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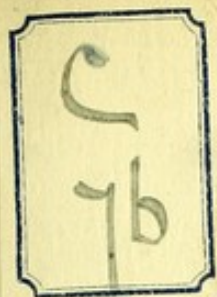
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With the Compliments of
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A CASE OF LEAD POISONING PRE-
SENTING SOME UNUSUAL
EYE SYMPTOMS.

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BY
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OF CHICAGO;

PROFESSOR OF OPHTHALMOLOGY IN THE POST-GRADUATE MEDICAL SCHOOL.

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**A CASE OF LEAD POISONING PRESENTING
SOME UNUSUAL EYE SYMPTOMS.**

By CASEY A. WOOD, M.D.,
OF CHICAGO;
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ALTHOUGH ocular symptoms, as a result of plumbic intoxication, have been recognized and studied for over two centuries attention has been chiefly directed to the visual defects arising from the fundus changes, especially inflammation or atrophy of the optic nerve. In recent years a transient amblyopia, not attended by any lesion visible to the eye and comparable to the temporary blindness of uremia or diabetes, has also been recognized and frequently reported. Various forms of retinitis, too, have been observed, and these are believed by Parisotti¹ to be due to a form of endovasculitis which he terms *endarteritis saturnina obliterans*.

Another class, possibly rarer than the foregoing, includes instances where the intoxication produces paralysis of one or more of the extrinsic ocular muscles. The following report is of such a case:

Jacob B., aged forty-nine, has steadily followed the occupation of painter for sixteen years, doing both inside and outside work during that time. He has enjoyed fair health and the only history of illness, apart from the attacks about to be described, is that of typhoid fever many years ago, followed by a large carbuncle in the lumbar region. He is married and has had five children, of whom one died at six months of age. There is not now any evidence of syphilis in his own person or that of his wife or

children. He has had no injury to the head nor is he the subject of any lesion of the brain or cord. He has, however, had several attacks of what seemed to be true painter's colic. The first of these come on five years ago, and the last one six weeks ago. There has never been any weakness of the extensors of the hands, approaching the condition of "wrist drop," but he speaks of having noticed a slight tremor of the hands and a weakness of grasp. There is no definite history of transient attacks of defective vision. Three years ago, on rising one morning, he became dizzy and fell. He says he remained unconscious for half an hour, after which he felt a dull pain which extended over the whole head, lasted two days, and then gradually disappeared. Since then he has had persistent double vision, and occasional attacks of vertigo. Three weeks after this last attack his left eye began to turn out, and the left lid has drooped so that the eye is almost closed.

His present condition is as follows: his left eye has vision $\frac{2}{5}$, not improved by glasses. With $+ 3$ D. he reads words of Jæger 1. There is complete *ophthalmoplegia externa*, with the exception of the external rectus and possibly of the superior oblique. The paresis of the levator labii superioris is most marked when he is not fixing his eyes. The pupillary reflexes of both eyes are sluggish to light and accommodation. With the ophthalmoscope very few fundus changes are visible, but the outlines of the papilla are not clean cut, and it has a woolly appearance. There are a few patches of disturbed choroidal pigment scattered over the fundus. There is nothing abnormal about the right eye except the lazy pupillary reactions and slight fundus changes resembling those of the left eye. $V = \frac{2}{3}$, and Jæger 1 is easily read with $+ 3$ D.

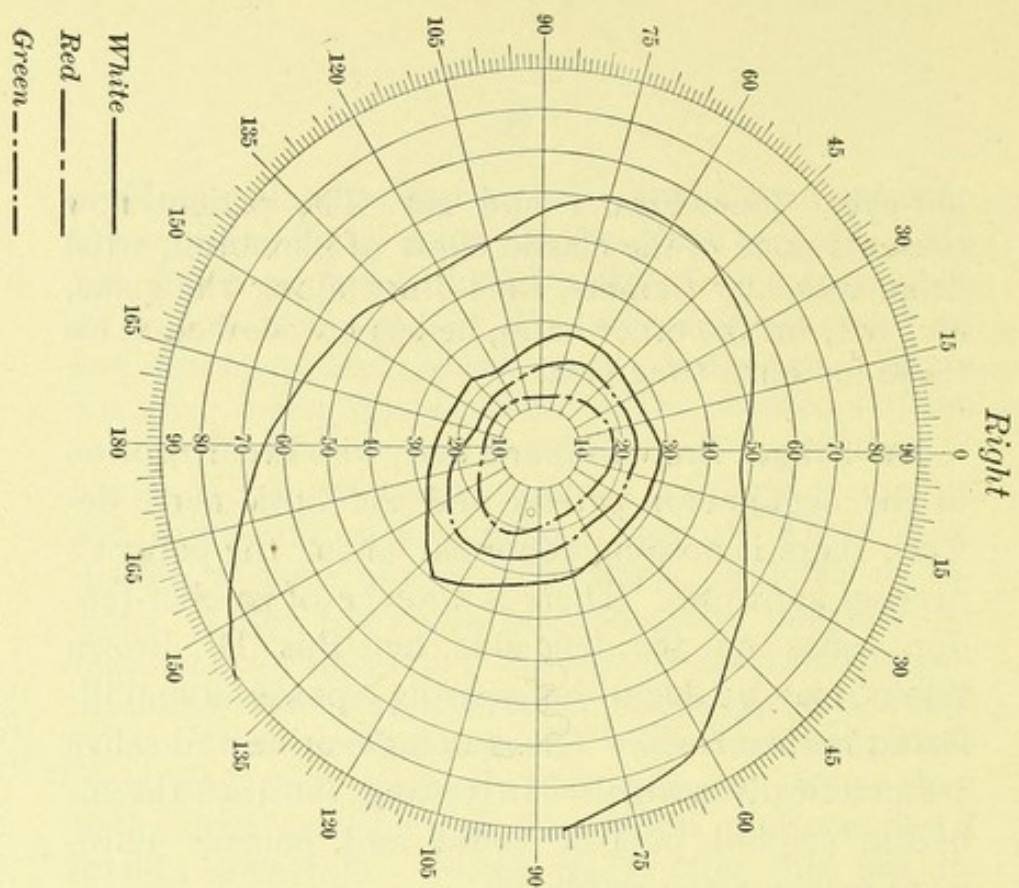
The visual field of each eye is decidedly contracted, the limitations being more marked in the

left eye. (See Figs. 1 and 2.) The patient now presents none of the classic signs of plumbism, wrist drop, muscular tremors, blue lines along the gums, etc, but, on the other hand, he has not worked at his trade for some time past.

Dr. Sanger Brown reports that, with the exception of the oculomotor paresis and the optic-nerve defects, there is nothing abnormal about the patient's nervous apparatus. There is absence of patellar-tendon reflex on the left side, but this, Dr. Brown thinks, may be due to his wearing a powerful and ill-fitting hernial truss. The patient's urine and saliva are practically normal. In testing for lead the dihydric sulphid, potassic iodid, and potassic chromate reagents are employed.

To me the most interesting signs of plumbic intoxication in this case are the unilateral pareses, and the fact that the optic-nerve atrophy set in without any of the transient attacks of amblyopia which usually precede the permanent disease. The treatment has consisted of hot baths, small daily doses of magnesian sulphate and potassic iodid; of the latter one gram three times a day, gradually increasing it until nine grams *per diem* were administered. The patient has not been working for the past six weeks. Some improvement in the muscular signs has already resulted.

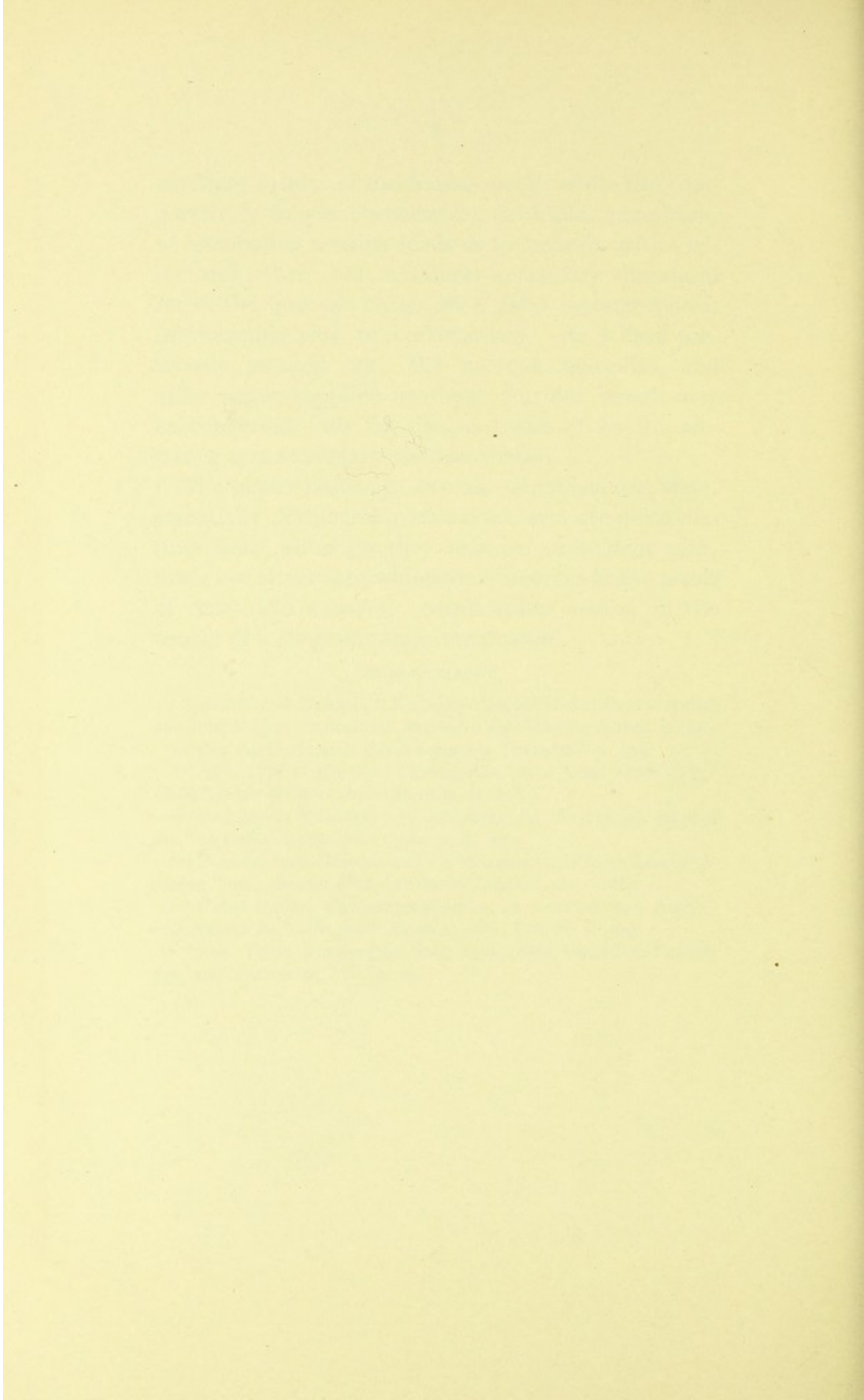
The above is the only instance of saturnism affecting the third nerve that I have observed among nearly 13,000 cases of ocular disease that have come under my immediate care, but I think it is quite possible that I have attributed to syphilis and other causes, examples of ocular paralysis that were really



Contraction of visual field as a result of chronic lead poisoning.

due to lead poisoning. When one thinks of the numerous sources of lead intoxication to which the public is exposed (from food and drink stored in leaden vessels) and of the many crafts in which lead is continually used, examples of oculomuscular defects due to plumbism must be far from rare. However that may be, the reported cases are not numerous. Galezowski² reports several of these and thinks they constitute a large percentage of saturnine ocular intoxications, and that they are particularly unsatisfactory to treat if all the branches of the third nerve are implicated. Incomplete pareses, on the other hand, are more amenable to remedial measures. Landesberg³ reports a case of bilateral paresis of the external rectus, while another patient observed by him had complete paralysis of all branches of the oculomotor nerve. Von Schröder⁴ also reports a case of typical neuroretinitis with bilateral abducens paralysis. Wadsworth⁵ gives a very instructive account of a boy, aged 9, in whose urine lead was found for many months, although the source of the poison was never ascertained. He had marked optic neuritis and paralysis of several ocular muscles, and eventually became blind from optic atrophy.

It is not difficult to understand why the eye muscles should occasionally suffer in chronic lead poisoning. One of the most characteristic signs of the disease is an affection of the voluntary muscles in other parts of the body, and there is no reason why the motor apparatus of the eyeball should escape. The case reported by Parisotti furnished, by means of the ophthalmoscope, an opportunity of studying the minute changes as they actually progressed in the



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