

THE DECAPSULATION OF THE KIDNEY, WITH REFERENCE TO THE CONCOMITANT INTRA-OCULAR COMPLICATIONS IN THE CHRONIC FORMS NEPHRITIS.*

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The object of this paper is to elicit the interest of the members of this Section in determining whether or not the decapsulation of the kidney can be accepted as a curative or as a palliative measure for the various intra-ocular complications accompanying either the diffuse, interstitial or chronic parenchymatous nephritis. The question of decapsulating the kidney¹ still attracts considerable attention,² not only from the standpoint of the surgeon but from that of the internist as well. The writer is well aware of the fact that it is rather early to make any definite statements or draw any definite conclusions as to the merits or demerits of the operation from an ophthalmologic point of view.³ But, if the paper should arouse enough interest in the matter so that before long we might have either a symposium or a more extended report on this subject, as is our wont in such matters, we would be in a position to decide a very important question which would be infinite in its reachings. In this particular, it is only necessary to call attention to the tremendous death rate in those forms of subacute or chronic nephritis associated with deep-seated fundus lesions, especially albuminuric retinitis.

The surgery on encapsulated organs, such as the kidney, liver and spleen, is of recent origin. The pioneers in this field are Reginald Harrison of England, Israel of Berlin, Pean and Pousson of France, Ferguson of Chicago, and Edebohls of New York. It is to be hoped, however, that with reference to the decapsulation of the kidney it will not be as it was with the splitting of the capsule of Glisson, namely, a nice experimental operation but of no practical value, as its results are not definite.

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1. Following is the main surgical literature on decapsulation of the kidneys. There are included references to two articles (by Ferguson and Whitacre), which have appeared in THE JOURNAL since the reading of the paper. Edebohls, G. M.: "Renal Decapsulation for Chronic Bright's Disease," N. Y. Med. Record, March 28, 1903; also "Question of Priority in the Surgical Treatment of Chronic Bright's Disease," N. Y. Med. Record, April 26, 1902, p. 651; also, "The Cure of Chronic Bright's Disease by Operation," N. Y. Med. Record, Dec. 21, 1901, p. 961; see also Brit. Med. Jour., Nov. 8, 1902, p. 1507. Ferguson: THE JOURNAL A. M. A., July 4, 1903. Whitacre: THE JOURNAL A. M. A., May 23, 1903. Primrose, A.: "The Operative Treatment of Chronic Bright's Disease," Canadian Jour. of Med. and Surg., 1902, xi, p. 143. Harrison, R.: London Lancet, Jan. 4, 1896, p. 18; also British Med. Jour., Oct. 19, 1901, ii, p. 1125. Rovsing, T.: "Operative Treatment of Chronic Nephritis," Mittheilungen a. d. Grenzgebieten d. Med. u. Chirur., 1902, vol. x, p. 288. Gultaras, R.: "The Surgical Treatment of Bright's Disease," N. Y. Med. Jour., 1902, lxxv, p. 847. Lyman, C. B.: "The Surgical Treatment of Chronic Bright's Disease," THE JOURNAL A. M. A., 1902, xxxviii, p. 1030. Pousson, A.: "De l'Intervention Chirurgicale dans les Nephrites Médicales," Annales d. Maladies d. Organes Genitourinaires, 1902, xx, pp. 513, 641 and 832. Israel: Deutsche med. Woch., Feb. 2, 1902. Gradle, H.: "Neuroretinitis Due to Disease of the Kidney," Chicago Med. Recorder, Nov. 15, 1902, p. 321.

2. About 200 letters were sent out by the writer to men of repute, both surgeons and ophthalmologists, in various sections of the country in reference to this question.

3. This is, perhaps, the first attempt that ever has been made to gather data and discuss this question from the standpoint of the ocular involvement in chronic nephritis and its relation to decapsulation.

It was Edebohls⁴ of New York who first split the renal capsule with the avowed intention of curing a case of chronic interstitial nephritis. Since then, a number of surgeons have performed the operation. However, no mention at all was made in most instances of the condition of the eye at the time of the operation, nor, for that matter, at a subsequent time. It is sad, but nevertheless true, that the general surgeon pays very little attention to the eye when it comes to operations on the kidney. This was a pertinent point brought out by an extensive correspondence on this very subject in order to gather data for this paper.²

The very fact that we, as ophthalmologists, are often the first to apprise the patient of any kidney lesion makes the subject of great interest to us. And, if the future will soundly establish the value of kidney decapsulation, it is our duty to follow up such cases now, with or without eye lesions, as submit to the operation.

True it is that we do not at present definitely know how the operation accomplishes the good it does,⁵ even in those cases without deep-seated fundus lesions. That is to say, the pathologists have been unable to give any definite data, and experimental work⁶ along this line does not seem to offer a very satisfactory solution.

The deep-seated fundus lesions usually accompanying an interstitial, parenchymatous, or diffuse nephritis of the chronic variety, are about the same in character. We know these to be a simple retinitis, neuroretinitis, optic neuritis (all with or without retinal hemorrhages), and some form of choroiditis. From the fundus alone we can not determine what form of kidney lesion is present. But when a retinal lesion is present we know that it materially adds to the gravity of the prognosis. The severity or extent of the fundus lesion in a measure indicates the amount of kidney and vascular involvement. The reverse of this statement is also a fact, in a limited degree.

The frequency with which the fundus is implicated in the various forms of chronic nephritis is startling. According to Lécorsche 20 per cent., according to Galezowski 33 per cent., and according to the writer at least 30 per cent. of all chronic Bright's disease patients present some fundus complication. It is not by any means such a

4. This was on Jan. 10, 1898. The first operation and the five preceding it which led up to the question of decapsulation are reported in the Medical News, April 22, 1899.

5. Harrison attributes the results to the relief of tension. Edebohls and others add to the relief of tension the establishment of a more extended vascular formation with a greater anastomosis. Ries, Bernays and others do not place any weight on the new vascular formation. Zeit of Chicago says the kidney regenerates function quite rapidly if tension is removed. Ries made sections of two kidneys and found no increased blood supply. Israel says concerning the mechanism of the operation that an inflamed tissue may be made to retrogress toward the point of health by splitting the affected structures. According to him, new venous anastomoses are formed between kidney and capsule.

In the eye, the operation causes a decrease in the amount of albumin in the lymph channels, thus reducing the liability of deep ocular complications in consequence of lymph and vascular disturbances. This idea is advanced by Oliver and is, in a measure, supported by the writer.

Perchance the free circulating albumin takes on a certain toxic tendency and in this way excites inflammatory reactions in the retina or other eye structures.

6. Albarran and Bernard found in rabbits a rapid regeneration after the splitting of the capsule. Within two weeks it was reformed and within two months as dense as the normal capsule and not as vascular. The cortex showed superficial lesions, not very extensive. See also La Semaine Médicale, 1902, vol. xxii, p. 215.

Tait of San Francisco, on everting the kidney from the capsule in numerous rabbits, found a dense adherent fibrous mass surrounding the kidney within two months afterward. Johnson, before the California Academy of Medicine and Surgery, in November, 1902, reported a series of experiments in corroboration of the work of Albarran and Bernard.

very doubtful statement that every one with a chronic nephritis will probably have some fundus implication before he dies (provided the nephritis carries him off). For we know that the fundus may be involved and yet the patient not be aware of it. There is no stated time during the nephritis for the retinal or other fundus lesion to appear. At times these lesions are the initial symptoms; again, they appear very late or very early in the attack of nephritis. And we are aware that frequently the very first diagnosis is made by an ophthalmoscopic examination. So much importance ought we to attach to this statement that a careful examination of the kidney should be made whenever a retinal lesion of an inflammatory nature is detected. This is also true with reference to the acute forms of nephritis on behalf of the retina.

The death rate for nephritis with fundus complications, especially retinitis, is exceedingly high. The majority of statistics of this kind were taken without reference to the fact whether the kidney lesion was unilateral or bilateral.⁷ That we can have a one-sided chronic parenchymatous, interstitial or diffuse nephritis for quite a period of time is to-day a well-recognized and accepted clinical fact. We also know that a small portion of healthy kidney can meet the demands of the economy, and that two kidneys are not necessary to maintain life. Clinicians now tell us that a part or the whole of one kidney may be involved in the aforementioned diseases. True, if one kidney is affected the other is liable to become involved, as the same etiologic factor is present. Often one kidney is rather extensively involved before the other is implicated. However, should only one kidney remain involved, the prognosis as regards life is very favorable, other things being equal. These cases, however, are very rare.

The age of the patient is an important factor as regards the prognosis. Whether a unilateral or bilateral nephritis, the death rate rapidly increases in an inverse proportion to the age, i. e., in the twenties and thirties the death rate is considerably higher than in the sixties or seventies. Again, the mortality is greater in hospital than in private practice.⁸

The fundus complication, too, may be unilateral or bilateral.⁹ However, the great majority are bilateral, just as perhaps the kidney lesions are more often bilateral. The fundus lesion serves as an indicator as to the extent of kidney involvement; it also serves the same as regards

the prognosis. In this particular it matters little whether the fundus lesion is unilateral or bilateral, though, naturally, the bilateral involvement is the graver. Hence it may be that those with retinal complications who outlive the classical two-year period have had but one kidney involved at the time, or one may have been considerably less involved. Nevertheless, postmortem examinations show ultimately an usual bilateral nephritis and retinitis. The nephritis may exist for some time before albumin is observed in the urine, and in these cases the retinitis is often the first observable diagnostic lesion.¹⁰ The retinal lesion may also be late in appearing, and then, as a rule, the disease makes rapid strides.

It is yet an open question in what manner the fundus and kidney lesions are associated. Traube says it depends on excessively increased arterial tension, yet in many cases there is no cardiac hypertrophy. Weeks and Samelsohn attribute it to a general vascular—both arterial and venous—disturbance which takes place in both organs at the same time. Cardiac hypertrophy¹¹ is an important factor in the production of the retinal hemorrhages. The theory of auto-intoxication is not to be lightly considered in this particular. Furthermore, we may have a chronic Bright's disease without retinal complications for some time, on top of which an acute attack supervenes with rapid retinal involvement.

We are now in a position to review the results of decapsulation of the kidney in this class of cases. The forecasted conclusions to be drawn from these cases are, to say the least, not encouraging. And, what is more, the cases strikingly illustrate that as yet we have found no cure or even a wholesome palliative measure for any form of chronic Bright's disease with retinal complications.

There are 15 cases, and all except two died in consequence of the nephritis within the two-year period. One of them died from tubercular peritonitis. Of the two still living, the history of one case (No. 14) is very questionable. The other is losing ground.¹² Between the ages of 20 and 40 there were six cases; between 50 and 60 five cases; between 60 and 70 two cases. The age was not given in two. These cases were nearly all in hospital practice and gave a higher death rate than the well-known Belt and Bull¹³ statistics (85 per cent. for the first year and 93 per cent. of the remainder for the second year).

It is not necessary to give the detailed history of any of the cases, as they are all about the same as in everyone's practice. In Gradle's case (operated on by Ries) the improvement in the vision was simply remarkable, from 5/200 to 20/90. In the writer's case (seen in consultation with Ries and operated on by him) the vision rose from 20/60 to 20/20 minus and remained so until death. The same in this particular is true of Gradle's case. In Ochsner's case the vision was improved after the first operation. In these 3 cases, which the writer had the privilege of examining, the retinal condition improved remarkably after the operation. In one case (No. 6)

7. In order to determine whether one or both kidneys are affected, a urethral catheter or a Harris' segregator must be employed. These methods of examination have been in vogue but a short time. No statistics of albuminuric fundus complications, as regards the mortality rate, have been compiled since. Radiography may, in some instances, determine which kidney is involved, but this method is not very reliable.

8. Bull, C. S. (Trans. Amer. Ophthal. Soc., 1886), cites a series of 103 cases, 87 per cent. dying within two years; 6 died in the third year, 4 died in the fourth year and one in the sixth year.

Miles, Miley ("The Prognosis of Neuroretinitis in Bright's Disease," Ophth. Soc. U. K., 1888), in a series of 51 cases in which the retinal lesion was determined early, 53 per cent. died within six months.

Haehnle, E.: ("Ueber die Lebensdauer der an Retinitis Albuminurica Erkrankten," Diss. Tuebingen, 1897), found that in a series of 98 cases 56 per cent. died the first year and 68 per cent in the two years. For further statistics see the following: Possanner: "Ueber d. Lebensdauer n. d. Auftreten v. Retinitis Albuminurica," Deutschmann's Beitrage z. Augenheilk., xv, s. 22, 1894.

9. Cases where one fundus only was implicated for a long time are cited by: Leber: Handb. d. ges. Augenheilk. (Graefe-Saemisch), 1 Aufl., s. 583. Eales, H.: Brit. Med. Jour., 1884, vol. ii, p. 1248. Bull, C. S.: Loc. cit. Weeks, J.: Arch. f. Augenheilk., xxi, p. 54 (Pathology of Ret. Albuminurica). Webster, D.: N. Y. Polyclinic, 1894, iii, p. 161. Werhli: Arch. f. Augenheilk., xxxvii, p. 173, 1898. Moore, O.: N. Y. Med. Jour., April 17, 1886.

10. Hirschberg, Abadie, Gand and Eyre speak of a retinitis before albumin appears in the urine as "prealbuminuric retinitis." (Quoted by Groenouw and Uthoff in Graefe-Saemisch Handb. d. ges. Augenheilk., 2d Ed., vol. xi, chap. xxii, p. 96.)

11. Bull, C. S., found, in a series of 18 cases, 12 with cardiac hypertrophy which had retinal hemorrhages (Amer. Jour. Med. Sc., 1873, p. 37). Litten has made the same observations as Bull. The writer has about the same experience.

12. Reported to have died in June, 1903.

13. The combined number of cases in the Belt and Bull statistics is 258. Of these 153 died within the first year, 88 within the second year, leaving only 17 cases to live longer.

before operation, the neuritis was so marked that the disc could scarcely be outlined, while after the operation it was plainly outlined. In the cases operated on by Ries and Ochsner there were no degenerative changes present in the retina. Whether this was so in the other cases could not be ascertained. However, slight degenerative changes did develop after the operation in Cases 6

gorged and the arteries very much attenuated. The retina was spattered with small flame-shaped hemorrhages. The macular region was fairly free. The optic disc was very imperfectly outlined, due to the neuritis and the swelling; it equaled about 2 mm. Both eyes were about alike as to the changes present. V.=20/60 in each eye March 29, 1902. The field of vision on this day concentrically contracted as seen in the chart (Fig. 1).

STATISTICS OF CASES OF DECAPSULATION OF THE KIDNEYS.

Number.	Age, Sex.	Date of Diagnosis.	Kidney Lesion, Single or Bilateral.	Fundus Lesions.	Date of Last Operation.	Vascular Condition and Heart.	Immediate Result.	Final Result.	REMARKS.	Surgeons.
1	M., 67	June, 1901	Bilateral interstitial.	Retinitis albuminurica.	6, 14, '02..	Mitral regurg., arteriosclerosis.	Died 48 hours later.	Fatal	Cause of death, uremia. Edebohls' case No. 39.	Edebohls
2	M., 62	Jan., 1902	As above . .	Ibidem	5, 26, '02..	Aortic murmur and mod' rate hyper.	Died within 12 hours.	Fatal	Acute dilatation of heart the cause of death. Case No. 34.	"
3	M., 56	May, 1901	As above . .	Neuroretinitis	5, 26, '02..	Cardiac hyper. and arteriosclerosis.	Improvement.	Died on Nov. 6, '02; lived 5½ months.	Both the eyes and kidneys were markedly improved; relapse set in. Case No. 35.	"
4	M., 21	Dec., 1900	Bilateral diffuse.	Retinitis albuminurica. V=1/200 in each eye.	4, 16, '02..	Cardiac hyper. and aortic stenosis.	Vision markedly improved, kidney also.	Died 1, 18, '03	Improved for 5 mos. especially, lived 9 mos. after operation. Case No. 27.	"
5	M., 50	Apr., 1901	Bilateral interstitial.	Optic neuritis, temporary blindness.	5, 26, '02..	Heart on verge of dilatation, hyper. also.	Died within 12 hours.	Fatal	Very desperate case—paralysis. (?) Case No. 36.	"
6	F., 22	Mar., 1901	As above . .	Neuroretinitis, hemorrhages. V=20/60.—	3, 30, '02..	Hypertrophy, cardiac.	Both organs improved and remained imp.	Died 8, 10, '03, tub. perito's, lived about 3 months.	Albumin disappeared entirely for a time, vision rose to normal, field enlarged, kidneys operated on at two sittings; albumin returned later on.	Ries & Suker, Chicago.
7	M., 36	Aug., 1902	As above . .	Neuroretinitis and hemor. V=5/200.	9, 1, '02..	Cardiac hypertrophy.	Marked improvement in eyes and kidney. V=20/90.	Died Dec., '02, lived about 4 months.	Cause of death uremia. (?) Dr. Gradle said field enlarged remarkably, so did the acuity. Segregator used in Nos. 6 and 7.	Ries and Gradle, Chicago.
8	F., 30	Apr., 1900	Bilateral interstitial.	Neuroretinitis more marked in one.	Oct., 1902.	Hypertrophic heart.	Eyes did not change, rather grew worse.	Died Jan., '03..	In this case a choked disc developed after first operation, vision decreased markedly.	O. Dodd and Ochsner, Chicago.
9	(?)	Prior to 4, 10, '02	Bilateral interstitial.	Neuroretinitis	About 4, 10, '02.	Not given. . .	Improvement.	Still living, 4, 22, '03.	General condition improved, recurrent hemorrhage into retina and more mosaics.	C. A. Oliver, Philadelphia
10	M., 50	Prior to Apr., 1902	Bilateral interstitial.	Albuminuric retinitis.	April, 1902.	Not given. . .	Eyes not improved much.	Lived 7½ mos. 11, 1, '02.	Was a desperate case from the beginning.	A. C. Bernays, St. Louis.
11	M., 20	About 1 year ago	Bilateral interstitial.	Mild optic neuritis with few hemorrhages.	11, 10, '02.	Cardiac involvement.	Temporary improvement.	Died 11, 17, '02.	Dr. O. performed a decapsulation on one kidney prior to 11, 10, '02; died of acute suppression of urine and uremia.	Ochsner and Suker, Chicago
12	(?)	As above	As above	Album. ret., glistening spots (not characteristic), V reduced.	As above	Cardiac hypertrophy. (?)	Died within a few days (4 days).	Fatal	Was a desperate case, almost moribund when brought to hospital.	Munro, Boston.
13	M., 29	Jan. 15, 1903.	As above . .	Silvery spots in macula, light streaks along arteries, V poor.	1, 27, '03..	Not given. (?)	Improved for a time.	Losing ground rapidly. Died in June 1903.	General condition improved some but eye symptoms not as a subsequent examination showed.	Munro.
14	M., 51	1885(?)	Bilat. chron. diffuse.	Failing eyesight, ret. album. (?)	6, 2, '02..	Varying am'ts of edema.	Good	Improved, living, 1, 19, '03, eyes no report.	This is Dr. Edebohls' case No. 37. (If this is a true case of retinitis albuminurica of so many years standing, it is one of the wonders.—G. F. S.)	Edebohls.
15	M., 23	Dec. 21, 1901.	Bilateral interstitial.	Sudden blindness, Dec., '01	4, , '92..	Mitral regurg., hypertrophy heart.	Not good; lived 8 days.	Died 4, 19, '02..	Died of uremia and exhaustion. Case No. 24.	Edebohls

1. The record "fatal" means that the patient died within 8 to 72 hours after the operation. The majority were bilateral decapsulations. Ries and Ochsner operated on one kidney and later on the other. Edebohls usually decapsulated both kidneys at one sitting.

2. C. A. Oliver is conducting a series of experiments to determine the effects of the operation on the amount of albumin in the aqueous humor.

3. Cases Nos. 6 and 14 can be excluded in drawing definite conclusions, because No. 6 died of an intercurrent disease and No. 14 has lived so long with a supposed albuminuric retinitis that perhaps the retinal trouble is of a different nature (advisedly stated).

4. The visual fields were taken in Ries and Ochsner's cases. In all (3 cases) it increased after operation.

5. The writer had the privilege of examining and watching the cases of Ries, Gradle and Ochsner, both before and after operation. These were typical cases in every particular, and therefore of great value in this statistic.

6. Some cases had blood casts, most of them presented hyaline and granular casts, a few had erythrocytes in the urine.

7. Ries has made many microscopic sections of the kidney after operation, but failed to find increased vascular anastomosis. Zeit, ibidem (personal communication).

8. The operation did not prolong life. The medical treatment offers a better percentage as far as prolonging life is concerned.

9. Since reading this paper the writer has obtained 4 more cases which were typical and submitted to the operation; they died within a few months thereafter.

10. Excluding the two cases, Nos. 6 and 14, the mortality rate is 99 per cent. What a showing in 17 cases!

and 7, as is usual in well-marked cases of this kind. The increase in the field of vision in Case 6 is not very marked, yet it shows a decided improvement (see Fig. 1).

NARRATION OF CASE.

Mrs. L. (operated on by Dr. Ries, Chicago).

Diagnosis.—Neuroretinitis albuminurica; chronic interstitial nephritis with rather marked cardiovascular implications.

Fundus Findings.—Disseminated albuminuric spots of the inflammatory type throughout the retina; the veins were en-

April 23, 1902 (not quite four weeks after the capsulotomy) the nerve head could readily be outlined. The retinal hemorrhages had disappeared and the whole retina was greatly improved. The field of vision was materially enlarged (Fig. 1) and the acuity was: O.D.V. 20/40+3. O.S.V. 20/40+2.

At time of death (August, 1902) she was reported to have had practically normal vision. The cause of death was peritonitis. (For further particulars as regards the disappear-

ance of albumin and its return after the operation in this case see reports of Ries.)

The period of time that is regarded necessary to pass before the operation is considered as having produced a cure is six months, according to Edebohls. At the end of this time the urine must be free from albumin and remain so. But few of the cases with retinal complications outlived this period. The majority improved to some extent, both as to vision and urine, still the relapse came on rapidly in both organs and the patients died. True, in a few instances the improvement of the eye condition was almost miraculous. We know that even with the medicinal treatment marked temporary improvements take place and may even continue for some time. All the cases with eye symptoms, except one, died within the first year. The greater share, if not all, had a bilateral kidney and eye lesion and perhaps always bilateral at death. The majority gave decided evidences of cardiac involvement. The kidney lesion in each instance was a chronic interstitial or diffuse nephritis, or an acute attack on top of a chronic condition. The successes so far reported for decapsulation were for the

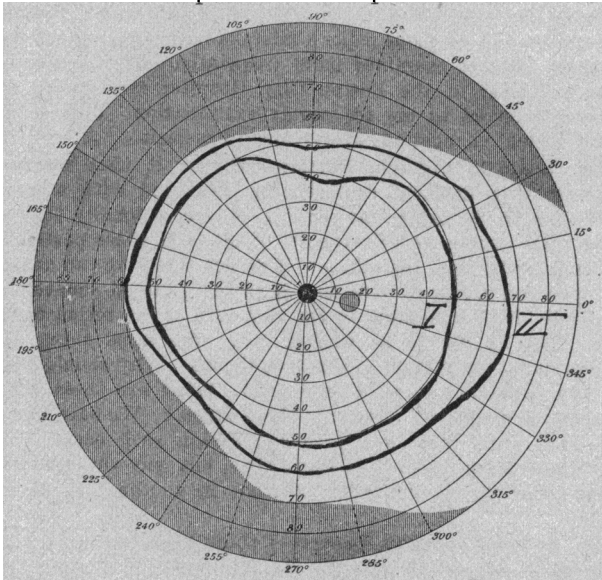


Fig. 1.—Mrs. L. I. Visual field on March 29, 1902, two days prior to the operation. II. Visual field April 23, 1902, about four weeks after the operation. The left field is practically the same as right. No scotomata in either field.

uncomplicated chronic nephritis. Whether or not the operation in any way hastened the exitus in the cases reported here is certainly very problematical, for they nearly all came, as it were, under the "two-year clause." The operation, in competent hands, is practically devoid of any dangers (kidney and life).

The mere fact that the general condition of the patient improved slightly after the operation does not establish its validity. For we know that often in not doing anything, excepting advising hygiene, marked improvements both as to sight and kidney take place.

If we could have an interstitial nephritis definitely limited to one kidney, though with more or less fundus involvement, the operation might, if performed early, be of great benefit. This can be partially supported by the fact that some uncomplicated cases of nephritis have survived the operation for a period of 3 to 10 years (see Edebohls). Knowing that the retinal manifestation indicates a very firm establishment of the nephritis and that a serious cardiac involvement is almost a constant factor, it does seem that the operation offers no

ray of hope, either as a cure or as a permanent amelioration. The statistics pertinently show this point to be true. As fundus complications are so frequent in interstitial nephritis, it may be asked whether kidney decapsulation will prevent them if done very early. This is problematical. The exact limits beyond which the operation in any case, simple or complicated, will or will not fail to accomplish any good has not been determined. As far as albuminuric retinitis is concerned, all that can be said is that the results have been decidedly temporary, and this in nearly every instance.

In conclusion, we may recapitulate as follows:

1. The operation of decapsulation so far is not warranted in all instances, nor does it offer any hope in cases of bilateral chronic nephritis with retinal complications.
2. Though there is an improvement in the eye ground directly after the operation, still it is decidedly temporary, judging from the present statistics.
3. The operation may be of service in unilateral nephritis with fundus lesions in the initiatory stages. These cases, however, are rare, and, if found, soon become bilateral. Therefore, these latter cases would come under the first remark.
4. The mortality rate for albuminuric retinitis in chronic nephritis has not been lessened by the operation.
5. The statistics show that retinal complications are a decided index as to the severity of the kidney involvement.
6. In nearly all the cases of nephritis chronica the cardiac involvement is present and is an important prognostic factor. With the retinal lesion, it is usually present.
7. Unilateral retinal lesions are the exception in chronic nephritis.
8. As yet the medical treatment of all these cases yields as good results, if not a trifle better, than the kidney decapsulation.
9. Statistics of the mortality rate of albuminuric retinitis based on urethral catheterization are indicated. This is to determine whether those that outlive the two-year period might be cases of unilateral nephritis or are late in becoming bilateral.

The writer wishes to take this opportunity to express his gratitude to the various gentlemen who answered the circular letter in reference to this subject. In particular does he wish to acknowledge the courtesies of Drs. Ries, Gradle, Ochsner and Dodd of Chicago, Oliver of Philadelphia, Munro of Boston, Bernays of St. Louis, and Edebohls of New York.

DISCUSSION

ON PAPERS OF DRs. STRICKER AND SUKER.*

DR. HENRY DICKSON BRUNS, New Orleans—Dr. Stricker is entirely right in holding that the proportion of urea excreted in the urine is of far greater significance than the presence in the same fluid of more or less albumin. The general practitioner was somewhat slow in recognizing this truth and making practical use of it; it is not to be wondered at, therefore, that the oculist should have been still slower. To-day, however, I believe that all physicians of the first rank accept and act on it in the study and treatment of their cases of renal disease. They all, too, hold to the belief that it is the retention of the excrementitious products, of which the urea in the urine is the expression, that produces the lesions found during disease of the kidneys; the escape of albumin into the urine being a symptom of more or less importance according to the presence or absence of a host of other phenomena. Nor

* The paper of Dr. Stricker appeared in *THE JOURNAL* February 20, p. 514.

have the ophthalmologists followed far behind, as the passage cited by Dr. Stricker from Professor Leber, published twenty-six years ago and containing a citation from Von Graefe, plainly shows. Dr. de Schweinitz,¹ quoting J. B. Lawford, says: "Uremic amaurosis may occur in any of the various forms of renal disease in which there is sufficient interference with the function of the kidneys to load the blood with those poisonous materials, whatever their exact nature may be, which induce the uremic condition." And, again, that retinitis, retinal hemorrhages, etc., may precede albuminuria by a considerable time. It is probable, as Dr. Sutton suggests, that these are examples of granular kidney, in which albumin is absent, or only present to a very small extent until a later stage of the kidney affection. On the other hand, the lesions which I have described as occurring in the blood vessels, particularly various types of perivasculitis, and of "silver-wire" arteries, producing compression on underlying and overlying veins, are often present for long periods before the renal lesions are manifest, or at least before the other signs of renal disease are fully established; that is, albumin and casts. These expressions certainly indicate a belief that the excrementitious products are the injurious agents. So in a paper by Dr. S. C. Ayers² we read: "The question of the diminished excretion of urea is not a new one. Dr. S. Marx,³ in a very interesting paper, states that this condition arises not from albumin, but from diminished excretion of urea. He states that many women with grave nephritic disease go to full term with little or no discomfort. He further states that albumin and casts have no meaning for him in the presence of a normal urea excretion, for with that he is in a position to know that sufficient of the effete products are being excreted and the condition of the safety valve in the engine of the body is evidenced by the behavior of this element. Whether this urea is really the offending agent or not, modern investigation does not allow us to state with any degree of positiveness. It is enough to this end to state that when there is a lessened secretion the patient suffers, and when hypersecretion occurs the patient is freed from those symptoms to us indicative of toxemia. Dr. E. E. Morse of Washington, D. C., endorses the theory of Dr. Marx. Dr. Stricker, then, deserves our thanks for bringing this subject to our attention at this meeting and emphasizing the necessity of observing whether or not the vision and other ocular symptoms vary with the fluctuations of the amount of urea excreted. His position as to the indications in the albuminuria of pregnancy seems eminently correct, but one must not be too sure that in every such case "the kidney disease will subside quickly after labor is terminated." I once saw a case where albuminuria had been recognized only after the pregnancy had begun. Shortly after the birth of her child the patient became quite blind, and I was called in. The eyes showed typical Bright's changes. The patient did well for a few days, but then fell into convulsions and coma, and died. The fact that vision may improve even to the normal in any form of Bright's retinitis, although there may be little, if any, change in the ophthalmoscopic appearances, may now be taken as established. A case (No. 11) reported by Dr. C. S. Ayers² would go to prove that even extensive degenerative changes may clear up, though this is unusual, if time enough be afforded. In Dr. Ayer's case the patient had had nine pregnancies. In the fourth, fifth, sixth, seventh and ninth there had been failure of vision. Premature delivery occurred in the seventh month of the ninth pregnancy, and her eyes were examined some time after; the right equaled 0.1 and the left equaled 0.7. There were characteristic alterations in each eye and numerous white patches in the retina of the left. Twelve years afterward he found the right eye perfect; L. E. V. equaled 20/xl, but with +0.75 cyl. ax. 110 degrees it equaled 1. The retina looked normal and healthy, and there was no trace of patches. While there is no etiologic

connection established between senile cataract and albuminuria in premature cataract—cataract occurring in middle life—the urine should always be examined. Finally, perhaps, I may be permitted to say that while pathologist of the Charity Hospital, as many as ten years ago, the fallacious nature of albuminuria as an evidence of renal disease was forced on my attention by the not infrequent finding of casts in urine showing freedom from albumin by the most delicate tests. In a series⁴ of 2,441 first examinations, casts without albumin were found 40 times—about once in 61 times. The kinds of casts were: Epithelial in 1 instance, granular in 4, hyaline in 14 and unrecorded in 21 instances.

DR. ALLEN GREENWOOD, Boston—A large number of these cases should be considered from the standpoint of arterial degeneration, rather than from the standpoint of kidney disease. I classify my cases in two groups: one, the aged people with general arterial degeneration, in whom you find degeneration of the arteries of the retina and choroid sometimes before there is evidence of kidney lesion; and the other, in the young and middle-aged, where you have distinct chronic parenchymatous nephritis with retinal changes and death in a few years. In the last twelve years I have seen 26 cases in private practice, and have followed all but 2 to the ultimate end. Out of the 26 cases only 3 are living, and none of the 3 living was seen prior to two and a half years ago. I think physicians and surgeons make a mistake in not having the eyes of their patients examined more frequently. All the cases of kidney disease at the Boston City Hospital are, during my service, referred to me for ocular examination. Just before death retinal changes are nearly always present.

DR. LEARTUS CONNOR, Detroit—Why is it that we have patients with deficient excretion of urea, and with albumin and casts in the urine, but yet it is only occasionally that we have these ocular complications? What is in these particular cases different from the others? I do not think we can answer the question except by referring it to the personal equation of the individual, something in the structure of the retina of the one. This is yet to be worked out. The same thing confronts us in many other morbid conditions. Why should there be this difference in urea excretion? What are these excrementitious products? What are these toxins? We have known for some time that in a pregnant person such ocular complications mean death in a short time, but why the complications occur in one individual and not in another, we do not know.

DR. EDWARD JACKSON, Denver—I think there can be no question as to agreement with Dr. Stricker that not albumin, but deficient elimination of urea, is the factor of importance. My experience would point out that why one patient has retinal lesions and another has not depends on the condition of the general vascular system. I have never seen a typical case of albuminuric retinitis without involvement of the vessels generally. But sometimes where I have found slight lesions of the disc and retina and decided changes in the vessels, careful and repeated examination has failed to show any changes in the urine. The retinal lesions are associated with vascular changes. This leaves a small loophole for the surgeons who urge decapsulation. The cases that present retinal lesions are evidently not saved, so far as our statistics indicate; but possibly there is a class of cases of chronic nephritis that do not have retinal lesions and are not liable to have them, that may be helped by the operation. But so far as those cases are concerned about which we can form a prognosis, his paper indicates that they are not permanently helped.

DR. G. E. DE SCHWEINITZ, Philadelphia—While it is true, in general terms, that ocular lesions may occur with any form of nephritis, the various types of retinitis are most frequently associated with chronic interstitial nephritis (that is, with renal sclerosis), whether this is an independent primary affection, or whether it is a sequel of arteriosclerosis. It seems to me undoubtedly true, as Samuel West has recently insisted, that a sharp distinction should be made between the degenera-

4. "Notes on Examination of the Urine," by H. D. Bruns, M.D., Med. News, Sept. 7, 1895.

1. "Ocular Manifestations in Chronic Bright's Disease," Proceedings of Phil. Co. Med. Soc., 1902.

2. Cincinnati Acad. of Med., April 14, 1902; Amer. Jour. Ophth., October, 1902.

3. Med. Record, April 20, 1901.

tive and exudative forms of albuminuric retinitis. The former indicate the presence of chronic granular nephritis, and the degenerative changes depend on vascular alterations. The latter indicate a chronic parenchymatous nephritis and probably depend on an inflammatory or toxic influence. To put this in other words, albuminuric retinitis of the degenerative type is a symptom of vascular changes, which in their turn are widespread throughout the body, and not necessarily a symptom of kidney disease itself as an isolated condition; that is, the nephritis and the retinitis are both caused by the vascular lesions. Doubtless, Dr. Jackson is correct in stating that in the great majority of instances it is possible, ophthalmoscopically, to see lesions in the retinal vessels no matter what the type of the so-called renal retinitis may be, but none the less I am persuaded that the distinction before referred to is worthy of consideration.

DR. O. A. GRIFFIN, Ann Arbor, Mich.—A recent case of albuminuric retinitis proved interesting because of its sudden development and early termination. A woman about 60 years of age had her hip fractured and her physician made repeated examinations of her urine with negative results. About five months later she became suddenly partially blind, the condition being attributed to a general debility. Two weeks later I was called and found that the vision could not be materially improved by lenses. An examination revealed the characteristic fundus changes, and on testing the urine a large amount of albumin was found. The case was then referred back to her physician, but treatment was of little avail, death occurring within seven months.

DR. LOUIS STRICKER, Cincinnati—I would not like to leave any one under the impression that I do not recognize that a certain number of cases are the results of vascular changes. They result from the general vascular fibrosis, which is present all over the body, but I believe that the majority, including most typical cases, are not those of vascular changes, but are consequent on toxemia. I might say that the fibrosis is in all probability due to toxemia. True, where we get closure of the vessels, it is but natural that necrotic changes and hemorrhages follow, but the typical conditions are due to toxemia. Recently a woman came to me with a pure neuritis. I found her urea quantity one-tenth normal, and put her on treatment for Bright's disease, sweating her freely. This was not a typical case of neuroretinitis Brightica, but while she was under treatment in the hospital, as I observed her from day to day, this typical picture did develop. In spite of the treatment, the necrotic conditions went on. This was not a vascular change, but was undoubtedly the result of chemical irritants in the blood, the pure toxemia. These toxins do not come from the kidneys. The supposition is that they are the result of an imperfect metabolism, a break in the chain leading up to a perfect conversion of proteids into uric acid and urea in the liver. Where this conversion is interfered with, the toxins develop. Just what these are we are unable to state, but one thing is sure: when urea fails of perfect excretion from the kidneys, either this as such or the toxins produced remain in the system and are the causative factor of inflammation. I would again repeat, greater stress is to be laid on the reduction of the urea below the normal in the urine than on the presence of albumin; and this is a point which all text-books on the eye fail to recognize. Albumin in the urine simply indicates that the kidneys permit it to escape; that they are diseased, but loss of albumin does not produce eye disease. These diseases, I take it, are not the result of anemia, but of chemical irritants or poison circulating in the blood. One of the latest works on obstetrics advances the idea that the Bright's disease of pregnancy is due to the impaired action on the part of the liver. I do not see how the taking off of the capsule and tearing the kidney tissue is going to do any good. I appreciate that the removal of tension can give the kidney a chance to regain its function, but this could only be of service in acute cases; beside, the operation is surely an extremely dangerous one, and I, for one, would hesitate to use it.

DR. GEORGE F. SUKER, Chicago—Dr. Edebohls is operating on a great variety of cases, including even puerperal eclampsia.

Whether or not the operation is indicated in all the classes of nephritic lesions is certainly an open question. One must carefully weight the advisability of suggesting the operation in any form of albuminuric retinitis. From the statistics given in the paper, one can hardly see any justification for the operation in chronic forms of nephritis with retinal lesions. The operation has not been tried in a sufficient number of cases of acute nephritis with retinal lesions to be of any value in deciding its justification. Again, the indication for the operation must to a certain degree depend on the fact whether the Bright's disease is a pure nephritic lesion or is due to a general arteriosclerosis. If the latter, the operation can accomplish little; but a good deal if due to the former cause. As yet, it is too early to render a final opinion whether the operation is justifiable in cases of nephritis with fundus lesions. Still, we have no right to discard the operation without giving it a fair and extended trial in well-selected cases.

SOME REMARKS CONCERNING THE OCULAR AND AURAL REQUIREMENTS OF RAILROAD EMPLOYEES.*

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Some three years ago the Interstate Commerce Commission published an interesting report of railway accidents occurring in the United States in one brief year. There is no reason to suppose that this was an especially unfortunate year, and as the figures are instructive and full of significance, I will quote some of them.

The total number of personal accidents was 51,743, and of these 7,123 were killed, 2,210 being railway employees; 334 employees were killed and 2,153 injured by collisions and derailment, and 82 passengers were killed and 1,557 passengers injured from the same cause.

The causes of these accidents are doubtless numerous, but have not been, so far as I am aware, statistically enumerated, although such tabulation would be most interesting and instructive. A small minority of the accidents were perhaps in the ordinary course of events unavoidable, but a large majority were probably preventable, and due to carelessness or physical defects on the part of train operators. Of such physical defects it is reasonable to suppose that ocular and aural defects, especially the former, played a most important part, for no physical condition, except the mental condition, is so intimately connected with the moving of a train as vision. The eyes are continually in service—watching for signals, both luminous and non-luminous, looking at the track and searching for approaching trains, familiar objects, etc., etc. In short, there is not a moment during the active operation of a train that the engineer and his fireman have not a vital interest in good vision, and the same is true in a mitigated sense of the conductor, brakemen, switchmen, bridge-men, etc.

I am confident that if statistics could be gathered of the accidents caused by bad eyes, even for a single year, the showing would be so appalling that every intelligent railway official would require no further incentive to perfect the ocular status of his men.

In order to ascertain what was being done to regulate this important essential to transportation security,

* Read before the American Academy of Railway Surgeons, Oct. 1, 1903.