda salicylate, by injection, and on the second day afterward he gave sixty grains of salicylic acid, in the same way. On the second day the dog became partially blind and failed to avoid objects about the room, — slight diminution in size of arteries. On the eighth day the dog was still partially blind; two days later the sight had evidently returned, as the dog no longer avoided objects. In three days more the discs were pale, the arteries distinctly contracted. De Schweinitz thought it probable the peripheric field was contracted.

In experiment No. 2 he injected sixty grains into another; the following day the dog was apparently blind. In several similar experiments partial blindness resulted.

The same author, after his experiments on dogs, concludes that it is possible to produce partial blindness with large doses of

salicylic acid or salicylate of sodium, and that the ophthalmoscopic picture in a minor degree is that produced by quinine.

The only ophthalmic sign in my case, two months after the intoxication, was fullness of the veins. The only sign in the fundus remarked at the time of the intoxication was "a possible pallor of the discs."

A recent communication from the surgeon who first examined this case tells me that he has found the fields, in April of this year, practically the same as I found them twelve months before; the main difference between his result and mine lies in personal equation. He did not, however, find the islets for green and red. The patient writes me he is no longer annoyed by his defect.

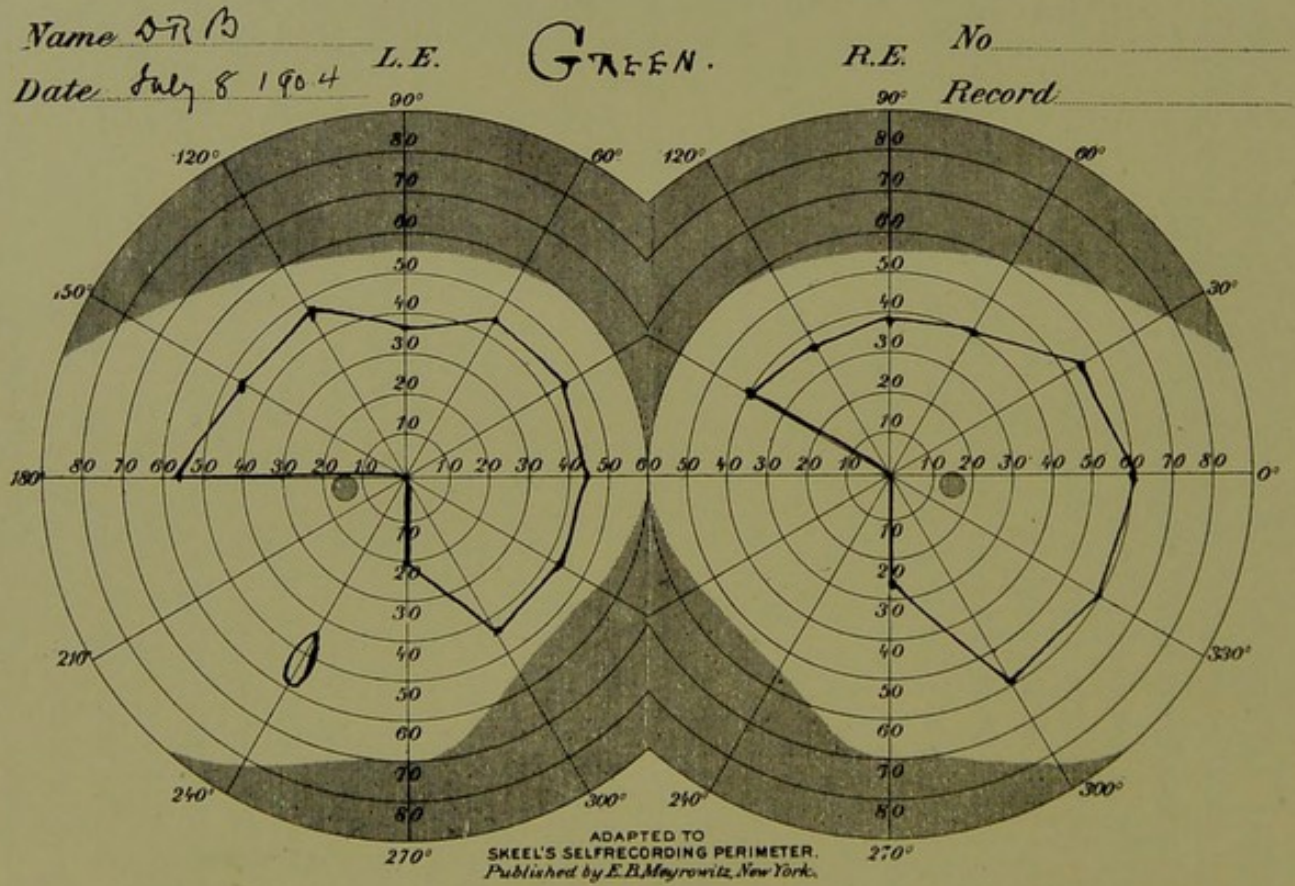
Brunner, in discussing the mechanism of quinine blindness, suggests the possibility that the blindness has a cerebral origin, the central ganglia of the cortex of the cerebri being affected, and that the loss of function depends upon circulatory disturbances or inflammatory processes in the cerebral cortex of the visual centers. He finally, however, dismisses this possibility, and accepts ischæmia of the retinæ, the ultimate result being secondary changes and thickening of the vessel walls.

De Schweinitz, in his study of quinine amaurosis, found complete atrophy of the visual path, optic nerve, chiasm, and tracts, as far as they could be traced.

Both Brunner and de Schweinitz admit that the original effect of quinine is upon the vaso-motor centers, and I believe all concur in this view. If this is true, then it is not impossible that there may be an acute endovasculitis or thrombosis at the cerebral centers, and that the degeneration of the retina and the visual paths, in quinine amaurosis, may be partly *descending*, if not entirely so.

Holden, in an experimental study on dogs, concludes that the pathological process, following quinine amaurosis, consists in a constriction of the retinal vessels, particularly of the arteries, followed by an albuminous serous exudation into the nerve fibre layer, and a degeneration of the ganglion cells, together with their axis cylinder processes.

Holden subsequently made other experiments, keeping the dogs



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for six months, but found nothing more; the degeneration of the nerve fibres proceeded as far as the basal ganglia, but he did not find at any time any cortical changes. He regards the nerve degeneration as distinctly an ascending one. His later experiments are unpublished, and these remarks are based on his statement to me in person.

As far as I knew, no one up to the present has examined the cerebral cortex of any person who has suffered from quinine amaurosis, and until that has been done, the burden of proof must rest upon the pathological findings after experimenting upon the lower animals, and upon the observations of clinical observers.

In Gatti's case 8 grammes (about 120 grains) was taken in ten hours. The girl fell asleep and awoke entirely blind. Gatti explained the result by the direct action of the salicylate of soda upon the retina and optic nerve — clearly an opinion.

Riess observed visual disturbances after 5-gramme doses (about 75 grains. De Schweinitz found visual disturbances in 60-grain doses injected into the dogs. The patient in the present case had about 80 grains in twenty-four hours. A pallor of the discs was observed about that time, and the complete physiological effect of the drug was experienced. Incidentally, the islets of vision for red and green in the blind areas for these colors are consistent with findings in quinine amaurosis; likewise the resultant good vision.

A careful search of the literature bearing on this subject fails to bring to light any observations on the fields in salicylic acid or salicylate of soda intoxication.

Uthoff, than whom there is no greater authority in the toxic amblyopias, remarks that in the matter of the fields, in salicylate toxæmia, scarcely anything definite has appeared.

In Willbrand's masterly exposition of the field of vision, I have not been able to find anything comparable to the fields herewith shown.

In many other papers on quadrantal defects in the field I have found nothing similar. They are apparently unique, in the first place, as quadrantal defects, secondly, as the first carefully

outlined fields which have been observed to follow salicylate intoxication.

The location of the lesion is interesting as a matter of speculation, notwithstanding the elaborate scheme of Henschen. It was probably not subcortical or in the capsule, since there was no history of hemiplegia or hemianæsthesia. The weak grip and the exaggerated knee-jerk on the left, I believe, are entitled to but little consideration. I believe the lesion was in the cuneus, probably the lower portion.

To apply the term *hemianopsia* to fields like these, is obviously incorrect. The proper name is homonymous left-sided, inferior tetartanopsia.