

oozing of blood and serum into the wound, so that it becomes rarely necessary to ligate any of the vessels. The accumulation of all collections of blood within the layers of the abdominal wall is thereby prevented. Stitch and other forms of abscesses become practically eliminated. With its use several advantages in its favor have appeared which were not apparent or expected at its conception. One is that the pressure of the metal guard promotes adhesion between the layers in the abdominal wall, and a distinct non-inflammatory induration may be felt running along on either side parallel with the edges of the wound. This, I believe, tends to the formation of a firmer and stronger cicatrix than any other method yet proposed. It also tends to prevent stretching apart of the edges of the skin, and results in a linear scar.

The writer, in conclusion, would say that he does not so much regard this as a new method of abdominal suture, as he does that it is another step in the technic of the closure of the abdominal wound, and one by which many of the disadvantages of the layer and the mass sutures may be overcome. It requires very little extra time for its application, it promotes union, and it gives additional strength and improved appearance to the abdominal wall. I do not expect its advantages to be appreciated without a trial, but I do believe, from my own experience, that after a fair trial others will be convinced that it has certain advantages in many cases.

Since this paper was begun the writer has been trying, in place of the metal shield, transparent plates of celluloid or pyrolin. With plates of this material, pads of sterile gauze are placed alongside either edge of the wound, but do not press directly upon the surface of the wound itself. Glass plates might be used in the same way.

This method possesses the attractive feature that the outer dressing may be removed daily if desired, and the healing observed without being obliged to disturb or dress the wound. The writer would merely mention this latter device as a suggestion here. He does not intend at the present time to advocate its permanent use without more extended trial.

ALBUMINURIC RETINITIS.¹

BY ALLEN GREENWOOD, M.D., BOSTON,

Ophthalmic Surgeon to the Boston City Hospital.

It is well known that frequently the presence of an optic neuritis will lead to the discovery of a previously unsuspected cerebral tumor, but it is even more frequent that the presence of a characteristic retinitis will lead to a diagnosis of chronic nephritis, in cases where renal disease was previously unsuspected. It is the frequent experience of every oculist to find in patients whose only complaint is a dimness of vision, the ophthalmoscopic picture of albuminuric retinitis, resulting in the diagnosis of a fatal renal disease. Out

of 26 cases I have seen in private practice, in only 5 was there any previous knowledge of kidney disease. It is in relation to the prognosis of such cases, however, that albuminuric retinitis is of the greatest aid; a prognosis, indeed, than which there is scarcely any more certain. By a very brief ophthalmoscopic examination, a diagnosis and prognosis may often be given which seem little short of miraculous to the friends and physician of the patient. That the possibilities of diagnosis are apparent to laymen as well to physicians is evidenced by the experience which probably happens to all oculists of having patients present themselves with the request that their eyes be examined to see if they show anything indicating kidney disease. Physicians certainly appreciate the prognostic importance of this condition, for I have many times been called in consultation in cases of well established chronic nephritis to see if the ophthalmoscope would aid in the prognosis. It is well known that the prognosis as to the length of life in most cases of chronic nephritis without retinal complications is very uncertain, while with them there is a certainty such as is established in but few chronic diseases.

The ophthalmoscopic appearances of albuminuric retinitis are so well known and described in all the modern textbooks that I will only mention them in part and very briefly. In Norris and Oliver seven forms are spoken of as capable of differentiation, viz.:

- (1) Typical albuminuric retinitis.
- (2) Degenerative albuminuric retinitis.
- (3) Hemorrhagic albuminuric retinitis.
- (4) Albuminuric chorio-retinitis.
- (5) Albuminuric neuro-retinitis.
- (6) Albuminuric papillitis.
- (7) Saturnine retinitis.

In actual practice these various forms merge into each other, and often a number may be nearly equally prominent, so as to make it impossible in many cases to name the variety. After having watched several cases from nearly the beginning to the end, one is inclined to consider that many of the so-called forms are but stages in this inflammatory and degenerative disease of the retina, choroid and nerve. In some cases and in some stages one or more of the pathological conditions predominates, hence the different varieties.

Besides the usual signs of neuro-retinitis, the most characteristic alteration indicating albuminuric retinitis is the grouping of white patches about the disc and macula. These are due to fatty degeneration of the retinal elements and exudates, and vary much in size and shape. In the macula region they often run together, forming lines radiating out from the fovea like star points with lines of pigment deposit between. In some cases of albuminuric retinitis they are absent, and there may be only a few hemorrhages or a papillitis to suggest the correct diagnosis, the papillitis in rare cases amounting to a choked disc, so that in every case of retinitis or neuritis

the urine should be examined. The retinitis is almost invariably bilateral sooner or later, though usually more advanced in one eye. It is, I think, generally conceded that the pathological conditions are due to degenerative changes in the blood vessels of the retina, choroid and nerve, resulting in the inflammations, hemorrhages, exudates and degenerations which go to make up the usual ophthalmoscopic picture. These vascular changes are in some cases largely a part of a general arterial degeneration, and in others largely the result of slow uremic poisoning. The latest researches into the microscopical changes show an even greater involvement of the vessels of the choroid than those of the retina, and it has certainly been my experience to find extensive choroidal changes in quite a number of cases. I find that several times lately I have made the diagnosis of albuminuric chorio-retinitis where there has been extensive choroidal change with destruction of the choroid and laying bare of the sclera with the surrounding embankments of pigment.

After seeing a few cases of albuminuric retinitis, I found myself grouping them in two distinct classes based, not on retinal differences, but on the supposed primary pathological conditions which cause the chorio-retinal changes. The cases here reported illustrate this very well. The first group of cases include those where the endarteritis of the chorio-retinal vessels, with its consequent changes in the choroid and retina, is largely a part of a general arterial degeneration of which the chronic interstitial nephritis is also only a part. These cases occur in the old and middle-aged where the whole vascular system has become more or less subject to degenerative changes, from age as well as from impure blood, and the involvement of the chorio-retinal vessels only shows an advanced stage of a degenerative condition, which has existed for some time, and which almost invariably advances to a fatal issue. Death usually results from extensive kidney impairment causing uremia, or else one of the arterioles in the brain more diseased than its fellows, yielding to that increased blood pressure which comes with cardiac hypertrophy, causes a fatal hemorrhage. In these cases the chorio-retinal degeneration is less extensive and often the vascular disease in the choroid and retina causes visible pathological changes before the vascular disease in the kidney has advanced enough to produce a constant albuminuria, as shown in the first and eleventh cases of this group. The prognosis as to length of life is a little better than in the second group.

The second group includes those cases which are more correctly designated as cases of retinitis albuminurica, from the fact that they are directly caused by uremic poisoning of the blood from a distinctly demonstrable acute or chronic lesion of the kidney, usually of a parenchymatous or diffuse type. They occur mostly in youth and early adult life, showing a greater retinal destruction with consequent greater impairment of vision, and the prognosis is extremely bad in all

but those resulting from the acute nephritis of scarlet fever and pregnancy. It is in the prognosis of both classes of cases of chronic nephritis associated with albuminuric retinitis that I wish to interest you particularly.

Regarding the length of life after the discovery of the retinitis, the following may help. Baroness Possaner found the following from the cases taken from 67,000 cases from the Zurich clinic and private practice of Professor Haab:

Men	from the poorer classes	all died in two years.
Women	" " "	68% " " "
Men	" " " better	59% " " "
Women	" " " "	53% " " "

The longest period in the poorer class was six years, and in the better class, eleven years.

In a report by Bell at the Ophthalmological section of the American Medical Association, 100 cases collected from the private practice of various ophthalmologists are given, and of these 73% died in one year and 94% in two years.

Rogers collected 419 reported cases which included those of Possaner and Belt, and of these 90% died within two years.

In my private practice in the past twelve years I have seen 26 cases, and am able to report the subsequent condition of all but two. Of the 26 there are six living to-day, but none of these was seen prior to three years ago. Of the 20 who have died only two lived over three years. I shall only give a brief history of the two or three most interesting cases and a synopsis of the others.

GROUP I.

1. Miss E., aged forty-two, called on account of failing vision Dec. 18, 1891.

O. D. V. = 20-70. O. S. V. = 20-20.

O. D. well-marked neuro-retinitis, with numerous flame-like hemorrhages and spots of exudate. There was a large irregular patch of degeneration at the macula involving the choroid, with consequent embankment of pigment.

O. S. Slight neuro-retinitis with a few hemorrhages and spots of exudate about disc. Macula not involved.

Examination of urine showed specific gravity 1.012, and only the most minute trace of albumin. Three casts in 8 slides. Chronic interstitial nephritis. Examination a few days later by an expert chemist, who reported that no albumin or casts were present. A few months later, however, albumin and casts were easily found.

March 17, 1892, O. D. V. = 20-200. O. S. = 20-70 more spots of exudate and degeneration. Patient died August, 1893, from uremic poisoning, a few months after a cerebral hemorrhage, one year and eight months from time of my first seeing her.

2. Mrs. F., Natick, age fifty-five. Seen March 14, 1892. Retinitis with hemorrhages and exudates. No subsequent history obtainable. Urine—chronic interstitial nephritis.

3. Mrs. F., aged seventy. Seen Nov. 3, 1897. Had had a cerebral hemorrhage a few weeks previously with hemiplegia and hemianopsia. Fundi showed neuro-retinitis with flame-like hemorrhages and spots of exudate. Chronic interstitial nephritis. Died March 27, 1900. Lived two years four and one-half months.

4. Mr. C. O. L., age sixty. Seen Jan. 6, 1898. Neuro-retinitis with exudates. Urine—chronic interstitial nephritis. Died Nov. 6, 1899. Lived one year and two months.

5. Mrs. H. W. P., age seventy-three. Seen April 11, 1897. Albuminuric retinitis. Chronic interstitial nephritis. Died comatose Oct. 5, 1899. Lived two years and six months.

6. Mrs. R. H., age sixty-seven. Seen July 12, 1897. Neuro-retinitis, with exudates about the maculae. Urine

— chronic interstitial nephritis. Died Dec. 5, 1897. Lived six months.

7. Mrs. M. B., age fifty-five. Seen July 30, 1897. Simply exudates about maculae. Urine — chronic interstitial nephritis. Died March 8, 1900. Lived two years eight and one-half months.

8. Mrs. F., age thirty-five. Seen Jan. 15, 1896. Fundi show chorio-retinitis with a few hemorrhages and spots of exudate and degeneration. Chronic interstitial nephritis. Died March 14, 1900. Lived four years and two months.

9. Mrs. F., age fifty. Seen June 3, 1898. O. U. show a few scattered hemorrhages, and a few spots of exudate about maculae, chronic interstitial nephritis. Died of cerebral hemorrhage, Aug. 11, 1900. Lived two years one month eleven days.

10. Mr. L. W., age eighty. Seen April 11, 1896, on which date I removed a cataract. The resulting vision being poor, I examined the fundus, finding albuminuric retinitis. The family physician found advanced chronic interstitial nephritis. The patient died Sept. 28, 1897, living one year and five months.

11. Mrs. W., age fifty. Seen June 12, 1896. Had neuro-retinitis, with abundant hemorrhages and spots of exudate. The family physician reported urine normal to the family, but in a year she developed chronic interstitial nephritis, and died in convulsions one year and six months after I saw her.

12. Mrs. H., age seventy. Seen July 6, 1894. Albuminuric retinitis, complicating incipient cataract. Urine of chronic interstitial nephritis. Died twelve months later.

13. Mr. T., age seventy-five. Seen Nov. 14, 1897. Fundi showed haziness of disc outlines, and in the region between the discs and maculae were many small round spots of yellowish-white exudate. No hemorrhages or degenerations. The family physician reported $\frac{1}{2}\%$ of albumin and existence of chronic interstitial nephritis. Died Feb. 26, 1898. Lived three and one-half months.

14. Mrs. S., age fifty-six. Seen July 12, 1902. Complained of dim vision and not being able to get glasses to help her. v. o. u. 20-200; incipient cataract o. s. Fundi showed neuro-retinitis with exudates about maculae and one hemorrhage below right disc. Urine shows chronic interstitial nephritis. Died Nov. 12, 1902. Lived four months.

Added to this group are the following cases still living:

15. Mrs. P., age sixty-one. Seen July 26, 1900. Her family physician had discovered albuminuria from chronic interstitial nephritis, and sent her to me for help in prognosis. Neuro-retinitis with spots of exudate. No hemorrhages. Is still living two and one-half years after.

16. Mr. C., age sixty-seven. Seen Feb. 16, 1901. Has incipient cataract and neuro-retinitis, with hemorrhages. Chronic interstitial nephritis. Still living one year and eleven months after.

GROUP II.

1. Susie W., age sixteen. Seen Nov. 15, 1893. Seamstress; came for failing vision. Degenerative chorio-retinitis. No hemorrhages. Vision much reduced. No papillitis. Maculae involved. Chronic diffuse nephritis. Died April 6, 1898, from uremic coma. Lived four years and seven months.

2. Miss G., age twenty-six. Seen Sept. 21, 1894. Watch-maker, came for failing vision. Degenerative chorio-retinitis o. u. Macula involved o. u. No papillitis or hemorrhages. Diffuse nephritis. Died Oct. 22, 1894, in uremic coma. Lived one month.

3. E. L., age sixteen. Seen April 12, 1899. Came for failing vision. Had to leave her work several weeks ago on account of poor eyesight. O. D. V. = fingers at two feet. O. S. V. = 20-100. O. U. the fundi show a large amount of whitish exudate about the disc, covering the edge and burying the blood vessels in places. Throughout the retina were numerous white spots of exudate. In the macula region O. D. there was the star appearance of radiating lines of degeneration with intervening lines of pigment deposits with a heap of pigment in the centre of the macula. No hemorrhages. In O. S. the same condition involving only half of the macula region. Chronic

diffuse nephritis with urine solid on boiling. Died in uremic coma April 19, 1900. Lived one year seven days.

4. Miss P., age twenty-two. Seen July 6, 1893. Worked in watch factory till six months before. O. U. showed chronic retinitis with complete destruction of retina and choroid over large areas including the maculae. Gray atrophy of optic nerves with arteries reduced to mere threads. No perception of light. Had uremic convulsions every day or two, and died in one six weeks later. Chronic diffuse nephritis, urine solid on boiling.

5. J. W. H., age thirty-four. His physician had diagnosed chronic diffuse nephritis. Two and one half years ago feet and legs began to swell. Six months ago his sight began to fail in left eye; right eye blind from injury. Seen May 1, 1901. Left eye showed a neuro-retinitis. The neuritis was so marked that the disc could not be seen. Region about disc and macula occupied by exudate. Few small hemorrhages. His physician informed that death would probably occur inside of a year. Died of uremic poisoning Nov. 29, 1901. Lived about seven months.

6. A. H., age thirty. Came to my office Aug. 27, 1901, for failing sight in both eyes, particularly the right one. Was not aware of any other physical disability. O. D. V. = 20-400. D. S. V. = 20-30. O. U. neuro-retinitis with hemorrhages about discs. Exudates and degenerations about maculae. Urine showed chronic diffuse nephritis with $\frac{1}{2}\%$ albumin. Died in New York, Nov. 19, 1901. Lived about three months.

Added to this group are the following cases still living or unheard from:

7. W. W., age twenty-two. Seen July 20, 1900. Complained of failing vision only. O. U. V. = 20-200. Fundi showed neuro-retinitis with exudates; and degenerations. His physician reported chronic parenchymatous nephritis. Left town soon after and I have been unable to trace him.

8. F., age twenty-six. Seen Aug. 7, 1900. Sent by his physician for help in prognosis, as he had a chronic diffuse nephritis. O. U. had well-marked neuro-retinitis with hemorrhages and exudates. The last report from him was to the effect that he had failed so much that he was obliged to give up all work.

To these should be added cases nine and ten of this group, which were cases of acute nephritis from pregnancy with complete recovery of the eyes and kidneys.

My cases are too few to deduct percentages from, but the following may be noted: Out of the sixteen cases of the first group, of one I have no subsequent history; four died within one year, four within two years and four within three years. Only one lived over three years, death coming in four years and two months. Of the two living neither dates back over two and one-half years.

Out of the ten cases of the second group, of one I have no subsequent history; four died within eight months; one died within two years; one died after four years and seven months: one was known to be alive two and one-half years after first being seen. The two cases occurring from pregnancy have recovered entirely.

From these cases and the many others of different observers, it is plain that the prognosis afforded by the discovery of albuminuric retinitis in cases of chronic kidney diseases is very grave, and that life is but rarely prolonged more than three years. It has been noticed that cases occurring in the better classes live the longest as a result of better hygienic surroundings and care. I feel sure that as we learn more about the body metabolism in such cases, and can give them the best diet and hygienic treatment, we shall

be able to retard the progressive decline, and materially lengthen life. How much capsulotomy may do in these cases will be told in the future. I realize that my clinical grouping of these cases is open to criticism, but I hope, in the near future, by post mortems and microscopical examinations to prove the grouping correct pathologically.

AS TO POPE LEO'S CASE — PLEURO-PNEUMONIA.

BY SAMUEL DELANO, M.D., BOSTON.

Now that Italy has poured a broadside into the diagnosis of the late Pope's case, the time may seem opportune for letting go with some guns of our own. It might be worth while, if merely for general principles' sake, to submit the diagnosis to the test of inherent probability; because general reasoning in a large percentage of cases furnishes so good a guide to follow. It must, however, be conceded that in a given instance this would not coincide with legitimate criticism, because one well-observed fact might show your most careful logic to be a non-sequitur.

Nevertheless it seems highly improbable that fibrinous pneumonia should be at work in an old man of ninety-three, sick some days before medical aid was summoned, lingering on some weeks, while in that time he could be up and dressed and give many audiences; with a temperature ranging irregularly over the lower fever scale, a respiration for much of the time in the neighborhood of 25 and the pulse around 85.

Inadequate as these items might seem, however, we should still have to keep silence, if only on the principle that in medical diagnosis the looker-on does *not* get the best of the game, had not there been a fatal flaw in the autopsy. Here, certainly, allegiance must waver, for, advancing a pathological diagnosis of gray hepatization of the lower lobe, this is made to rest upon the statement in the commentary that "from the cut surface an abundant dirty white fluid without gas oozed out, showing complete hepatization." Of course, as a matter of fact, nothing could be much drier than the cut surface of gray hepatized lung and we thus not only have to do with an absolute inconsistency of statement, but the description fits no stage of pneumonia, — on the contrary comes very much closer to an edema in a compressed lung.

Yet the paramount reason for taking exception to the diagnosis of pneumonia in the Pope's case is that pleuritic effusion in sufficient quantity to aspirate is next to never associated with fibrinous pneumonia. And this furnishes the real object of this communication as well as the moral that the case points. That this is not the prevailing view and that clinical evidence might be put forward to the contrary, I am well aware, but my firm belief is that owing to a confusion of physical signs a great deal of error has crept into our conceptions. The point has obviously an important practical bearing, inasmuch as the prognosis and treatment of effusion and pneumonia are quite

antipodal and because the early detection of fluid is necessary to a wise supervision.

To begin at the end, viz., the autopsy, that great controller of clinical observation, — autopsy findings offer next to no support to the idea of effusion combining with pneumonia; that is to say, personal observation of a goodly number of autopsies and a considerable search of records confirm the principle that pneumonia is not associated with effusion sufficient to influence the physical signs or yield on tapping; a teacupful of fluid there may be, perhaps, but this is no more than the agony or gravity would account for.

This fact if confirmed, would indicate at least that the tendency of pneumonia was not towards the establishing of effusion and it would only remain to determine how far the trend may be influenced by a prolongation of the case or by recovery.

How then account for the impression prevailing that many cases come in as pneumonia and end up with simple effusion or empyema? It goes back to nothing except the perversion and consequent confusion of physical signs. I leave out of account the rational signs which in these cases, especially of empyema, might cut a considerable figure in correcting the error.

The crux of the matter is that pleuritic effusion commencing as such idiopathically may in a certain good percentage of cases be characterized by a bronchial or sniffling respiration that easily passes for the respiration originating in solidified lung. The sophistication is especially apt to be the more successful because it is in very moderate effusions that the most suspicious signs occur, while towards the top of the fluid the sniffling respiration and the voice sounds of like character may be very near the ear. The discrimination may be a delicate task, but into that it is not my purpose to enter. Finally, if the chest fills up we may have a blowing respiration over the whole chest, the sound, however, being fuller and more amphoric than in the case of the smaller amounts.

In my experience empyema is especially apt to include the bronchial element in its signs and that, too, from the outset. I have observed a considerable number of empyemas starting with an all-round bronchial character in the lower back, this mounting higher till extended over the whole chest. The diagnosis of pneumonia was persisted in by a number of observers. Finally, that the chest was full could no longer be denied and operation was invoked. Cases such as these are undoubtedly reported as empyemas following pneumonia, yet there was no distinct demarcation in the physical signs; furthermore, the diagnosis of effusion at the outset was made as well, and it would seem justifiably. In the writer's opinion empyemas, as a matter of fact, are much more often effusion from the start and much more rarely associated with pneumonia than supposed. In the first place, if autopsy shows the marked tendency of pneumonia away from effusion, this fact emphasizes the unreasonableness of linking empyema to pneumonia through the medium of a large serous effusion, to become purulent