illumination, can be seen to have a well-defined and somewhat thickened, irregular border. Slight hazy opacities can be seen in the center of the disk, and the same to a less degree outside of same in the posterior cortical plane of the lens.

That this case is exactly similar in type to glass-blower's cataract is proved by the words of Herbert Parsons and Marcus Gunn, who, in 1908, visited some of the important glass-works in England for the Royal Society, to gather statistics of industrial diseases for the framing of the Workmen's Compensation Act. They say:

Bottle-makers are subject to a form of cataract which appears to be characteristic, and is unlike other forms of cataract commonly observed. In its typical form there is a dense, well-defined disk of opacity in the center of the posterior cortex. Not infrequently slighter hazy opacities are seen around this posterior cortical disk.

Hirschberg describes it in a similar way, and in 1898 exhibited five cases of glass-blower's cataract. At the same time he drew attention to the peculiar color of the faces—the effect of heat on the skin. Most of the German literature on the subject seems to concede that heat is the factor that produces these changes in the lens and skin.

Weyl* mentions fire-workers, blacksmiths, puddlers, bakers and cooks as subject to this same change in the lens.

In a volume on industrial diseases, 1909, Greer says of bottle-maker's cataract: "It commences as a saucer-shaped opacity like a cobweb in appearance, and situated immediately within the posterior capsule of the lens."

The treatment which I am using is the method of von Pflugk and Verderau—instillations of 2 per cent. solution of potassium iodid, to be followed later by sub-conjunctival injections of the same. According to Hirschberg, these cases progress very slowly until 20/200 vision has been reached, when often a more rapid decline follows. When ready for operation they present a most favorable type for successful extraction.

34 Washington Street.

THE DIAGNOSTIC VALUE OF THE PARADOXIC REFLEX

AN ADDITIONAL ANATOMIC PROOF OF ITS PRACTICAL
IMPORTANCE

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In December, 1904, I described¹ a new reflex under the name of paradoxic flexor reflex. I found it then present in cases the history of which pointed to organic diseases of the nervous system and especially of its motor tracts, and always associated with exaggerated knee-jerks. As is well known, Babinski's toe phenomenon is the clinical expression of a disease of the motor pathway.

The reflex can be elicited in the following manner: The patient may either lie on his back or sit on a chair with the feet (not the legs) on a stool. The feet must be slightly rotated externally; in this position complete relaxation of the muscles of the leg is obtained. The examiner must always place himself at the outer side of the leg; the thenar and hypothenar muscles of the right hand must be placed on the inner surface of the

tibia of the patient and the fingers press deeply on the middle or the lower portion of the calf muscles. I say "deeply" because the pressure must be transmitted to the flexor muscles of the deep layer; sometimes pressure must be combined with lateral movements of the superficial muscles. If the reflex is present, extension of the great toe, or of all the toes, will be noticed. There is evidently no other muscle except the flexors that could be brought into display. The tibialis posticus, the peronei have an entirely different function from flexion or extension of the toes.

During my investigations I observed a peculiar relationship between these two reflexes. Babinski's sign was sometimes present and sometimes absent, but in the majority of my cases the paradoxic reflex was present on the side on which the Babinski sign was either absent or slight. It was present in those obscure cases in which the Babinski reflex was not obtainable.

My first series comprised thirty organic cases, in nineteen of which I observed that Babinski's sign and the paradoxic reflex showed a tendency to replace each other; viz., when one was slight, the other was marked and *vice versa*. There was a species of antagonism between the two reflexes.

My observations at the time of the first report were purely clinical. I had no anatomic proof to offer.

In December, 1908, I made a second report to the Philadelphia Neurological Society on fifty-eight cases. The diseases in which the reflex was studied were as follows: transverse myelitis, amyotrophic lateral sclerosis, hemiplegia, Jacksonian epilepsy, disseminated sclerosis, cerebrospinal syphilis and essential epilepsy. Here again I observed the same antagonism between the Babinski and paradoxic reflexes, viz., when one was present and pronounced, the other was absent; when one was mild, the other was pronounced. The latter fact was particularly conspicuous in seven cases of transverse myelitis: at first Babinski's sign was absent on one or both sides and the paradoxic reflex was present; two or three months later Babinski's sign made its appearance, and the paradoxic reflex completely disappeared or was faintly present. In one case of cerebrospinal syphilis in which the spinal symptoms were the most conspicuous, energetic mercurial treatment was instituted. The patient's mental functions, the ataxia and sphincters improved considerably. The knee-jerks, however, remained markedly exaggerated; ankle-clonus was present on one side, but Babinski's sign disappeared on one side and was only slightly preserved on the other; the paradoxic reflex remained intact and was easily obtainable.

As another illustration of the antagonistic relationship of the two reflexes may be mentioned six cases of Jacksonian epilepsy of the same series. In all there was paresis on the same side. When the examination was made immediately or shortly after the attacks, the knee-jerk was exaggerated on the affected side and the paradoxic reflex was easily elicited in all the cases, while Babinski's sign was in three cases only slightly present and in two others absent.

An analysis of all the facts observed and followed up led me to the conclusion that the paradoxic reflex is a valuable sign for diagnostic purposes at the beginning of an organic affection showing that the motor tract is being irritated; while Babinski's sign is of inestimable value in cases of a well-established lesion of the motor tract, the paradoxic reflex is a sign, to say the least, of a transient irritation or of a slight lesion of the same tract.

* Weyl: *Handbuch der Arbeiterkrankheiten*, 1908.
1. *Am. Med.*, 1904, viii, 971.

That a transient irritation may be the cause of the reflex can be seen among other facts, especially from the following case:

CASE 1.—A lad of 12, in the course of typhoid fever, developed meningeal symptoms. He then had an attack of epileptiform convulsions on the right side which were followed by paralytic symptoms on the same side. I had an opportunity to examine him twice at an interval of two weeks. On both occasions the right knee-jerk was markedly exaggerated, there was no ankle-clonus and no Babinski sign, but the paradoxical reflex was present and easily elicited. The patient made a complete recovery; the paralytic symptoms, the increased knee-jerk and the paradoxical reflex disappeared.

My systematic study of sixty cases of parietal dementia² with reference to the reflexes of the lower extremities demonstrate the great rarity of the Babinski sign (on both sides in three cases and on one side in four cases), but the great frequency of the paradoxical reflex (in forty-two cases) in this disease. According to the pathologic reports in cases of paresis, the motor pathway is involved only to a very slight degree in the majority of instances. As Babinski's sign is usually the clinical expression of a well-defined degenerative lesion of the motor tract, the very moderate degree of pyramidal involvement will perhaps explain satisfactorily the rarity of Babinski's phenomenon in paresis. On the other hand, the presence of the paradoxical reflex in the largest number of my parietal cases finds its logical explanation in the slight degree of pyramidal involvement. This observation in paresis is therefore analogous to, if not identical with, the observation made in other organic nervous diseases, and tends to show, I believe, the correctness of my contention made in my earliest study of the significance of the paradoxical reflex.

From the clinical standpoint the diagnostic value of the paradoxical reflex appeared therefore to be established; it remained for me to corroborate it by anatomic proofs. That is to say, it was necessary to show that a real anatomic lesion was present in those cases in which during life the paradoxical reflex was the only or the most conspicuous sign and in which operative procedures were undertaken for this reason with satisfactory results so far as the finding of the lesion is concerned. Soon such opportunities presented themselves.

In 1906 and 1907, I had the opportunity to observe two cases.³

CASE 2.—In this case, in which a localized hemorrhagic pachymeningitis was found in the right motor area, the paradoxical reflex was very distinct on the left together with a slightly exaggerated knee-jerk on the same side, but there was total absence of Babinski's and Oppenheim's toe phenomena and of ankle-clonus. The paradoxical sign was the most prominent symptom. When the blood was removed by the surgeon from the right hemisphere, the patient regained consciousness (he was deeply comatose before the operation), improved considerably and on the second day the patellar tendon reflex became normal and the paradoxical sign totally disappeared. Four days later, the same phenomena returned and the patient again became stuporous; the abnormal reflexes returned. The patient died two days later. Autopsy showed a reformation of the blood clot in the right hemisphere over the Rolandic area.

CASE 3.—This concerns a man who complained of pain in the right side of the head in the same place where he received an injury six months previously. There was also marked mental hebetude and somnolence. The left knee-jerk was exaggerated and the paradoxical reflex was obtained with the greatest facility. There was no Babinski reflex, no Oppenheim sign, no ankle-clonus. The contrast between the flexion of the toes on the

right side and extension on the left on pressure of the calf muscles was most striking. Trephining of the skull with exposure of the upper part of the Rolandic area was performed. As the opening was made, the cerebral tissue bulged out; it was evidently under high tension. The surgeon immediately closed it up. Rapidly the patient regained his intelligence; the somnolence and headache totally disappeared. At the end of forty-eight hours the paradoxical reflex became weaker and on the fourth day could no more be elicited. The patient was reexamined six weeks later; the paradoxical reflex and the increase of the knee-jerk were totally absent.

These two cases with the anatomic proofs corroborated in the most forceful manner the conclusions which I reached at the beginning of my studies from purely clinical observations, to wit, that the paradoxical reflex is a sign of value in the beginning of an organic affection of the motor system or in cases of its irritation, while Babinski's sign is of inestimable value in cases with a definitely established lesion of the motor pathway.

I wish to add a third anatomic case which, like the previous two, presents an exceedingly definite illustration of the diagnostic value of the paradoxical reflex and of its practical bearing.

CASE 4.—*History*.—An Italian laborer, aged 23, of excellent health, during a quarrel sustained an injury over the left frontoparietal region at its upper level. He was struck with a piece of heavy iron. Laceration of the scalp ensued and a swelling of the parts of the size of a silver half dollar followed. There was no loss of consciousness, but mental hebetude and distinct motor aphasia developed. All the man could say was "yes" or "no." The patient came under my observation through the courtesy of Dr. I. Cortese about ten days after the trauma. On examination I found the above-mentioned hebetude and the motor aphasia. The patient could comprehend fairly well when spoken to, but was unable to articulate. Various objects were shown him and wrong names given them; when asked whether they were the proper names of the objects, he answered correctly "yes" or "no." Voluntarily, he could not articulate other words, or repeat them when spoken to him. There was no word-blindness or word-deafness.

Examination.—Physical examination showed good station and gait. The power of the right lower extremity was very slightly diminished, so slightly that the defect could be easily overlooked if the patient were not very closely examined. This was done by testing the resisting power of the thigh, leg and foot and comparing it with that of the corresponding segments on the opposite side. The right knee-jerk was also slightly more marked than the left; nevertheless the difference was distinct on repeated examinations. There was no ankle-clonus, no Babinski reflex, no Oppenheim sign. The paradoxical reflex was present on the right side in the most conspicuous manner; it could be elicited, with the greatest facility, during the entire fifteen days before the operation. The scalp presented the above-mentioned elevation over a small area at the uppermost portion of the left fronto-parietal region. The thickness of the lacerated surface prevented an exact determination of the condition of the underlying bony tissue; pressure provoked pain, however.

Treatment.—After the patient was placed in the Northwestern General Hospital his mental condition began to improve and the aphasia gradually disappeared. Both the paradoxical reflex and the slightly increased knee-jerk persisted with the greatest obstinacy and at no time could either Babinski or Oppenheim sign be elicited. The condition, in my opinion, therefore, indicated an involvement of the left motor area, probably only an irritation, but not a distinct lesion, in view of the absence of Babinski's sign. I advised an operation over the injured region of the scalp. Before operating an x-ray picture was taken, and a distinct involvement of the bony tissue was detected on the skiagraph. Dr. J. Thompson Schell performed an osteoplastic operation. The upper half of the Rolandic area, including the lacerated region, was exposed. A small fragment of the inner table of the skull was found imbedded in the dura, producing a subdural hemorrhage lying over the

2. Jour. Nerv. and Mental Dis., July, 1907.

3. Rev. Neurol., No. 22, 1906; and Am. Jour. Med. Sci., August, 1907.

uppermost portion of the left motor area. The outer surface of the skull was intact. The opening of the lacerated dura was enlarged, the coagulated blood removed and the cortex exposed. The latter's surface was found intact; no laceration could be seen.

Result.—The patient gradually improved and finally recovered. The mental hebetude completely disappeared. During the first two days the knee-jerk remained slightly exaggerated, but on the fourth day became normal. On the sixth day, the paradoxical reflex became fainter and on the twelfth day after the operation it could no more be elicited. It was interesting to observe its gradual diminution and disappearance and it was also interesting to notice its conspicuous presence on the morning before the operation and its gradual obliteration after the operation. The patient was subsequently seen by me several times during a period of two months and at no time could I detect the presence of an abnormal knee-jerk or of the paradoxical reflex.

This case, I believe, is the most striking illustration of my contention concerning the practical value of the reflex. Added to the first two cases published in 1906 and 1907,³ it furnishes decisive anatomic proofs for the *a priori* deductions made from clinical observations. The reflex has its diagnostic value and deserves a place in nosology of the nervous system.

In conclusion, I may mention that a systematic investigation was made by me in 800 individuals free from diseases of the nervous system, also in 250 cases of functional nervous diseases and at no time was extension of the toes elicited by the method employed by me for the detection of the paradoxical reflex.

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THE AUTOLYSIS OF THE CRYSTALLINE LENS

A PRELIMINARY REPORT

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In the study of traumatic cataract and the needling operation, I was struck with the very scant description of the manner in which the lens becomes opaque and the causes for its opacity and final absorption.

Parsons¹ states that after exposure of the lens fibers to the action of the aqueous they swell up, become opaque, protrude through the capsular wound, and finally break up in the usual manner, absorption being largely due to the leukocytes, which become swollen and filled with granules.

Schlösser² (1887) in an article on traumatic cataract, and Schirmer³ (1889) have studied the microscopic changes very thoroughly, but without throwing much light on the cause of these changes.

Fuchs⁴ states that the fibers swell up and become opaque through absorption of water; some are broken off and drop into the anterior chamber and are absorbed. But he also says that concussion without rupture of the capsule may cause the development of complete opacity of the lens. He, however, fails to state just why this occurs.

De Schweinitz⁵ says that the opacity and liquefaction are explained by the action of the sodium chlorid of the aqueous on the globulin of the lens substance, as globulin is normally soluble in weak sodium chlorid solution.

This theory was, I think, first suggested by Marcus Gunn in 1899 in the *Ophthalmic Review*.

In further search of the literature on the subject I have found no elaboration.

In order to understand the subject it is necessary to have a knowledge of the chemistry of both the aqueous and the crystalline lens.

Halliburton gives the composition of calves' aqueous as in Table 1.

TABLE 1.—COMPOSITION OF THE AQUEOUS

Water	986.87
Solids	13.13
Albumin	1.12
Extractives	4.21
Inorganic salts	7.70
Sodium chlorid	6.89

Thus it will be seen that it consists practically of normal saline solution with a trace of albumin and extractives.

The composition of the lens as given by Neumeister is set forth in Table 2.

TABLE 2.—COMPOSITION OF THE LENS

	Per cent.
Water	63.50
Solids	36.50
Albumins	35.00
Insoluble albuminoids	17.00
Crystalline (b)	11.00
Crystalline (a)	6.80
Albumin	0.20
Fats	.29
Lecithin	.23
Cholesterol	.22
Salts	.80

It will be seen that the lens contains 35 per cent. of protein, the highest protein percentage of any tissue of the body. Of this 35 per cent. protein, 17 per cent. is in the form of an insoluble albuminoid which is insoluble in normal saline or water.

It is a true albumin and constitutes most of the lens fibers, its amount increasing from without inward in accordance with the increasing age of the lens fibers. The remaining protein consists of a small amount of serum albumin and two vitellins which are soluble in dilute saline or in water.

If these analyses are correct, we have great difficulties in explaining the foregoing theories.

I can hardly agree with Parsons' statement that most of these fibers are carried off by the leukocytes. In the first place, if there is no infection there should not be many polymorphonuclear leukocytes about, as they occur chiefly in inflammatory troubles; and secondly, it would not explain the traumatic cataract with absorption in those cases of concussion without rupture of the capsule.

Fuchs seems to keep on the safe side by simply stating that the fibers become opaque and are finally absorbed, and by giving no explanation for the cause of the concussion cataract.

De Schweinitz's promulgation of Marcus Gunn's suggestion is, if the chemistry be correct, entirely untenable. To be sure the (a) and (b) crystalline bodies would be dissolved out either by water or the aqueous, but the insoluble albuminoid which makes up the greater portion of the protein, and which occurs chiefly in the lens fibers, is insoluble in either the water or the saline.

It is easily seen from a study of the chemistry of the lens why it becomes opaque either after injury with the entrance of aqueous or even from concussion. In case of rupture of the capsule and entrance of the aqueous there would occur an immediate precipitation of this insoluble albuminoid, while the remaining portions

1. Parsons: The Pathology of the Eye, 1904.

2. Schlösser: Experimental Studies on Traumatic Cataract, 1887.

3. Schirmer: Arch. f. Ophth., 1889.

4. Fuchs: Text-Book of Ophthalmology, 1908.

5. De Schweinitz: Diseases of the Eye, 1910.