interfibrillary spaces of the pars papillaris are distended with fluid and small round cells (Figs. 11 and 12).

To recapitulate briefly the series of changes in a chancre: When the causal agent of syphilis enters through the skin or mucous membrane, it excites local leucocytosis and exudative inflammation, with more or less necrosis; there are also proliferation of the connective tissue cells, a propagation of proliferating cells along the perivascular lymph-spaces, and later a wall of infiltration and edema of the upper corium layers formed about the periphery of the ulcer corresponding to the stage of indurating edema.

In the light of these studies it is easy to understand cases of early indurating edema symptomatic of active primary or early secondary infection. The cases which puzzle us are those in which traumatism and various phlegmasia of the genitals seem again to set up this sclerosing process. The pathogenesis of these later cases is yet to be worked out.

PATHOLOGY OF CHRONIC HYPERPLASIA OF THE VULVA.

In chronic hyperplasia of the vulva, as the name implies, there is also a productive inflammation, a development of new connective tissue through the agency of the connective-tissue cells of the derma. The connective tissue cells proliferate and, lengthening out, become fibroblasts; the fibroblasts in turn are slowly converted into new fibrils of the derma, so with each division and focus of connective tissue proliferation the ground is laid for a future addition to the fibrillar substance of the derma. The process is exceedingly slow, and takes place by small increments; that is, groups of connective tissue cells in certain portions of the vulvar regions proliferate and become the forerunners of new connective-tissue fibers. This may go on simultaneously over quite an extent of the vulvar region, and in the course of time produce more or less evenly a hyperplasia about the pudendum, or it may be limited and accentuated in some one particular spot and give rise to localized fleshy tabs, excrescences and nodules. Between the two sets and forms of this activity of the dermal cells and their combinations are such a variety of intermediate gradations as to well merit the characterization of protean. But this should give rise to no confusion or temptation to classify them into different diseased processes, since however great the external configuration and apparent differences in their aspect, the underlying process is one and the same.

As a particular illustration of this process of chronic cellular inflammation of the derma Figure 13 may be consulted. This shows nearly all of the phases of connective tissue cell proliferation. And it also shows the participation of plasma cells, and possibly small lymphocytes, especially in a lymph crevice about a small vein. The obscure origin and differences of these latter cells do not concern us, as their destiny is much the same for all —the increase of connection in the derma, namely, hyperplasia.

142 West Forty-eighth Street.

DISCUSSION.

DR. JOSEPH ZEISLER, Chicago, stated that what little he has seen of kraurosis vulvæ did not suggest at all a hyperplastic condition such as Dr. Taylor described. Kraurosis seems to him to be more closely allied to atrophy, and apparently has no clinical relationship to the condition described in the paper.

DR. WILLIAM A. PUSEY, Chicago, said that in the last year he has been able to study two cases of kraurosis vulvæ in elderly women, each with a history of long-standing, superficial atrophy of the labia, with patches of leucoplakia of the mucous membrane, such as are seen in the mouth. In both of these epitheliomata had developed on the leucomatous patches. Dr. Pusey has never seen any atrophic conditions of the vulva that were the result of previous hyperplasia, but he can readily see how some of the cases might have that etiology.

DR. WILLIAM S. GOTTHEIL, New York City, declared that in the City Hospital in New York more of these cases are seen than in most institutions in this country. He has seen some cases that were hypertrophic and others that were atrophic followed by sclerotic lesions, such as Dr. Taylor described. He thinks that the processes are identical. Connective tissue hyperplasia in the later stages shrinks and becomes atrophic. The photomicrograph which Dr. Taylor exhibited showed a very late stage of an intense inflammatory process, such as might follow sclerotic or ulcerative processes of various kinds.

DR. R. W. TAYLOR, New York City, in answer to a question by Dr. Baum, stated that the disease, as he saw it, was a sequel of syphilis, and of syphilis alone. In his cases the extension of the edema was coincident with the hard chancre. In some cases of progressive syphilis, in which the diathesis is still active, the edema sometimes occurs from simple lesions, sometimes from condylomata lata or from the hyperplasia set up by staphylococcic irritation following vaginal discharges. Dr. Taylor recalled the fact that his paper was divided into two parts: The first dealing with hyperplasia and the second with its sequelæ. He does not claim that the cases reported were cases of kraurosis, but that they resemble that condition. Case No. 7, he declared, was one of the most typical he has ever seen, the patient's vulva resembling a rubber tobacco pouch. All these cases occurred in his service at the Charity Hospital, many years ago, and were carefully observed for a long time. In every case sections were removed and submitted to Dr. Ira Van Giesen for microscopic examination, and his findings were verified by Drs. Prudden and Delafield.

LIGATION OF THE COMMON CAROTID ARTERY FOR MALIGNANT RECURRENT HEMORRHAGE OF THE VITREOUS.*

GEORGE S. DERBY, M.D.
Ophthalmic Surgeon, Carney Hospital.
BOSTON.

The object of this communication is to bring before this Section the subject of ligation of the carotid for the relief of a disease other than the vascular tumors of the orbit and lid in which this procedure is commonly employed. I refer to the rather infrequent cases of malignant, recurring vitreous hemorrhage.

The following example came under my observation a little over a year ago:

Patient.—B. K., 51, a healthy married woman of German birth.

History.—On the morning of Jan. 18, 1906, the patient noticed that the vision of her left eye was somewhat blurred. On the following day she applied for treatment at the Carney Hospital. Her family and previous history were negative. There was no evidence of hemophilia or of syphilis. There had never, to her knowledge, been any disease in her family and, as far as she knew, her eyes had always been normal. She had given birth to seven healthy children and her menses, always normal in character, had ceased four years previously.

Examination.—For the sake of brevity I will say here that several physical examinations made by members of the medical staff, at this time and during the succeeding year, were all absolutely negative. The heart and kidneys appeared entirely normal and there was no evidence of vascular disease. The

*Read in the Section on Ophthalmology of the American Medical Association at the Fifty-eighth Annual Session, held at Atlantic City, June, 1907.

blood pressure (Riva-Rocci) equalled 145 mm. The blood contained 5,568,000 red cells, 9,600 white, and 85 per cent. of hemoglobin. The patient was a stolid, well nourished woman with a good appetite and digestion.

Ocular Examination.—The left eye was white, cornea, iris and lens normal, the pupil slightly dilated. The vitreous contained numerous small and large floating opacities. Situated about in the optic axis and best seen with a plus 12 glass was a peculiar membranous looking structure roughly square in outline, and suggesting an inverted pyramid in shape (base forward) rather than a flat septum. In color it was white, with irregular areas of black pigment along its borders, and on its surface were large splashes of fresh blood. Along the lower temporal border was a small hemorrhage extending into the vitreous. The retina and nerve head could not be seen. Vision: Counts fingers at one foot. Examination of the right eye showed a similar but somewhat smaller structure, situated in the same relative position and connected with the disc by a fine strand of grayish tissue. No vascularization could be made out. The vitreous was clear, and except for this structure nothing abnormal could be seen in any part of the eye. V. O. D. = 6/12 —, not improved by glasses. Increasing doses of mercury and iodid were prescribed, but seemed to have no lasting effect on the process. There was an occasional slight improvement and then would come a sudden diminution in vision caused by fresh hemorrhage on the membrane and increased clouding in the vitreous. By June the vision was reduced to the perception and localization of light, and the red reflex had entirely disappeared. On October 12 the patient again came to the clinic saying that two days previously the sight of the right eye had suddenly failed. Examination showed vitreous hemorrhage and large splashes of blood on the membrane. Vision = 3/60. The retina could barely be distinguished. Barring a greenish tinge to the iris the state of the right eye had not changed. She was taken into the hospital for observation and treatment. The course of the disease did not differ from that in the other eye. Under large doses of iodid and mercury the vision occasionally showed slight improvement only to fall off again from a fresh hemorrhage, which in each instance appeared to come from the membranous structure. Mercury and iodid gave place to lactate of calcium and large doses of gelatin,[1] and later to other drugs, over a period of three months. There was a steady loss of vision down to counting fingers at one foot, and it became increasingly evident that complete blindness would ensue were not energetic measures taken. Meanwhile, there was no sign of returning vision in the left eye.

Operation.—Remembering that in two somewhat similar cases, one reported by Mayweg[2] and one by Axenfeld,[3] ligation of the common carotid had apparently checked the disease, I explained to the patient the dangers of such an operation and the uncertainty of the result, and on the other hand the probability of blindness under medical treatment alone. Her good health and absence of all signs of vascular changes were in her favor. She chose to take the risk, and on December 10, the right common carotid was ligated by Dr. H. H. Germain under ether anesthesia. At this time the vision equalled counting fingers at one foot. The patient made an uncomplicated recovery from the operation save for a slight rise in temperature. She was kept in bed for two weeks.

Postoperative History.—December 11: V. O. D., counts fingers at five feet.

December 12: V. O. D., counts fingers at fifteen feet. With the ophthalmoscope the red reflex was perceptibly brighter.

December 14: V. O. D., counts fingers at twenty feet.

December 17: V. O. D. = 6/30. It was possible to distinguish the retina with both the indirect and the direct methods. The vitreous had cleared up markedly and the membrane was clearly visible. No fresh hemorrhage could be seen.

December 27: Patient discharged from hospital. Vision = 6/30+. There were no fresh hemorrhages and the vitreous was clearing slowly.

To our great disappointment, two days later there was a sudden diminution of sight and examination showed the membranous structure to be almost covered with fresh blood and the vitreous very cloudy. V. O. D., counts fingers at one foot. The attack came on spontaneously and did not follow exertion or excitement. Since that time the course of the disease has been about the same as before the operation and the vision on March 8 was limited to hand movements directly in front of the eye. The red reflex has almost entirely disappeared, but it is still possible to see the membrane faintly and to note that it is covered with blood.

PREVIOUS CASES.

Mayweg,[2] at the twentieth meeting of the Heidelberg Society, reported the following case:

CASE 1.—A 17-year-old peasant had lost the sight of his left eye from repeated vitreous hemorrhages, although under capable treatment. Three years after the left eye first became involved, a similar process affected the right, and recurred four times. At the fifth attack he came to Mayweg, who found extensive hemorrhage into the vitreous and vision reduced to 20/200. There was no evidence of organic disease. In spite of energetic treatment there were six further recurrences and finally, ligature of the carotid was resorted to. This checked the process and eighteen months later the vitreous was clear and the vision = 20/40.

CASE 2.—Axenfeld[3] saw a healthy girl, 24 years old, who had lost the sight of her right eye from persistent vitreous hemorrhages followed by secondary glaucoma. Several years later the left eye became affected and the vision reduced to hand movements. Ligation of the common carotid controlled the disease and three months later there had been no further hemorrhage. The vitreous cleared up very slowly. I have been unable to obtain the later history of this case.

Before proceeding to consider the operative treatment involved there are one or two interesting features of the case reported above which deserve a few words. As to the nature of the membranous structures there is an element of uncertainty. On the one hand they may have been the organized remains of vitreous hemorrhage which took place at an earlier period in the patient's life, and very likely this would have been the assumption had the patient been seen only after the involvement of the second eye. It does not appear, however, that such was the true nature of the case for the following reasons: The structures resembled each other closely and were symmetrically placed. They were situated in the line of Cloquet's canal and, in the right, a fine strand of grayish tissue could be seen (before blood obscured the vitreous), running back from the structure to the disc. There was no vascularization and absolutely no sign of disease in the chorioid or retina (O.D.). It seems probable that we have in this case the remains of embryonic structures. Vitreous hemorrhages are occasionally observed in connection with a persistent hyaloid artery, as, for instance, in the case reported by Hickman.[4] Furthermore, it is well recognized that undeveloped organs form a locus minoris resistentiæ. The assertion of the patient at her first visit that the sight of the right eye had never been more acute favors this view.

It is worthy to note that in this case each hemorrhage appeared to take place from the substance of the membrane.

The rapid improvement in vision from 1/300 to 6/30+ in one week is striking evidence of the temporary efficiency of tying the carotid in these cases. On the other hand, the recurrence, ten days later, shows

1. We were unable at this time to estimate the coagulation power of the blood and treated her empirically on the assumption that it was retarded.

2. Bericht d. Ophth. Gesellschaft zu Heidelberg, 1889, 92.

3. Oberrhein, Aerzt., July 6, 1905; Münch. Med. Wochschr., 1905, lii.

4. Ophthal. Rev., 1902, 85.

how quickly the circulation can be re-established from the collateral supply. That it was the operation which caused the improvement is hardly open to question.

For a time the patient was treated with lactate of calcium and gelatin on the theory that the coagulating power of the blood was diminished. As we were unable to determine then whether or not this was the case, and as there was no apparent improvement, this treatment was discontinued. Recently through the kindness of Dr. F. T. Lord, whom I here wish to thank, an estimation was made by Wright's method, and it was found that coagulation took place in 2 minutes and 50 seconds, the normal by this method being 1 minute 45 seconds to 2 minutes 10 seconds. Thinking that possibly the former preparation of calcium may not have been efficient, the patient has again been given this drug.

This method of determining the time of clotting is as yet in its infancy, but its great simplicity and availability give great promise. Although Wright and his assistants have indicated that retarded coagulation may be responsible for many and various lesions, there has as yet been little confirmatory evidence, still as in this case we have no other clue to the etiology, and as many other similar cases occur in which the causation is likewise obscure, the possibility of retarded coagulation as a cause is worthy of consideration in the future. Therefore, it would seem to be advisable to ascertain the clotting time in all such cases, and if it be slow the administration of calcium salts is indicated and may prove of marked benefit to the patient.

Whether we are justified, after medical treatment has failed, in proposing ligation of the carotid to these patients is the question which more nearly concerns us here. Our first consideration may well be the danger of the operation. We have a large mass of statistics on the subject of carotid ligation and they are somewhat complicated, owing to the fact that in pre-antiseptic days the danger of sepsis and of secondary hemorrhage had to be seriously considered, and added very materially to the mortality of the operation. Moreover, the disease for which the operation is performed has a marked influence on the mortality. It would seem fair to assume about the same rate of mortality after ligation for the cure of vitreous hemorrhage as that after the same operation for the relief of pulsating exophthalmos. Keller,[5] from the Zurich clinic, gives 4 per cent. in 31 cases. Slomann,[6] from 95 cases, some of the patients of which were operated on in the pre-antiseptic era, gives a mortality of 10 per cent. Werner[7] 10 per cent., Le Fort[8] 12 per cent., Murray[9] 10 per cent., de Schweinitz[10] 10 per cent.

For other diseases the mortality is much greater, as would be expected when we take into account the serious character of the lesions which call for this operation. Keller[5] gives 34-43 per cent., Le Fort[8] 43 per cent., Murray[9] 25 per cent. The total results of carotid ligation as quoted by Barnard and Rugby[11] are as follows: Tillman 31 per cent., Zimmermann 31 per cent., Pilz 18 per cent., and Friedlander 13 per cent.

As regards the dangers and complications of the operation, Erichsen, quoted by Barnard and Rugby,[11] says that cerebral symptoms occur early or late in 25 per cent. of the cases. The dangers of sepsis and hemorrhage may now be disregarded.

Blindness, from obstruction of the arteria centralis retinæ, has been noted by Elschnig,[12] Siegrist,[13] Gifford[14] and Uhthoff,[15] and has apparently in the majority of instances been caused by lodgment in this vessel of a fragment detached from the thrombus in the carotid. In any event this is an uncommon accident, for in the ordinary case closure of the carotid causes but a temporary disturbance of the retinal circulation. Uhthoff[15] and Hansell[16] have observed the temporary obliteration of the retinal vessels at the moment of ligation.

Sattler,[17] Wagenmann[18] and Silex[19] have seen lesions of the cornea, which have been referred both to trauma and to trophic disturbance. Axenfeld[20] has reported homonymous hemianopsia following ligation, and Michel[21] has seen cataract following thrombosis of the carotid.

In the case of a patient operated on in Boston hemiplegia and death followed carotid ligation and the autopsy showed the absence of the circle of Willis. All these complications are, however, of rare occurrence and need not be considered here.

Having spoken of the dangers and mortality of the operation, we must now consider what chances the patient has of benefiting by it, and here we have but little information to guide us. In pulsating exophthalmos we may assume from the statistics 50 to 80 per cent. of cures from the operation, but in recurrent vitreous hemorrhage we have only the three cases above on which to base an estimate. Mayweg's was successful, Axenfeld's successful up to the end of three months (later progress unknown), and the case reported here was a failure. The two favorable ones occurred in youth, mine in middle age.[22] In all three it was done as a last resort. On the one hand stands an operation of considerable risk and of uncertain value; on the other is almost certain blindness. In making a decision, the age of the patient and the condition of the vascular system are of great importance. In my own case no lesions could be found, but her age made it possible that they were present. We estimated her risk at from 15 to 20 per cent. In all cases with healthy vessels and no complications, and where the patient has chosen to run the risk after an impartial statement of the facts has been given, I believe that the operation is justifiable. Naturally this refers only to those cases in which the vision of one eye has already been lost and in which medical treatment offers small hope of saving the second eye.

Additional Note (June 1, 1907).—Since April 1 the condition of the patient has remained practically unchanged. V. O. D., hand movements at one foot. The second course of treatment with calcium lactate was no more successful than the first. Numerous estimations of the coagulation time showed a minimum of 2 min. 30 sec., which did not decrease even when large doses were given.[23]

5. Inaug. Diss. Zurich., see Michel's Jahrs., 1898, 606.
6. Doctordiss. Copenhagen, see Michel's Jahresbericht, 1898, 612.
7. Inaug. Diss. Tübingen, see Michel's Jahresbericht, 1898, 617.
8. Rev. de Chir., 1890, May and June.
9. Ann. of Surg., 1904, 421.
10. Quoted in Modern Ophthalmology; Ball, 640.
11. Ann. of Surg., 1904, 649.
12. Arch. f. Ophth., 1893, xxxix, 151.
13. Ber. d. Ophth. Gesell. z. Heidelberg, 1898, 10.
14. Ophth. Rec., 1899, 595.
15. Ber. d. Ophth. Gesell, 1898, 21, 22.
16. THE JOURNAL A. M. A., 1905, 536.
17. Quoted by Siegrist (13).
18. Quoted by Siegrist (13).
19. Ber. d. Ophth. Gesell., 1898, 23.
20. Ber. d. Ophth. Gesell., 1898, 22.
21. Quited by Siegrist (13).
22. Pollack (Michel's Jahresbericht, 1900, 368), has reported a case in which the artery was tied in a patient 66 years of age, and the second one ligated five years later without accident.
23. The technic used was that described in the Lancet, 1905, 1906.

In this connection I find that in four cases of vitreous hemorrhage Paton[24] and Paramore[24] found diminished clotting time, and Wright suggested that in these instances the hemorrhage might be due to thrombosis taking place in a small vein. Clearly the part played in this disease by abnormal coagulation of the blood is purely a matter of speculation at the present time and remains for future investigation to solve.

Professor Axenfeld has kindly written me that the progress of his case is still satisfactory. There have been no fresh hemorrhages and the vision two months ago was 6/12. I understand that he has had the carotid tied in a second case, but that it is too early yet to judge of the result. A detailed history of his first case may be found in the dissertation of Fraülein Bieber,[25] and from her paper I take this occasion to quote Siegrist's figures of 6 per cent. mortality in 997 cases of carotid ligation gathered from the literature. This writer (Siegrist) is of the opinion that if the patient's condition be good and if compression of the artery be carried out methodically before the operation, then ligation may be performed without serious danger to the patient.

[FOR THE DISCUSSION, SEE THE DEPARTMENT OF SECTION DISCUSSIONS IN THIS ISSUE.]

METASTATIC CONJUNCTIVITIS IN GONORRHEA.*

JAMES J. CARROLL, M.D.
BALTIMORE.

Although gonorrhea was known to the ancients and ophthalmia neonatorum to the early Greeks and Arabians,[1] the true relation of the parent disease to its affiliated inflammations of the eye was still very imperfectly understood even at the beginning of the eighteenth century. While many of the primitive physicians appear to have fully recognized the ophthalmia of infants, it was not until 1750[2] that it was believed to depend on the vaginal discharge of the mother. Before this the theories as to its origin were rather vague, the old physicians of India, for example, ascribing it to the bad character of the mother's milk. In later years the ophthalmia of adults was thought by some to be due to gonorrheal virus seeking another outlet in consequence of suppression of the urethral discharge. Acting on this view Jüngken[3] recommended, in his text-book, the reestablishment of the flow from the urethra as the proper treatment for the ocular disease.

Without citing all the views and opinions in regard to gonorrheal affections of the eye which were in vogue before 1700, it will suffice to state that nearly all of them contained a belief in an internal or indirect route by which the venereal virus reached the eye in contradistinction to a direct or external route. This endogenous conception of the origin of gonorrheal affections of the eye prevailed until the time of Astruc,[4] who, in 1736, published the report of a case of purulent conjunctivitis which he attributed to direct infection, by the hands, from the urethra. This new theory made very slow progress. Although it was greatly strengthened in the beginning of the nineteenth century by the support of Jaeger, by the publication of Allan[5] and the lectures of Mr. Abernethy[6] at St. Bartholomew's Hospital, it did not supersede the old belief entirely until the experiments of Piringer[7] firmly established the direct causative relationship between the infecting material of the urethra and the inflammation of the conjunctiva.

After the convincing proofs of this investigator were known, the pendulum soon swung to the extreme, and it was not long before Ruete[8] and Arlt[9] maintained that all gonorrheal affections of the conjunctiva were the result of direct inoculation. While this was the prevailing belief, Fournier,[10] in 1866, described a type of conjunctivitis, occurring in a gonorrheic with inflamed joints, which he regarded as metastatic. In 1881, Haab[11] reported a case of bilateral conjunctivitis, which he saw in a patient with gonorrhea and which he thought was not due to direct infection on account of the absence of gonococci in the conjunctival secretion. In 1885, Haltenhoff[12] reported five cases of gonorrheal conjunctivitis without inoculation. In the following decade and a half appeared similar publications by Rückert,[13] Liebrecht,[14] Vanderstraeten,[15] Parinaud,[16] Morax,[17] Lipski,[18] Nobl,[19] Gielen,[20] Becker,[21] Lichtenstern,[22] Fage,[23] Morton[24] and others. In 1899, the subject was brought before the Ninth International Congress of Ophthalmology by Van Moll,[25] of Rotterdam, who reported six cases of his own observation. Kurka,[26] in 1902, published an account of twenty-two cases, two of which were seen by him in Professor Fuchs' clinic in Vienna and the others were taken from the literature. Later cases have been reported by Lesser,[27] Apetz,[28] Thorner,[29] Sym,[30] Paul,[31] Burnett[32] and others.

24. Lancet, 1905, 1249.
25. Inaug. Diss. Freiburg, Berlin, 1906.
*Read in the Section on Ophthalmology of the American Medical Association at the Fifty-eighth Annual Session, held at Atlantic City, June, 1907.
1. Hirschberg (J): Geschichte der Augenheilk., 1899, xii, 397, of Graefe-Saemisch Handbuch der gesamten Augenheilkunde, 2d Ed.
2. Guellmalz: Ref. in Graefe-Saemisch Handbuch, etc., 2d Ed., v, chap. iv, p. 238.
3. Jüngken: Die Lehre von den Augenkrankheiten, Berlin, 1832, p. 252.
4. Astruc: De morbis Veneris, 1736, 192. Lutetiæ Parisiorum.
5. Allan: System of Pathological and Operative Surgery, Edinburgh, 1819, i, 153.
6. Abernethy: Lancet, London, 1825, p. 5.
7. Piringer: Die Blennorrhoe am Menschenauge. Gratz, 1841.
8. Ruete: Lehrbuch der Ophthalmologie. Braunschweig, 1845.
9. Arlt: Die Krankheiten des Auges, Prag, 3d Ed., 1854, i, 45.
10. Fournier: Nouv. Dictionaire de Méd. et Chir. pract., réd. par Jaccoud, Paris, 1866.
11. Haab: Cor.-Bl. f. Schweiz. Aerzte, 1881, No. 4, 105.
12. Haltenhoff: Ueber Conjunctivitis gonorrhoica ohne inoculation. Archiv f. Augenheilk. von Knapp u Schweigger, 1885, xix, 103-120.
13. Rückert: Ueber Iritis, Conjunctivitis, Poliarthritis gonorrh. Klin. Monatsb. f. Augenh., 1886, xxiv, 339-358.
14. Liebrecht: Kasuistiche Beiträge aus Prof. Schöler'schen Augenklinik, Klin. Monatsb. f. Augenh., 1891, xxix, 176-179.
15. Vanderstraeten: Des affections oculaires de nature blennorrhagique. Arch méd. belges, 1891, xi, 300.
16. Parinaud: Congr. d'Opht., Paris, 1891.
17. Morax: Recherches bactériologiques sur l'étiologie des conjonctivites aigues et sur) l'asepsic dans la chirurgie oculaire, Thèse de Paris, 1894.
18. Lipski: Des complications oculaires métastatiques de la blennorrhagie, Thèse de Paris, 1895.
19. Nobl: Ueber seltenere Komplicationen der Blennorrhoe. Allg. Wiener med. Zeitung, 1895, No. 33.
20. Gielen: Ueber gutartige doppelseitige Conjunctivitis nach Affectionen der Urethra. Diss. Bonn., 1897.
21. Becker: Die metastatische gonorrhoische Augenbindehaut-Erkrankung. Jahresb f. Natur- und Heilkunde, Dresden, 1897-98.
22. Lichtenstern: Zwei Fälle von gonorrhoischer Allgemeininfection. Prager med. Wochschr., 1898, No. 43.
23. Fage: Conjonctivite blennorrhagique métastatique, Recueil d'Opht., April, 1900.
24. Morton: Relation of Certain Catarrhal and Suppurative Inflammations of the Conjunctiva to Urethritis and Arthritis. Ophthalmic Record, June, 1900.
25. Van Moll: Giebt et eine metastatische Conjunctivitis? Ix Internat. Congr. of Ophthal., Utrecht, 1899.
26. Kurka (A.): Ueber metastatische Bindehautentzündung bei Gonorrhoe. Wien. klin. Wochschr., 1902, No. 40.
27. Lesser: Conjunctivitis duplex rheumatica nach Gonorrhoe. Münch. med. Wochschr., 1902, No. 29.
28. Apetz (W.): Ueber gonorrhoisch-metastatische Entzündung am Auge Erwachsener. Münch. med. Wochschr., Aug. 4, 1903.
29. Thorner: Ueber metastatische Conjunctivitis gonorrhoica. Charité Ann., Berlin, 1904, xxviii, 340-347.
30. Sym (W. G.): Metastatic Gonorrheal Ophthalmia. Edinburgh Med. Jour., 1904, 126-129, vol. xvi.
31. Paul: Metastatische-gonorrhoische Augenaffektion. Berl. klin. Wochschr., 1905, No. 9.
32. Burnett (S. M.): Inflammation of the Eye Due to the Toxin of the Gonococcus. THE JOURNAL A. M. A., 1905, xlv, 1926-1928.