ABSTRACT OF DISCUSSION

Dr. E. R. LEWIS, Dubuque, Iowa: In considering the pathologic conditions of the nasal and accessory nasal tissues, it must be remembered that a majority are due directly to infections, and of the remainder a large number are due indirectly to preexistent or coexistent infections. The subject of Dr. Walter's paper is, therefore, of practical interest to all who have to deal with these conditions. Inasmuch as there are involved two widely differing antagonists, the invading organism and the invaded tissues, analysis must proceed from two widely differing standpoints, that of the bacteriologist and that of the clinician. Observations which are to form the basis for deductions as to the bacteriologic or the clinical side of the matter must be made by experts and the bacteriologic expert should not dwell in the body of the clinician, and *vice versa*.

Among the deductions, certainties may or may not present; uncertainties are bound to. Undue emphasis should be spared the former and accorded the latter.

The fundamental reasons underlying the immunity of certain tissues and the susceptibility of other tissues apparently similar, in the same individual, to the same infection, may not be forthcoming. For instance, a gonococcus, bathing in mucous emulsion the newly born head, harms not the oral mucosa, or the nasal mucosa, but does attack the ocular mucosa. Yet that same oral mucosa, scatheless from its encounter with the gonococcus, falls easy prey to the leprothrix or saccharomycetic infections, which in turn do not attack the conjunctiva; the same nasal mucosa offers but weak resistance to pneumobacillus or streptococcus. Why? The Morax-Axenfeld bacillus conjunctivitis proceeds uninterrupted in the presence of bichlorid of mercury or silver salts but succumbs to zinc sulphate, whereas the more virulent gonococcus conjunctivitis subsides under the influence of silver salts but is not affected by zinc sulphate. Why? Questions like these must remain unanswered until careful investigations, patiently pursued and zealously guarded against error, shall have built up little by little a knowledge of the subject founded on truths proved by oft-repeated observations. I wish to express the appreciation which I am sure we all feel in regard to Dr. Walter's patience and energy.

I want to voice a suggestion concerning nomenclature which may be of some practical value. In speaking of the pharyngitis, tonsillitis, or rhinitis due to the *Bacillus diphtheriae*, we are wont to use the terms nasal diphtheria, diphtheritic tonsillitis or diphtheritic pharyngitis. In speaking of other forms of pharyngitis, tonsillitis or other inflammation specified according to site, of known etiology, should we adopt the custom of using etiologic terms, it seems to me we should have made a step in advance, albeit a small one.

Dr. W. S. ANDERSON, Detroit: Why do infections of the mucous membrane of the lower respiratory tract occur? We all have patients who very seldom have an acute cold, and we have other patients who repeatedly have acute colds. We know that the lower animals are comparatively free from these infections. Why? What is there in the normal mucous membrane of some persons that resists infection while that of others does not? As I understand it, the normal respiratory tract is sterile, or nearly so. In the nose there are always a few micro-organisms but they do not thrive on a healthy mucous membrane. Going downward we find fewer and fewer germs until the tract below the vocal cords is always sterile in a healthy subject. But, in the presence of an acute cold there will be an abundant growth of organisms along the whole respiratory tract. Why? I have done some work in animals and it is easy to show that the lower respiratory tract is sterile but in many conditions it becomes infected. The mere closing up of the nose of a dog will be followed by a copious growth in the lower respiratory tract. Now we are coming to believe that many of the infectious diseases gain entrance through the mucous membrane of the respiratory tract, meningitis, diphtheria and so forth. In many of these there are local lesions of the mucous membrane, and yet if you attempt to reproduce these lesions in the lower animals you will fail. I have tried over and over again with the Klebs-Loeffler bacillus and have always failed,

and yet if we inject the organisms into the blood of the animals, they will die immediately or in twenty-four to forty-eight hours. The same thing occurs with the organism of pneumonia. If you rub it on the mucous membrane of the respiratory tract you will get no results at all. Why is the membrane in some cases so resistant? If we could learn what the local condition of the respiratory tract is that favors infection it would explain a great many conditions.

Dr. WILL WALTER, Chicago: Infection of the normal mucosa was not investigated but I reported the investigations of others who compare the normal with the abnormal. As to resistance to infection, of course there is the local resistance and the general immunity to be considered. There is also a variation in virulence of infectors and doubtless a specific local resistance. These are the problems which the laboratories of the world are trying to solve and which may not be answered offhand. In obstructed noses we have from mechanical causes practically a stasis and hence lowered local resistance. The opsonic index may be as high as 2 or 2.50 and still you get infection because of this stasis and the consequent poor local resistance. If the nose is shut off from its function by such abnormalities or by being held closed infection naturally occurs lower in the tract. Protection against such infection is one of the reasons for the existence of the nose.

A CLINICAL STUDY OF A CASE OF PSEUDOLEUKEMIC ANEMIA OF INFANCY (VON JAKSCH) *

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In 1889, von Jaksch proposed the term "anemia pseudoleukemica infantum" to designate a pathologic picture characterized by oligocythemia, oligochromemia, considerable permanent leukocytosis, enlarged spleen, slightly enlarged liver, and, sometimes also, enlarged lymph-nodes. Its identity as a distinct disease has been seriously questioned by some authorities on diseases of the blood. Notwithstanding, however, we find in a certain number of infants suffering from a severe form of rachitis or hereditary syphilis, a symptom-complex which is entirely apart from the pre-existing disease.

A number of German authors, Japha, Fischl and Geissler and also some Italians include the grave infantile anemias in the secondary anemias with a toxic basis, practically as a middle condition between anemia and leukemia. Japha does not regard the spleen as the primary focus, and is of the opinion that it has nothing to do with the regeneration of the blood; this is based on the fact that the enlargement frequently becomes reduced before the blood-picture has become normal, while the extent of the enlargement does not correspond to the gravity of the anemia. Japha also believes that deficient new formation of blood constitutes the nature of the affection. At the same time, there may be an exaggerated destruction; but there are only a few cases in which the anemic (granular) degeneration of the red corpuscles is found as a positive evidence of blood poison.

A very notable work on this subject by Banti appeared in 1883. He believed that anemia splenica was a primary disease of the spleen, but he also considered it a splenic form of leukemia. In the case studied and described by him, there were marked fibrous changes in

* Read in the Section on Diseases of Children of the American Medical Association, at the Sixty-first Annual Session, held at St. Louis, June, 1910.

the trabeculae and the follicles of the spleen; and a great diminution of the cells. There were many phagocytic cells containing red corpuscles; the bone-marrow was red and showed a lymphoid condition, but the lymph-nodes were enlarged. The spleen weighed 2 pounds, 7 ounces; the liver weighed 2 pounds, 12½ ounces. The blood showed a diminution of leukocytes and the red corpuscles were between 2,000,000 and 3,000,000 per cubic millimeter. Koplik reports nine cases in which the blood-picture was as follows:

The hemoglobin ranged from 28 to 65 per cent.; the count of red blood-cells or erythrocytes fell as low as 1,400,000 and in others was as high as 4,448,000; and the leukocytes ranged from 5,200 to 40,000 and 80,000 to the cubic millimeter. In all cases there were nucleated red blood-cells, normoblasts and megaloblasts from 7 to 15 per cent. In some cases the white cells varied from 10,000 to 80,000 to the cubic millimeter in a given case, with erythrocyte count of 2,699,999 to 3,799,999.

He regards von Jaksch's anemia as a severe secondary anemia with or without marked leukocytosis.

Monti and Berggruen report 20 cases. In six of these the red cells were below 1,600,000. Four cases proved fatal; one recovered in which the proportion of leukocytes to the red cells had been 1 to 12.

History.—The case which I observed was that of a male child aged 13 months, admitted to the children's ward of the Allegheny General Hospital, Pittsburg, Aug. 4, 1909. The father and mother are both of German parentage and are apparently in good health. There is no history of tuberculosis or syphilis. The patient was one of twins; the other died at the age of 2 months from diarrheal affection. The patient had always been a weak and delicate baby. He was breast-fed for a few weeks only, and then fed on various foods, principally condensed milk. He had always been pale and cries a great deal. He had never been known to smile.

Examination.—The child was very poorly nourished and anemic. His expression was often anxious; but, as a rule he seemed apathetic. The skin was dry and flabby. Numerous purpuric spots were seen in various parts of the body, particularly in the legs and over the right iliac region. There was considerable enlargement of the lymphatic glands in the cervical, axillary, and inguinal regions. The abdomen was distended. The anterior fontanel was open and about the size of a twenty-five cent piece. The child had but six teeth. The epiphyses of the forearms were enlarged. Rachitic rosary, well marked. Examination of the chest revealed a subacute bronchitis. The heart-sounds were clear and distinct. The liver was found to be enlarged, extending several finger-breadths below the costal cartilages. The spleen was enormously enlarged, the tumor extending far below and to within half an inch of the umbilicus. The notch of the spleen could be felt plainly. There seems to be some pain on palpation. Examination of urine was negative. The examination of the blood showed 27,500 whites, 2,500,000 reds and 45 per cent. of hemoglobin.

Course of Disease.—The foregoing is a description of the condition of the patient on admission to the hospital. He remained in the institution until the time of his death, Aug. 26, 1909, twenty-two days after admission. The progress of the disease was rather rapid and influenced to a considerable degree by intercurrent affections and conditions.

Temperature: On admission, his temperature was slightly above normal, but during the course of the disease varied from 101° to 104° F. partly due to a bronchopneumonia and a purulent inflammation of the middle ear, which occurred on the tenth and twelfth days, respectively, after admission.

Pulse: The pulse was rapid, never below 120, but, until shortly before his death, of fair quality.

Weight: From 15 pounds and 9 ounces, the weight fell rapidly to 13 pounds during the first week. Later there was a gain of about half a pound, which was followed by a rapid decline, reaching 11 pounds on the day of the patient's death.

Skin: In addition to the purpura, which remained and increased throughout the course of the disease, marked icterus was present shortly before death.

Lungs: While, on admission, only a few coarse râles could be heard over his chest, evidences of a bronchopneumonia were present on the tenth day and continued up until the time of his death.

Digestive System: Vomiting and diarrhea occurred frequently. It was extremely difficult to find any food which would agree with the patient. Whey and barley-water were about the only things which he retained.

Urine: At first the urine was normal, but later after the administration of arsacetin¹ it contained a large amount of albumin, also a few hyaline and granular casts.

Blood: As stated above, at the time of admission, the patient had 27,500 whites, these gradually became reduced to 16,000; the reds were 2,500,000 and were reduced to 2,287,000. The hemoglobin remained about the same, 45 per cent.

Differential count:

Neutrophils	6.50 per cent.
Small lymphocytes	61.00 per cent.
Large lymphocytes	18.63 per cent.
Transitionals	1.47 per cent.
Eosinophils	0.28 per cent.
Basophils	0. per cent.
Neutrophils myelocytes	1.18 per cent.
Eosinophils myelocytes	1.18 per cent.
Normoblasts	9.76 per cent.
Polkilocytes	 per cent.
Polychromatophile red cells	 per cent.

Therapy.—The treatment at first was directed entirely to the digestive organs, calomel and bismuth being employed. Later the patient was given arsacetin hypodermatically in doses of 0.05 (gr. ¾) but, on account of the occurrence of albuminuria had to be discontinued. In addition, iron and stimulants were given.

Summary of Clinical Symptoms.—Rachitis, enormous enlargement of the spleen, moderate enlargement of the liver and lymphatic glands, purpura, icterus, bronchopneumonia, otitis media, gastro-intestinal indigestion, albuminuria.

Autopsy.—This was made by Dr. F. Proescher, Pathologist, Allegheny General Hospital.

Spleen, Macroscopic Examination: The spleen measured 10 cm. (4 in.) in length, 8 cm. (3.2 in.) in breadth, and 5 cm. (2 in.) in thickness. It weighed 163 gm. (5 oz. 15 grains) and was therefore about seven times the natural size of the spleen of an infant at that age. Its consistency was firm and it was deeply notched at the lower end. The surface was mostly of a light red color, but in some parts, a dark blue discoloration was seen. On section it was found to be of tough consistency. The follicular markings were obliterated. Numerous irregular but not sharply-defined areas were to be seen. These were about the size of pin-heads, of a grayish white color.

Spleen, Microscopic Examination: The capsule, as well as the trabeculae, was thickened. The connective tissue was deficient in nuclei and hyaline in appearance. The follicles were completely obliterated. Most remarkable was the great interstitial fibrous induration, which had caused almost complete obliteration of large areas of the pulp. This induration was seen to be partly in the shape of large, irregularly scattered areas of greater or less thickness, with remnants of pulp tissue, still enclosed; or, in the form of diffusely scattered, isolated fibers, traversing the entire pulp and passing between the pulp cells. These were exceptionally well defined by the Van Gieson staining. Both the smaller and larger vessels also showed considerable fibrous induration, especially the tunica adventitia, which was enormously thickened. In some places there was also a proliferation of the intima, which led to complete occlusion of the lumen. The enormously thickened adventitia presented a marked tendency to hyaline degeneration. The fibers of the connective tissues were conglomerated into hyaline masses

1. Arsacetin is sodium acetyl arsanilate, a compound derived from sodium arsanilate. It is claimed to be less toxic than sodium arsanilate and to possess the advantage of keeping well and being sterilizable without decomposition. Adult dose.—0.1 gm. (1½ grains) to 0.05 gm. (7½ grains). For internal administration 0.05 (¾ gr.) 3 to 4 times daily. See N. N. R., 1910, page 30.

containing no nuclei. The lumina of the majority of the blood-vessels were empty, but, where they were filled with blood, erythrocytes pale and indistinct were found, beside which were seen many mononuclear and a few polynuclear cells.

In addition, there were seen cavities, either scattered or in groups, or even long and winding, and lined by a single layer of spindle-shaped endothelial cells. They were enclosed by a dense ring of round cells, some of which were surrounded by a thick hyaline non-nuclear tissue. That we were not dealing with blood-vessels was established by the fact of the uniformity of structure of the surrounding tissue, which allowed of no distinction of the different layers of the blood-vessels. We were in all probability dealing with a cystic dilatation of the lymph spaces. The pulp consisted of numerous blood-cells and round-cell elements of varying sizes, averaging from 3 to 6 microns in diameter. These mostly showed a round or slightly lobulated nucleus, with scanty protoplasm. Some of the nuclei were rich in chromatin, others showed only a framework of chromatin; many of the large mononuclear cells showed eosinophil granulations. In addition, a few spindle-shaped cells resembling endothelium cells were found. Large giant-like cells with phagocytic enclosure of red blood cells were scarcely seen.

Liver, Macroscopic Examination: The liver was 9 cm. ($3\frac{1}{2}$ in.) in length, 16 cm. ($6\frac{1}{2}$ in.) in breadth, 4 cm. ($1\frac{1}{2}$ in.) in thickness and weighed 345 gm. (11 oz. $\frac{3}{4}$ drams). It was of firm consistency, its edges were sharp and the color a yellowish red in some places, while in others it was more of a yellowish color. On section it was of a bright yellow or red color. The acinous delineations were quite plainly visible. At the periphery the acini were of a sulphur yellowish color; but, in the center more of a yellowish red and cloudy. Throughout the entire parenchyma there were numerous punctiform hemorrhages. The vessels were not enlarged. The tissues were moist and on pressure a thin, bloody fluid exuded copiously. The gall-bladder contained a large quantity of tough yellowish green bile.

Liver, Microscopic Examination: The parenchyma of the liver showed extensive tough infiltrations, especially the middle acinous zone. The liver cells in the center of the acini showed parenchymatous degeneration, partly sclerotic, partly necrotic. Irregularly scattered were seen larger and smaller hemorrhages (extramedullary blood formation) as well as larger and smaller round cells, with deeply stained nuclei. Some of these were round, others lobulated. The periportal connective tissue was very much thickened and infiltrated with round cells; also the walls of the hepatic artery and portal vein. Van Gieson's stain showed regularly distributed interstitial induration.

Kidneys, Macroscopic Examination: The two kidneys together weighed 100 gm. ($3\frac{1}{2}$ oz.); adrenals 6 gm. (90 gr.). The renal capsule was easily detached. The surface was lobulated and of a pale yellow, with numerous punctiform hemorrhages. On section the cortex was found to be enlarged, of a pale yellow color and slightly protuberant. The cortical marking was only present in form and was very much obliterated. Small white stripes were visible. The pyramids were somewhat more sharply defined by a more yellowish color. The left kidney presented practically the same appearance as the right one, but was somewhat redder in color. The mucous membrane of the renal pelvis was pale, with numerous punctiform hemorrhages.

Kidneys, Microscopic Examination: Both kidneys showed severe parenchymatous degeneration, with extensive necrosis of the epithelium. Slight hemorrhages into the glomeruli and uriniferous tubules were found. Hyaline masses were found in some of the canals, especially in the collecting ducts. On frozen section and with the Sudan stain fatty morphosis was found in some of the tortuous and straight uriniferous tubules.

Heart: The heart weighed 50 gm. ($1\frac{1}{3}$ oz.) and was well contracted. The musculature was of a cloudy, gray, yellowish color. Numerous small hemorrhages were found beneath the epicardium. The valvular apparatus was intact, the for-

men ovale closed. The intima of the large vessels, as well as that of the valves, was slightly discolored, a yellowish green.

Lungs: The lungs weighed 175 gm. (5 oz. 125 gr.). **Right lung:** In the upper lobe there was an irregular slightly depressed circumscribed spot about the size of a twenty-five-cent piece. The spot was discolored and dark gray in appearance. In the lower lobe there was also a spot about the size of a ten-cent piece, which was also depressed and dark gray in appearance. On section the entire pulmonary parenchyma seemed to be studded with the above-described areas. It was entirely airless, and on pressure a dark red turbid fluid exuded. **Left lung:** In the upper and lower lobes similar areas were found.

Stomach: The stomach contained a small quantity of fluid. The mucous membrane was atrophic and showed numerous punctiform hemorrhages.

Bowels: A small quantity of greenish white masses were found in the duodenum. The mucous membrane of the jejunum was also atrophic. In the colon a large quantity of fluid and dark green feces were found. The mucous membrane of the transverse colon was very much injected; that of the ascending and descending colon was pale.

The autopsy findings in this case correspond closely to those in the case described by Banti. Here we also find the fibrous changes in the trabeculae and the follicles of the spleen, and a great diminution of the cells. We also have a diminution of leukocytes and the red corpuscles.

The diagnosis of this case as belonging to the class of cases described under the term of pseudoleukemic anemia of infancy can scarcely be questioned. The low leukocyte count, the diminution of the red blood-corpuscles below half the normal, together with the enlargement of the spleen, moderate enlargement of the lymph-nodes distinguishes it from leukemia and simple or pernicious forms of anemia. Whether this case is to be considered a secondary anemia in connection with the rachitis with a toxic basis; or, as a distinct organic disease, it is difficult to decide. The organic changes in the spleen and liver would indicate a primary diseased condition of that most important blood-making organ, the spleen. The evidence seems about equally divided and in view of the fact that so far but few autopsies have been recorded in these cases, it is impossible to come to any definite conclusion in this single case. I hope that by having carefully studied and recorded the findings of this interesting case I have added a small mite to the literature of this important disease.

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ABSTRACT OF DISCUSSION

DR. J. F. HULTGEN, Chicago: In the last few years I have had a number of similar cases which have made me think a good deal of this question. The case just reported undoubtedly belongs to that class of anemias of infancy which we call, for want of a better name, pseudo-leukemia. Some of my recent cases have shown a combination of leukemic and leukanemic changes; that is, not only very marked qualitative and quantitative changes of the leucocytes, but also marked alterations in the number and form of the red blood-corpuscles. Others run into a true leukemia and end in death. Dr. Elterich's case does not belong to this last special class and I am glad he mentioned that. Assuming that all the leukocytes develop from the small lymphoid cell, those infectious characterized by specific changes in the blood must be due to an irritation of the leucogenic system. Whether or not this condition is recognized at an early stage, or advances to a splenic anemia or Banti's disease before it is diagnosed makes no difference. It is the same disease, only a later stage. I think we are almost smothered by a multitude of names in the description of the so-called pseudo-leukemia of childhood. Any severe dis-

turbance of the infantile leucopoietic system may in the first place produce partial or complete leukemia. Whether we have a polynuclear or a mononuclear or a lymphatic leukemia, does not alter the case. It simply depends on the hastening or the slowing of the processes of maturation of the lymphocytes. Later on, after months or years the patients come to our offices, and finding a large spleen, ascites, etc., we call it Banti's disease. The pathogenesis of infantile pseudo-leukemia does not reside in the spleen alone. It depends on infectious, or toxi-infectious lesions of the lymphoid tissues. I think it is easy to believe that as long as there is some leucopoietic tissue left there may be an acute leucogenic crisis. The blood examination then would show a leukemia. A blood examination made later on will likely show a splenic anemia, or an *anémie primitive* of French writers, a leukanemia, or Banti's disease, etc. Or the patient may go on to a pernicious anemia. This is also easy to understand if we consider that in irritation of the leucopoietic cells we must also have a hematopoietic irritation. I think that all these anemias should be looked on as the results of an infection of the lymphoid or leucopoietic system in the gastro-intestinal canal, which may or may not be noticed at the time of its occurrence. Its various forms are nothing but the variations common to all individuals. In childhood the death of these patients is due to exhaustion. Perhaps there are toxic symptoms, but they usually die with the clinical picture of asthenia, though this is often preceded by bronchopneumonia. I think the abdominal symptoms are always at the bottom of this condition, though they are not usually recognized.

DR. C. F. WAHRER, Fort Madison, Iowa: I have seen three cases that would come under the author's name of the infection. The first patient, after five months, died. The second after three months died, and the third recovered. The recovery may throw a little doubt on my diagnosis, but I did the best I could with the means at hand. We had a symptom-complex of an affection that we may class under the diagnosis named by the essayist. The children all had the symptoms that have been named briefly by the author. There was anemia, there were purpuric spots and a history of syphilis in the ancestry of the first two; in the last one, a gastro-enteric trouble with numerous and profuse hemorrhages from the intestinal canal. The treatment in the first two cases was addressed chiefly to the origin of the disease, i. e., the syphilitic condition, and in the last case it consisted mainly in the administration of Fowler's solution, iron and remedies addressed to the gastro-intestinal tract. This child, after ten years, is still in perfect health.

DR. THEODORE J. ELTERICH, Pittsburg, Pa.: I am not in a position to classify these cases according to their pathologic findings. I had only one case in which the diagnosis seemed positive. In one or two other cases no blood count was made and the patients died before they could be removed to the hospital for observation. The symptoms were those of rickets, with an enlargement of the spleen and anemia.

CHOKED DISC IN ITS RELATION TO CEREBRAL TUMOR AND TREPHINING *

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When I was invited to be the guest of the Section on Nervous and Mental Diseases of the American Medical Association, and to read a paper before this famous body, I felt highly honored, and it was with the greatest pleasure and thanks that I accepted this invitation. Having leave to choose the subject I did not hesitate long and selected the important theme of choked disc in its relation to cerebral tumor and trephining.

There is hardly any point which has for so long a time been subject to as much controversy as the patho-

genesis of choked disc. In spite of numerous works, experiments, clinical observations and pathologic-anatomic investigations, we have not succeeded in bringing about an agreement between the various investigators on this topic. Just lately the old quarrel on the pathogenesis of choked disc has become active again in Germany.

Surgical experience being, to my mind, of great importance for clearing up the origin of choked disc. I was convinced that America would be the best forum for discussing this question, because America possesses a greater experience on the effect of and indications for trephining in cerebral tumor than we do in Germany, thanks to the studies and works of Taylor, Mills, Spiller and Frazier, De Schweinitz, Weisenburg, Putnam, Knapp, James Bordley and also to the new methods of Harvey Cushing, not forgetting, however, England's contributions, by Horsley, Gowers, Gunn and Leslie Paton.

The fact that I was an ophthalmologist at first and devoted myself to neurology later has brought it about that I have been occupied for twenty years with studying the changes of the fundus of the eye in diseases of the brain.

By request of Professor Uthoff of Breslau, I made a report on the pathogenesis of choked disc before the seventy-sixth *Versammlung Deutscher Naturforscher und Aerzte* in Breslau in 1904. Since that date so much new work has been published, that I deem it necessary to give you a brief outline of the stand we take on this important question in Germany.

REVIEW OF WORK AND THEORIES ON CHOKED DISC

Tuerck was the first, who in 1853, when describing a case of cerebral tumor with retinal hemorrhage expressed the idea, that the changes in the fundus of the eye were due to increase of intracranial pressure. However, only in 1860 the scientific world became acquainted with the so-called choked disc through Albrecht von Graefe's work on complications of optic nerve-inflammation with brain diseases. Von Graefe saw the cause of choked disc in brain pressure, due to cerebral tumor, the sinus cavernosus being thus compressed and the voiding of both the vena ophthalmica and the vena centralis retinae being inhibited; bringing about dilatation and twisting of the retinal veins as well as edematous swelling of the optic disc. He explained the inflammatory changes, first, as originating from retinal extravasations of blood; secondly, from the irritating effect of swelling of the optic disc, still increased by toughness of the scleral ring.

Von Graefe's hypothesis, however, soon encountered opposition. Hughlings-Jackson took the optical affection for a reflex symptom, but it was the anatomic facts found by Sesemann which principally disproved the theory. It was he who proved that the central retinal vein has an off-flow into the facial veins, quite independent of intracranial pressure. Moreover, Sesemann argued that compression of the sinus cavernosus could not be effected as easily as one might think. Later these statements made by Sesemann in 1869, though accepted everywhere, even by von Graefe himself, have met opposition from the well-known anatomist, Merkel, and from Gurwith, who, on the basis of extensive anatomic investigations, argued that the greatest part of the orbital venous blood flows into the sinus cavernosus. Quite recently J. Tudeich and Krüdener have endeavored to uphold the old theory of von Graefe

* Read in the Section on Nervous and Mental Diseases of the American Medical Association, at the Sixty-first Annual Session, held at St. Louis, June, 1910.