

## **An international system of ophthalmic practice / edited by Walter L. Pyle.**

### **Contributors**

Beard, Charles H., 1855-1916.  
Pyle, Walter L. 1871-1921  
Goulden, Charles  
University College, London. Library Services

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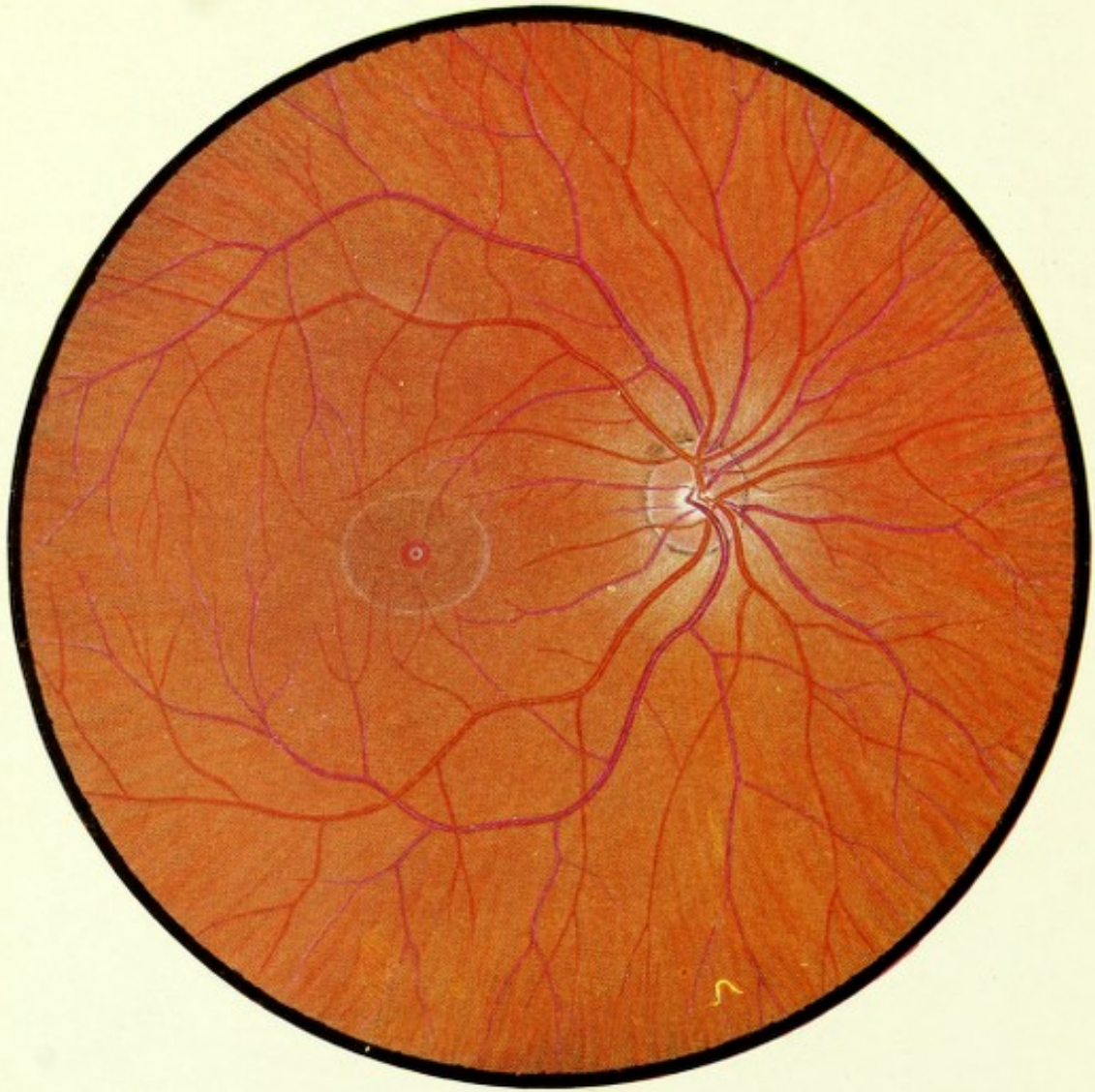


FIG. 1.

AN INTERNATIONAL SYSTEM  
OF  
OPHTHALMIC PRACTICE

EDITED BY

WALTER L. PYLE, A. M., M. D., Philadelphia  
MEMBER OF THE AMERICAN OPHTHALMOLOGICAL SOCIETY

OPHTHALMIC SEMIOLOGY AND  
DIAGNOSIS

BY

CHARLES H. BEARD, M. D.

SURGEON TO THE ILLINOIS CHARITABLE EYE AND EAR INFIRMARY (EYE  
DEPARTMENT), OCULIST TO THE PASSAVANT MEMORIAL HOSPITAL  
AND THE NORTH STAR DISPENSARY (CHICAGO), MEMBER  
AND EX-PRESIDENT OF THE CHICAGO OPHTHAL-  
MOLOGICAL SOCIETY, MEMBER OF THE AMER-  
ICAN OPHTHALMOLOGICAL SOCIETY, ETC.

WITH THIRTEEN COLORED PLATES AND SEVENTY-ONE  
FIGURES IN THE TEXT



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## PREFACE.

This book is the outcome of a long-standing conviction on the part of the writer that the rank and file of the medical profession would welcome a treatise of this nature: one that would deal systematically, succinctly and solely with questions of symptomatology and diagnosis as concerns the eye. Thus to get, in a single small volume, information not otherwise to be obtained save through research that, to the vast majority, would be impossible for lack of both time and opportunity.

There is no other separate volume, in any language, devoted exclusively to differential ocular semiology. The nearest approach to a work of this description is to be found in a limited portion of the fourth volume of the French Encyclopedia of Ophthalmology. And the author desires to state, in grateful acknowledgment, that he is more indebted to that and other volumes of this, the greatest of all works upon the eye, for help in the preparation of what the following pages contain, than to any other one source.

The original drawings of the illustrations for the Second Part are from a large collection, all in color, the making of which has most agreeably occupied certain hours of the author's life from the year 1884 down to the present moment. That they have been generously praised by numerous colleagues, and have been awarded a diploma of honor by the American Medical Association are things gratifying to one's vanity if not proof positive of intrinsic worth. They do possess the merit, at least, of having been all painted directly from the living human fundus, and with whatever faithfulness to nature, in every particular, their delineator was able to bestow. To have rendered all the reproductions in color would have too greatly increased the cost, hence the price, of the book.

CHARLES H. BEARD





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# OPHTHALMIC DIAGNOSIS AND SEMIOLOGY.

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## CHAPTER I.

### INTRODUCTORY.

Notwithstanding all the modern methods and appliances for research and verification, it is doubtful whether diagnosis, which constitutes the key to the successful practice of medicine, is more a science than an art. What is true in this particular of medicine in general would seem to be truer still of a specialty in medicine. The study of disease in the text-books and in the laboratories is quite different from that in the living human subject; yet the two kinds of investigation are absolutely and hopelessly interdependent.

Our profession is awakening to some of the evils of specialization, which will, in time, doubtless be overcome. Too often the student of medicine is led into the fallacy of *preparing* himself, from the beginning, for a chosen specialty. His progress through not only the medical school, but often also through previous ones, has been by the elective route, and always with his chosen specialty in view. Unless he is well advised, or excels in judgment, the scope of his studies will be too limited. He risks becoming more of an intricate scientific automaton than a thinking, reasoning individual. His power to see into things with the mind's eye may remain underdeveloped, and he may find himself in that class whose members are rapidly increasing, wherein perspicacity, personal equation and common sense do not count for their true value. It would be better for the student to lay the widest



possible foundation, then practise long enough to demonstrate the possession of any peculiar aptitude, and thus allow a specialty to choose *him*.

Affections of the eye are, in the main, but the local manifestation of a morbid process elsewhere in the system, hence the necessity for broad grounding, and of keeping up to date in matters relative to the whole domain of medicine. On the other hand, the purely practical physician is yet more to be deprecated. Leftwick refers to two kinds of diagnosticians that are to be avoided. The mind of the one, through recent observation and reading, is obsessed with the idea of a certain rare and interesting disease. In the mind of the other conservatism amounts to prejudice. The first is, in most instances, young and enthusiastic, and he is able in a surprisingly short time, to find a whole "series of cases" of his favorite disorder. The second, more often further along in years, finds only the commoner diseases. The victim of obsession sees too many unusual affections, while the victim of perspective and assumption finds too few. Of the two the first, so long as he acts according to his best reasoning and is only misled by his fallacies, is probably the lesser evil; but when he allows himself to build upon mere sophistries, making facts fit theories, he becomes far the worse.

**The Interrogatory.**—The qualifications requisite in a good diagnostician are not all acquired. Those inestimable subtler attributes are, in great measure, innate, though they are all susceptible, through proper thought and training, of wonderful development. To meet, professionally, the infinite variety of character and disposition of mankind his equipment must be physical and moral as well as mental. He is at once sleuth, advocate and judge. Toward the secretive and suspicious he is like a detective to ferret out the facts and to trace the sequence of things; to the deceitful, the over-voluble and the taciturn he is like a legal inquisitor, to draw forth the whole truth from patient and patient's friends; then, like a justice he sifts, weighs and interprets.



Throughout he must be patient, considerate and dignified. Severity of manner, as affected by many of our old-time physicians, is rarely excusable now; while flippancy and levity are faults not to be condoned. Any amount of zeal and interest he may evince is welcomed by his client, provided it is not too plain that he has back of it some selfish motive—that, for instance, his interest is purely from the scientific standpoint. But the slightest signs of indifference on the part of the examiner, such as yawning, asking the same questions over again, or other evidences of abstraction will not be tolerated or overlooked.

With the view to inducing candor and insuring freedom from embarrassment the interrogation is best conducted in private or, at least, out of hearing of others. The inquiry is begun, not by asking what the matter is, for that is precisely what the patient, in most instances, would like to find out, but how the consulting party has been troubled, or what he has observed in connection with his ailment. It is especially desirable to know approximately the date of the very beginning of the trouble and the qualities of those earliest signs; and, also, whether or not there had previously been similar attacks. We are all aware how important it is to regard with wholesome skepticism the declarations of patients and their acquaintances. It is a curious fact that even the more intelligent are rarely able to give a clear and concise description of the character and progress of a disorder of the eye; what then can be expected of the more ignorant? Equally unreliable are the statements of their relatives and friends. Bearing this in mind, many evidently think it smart to break brutally into the patient's narrative from time to time with such expressions as, "I don't want to hear that," or "I don't care what someone else has said." This is not only belittling to the examiner and uncivil to the patient, but it is impolitic and unwise, for it prejudices the attitude of the examined and tends to prevent that reciprocity of interests which is so desirable. Besides, in a mass of this sort of information are apt to lie scattered



precious gems of enlightenment. It were better adroitly to steer the recital of the views and conclusions of his doctor, his acquaintances and of himself concerning his malady as nearly as possible in the shortest road of relevancy, but, by all means, to permit the recital.

One of the greatest difficulties in conducting an interrogatory is to avoid suggesting too plainly what should or should not be the reply. This applies peculiarly to such matters as the rainbow rings in glaucoma, the wavering vision in intra-ocular syphilis, etc. It is always advisable, when practicable, to question the near relatives and the consorts of those who consult us. Of still greater value than evidence given by those persons already mentioned is that given by a physician who may have had the case in charge, and this should be obtained whenever possible. The examination is divided into **objective** and **subjective**. By far the more important is the first, for here we rely for our knowledge of the nature and progress of the affection chiefly upon the evidences of our own senses.

**Personal History.**—This branch of the inquiry is divided into two parts, viz., that which refers to the period of childhood and adolescence, and that pertaining to adult life. Something is to be gained by inquiries relative to the term of gestation, to the state of the mother's health during that period, to the character of the process of parturition, and whether forceps were employed; also to the time of appearance of the first teeth, and to the age at which walking and talking were first possible. Above all things in this connection, as a causative factor in abnormal conditions of the eyes, is **hereditary syphilis**. This disease affects particularly the cornea and the uveal tract, and its scars and stigmata are borne through life. These are evident in hair, nails, teeth, contour of head and face and affections of the joints. Were it not slightly complicating matters one might add a third period, viz., that of **advanced age**, or the period of degeneration. Senile changes in the eye and its appendages are usually apparent soon after middle life, and are readily



accessible for study. They are most easily observable in the skin of the lids, in the conjunctiva, in the cornea, in the lens, in the chorioid and in the retina.

For information as to the diseases in the first part of life we must call largely upon the parents and other older relatives. Of special moment also in this category are such general diseases as diphtheria, because of the ocular paralyses that accompany it; meningitis and scarlatina, because of the ocular metastases they occasion; convulsions, on account of their bearing upon soft cataract, etc. Of the local eye-diseases of that period most likely to influence the diagnosis are **ophthalmia neonatorum**, chiefly by reason of the resultant corneal and lenticular opacities and irregularities; phlyctenular keratitis, with or without cervical adenopathies; the existence of convergent squint, with special reference to a monolateral amblyopia; and as to any evidence of other acute inflammations of the conjunctiva, such as trachoma or vernal conjunctivitis. Inquiry must be made as to injuries of the eye and its adjacent territory, for these have an extensive field of application in seeking the causes of both the external and the internal diseases of the organ. It may be that the memory of some of these early traumatisms is a source of humiliation and embarrassment to the parents or guardians, hence they may tell of them either grudgingly or not at all. The consciousness of guilt or neglect in this connection, or the fear of the imputation of guilt or negligence, would act in the same manner to prevent a confession.

Of the diseases and accidents of adult life that influence one's opinion as to a given affection of the eye, just as in relation to those of the earlier years, **syphilis** both inherent and acquired, stands first as a cause. There is no part of the eye or its appendages exempt from its noxious influence. Next, perhaps, comes **manner of living**, as, for example, the use of alcohol and tobacco, leading to affections of the optic nerve; while over-eating and other vicious practices, and habitual idleness, with their attendant phases of auto-intoxication and infection, are often agents to be considered.



Then, the rôle of **occupation** in the causation of affections of the eye is a most important one. Instance the uveitis of brass-workers, the conjunctivitis of cigar-makers and the cataracts of glass-blowers and handlers of naphtha.

**Hereditary Antecedents.**—In this case the effects are either **direct** or **immediate**, meaning some physical abnormality that is imparted to the child from a parent, or **indirect**, implying that the transmission of the defect has skipped at least one generation. The last is known as **atavism**, another phase of which is **homochronism**, a term used to denote the occurrence of the same disorder in one or more members of the same family at the same period of life in more than one generation. Myopia and juvenile glaucoma perhaps furnish the most conspicuous of the homochronous traits. Among the more frequent of the hereditary and atavistic affections of the eye are anomalies of the lids (ptosis and epicanthus), of the muscles (squint and nystagmus), of the globe (microphthalmos), of the lens (luxations and cataract), of the uveal tract (colobomata of iris and choroid), etc.

**Consanguinity.**—Blood-relationship on the part of parents plays a certain rôle in the pathology of a few diseases of the eye, notable among which are **retinitis pigmentosa** and that unique form of retinal degeneration seen in connection with **amaurotic family idiocy**.

**Familial Affections.**—A more extensive category of abnormalities of the eye is comprised in this class. Both heredity and consanguinity may sometimes influence their origin, yet as they are not, in most instances, the result either of direct heredity or of kinship of the subject's parents they are placed in a class by themselves. To it belong infantile glaucoma (**hydrophthalmus**), hemeralopia (without retinitis pigmentosa), atrophy of the optic nerve, chorio-retinitis, color-blindness (**Daltonism**), congenital cataract, and a certain rare variety of corneal opacity.

**Errors of Assumption.**—It is hardly necessary to insist that the examination of the eyes be thorough and complete, and that it include *both of these organs* in every instance. No



matter if the patient positively affirms that only *one* eye is at fault, careful investigation as to the state of *both* is none the less imperative. Nor is one to be influenced by the fact that the patient has made his own diagnosis. Even though he be so explicit, for example, as to state that at a certain hour of the day while standing on a certain street corner he felt a cinder fly into the right eye; that it is lodged beneath the upper lid; that the eye is very painful, and especially so when the lid moves over it; that he has come to have the foreign body removed, and for no further examination. Certainly the finding of a cinder at the spot indicated would be strong corroborative evidence that nothing else ailed the eye, but it would not be conclusive; for, on the one hand, we have known small foreign bodies to be lodged on the cornea or beneath the upper lid without causing any discomfort, and, on the other hand, have had patients describe with equal minuteness the symptoms of a foreign body in the eye when they were, in reality, those of an ulcer of the cornea, or of an acute inflammation of the iris.

**Error of Precipitation.**—Not infrequently the signs of a disease are not sufficiently pronounced for one to make from them a positive diagnosis by a single examination. It is far better to postpone the giving of a definite opinion than to venture upon one while standing on uncertain ground. If the malady be an acute one, and in the incipient stage, a few hours wait will furnish the needed clues and be to the advantage of all concerned. If it be a case of longer standing further examinations and diligent study will usually serve to clear away doubts as to the true nature of the affection. Meanwhile, the patient, or his spokesman, may be frankly told that matters have not advanced far enough to warrant a confident decision—a bit of candor, however, which is not always appreciated to the full—or one may resort to a little strategy, and put off the parties most interested with some innocent pretext, and with the possible aid of a placebo.

If a given sign or symptom were invariably conclusive



evidence of the presence of a given anomalous or pathologic condition the greater portion of this book would be superfluous. Since, however, the same symptom may be common to two or more affections, it is of the utmost importance not only to know its individual meaning, but to interpret the significance of its associated symptoms. Indeed, the presence of the symptom of itself is often unintelligible save through the aid of these associated signs. Again, appearances in or about the eye are prone to be deceptive or illusory; for example, a visible abnormality may puzzle one as to its exact location. Or one symptom may closely simulate another, and the two be utterly unlike as regards their signification. And yet again, the differences between conditions that are pathologic and those that are physiologic are, in many instances, so slight as to mislead even the trained observer. The following pages are offered to those who need such help, in differentiating these things, as the subject matter affords.

The grouping of diseases according to their lesions or symptoms is, in the present state of etiologic science, no longer practicable. Their classification in the present volume will, therefore, be regional and anatomic as regards the objective symptoms. The subjective symptoms will be mentioned only incidentally. For it is how the case *reads*, and not how the patient *talks*, that counts most with the diagnostician.



## CHAPTER II.

### THE LIDS.

The **palpebral region** is comprised within the area immediately overlying the orbital rim, and includes the supercilia, or eye-brow. Exclusive symptoms and those that are common to more than one disorder will be given, also a brief description of them, their several causes, and their variations according to the cause.

**Alopecia**, or **Alopecy** ( $\alpha\lambda\omega\pi\eta\hat{\varsigma}$ , a fox).—This name originated in the fact that manginess and loss of hair is common among foxes. Baldness affecting the supercilia is not over-rare. In most cases it is due to syphilis, and women are more subject to it than men. It is a conspicuous sign—"one to be read in an omnibus," as the French say. This form may be diffuse and show a general thinning of the hairs of the brow, while those that are left no longer lie smoothly, with their points toward the temple, but are mussed and scraggy. Or the loss of hair may be circumscribed, and when this occurs at a point or points away from either end of the brow it imparts a broken look.

Next in frequency is **alopecia (or keratosis) pilaris**, a disease characterized by a horny cone of the skin around each hair, and usually congenital. There is a diffuse thinning of the hairs or, rarely, complete absence of them, and the skin of the region is roseate and wrinkled. Then there is **alopecia areata**, or **pelada**, which when occurring in the head causes baldness in spots. In the brow, however, the loss of hair in this form is usually complete. More rare is alopecia of the brow from **seborrhea**. Another form, and one that concerns us little, is that from **leprosy**.

**Ciliary Alopecia or Madarosis**.—The eye-lashes are most



frequently lost from chronic **blepharitis marginalis** and **seborrhea marginalis** (See "Marginal Affections"). In proportion as the disease is severe and prolonged is the destruction of the hair follicles complete. A transient ciliary alopecia is sometimes noticed in syphilis at about the period of the roseolous skin eruption. Like syphilitic epilation elsewhere it may be permanent as regards the lashes.

**Superciliary Tumors.**—These are either congenital or acquired. The first are non-inflammatory, and the most frequent among these is the **dermoid cyst**. Its favorite site is at the temporal end, or *tail*, of the eye-brow. These cysts may vary in size from that of a pea to that of a large filbert, are moderately firm, non-fluctuating, and are attached to the periosteum, but not to the skin. If very small the growth may escape notice for a number of years, when it is supposed by the parents to have been acquired. Of extreme rarity is congenital tumor in this region from **encephalocele**, or hernia of the dura-mater, with or without cerebral contents. The suture most often at fault is that between the frontal and the ethmoid bones, hence its habitual seat is at the supero-internal angle of the orbit, or at the *head* of the eye-brow. It may be of any size up to that of a hen's egg, is fluctuating and is not at all or only partially reducible. A still rarer congenital form is the **angio-fibroma**, whose point of origin is the periosteum of the orbital rim.

Of the **acquired tumefactions** of the superciliary region, and closely resembling in some respects the kinds just described, is **mucocoele of the frontal sinus**. It, too, is fluctuating, non-inflammatory, and is not reducible. If the tumor communicates with both sinuses its situation is more median, if with only one it is a trifle above the supero-internal angle of the orbit of the corresponding side. A small exploratory incision will evacuate reddish, mucoid fluid, containing perhaps crystals of cholesterin and globules of fat. The swelling may become inflamed and rupture spontaneously. It is important to make an early diag-



nosis in these cases to forestall deeper infection, such as meningitis.

The most frequent, however, of the non-inflammatory acquired tumors of the superciliary region is the **hyperostosis of syphilis**. It is most often located beneath the tail of the eye-brow, on that part of the rim of the orbit which covers the lacrimal gland and has been mistaken for a tumor of this organ. It is neatly circumscribed, fixed, and of almost or quite bony hardness. Its growth is rapid, and it is free from inflammatory symptoms or tenderness. Vigorous anti-luetic treatment will dispel all doubt as to its nature as the tumor disappears. Such tumors have never the ivory-like consistency and racemose formation of ordinary exostoses. The affection is sometimes symmetrically bilateral. My first observation of the condition was in the case of a negro pugilist. A growth the size of a pigeon's egg was situated over the site of each lacrimal gland. Possibly blows from the fist figured in their origin. The man insisted on their removal as they encroached on his temporal fields of vision, and were unsightly. One was removed. It was like a dense fibroma. The other soon disappeared under mercurial inunctions and iodides.

**Anesthesia of the Lids.**—The sensory nerves of the lids are branches of the fifth, or trigeminal nerve. Those of the upper lid come through the intermediary of the ophthalmic nerve of Willis; those for the lower, through that of the superior maxillary. Sensibility is referred to as of three kinds, viz., **tactile**, **thermic**, and **algescic**, meaning respectively that to the **touch**, to **heat**, and to **pain**. The tactile and the algescic are those commonly tested—the former by means of a hair or shred of paper, the latter with the aid of a pin. Disturbances of sensation in the brow and lids when circumscribed, or, at most, when confined to one lid, are of peripheral origin; when both lids are involved the cause is usually central. Superciliary and palpebral anesthesia are most often met with in **herpes zoster** of those regions, and appear *after the appearance of the characteristic eruption*.



In this disease it is the area supplied by the nerve of Willis only that is usually the site of the anesthesia, etc., the lower lid being seldom affected.

The ophthalmologist is also interested in other forms of deep paralysis of the fifth nerve because of their relation to that form of corneal ulcer known as **neuro-paralytic keratitis**.

Palpebral anesthesia is present in **leprosy**, and, sometimes, at the beginning or later in **tabes**. It is also observed in fractures of the roof or apex of the orbit, and in osteo-periostitis (syphilitic) in the vicinity of the Gasserian ganglion.

**Hysteric Anesthesia of the Lids.**—This variety simulates both the local, or palpebral, and the more general forms, such as hemianesthesia, the chief points of differentiation being the fact that the lack of sensation is apt to be more strictly regional, that is, restricted to the orbito-palpebral area of one or of both sides, and the absence of any signs of skin-eruption and of trophic disturbance in the cornea.

**Blepharospasm.**—Under this heading will be considered any variation of reflex action of the orbicularis, from normal winking to intense tonic spasm, exclusive of the **tics**. Normally there are from two to four nictations of the lids every minute. The rapidity of the movement is increased in proportion as there is fatigue of the accommodation or of convergence, or of the retina itself, and when there is irritation of the cornea and conjunctiva. **Neurasthenic** subjects wink oftener than normal persons, while the eyes of those afflicted with **paralysis agitans** and **exophthalmic goitre** are characterized by long intervals of fixed staring. In the last named disease this peculiarity is called **Rosenbach's sign**. Blepharospasm when it is merely an exaggeration and prolongation of nictation is spoken of as **clonic**, and the persistent and forcible closure of the palpebral fissure as **tonic**. The first is peculiar to the slight irritations and excitations of the eyes of children between eight and ten years of age, and is often mistaken for a tic, or for incipient chorea. Given a child with a mild follicular conjunctivitis



and a neuropathic disposition and there will be excessive nictation. Errors of refraction are also conducive of the trouble.

**Tonic Blepharospasm.**—This is a prominent feature in most all the severe inflammations of the conjunctiva, the cornea, and of the iris. It is especially pronounced in children and young adults who are suffering from phlyctenular ophthalmia and from interstitial keratitis, and in subjects of active trachoma, whatsoever may be their age. The fibres of the orbicularis become abnormally developed and through a sort of habit the blepharospasm may continue after the disease which induced it has disappeared. This form of blepharospasm is to be distinguished from the **hysteric** type. The first ceases while the eye is anesthetized by cocain, the second only in narcosis. In the hysteric type there is an absence of lesions adequate to cause the spasm, and palpebral anesthesia may be present; and, besides, in hysteria the lids of only one eye may be affected, and anesthesia may be present in the skin of one or both pairs of lids.

**Ptosis, or Blepharoptosis, is derived from** πίντείν, to fall. Literally the word ptosis, πτώσις, means a falling down, and βλέφαρο-πτώσις, a falling down of the lid. Blepharoptosis is the more comprehensive and explicit term, but it might as well apply to a dropping of the lower lid as to that of the upper—as, for example, in non-cicatricial ectropion of the lower lid. Both usage and convenience make ptosis preferable for the designation of the condition under discussion, which is partial or complete closure of the palpebral fissure through falling or dropping of the upper lid; and the ptosis is accordingly described as **partial** or **complete**. Further classifications are into **congenital** and **acquired**, into **paralytic** and **non-paralytic**, and into **unilateral** and **bilateral**, or **double**.

**Congenital Ptosis.**—The most frequent form of that which is present from birth is decidedly distinctive in character. It is bilateral and partial. The lids look incomplete, insig-



nificant and lacking in detail. The palpebral fissures are mere narrow openings, wanting in crispness, curvature and finish. The degree of closure is usually unequal as regards the two eyes. In most instances there are present other ocular anomalies, such as epicanthus, squint, microphthalmos, nystagmus, colobomata of the uveal tract, ectopia and opacities of the lenses and subnormal and incorrigible acuity of vision. Not infrequently there is underdevelopment of the entire organism—mental and physical—and, in rare instances, such additional connate irregularities as supernumerary digits and dislocation of the hips. In many cases there are no accompanying defects. This is particularly true of unilateral congenital ptosis, in which there is apparently full development of the palpebral structures, or, at least, but slight departure from the normal. The facial aspect presented in bilateral congenital ptosis is, in the highest sense, characteristic; the brow is evenly and permanently corrugated, the supercilia are situated abnormally high and the curve of their arc is exaggerated. In the higher degrees, besides the features just mentioned, the head is thrown backward, imparting what Hirschberg once called "the air of an astronomer." These constitute the **syndrome of Hutchinson**—that is, wrinkled forehead, lifted eye-brows, smooth or unfurrowed upper lids, and backward inclination of the head, and they are merely the vicarious resources for uncovering the pupils. In the milder degrees the subject has merely a sleepy or a stupid look, and the ptosis is not quite apparent except in looking upward.

The movements of the globe are usually restricted, particularly as concerns abduction and superduction, partly, perhaps, as a result of the general lack of development, but chiefly because of non-use. The drooping lid cuts off the field both in the upward and outward directions, hence the superior and the external recti are left idle. By patient efforts, however, one can usually demonstrate the fact that these muscles are capable of function. This is an important point to remember in the choice of a surgical measure for



ptosis, as, for instance, whether or not it is feasible to resort to the Motais method in a given case. For here, to obtain the best results, the superior rectus must not be devoid of power to contract.

Congenital ptosis is also hereditary and familial to a remarkable degree, and has in a number of instances been observed in several successive generations of the same family. In hereditary ptosis, especially in the lower and more moderate degrees, there are sometimes presented curious associated anomalies of movement of the upper lid—for example, it is momentarily lifted with certain efforts of mastication and of deglutition.

A case of this kind has recently come under my observation. The patient was a Jewish girl, fifteen years of age. The parents were not relatives, and there were seven other children, all normal. There was unilateral, left, ptosis, also deorsumvergens of the left eye, the downward deviation amounting to 15 degrees. This was the result of non-use and relaxation of the superior rectus, though it was evident that this muscle was, in a measure, capable of contraction. In all other respects the girl was fully developed. The peculiar feature was that although the levator was ordinarily incapable of lifting the lid, the eye opened with every downward movement of the lower jaw in mastication, and closed with every upward one. This anomaly was so pronounced as to attract the notice of the parents when the girl was a mere infant. A carefully performed Motais operation corrected *both the ptosis and the downward squint*, and put an end to the phenomena associated with mastication. It has often been asserted that the tendency of the Motais operation for ptosis is to cause downward deviation of the globe. In this instance, the downward squint, instead of having been increased by dragging of the upper lid upon the transplanted tongue of the tendon, was actually decreased because of the tonus thus imparted to the superior rectus. The writer has made fourteen Motais operations none of which resulted in downward deviation.



Among the **anatomic causes of congenital ptosis** are:

- A. Absence or partial development of the corresponding nerve-centre.
- B. Anomalous distribution and destination of the muscular and tendinous fibres of the levator.
- C. Absence or arrested development of the levator.
- D. Absence of the conducting neurons between the nuclear centre and the levator fibres.

Other kinds of congenital ptosis will be referred to later.

**Acquired ptosis** is divided into **paralytic** and **non-paralytic**.

Paralytic ptosis, in turn, is either **idiopathic** or **traumatic**. The lesions that give rise to idiopathic or spontaneous paralytic ptosis—or through which the function of the motor nerve of the levator is suspended or its substance destroyed—are either peripheral or central. Prominent among the peripheral lesions are tumors, hemorrhages, inflammations and abscesses in the orbit, or extension into the orbit of disease processes primarily of the accessory sinuses, especially those that involve the vicinity of the sphenoidal fissure. Affections of the nucleus of the third nerve, or those of the tissues immediately surrounding it, constitute the central causes of paralytic ptosis. Of the primary causes of this form of ptosis syphilis stands at the head, whether the lesion is peripheral or central. Syphilitic ptosis may appear three weeks or three decades after the initial lesion. It is nearly always accompanied by paralysis of other branches of the third nerve, though that of the levator branch is sometimes isolated, and is often seen in connection with such phases of the disease as tabes and general paresis. The manner in which leutic ptosis is produced varies greatly, though it is nearly always through direct or indirect lesion of the third nerve. Such lesion may be situated anywhere between the terminations in the levator to the cerebral cortex—as from orbital gumma and osteo-periostitis, from gummatous inflammation at the base, from alterations in the roots of the oculo-motor or of its nuclei, or from syphilis of the cortex itself. Tabetic ptosis is occasionally transitory, and



may be recurrent. Isolated paralysis of the levator branch of the third nerve is strongly suggestive of a **central lesion**. Other affections of which ptosis may sometimes become a symptom are syringomyelia, Basedow's disease, acute polio-encephalomyelitis, cerebral tumors, multiple neuritis, Landry's disease, polymyositis, zona, and simple senility. Exceptionally it is seen in such neuroses as hysteria, ophthalmic migraine, Gerlier's disease, and in certain toxemias, such as diphtheria, botulism (ptomain poisoning), saturnism (lead-poisoning), poisoning from the oxide of carbon, in uremia, and in general infections, such as the eruptive fevers and typhoid. Bilaterality is rather infrequent in all forms of paralytic ptosis.

**Traumatic Paralytic Ptosis.**—This variety has been observed in the newly born, consequent upon the use of obstetric forceps in the delivery, and it may be mistaken for congenital ptosis. **Direct injury** of the levator or of its motor nerve may occur in numerous ways, as by section, laceration, etc., or the traumatism may be **indirect**, as of the adjacent tissues, giving rise to pressure upon these structures (levator or its nerve). Especially is this true of those injuries that involve the sphenoidal fissure. Hystero-traumatic ptosis comes under this head.

Complete paralytic ptosis is characterized by total closure of the palpebral fissure, and absolute immobility of the lid. If there is no function whatever remaining in the levator the fact may be ascertained by fixing the patient's superciliary region by pressing the eye-brow tightly against the orbital rim with the thumb to eliminate the action of the frontalis, then telling him to close and then to open the eyes. There is no movement in the upper lid at the last command if the levator is paralyzed. The subject must not be told to look upward, for then the lid may be slightly raised by the action of the superior rectus. Of course, there are all grades of both ptosis and of movements of the lid in the partial levator paralyse. Diplopia, either latent or manifest, is present in most cases of syphilitic ptosis, whatever the degree of the



paralysis or of the ptosis. Such is true also of some of the paralytic toxic ptoses. Congenital ptosis, when it seems paralytic, may be recognized by the absence of secondary contractures and deviations, by the absence of diplopia, by the existence of nystagmus, and by the maintenance of convergence until it is lost in lateral movement, that is, by carrying the test-object beyond the point where binocular fixation is possible.

**Ptosis of Sympathetic Origin.**—A peculiar form of paralytic ptosis is that caused by lesions that affect indirectly or directly the cervical sympathetic and, in consequence, the involuntary muscle of Mueller. Since the experiments of Pourfour du Petit, in 1712, and those of Claude Bernard in 1859, demonstrating the relation between the sympathetic and the ocular apparatus, many observations of this form of ptosis have been reported, and the recent vogue of Jonnesco's operation for chronic glaucoma have furnished many more. Any lesion or affection that involves or causes pressure upon the cervical sympathetic ganglia is capable of producing a certain degree of ptosis. Among these are cervical adenopathies, goitres, neoplasms of the esophagus, aneurysm of the aorta, intra-thoracic tumors and abscess of the mediastinum. These constitute some of the **idiopathic** or **spontaneous** causes, in contradistinction to the **operative** causes, or those from excision of the superior cervical ganglion. All are accompanied in the earlier stages by other phenomena incident to sympathetic paralysis, viz., hypotension of the globe, slight enophthalmos, myosis, hyperemia of the conjunctiva, flushing, anesthesia and elevation of the temperature, and increased secretion of the skin of the corresponding side of the face. This entire category may be observed in the recent cases, particularly in the operative ones.

The spontaneous cases are apt to be brought to one's notice at so late a period that their aspect has become considerably changed. While the ptosis, the enophthalmos, and the myosis may still be present in the later stages, the phenomena relative to the skin of the face may be replaced



by atrophy, local lowering of the temperature, diminution of the sudorific secretion and pallor. Ptosis due to sympathetic paralysis is as a rule of slight or moderate degree. Attempts of the subject to elevate the lid are partly successful owing to the fact that the levator retains its function. The pupil is seldom entirely covered. The accompanying myosis is not very pronounced save in the acute stages—as, for instance, immediately after excision of the ganglion; though the pupil is always sensibly smaller on the affected than on the sound side; and it retains its normal reactions, though to a lessened extent. One may readily understand that it would be easy to overlook a case of double or bilateral sympathetic ptosis. The condition is almost habitually unilateral, hence readily recognized as sympathetic ptosis by the myosis.

**False or Pseudo-ptosis.**—Spasm of the orbicularis, especially when tonic, may be mistaken for ptosis. Under this influence the lids may remain closed for weeks, or even months, at a time. The condition is, as a rule, unilateral. It must be understood that cases of **symptomatic** blepharospasm, from acute inflammatory processes, as of the conjunctiva, the cornea, and the iris, which are usually bilateral and largely under control of the will, are not included in this discussion. The spasm in question may, however, be primarily excited by some irritation of the eye—by a foreign body, by a superficial keratitis, or other cause. An important variety of tonic palpebral spasm is that associated with hysteria—**essential blepharospasm**. It is most often bilateral, and is frequently recurrent. Usually it is accompanied by other ocular signs of hysteria, such as photophobia, contraction of the visual field, inversion of the color fields, amblyopia without apparent cause, and monocular diplopia. On forcible opening of the lids by the examiner the globe is seen to spasmodically deviate in various directions, due to the impact of the light. Other accompanying phenomena are peri-ocular anesthesia and contractures throughout the region supplied by the facial nerve.



The fact that normal closure of the palpebral fissure is accomplished almost wholly by the upper half of the orbicularis leads to the deception in these cases of false ptosis. Most all the other signs of true ptosis are lacking. The brow is not corrugated, the supercilia are apt to be depressed and straightened, rather than elevated and over-arched, and the palpebral skin is less smooth and lax. Hysteric ptosis may even simulate the paralytic form. Here, while the fallen lid is not ordinarily associated with a wrinkled brow, the frontalis is strongly contracted, as in true ptosis, when the patient is told to open the eyes. The upper lid is lax, but when it is raised by the finger it falls much more quickly than in paralytic ptosis. If the patient's attention is suddenly and forcibly diverted by some unforeseen circumstance, or, if observed in the evening, when the light is not intense, the lid may be raised by its own power. In many instances of hysteric blepharospasm and ptosis it is possible to find a spot, usually somewhere in the regions supplied by the trigeminus, or the spot may be situated in a part of the body more remote, where pressure with the finger will cause the eyes to open on the instant, as if by magic. These are called **hysteric pressure spots**. Senile blepharospasm is sometimes of a prolonged tonic character and may even be permanent. It is, as a rule, related to a form of **tic**, either a **convulsive**, or a **painful tic**, or both, and is accompanied by general facial spasm. Here also may occasionally be found pressure spots that may be utilized to relieve or to modify the spasm.

**Ptosis from Alterations in the Palpebral Tissues. Trophic Ptosis.**—This is a large and comprehensive class. To it belong all those cases of non-paralytic ptosis, both congenital and acquired, in which the fallen lid is characterized by an excess of some part or of all of its tissues, or by certain degenerative changes in its structures. Either the contraction of the levator muscle fails, from laxness and stretching of the various ramifications of its tendinous expansions, to be communicated to the lid—**degenerative ptosis**—or the



lid has become so enlarged as to overcome by sheer weight the effort of the levator to lift it—**hypertrophic** or **mechanical ptosis**. When the alterations causing the ptosis concern only the skin of the upper lid the affection is known as **cutaneous ptosis**. Laxity and redundancy of the skin in this location naturally increase with years, and when the condition is so pronounced as to prevent normal elevation of the lid, the condition is designated as **senile ptosis**. The same term has often been applied to a **pseudo-ptosis** (cutaneous); that is to say, while the levator is able normally to raise the tarsus, the folds of skin are so voluminous as to fall below the free border and partially occlude the palpebral fissure. Sichel assumed that this condition was due to an excessive accumulation of fat in the folds in question, hence called it **ptosis adiposa**.

Typical senile ptosis is perhaps best represented by the form so designated by Fuchs. Its development begins with middle life. It is mostly limited to women and is nearly always bilateral. The first symptom is a sense of impotence in attempting to open the eyes, and the fingers are often called upon to aid in the act. It is the result not of lost function in the motor nerve of the levator, but of a primary atrophy of the muscle itself. It is true, however, that a certain degree of ptosis is necessary to a complete picture of the countenance of age, for weakness of the levator here but keeps pace with that of the muscular system at large, and this form of ptosis is as certainly senile as are the drooping shoulders and the bended knees. In reality it depends upon the fact that the fibrous bands connecting the palpebral skin with the tendon of the levator and with that of the superior rectus have become stretched and flaccid, and no longer act to hold up the integument. Such at least is the opinion of Fuchs, who has studied the pathology of these cases, and described them under the name of **blepharochalasis**. According to the researches of others, however, notably those of Lodato, the essential features are atrophy of the derm and of the elastic fibres, with enlargement of the



blood-vessels; therefore, it has more recently been termed **angiomegaly of the lids**, precisely what has also been denominated **dermatolysis**. Indeed, there seems to be considerable confusion as to what constitutes cutaneous ptosis—how much is due to overgrowth and how much to degeneration; and observers are far from being in accord as to any dividing lines between this and the forms yet to be mentioned. Under the names blepharochalasis, angiomegaly, and dermatolysis have been described seemingly identical conditions, and again, very different, not to say distinctive, affections of the upper lid giving rise to ptosis in some form or degree.

Mention may also be made of a cutaneous disease that has been known to affect the upper lid to the extent of producing ptosis, namely, **cutaneous geromorphism**. It is characterized by extreme relaxation and flaccidity of the integument in divers parts of the body, which assumes a bagginess that gives it, even in young subjects, precisely the appearance of the skin of the aged. The condition may become general over the entire body.

**Hypertrophic Ptosis.**—According to the views of certain clinicians there exists a distinct variety that may be thus styled. Briefly described, it is bilateral, peculiar to neuro-pathic females, young or middle-aged, and capable of being produced by external irritating causes, such as exposure to cold. Its development progresses by successive attacks of edema and vascular congestion. In the intervals between the attacks, the skin of the affected lid is shiny, of a grayish rose tint, or slightly blanched, and falls over the free border like an empty pouch (Hotz).

**Ptosis Elephantiastica.**—The forms last mentioned are not always easily distinguished from those of the congenital and acquired varieties that are found in connection with elephantiasis in its numerous phases. Elephantiasis of the upper lid is extremely rare, and here, in turn, the lines separating this condition from those of lymphangioma and lymphangio-sarcoma are far from being sharp. Congenital elephantiasis of the lid is more frequent than that which is



acquired. The hyperplasia, small at first, later increases, and sometimes attains enormous proportions. It progresses by alternating periods of activity with hyperemia, edema, etc., and of quiescence, each crisis resulting in augmentation of volume. The process may extend to the scalp and to the whole side of the face. Blood-vessels, lymphatics, muscles, nerves, connective tissue, and even the underlying bone (**fibromatosis**) may share in the enlargement. It is, as a rule, unilateral. In the soft forms the skin is little altered.

**Acquired Palpebral Elephantiasis** develops spontaneously, without apparent cause, and, in most instances, by crises of inflammatory manifestations resembling erysipelas. It may begin at most any period of life and in males and females alike. It is unilateral in the vast majority of cases. Exceptionally the lower lid is also affected. Heredity plays no rôle in the affection.

**Neurofibroma** of the upper lid causes ptosis which might be mistaken for beginning elephantiasis or one of the other forms. The skin of the lid may be abnormally pigmented, or singularly free from pigment—most often the former holds. The growth is congenital or noticed in the earlier years. Its progress is very slow and free from inflammatory symptoms. It is unilateral. What constitute the great distinguishing feature are nodules along the courses of the nerve branches beneath the adjacent skin that on palpation feel like small coils or bunches of hard string—as has been said, “like knotted fiddle strings.” Because of these the affection is also called **plexiform neuro-fibroma**. When the nodules concern the fifth nerve they are likely to be painful and sensitive, particularly in their earlier stages.

**Cicatricial Ptosis.**—This is, in some respects, allied to non-paralytic traumatic ptosis. The class includes ptosis from long-standing trachoma—**ptosis trachomatosa**—and that from **symblepharon**. The partial falling of the upper lid resulting from the combined effects of the long-continued blepharospasm, from the ultimate atrophy of the conjunc-



tiva and degenerative changes in the tarsi consequent upon chronic trachoma constitutes, by far, the commonest of all forms of ptosis. It is usually bilateral and easily recognized upon everting the drooping lids. Symblepharon, especially that which concerns the upper fornix and cul-de-sac, is not infrequently the origin of cicatricial ptosis.

**Coloration and Discoloration of the Lids.**—The skin of the lids varies greatly in color even within physiologic limits, that of some individuals being deeply pigmented, especially after middle life, that of others being of a delicate "black and blue" tint. Dissipation in the young of both sexes, and menstruation in young women, will sometimes cause discoloration of the lids, particularly in the infero-nasal sulcus. Small circumscribed spots of bright red in the skin of the lids are, for the most part, **arterial angeomata**. They are closely resembled by the tiny blood stains of **purpura hemorrhagica**. Both are clear red and do not vanish on pressure. The angiomas are of a brighter tint and are congenital. Small **venous angeomata** are of a still darker, bluish-red, are deeper seated and more diffuse in outline.

**Ecchymosis of the Lids, Subdermal Hemorrhage or "Black Eye,"** sometimes also called **suggillation**, is notably the result of traumatism. The injury is usually from without, as from a blow or fall, but may be from a strain, as in elderly subjects, from sneezing or vomiting or efforts at stool; or in children, from whooping cough. Ecchymosis at the nasal side of the lids is diagnostic of fracture at the base of the skull, if occurring within a few days after injury to the head. It occurs in certain subjects after even slight surgical operations upon lids or globe, and spontaneously in some cases of **scurvy** and **typhus**.

**Edema of the Lids.**—This term indicates simply a watery infiltration and swelling of the more lax tissues of the lids. Except when accompanied by severe inflammation, the color of the puffed lids is likely to be paler than the normal skin, and the accumulation of water often imparts a translucent look to the swelling. The edema may be **unilateral**



or **bilateral, acute or chronic**, and arises from an infinity of causes, both local and remote. It is not difficult to recognize, chiefly for the reason that *the swelling always pits on pressure* with the finger. It is not always easy, however, to determine its origin. It comes sometimes as a precursor, or, at least, before its cause can be ascertained, or it is the result of the bite of an insect received in sleep, the point where the poison entered bearing no mark. An alveolar abscess has been known to cause a bilateral palpebral edema before it was known that there was any dental trouble. Monolateral edema of the lids is much more indicative of a local affection than the bilateral form; that is, of a cause situated in the eye, in the appendages of the eye, or in the orbit. Therefore, unless there exists some acute inflammatory or infectious condition of the skin, such as erysipelas, or of the conjunctiva, or of the globes, such as double iridocyclitis, the presence of a recent bilateral edema of the lids often points to some disease or disturbance of remote origin. It may be the **nephritis** following scarlatina or, especially in conjunction with suppuration of the middle ear, it may be septic **phlebitis or thrombosis** of the cavernous sinus.

**Chronic nephritis** and the **ingestion of arsenic** produce bilateral edema which is more marked in the lower lids. **Sarcomata** and **septic processes** originating in the **median sinuses** of the skull may cause bilateral palpebral edema, but there is almost sure to be in addition exophthalmos, or cellulitis of the orbit, etc. Monolateral palpebral edema is of greater frequency and is secondary to a greater multiplicity of affections than the double type. The primary disease may be in any **accessory sinus**, in the **walls of the orbit**, as **syphilitic osteoperiostitis**, in the orbit, in the globe, in the conjunctiva, or in the lids themselves. Sometimes the edema of the lids, whether of one or both sides, is out of all proportion to any obvious lesion. Such conditions may be the result of poulticing.

**Differential Diagnosis.**—Unilateral edema of the lids may be confused with chronic inflammation or dislocation of the



lacrimal gland, but here the characteristic pitting of the skin on pressure will be lacking. Palpebral edema cannot well be confounded with emphysema of the lids on account of the crepitation obtained on pressure when the tissues are infiltrated with air. Myxedema often shows itself, first in the lids by imparting to them a puffy look, but, even in the absence of the other signs of the disorder in the face and hands, edema can be excluded by the greater firmness on palpation, the absence of pitting upon pressure, the perfect bilateral symmetry and the chronicity. Abundance and prominence of the orbital fat, particularly when the fascia overlying it is lax, may simulate bilateral palpebral edema, but pressure with the finger will at once show the deception. **Giant urticaria** of the lids is, perhaps, the one affection that is most difficult to tell from edema of those parts. It, too, may occur on one or both sides. The chief distinguishing features are its regular passing and recurrence, its simultaneous appearance on other parts of the body, and the rather limited area of the lids that is involved as compared with edema.

**Marginal Affections.**—The skin of the free border of the lids is extremely delicate and sensitive, and is not only highly susceptible to irritating influences from without, but readily participates in the morbid processes of its immediate neighbors—the conjunctiva on one side and the skin of the lids on the other. The objective signs of marginal troubles are **redness** and **thickening**, the occurrence of **yellowish-white scales** around the bases of the cilia, and the presence of **hard, yellow-brown crusts** along the free borders. These various conditions are all commonly referred to as **blepharitis-marginalis**. Simple redness of the lid borders is usually due primarily to exposure to acrid substances in the atmosphere, to wind and dust, to dissipation, or they are secondary to eye-strain from errors of refraction, from improper use of the eyes, or to inflammations of the conjunctiva by propinquity and extension, and to dacryocystitis, because of the accumulation and overflow of tears. When the latter



conditions have lasted for a considerable length of time crusts of dried secretion and blood form along the cilia, the margins of the lids become thickened, and we have **chronic catarrhal blepharitis**. An exaggeration of the process becomes **blepharitis hypertrophica**, and leads to **mucous ectropion**. This form is called **blepharitis vasomotoria** and **hyperemia marginalis**. It is most prevalent among the younger adult blondes.

The variety distinguished by scales, or "**branniness**" among the eye-lashes is known as **blepharitis squamosa**, **blepharitis sicca** and **pityriasis marginalis**, but is more properly designated **seborrhea marginalis**. The irritation of the free border in this form, especially in the early stages, causes hypersecretion of the lubricating glands of lids and conjunctiva, and these drying around the cilia cause crusts to form, particularly during sleep. The third type, that marked by the hard crusts and the swollen and infiltrated lid borders, is called **blepharitis eczematosa**, or **blepharitis ulceroza**. Both names are equally applicable, for it is a true eczema, and the pathognomonic features of the disease are the deep ulcers that are to be found beneath the crusts close to the roots of the cilia. In most cases it is coexistent with eczema of the face and scalp, and it is peculiar to puny and neglected children.

**Blepharitis Parasitica.**—The lid margins are subject to the invasion of parasites, both vegetable and animal. Of the vegetable parasites the **trichophyton fungus** is the one most often to cause blepharitis. Recent observations have demonstrated that this marginal infection is not as rare as formerly supposed. Another fungus, the **acharion** of **Schonlein**, produces the disease known as **favus**. The literature contains probably some eight or ten reports of cases of blepharitis in some form caused by this infection. Among the animal parasites to attack the edges of the lids the most common is **pediculus pubis**, or the **crab-louse**. Of the subjects infested with the invasion of this insect in the regions of the cilia are children who are under the age of puberty and



devoid of pubic hair. **Pediculus capitis** has also been known to produce blepharitis. A yet rarer variety is blepharitis from **demodex-folliculorum**, or **pimple mite**. So closely can some of these parasitic kinds of blepharitis simulate the other forms that microscopic investigation is sometimes necessary to a diagnosis. This is especially true concerning the vegetable forms, as the pediculi are visible to the naked eye. The pediculi produce appearances at the margins easily confounded with the simple inflammatory and the seborrheic varieties of blepharitis, while the fungi may furnish many of the clinical aspects of blepharitis eczematosa. It is well known that the last disease has often erroneously been called **sycosis**, or a form of "barber itch." This is an infection of the hair-follicles by the **staphylococcus** and occasionally affects the lid-margins. Eczematous blepharitis is chiefly distinguishable by the exudative pits and the ulcers underlying the crusts, also by the tender age of the subjects. Cases of both sycosis and trichophytosis lack the ulcerations, and they are seen mainly in adult males.



## CHAPTER III.

### THE LACRIMAL APPARATUS.

In this connection we are concerned mainly with the secretory disturbances of the lacrimal glands and but little with the excretory, or those of the lacrimal canal. Ordinarily the secretion of the orbital portion of the gland—the tears—is in abeyance, the moisture which serves to lubricate the conjunctival sac being furnished by the palpebral portion and by the accessory subconjunctival glands of Krause. Departures from the normal in the amount of secretion of tears relate to the function of the orbital lacrimal gland. The palpebral portion may be ignored because of its insignificance, and the fact that it is often lacking entirely. There is then to be considered either **hypersecretion** or **hyposcretion**.

**Hypersecretion of the Lacrimal Gland.**—When tears are secreted in perceptible quantity, that is, sufficient to cause “swimming” of the eye, or to overflow upon the cheek, it is through the agency of one of three causes—(a) **irritation** of some part of the ocular or nasal system, (b) **an affection of the nervous system**, or (c) **the psychic phenomena** in connection with the emotions. The first may be called **lacrimation**, the second **watering** and the third **weeping**. The term lacrimation seems to have been a favorite one of ophthalmologists in all lands and from time immemorial. By usage it has a different signification from either the weeping or the watering of an eye. By lacrimation we understand that excessive production of tears which is the **result of irritation** of the terminations of the first branch of the fifth (trigeminal) nerve, whether it be in the eye, or in its appendages; in the nose, as from pungent fumes in the air; exposure to cold wind, a foreign body or strong light on the cornea; or an acute inflammation of the conjunctiva or of



the nasal mucous membrane. It is nature's method of irrigation or lavage of the conjunctival sac, and is a valuable sign of warning, for example, in the after-treatment of cataract-extraction. For the dressings to become wet with tears usually means a threatening complication. The overflow of tears resulting from disease and obstruction of the lacrimal canal come properly under this head for the reason that the said overflow is the result either of the inflammatory processes in the canal itself, or of exposure in the open air. This, however, is the form of lacrimation that is usually meant by the word **epiphora**. In pure stenosis of the lacrimal canal so long as its mucous lining is free from disease, there is, as a rule, no epiphora save when external influences act to excite the function of the lacrimal gland. Such eyes do not, as a rule, water indoors.

Among the **affections of the nervous system** in which lacrimal hypersecretion has been noted as a symptom are **trigeminal neuralgia, tabes dorsalis, exophthalmic goitre and hysteria**. In tabes it has been noted principally in the early stages, and has even been termed a premonitory sign of the disease. Paroxysms of tabetic lacrimation have been described as sudden crises of abundant lacrimation unaccompanied by any disturbance of the mental state. Its presence in the early stages of exophthalmic goitre and its absence in the later stages argues strongly in favor of the theory that it is here due in great measure to the exophthalmos and the infrequent nictitation; though the fact that here also it is often paroxysmal is fairly conclusive that it is not wholly due to local causes. It has even been observed in adolescent girls as a **premonitory symptom** of exophthalmic goitre. In **hysteria** epiphora is often associated with the photophobia and the blepharospasm, and is, in all likelihood, partly due to local irritation and partly to **associate psychic phenomena**.

Hypersecretion of tears has also been observed in **anemia** and, as a transient symptom, in **myxedema** and **pregnancy**. When observed in conjunction with other abnormal conditions epiphora is usually **intermittent** or, to a degree, **periodic**.



It occurs, for instance, on rising, or at twilight or during mastication. Unlike epiphora from stenosis of the lacrimal canal these subtler forms are likely to be more pronounced indoors than when the subject is in the open air.

**Emotional or associated psychic weeping** which accompanies sorrow, pain, joy and exultation needs only to be mentioned here.

**Monolateral lacrimation or epiphora** is significant of a cause to be found on that side. Epiphora of one eye without other symptoms may be set down as due to obstruction of the lacrimal canal of the corresponding side, otherwise the symptom would be bilateral.

**Hyposecretion** of the orbital lacrimal gland does not necessarily result in abnormal dryness of the conjunctival sac, as the compensatory action of the mucous and the accessory lacrimal glands usually prevent this. Lack or absence of moisture here is known as **xerosis**, and is most often the result of the extensive atrophy that follows chronic trachoma which extends to all the glands whose ducts perforate the conjunctiva. According to Morax, xerosis of the conjunctiva is almost always present in the poorly nourished infants and children under two years of age, with lesions of the cornea—especially **keratomalacia**—and who are, for the most part, victims of **hereditary syphilis**. In adults, xerosis occurring in small plaques on the bulbar conjunctiva, has often been cited as a symptom in affections of the liver. This and a moderate hemeralopia constitute a syndrome of primary hepatic disease. The secretion of tears is suppressed in peripheric paralysis of the facial nerve. The eye in the affected side neither weeps in response to the stronger emotions, nor can lacrimation be provoked by chemical or mechanical irritants, such as the inhalation of ammonia, touching the cornea, or tickling the nostril. Dryness of the eye and that of the mouth (salivary) go hand in hand. Both are present in **alcoholic intoxication**, in **diabetes**, in **Mikulicz disease**, and other grave diseases such as **cholera** and **typhoid**.



## CHAPTER IV.

### THE CONJUNCTIVA.

The importance of studying the normal and physiologic appearances of any visible portion of the living body with the view to a better comprehension and interpretation of its various anomalies and pathologic conditions cannot be too persistently urged. What is true in this respect in a general way is peculiarly true as regards the conjunctiva. This membrane is divided for purposes of study, according to the region it occupies, into three principal parts, viz., the **palpebral** or **tarsal**, the **retro-tarsal** or **transitional folds**, which also forms the cul-de-sacs or fornices, and the **bulbar**, or **scleral conjunctiva**. Although the conjunctiva is commonly referred to as the ocular mucous membrane, it is only the palpebral portion that actually possesses the properties of a true mucosa. Hence it is that diseases which are peculiar to mucosal tissue when they affect the conjunctiva always have their seat primarily in the lining of the lids. The bulbar portion, including as it does the anterior layer of the cornea, has more the qualities of epidermis, therefore dermal lesions which concern also the eye, first attack the scleral or the corneal conjunctiva.

**Color and General Aspect.**—The normal tint and topography of the conjunctiva vary with the location. The palpebral part presents a delicate scarlet tinge. Its ground is of an even yellowish tint and perfectly smooth, save near the lateral extremities of the tarsus, where it is naturally somewhat red and the surface is finely granular. One can distinguish through it the outlines of the Meibomian glands. It is the numerous small blood-vessels that cross it vertically and closely parallel to form tiny ramifications and anastomoses near the free border that impart the scarlet tint. The tint of the retro-tarsal part is roseate, and the membrane is here



thrown into large horizontal folds—more pronounced above than below—though the immediate surface is smooth except along the upper or convex border of the superior tarsus, where it has a slightly paler *furred* look. The retro-tarsal folds constitute the fornices of the cul-de-sacs. The bulbar or scleral portion is practically colorless and transparent, and one sees only the small blood-vessels that traverse it. At the inner angle, where it joins the lacrimal caruncle, it folds upon itself vertically, making the **plica semilunaris**, which is reddish-pink in hue. The bulbar conjunctiva varies in color chiefly because of corresponding variations in the tint of the sclera, which shows plainly through it. When one says that the eye is white or pearly, as in phthisis, or yellow, as in icterus, or blue, as in thinning of the sclera, it is the sclera and not the conjunctiva that imparts the tinge.

**Pinguecula.**—Certain circumscribed modifications in the color of the bulbar conjunctiva are to be differentiated as to their nature. All are more or less familiar with the pinguecula, a small, oval, yellowish elevation, situated slightly below the horizontal meridian, near the limbus of the cornea, and usually at the nasal side. It is very common in adults and the aged. It is a benign lesion, is not progressive, has no pathologic significance, and requires no professional attention; though, when it is the cause of particular anxiety, as sometimes happens, it may be removed by excision or by a ligature. It received its name because it was formerly thought to be composed of a mass of adipose cells. Fuchs, who has studied the affection, considers it a hyaline degeneration of the fibrous tissue of the conjunctiva, with proliferation of the elastic tissue. Its yellow color is due to the abundance of these elastic fibres. On close scrutiny the mass is seen to be made up of a number of closely packed, small elevations. It is the result of irritation from external causes; and injection of the surrounding conjunctiva renders it more apparent by contrast. It is movable with the conjunctiva. A pinguecula is not to be confounded with a



rare congenital tumor which occurs in the same location. The growth in question has not the regular sharp oval outline of the pinguecula. It is of a duller yellow, larger and more elevated. This is the **dermo-epithelioma of Parinaud**. It also is benign, and is of very slow growth.

**Spotches of dark brown pigment** in the sclera and ocular conjunctiva are common in negroes and are sometimes seen in the eyes of brunettes of the white race. They are flat and irregular of outline and remain unchanged in size, and must not be confused with **melano-sarcomata**, which are of round or oval outline, are apt to grow rapidly larger, and are distinctly raised above the surrounding surface. A casual or untrained observer might mistake a circumscribed **ectasia of the sclera** for such a tumor. This slight elevation is the result of thinning of the sclera, and its blue-brown color is imparted by the choroidal pigment which shows through. Small **ecchymoses** of the bulbar conjunctiva are extremely frequent. They are of a bright carmine tint, flat and sharply defined, and of rather regular shape when recent, then taking on a yellow tinge, becoming smaller and of more fantastic and less clearly defined shape and disappearing after a few days. One is often able to elicit the fact that there has been a slight injury or a sudden strain, such as a cough or sneeze, to account for the blood. They may be mistaken for **nevi** of the conjunctiva, which are exceedingly rare, are congenital, and not given to changes in size; or they may resemble a spot of circumscribed inflammation of that membrane. A **phlyctenule** is sometimes surrounded by an area of bright redness and may be so small or so nearly to the stage of disappearance as to add to the deception. Such redness, however, is not a uniform stain, and on close inspection is seen to be made up of a number of tiny blood-vessels. The more extensive subconjunctival ecchymoses, not infrequently covering the entire sclera and reaching even to the borders of the tarsi, are of a much darker red. They present a rather startling ocular picture, but the absence of all signs of inflammation in lids,



cornea and iris, in conjunction with the almost inevitable information as to the cause, makes the diagnosis easy.

**Hyperemia** or redness of the conjunctiva from injection, is of no special semiologic value, but it is a symptom to be differentiated. It is caused by local irritants, by foreign bodies in the conjunctival sac or on the cornea, by excitation of the ocular branches of the fifth pair of cranial nerves, by any deeper inflammation of the eye, as well as by conjunctivitis. It is the redness of the bulbar conjunctiva that first attracts attention, and here the color is truly imparted by the conjunctiva and not by the sclera beyond. It varies in tint from pale rose, through shades of deeper rose, scarlet and crimson, to bluish-red, or magenta. At a glance, the redness may seem to be uniform and homogeneous, but on close inspection it is seen to be composed of a network of tortuous vessels which overlie the sclera, and which are movable with the conjunctiva. It is not to be confounded with the deeper kinds of ocular redness such as those of scleritis and cyclitis. In these, the injected vessels lie deeper, they are not movable and their trunks are straighter. In scleritis, the redness is circumscribed. In iridocyclitis, the ciliary region, or zone, is often of a rose or rose-violet tint. The redness of acute inflammatory glaucoma so closely resembles that of acute conjunctivitis that there is danger of confusing them. When in doubt as to the presence of these more serious maladies, confirmation lies in seeking for the other objective signs of those diseases—in the iris, in the cornea, in the intra-ocular tension, and for the characteristic subjective phenomena.

**Chemosis.**—This is a specific name for the edema that affects the bulbar conjunctiva and that of the cul-de-sacs. It can occur in connection with almost any acute inflammation of the conjunctiva or of the globe or even of the lids. It begins as a pearly or slightly opalescent rising or puffing up of the scleral conjunctiva, without redness, and as it becomes more intense, rising higher in a central direction, extending to the limbus of the cornea, surrounding and



overlying that membrane, and to the cul-de-sacs in an outward direction, until, in the extreme cases, all the looser portions of the conjunctiva protrude from the palpebral fissure, like a lobulated giant bleb. The first opalescent appearance in the swelling of the ocular conjunctiva soon gives way to a yellowish, translucent look, and this, in turn, to an increasing redness. So long as the lids remain free from swelling, the ocular conjunctiva will tend to emerge from the palpebral fissure; but as the upper lid swells more and more and becomes harder and harder, as is usually the case, it extends mainly downward, pushing the edema before it, as it were, and forcing the watery infiltration into the lower portions of the conjunctiva, so that, in the moderately severe instances, it is that part of the membrane which constitutes the lower fornix that is left extruding between the lids in the form of a plump, red, horizontal fold. Through exposure to the air, the most prominent part of this fold becomes dry and hard, then necrosis and ulceration take place.

Chemosis of the bulbar conjunctiva is an early symptom in many acute ocular affections. It is present with the deeper inflammations of the lid such as **hordeolum** and **chancre**; and with those of the globe, like **iridocyclitis** and **suppurative panophthalmitis**, as well as in most all affections of the cornea. It is sometimes the first warning of infection of the wound after extraction of cataract. It is an illusory symptom, inasmuch as it is not, by its intensity, an index of the severity of the ailment which it accompanies. It is as apt to occur in conjunction with slight **injuries**, **traumatic** and **chemic**, as in the severe ones; and it is as common with the local poisonings by insects, drugs and plants, and the milder forms of conjunctivitis, as in the graver ones. It may be experimentally and safely produced and readily observed by instilling a drop of the 5 per cent. solution of **dionin** into the conjunctival sac of the normal eye.

**Cicatrices of the Conjunctiva.**—The scars left from former diseases and even from those of long standing that are still active, and also those that are the result of injuries such as



burns, cauterizations and perforating wounds have a certain etiologic value. The record they leave is more often traceable in the tarsal than in the other parts of the membrane. The extensive scars often consequent upon squint-operations are readily seen in the bulbar conjunctiva, but smaller ones, as, for example, those from flying particles of iron or steel that penetrate the sclera are more difficult or impossible to find. When invisible, the scars may sometimes be found by cocainizing the eye and picking up the conjunctiva all about over the sclera by means of delicate mouse-tooth forceps and noting the point of adherence. Cicatrices in the tarsal conjunctiva show themselves as lines or irregular patches of much lighter color than the surrounding tissue, and they have a sort of drawing or puckering look.

Scars due to the ravages of **trachoma** are by far the kind most frequently seen. A scar peculiar to this disease is one that extends horizontally through the entire central portion of the inner surface of the superior tarsus. It causes an incurvation of the tarsus that is called the **trachomatous groove**. The beginning of the stage of contraction of the upper tarsus in trachoma is marked by the advent of tiny isolated irregular scars scattered mostly over the upper two-thirds of the tarsal conjunctiva. The scars from trachoma are usually bilateral and occur in conjunction with other sequela of the disease, such as absence of the inner angle of the upper lid-border, trichiasis, atrophy of the retro-tarsal folds, etc. The cicatrices that result from burns, as of molten metal, lime, etc., are denser, more uneven, and are situated most often in the lower cul-de-sac, though they are not infrequent at the inner angle and in the middle of the upper tarsal conjunctiva. A large percentage of them are complicated by symblepharon. The scars that follow vernal catarrh of the tarsal conjunctiva have a peculiar pitted and trabecular look.

**Pemphigus of the conjunctiva**, when not too severe, is said to leave scars that are distinguishable by reason of



the stellate shape they assume. The worse cases leave complete atrophy of the conjunctiva, with xerosis. The marks of **herpes zoster seu ophthalmicus** can usually be recognized at a glance, not perhaps so much because of their individuality as regards the conjunctival scars, though these are isolated, small, round and dense, but particularly by the aid of lesions that are almost sure to be present on the forehead and upper lid in the same case. These consist of deep round pits, interspersed with larger irregular, cicatricial concavities, and not extending beyond the median line of the face. Cicatrices from the contagious diseases of the conjunctiva are, as a rule, bilateral, as also are those from acids, lime, etc.

**The Semiologic Value of Contagiosity.**—According to Morax **bilaterality** of a conjunctival affection on the one hand and previous **history of the case** on the other—aside, of course, from the more precise data furnished by the microscope—are the two points upon which one can establish the contagious nature of the conjunctivitis. Bilaterality alone is ground for strong presumption that the disease is contagious. In our inquiries, however, it is not enough to ascertain merely whether or not both eyes were affected, but one must go further and find out, if possible, whether the two eyes were similarly affected at the same time; or, if successively affected, what was the length of time between the first appearance of the disease in the two eyes. It is rare, except in ophthalmia neonatorum, that both eyes are exposed simultaneously to the identical contaminating influence. Most often it is the secretion from the contaminated eye that affects its fellow. Since this second contamination habitually occurs about the time that the symptoms were first noticed in the first eye affected, it will be found that the lapse of time between the successive attacks of the conjunctivitis in the two eyes will correspond to its period of incubation.

Bilaterality is the rule in most all the contagious forms of conjunctivitis—the more severe the disease, the more con-



stant is this symptom, though plentiful exceptions are not lacking. The fact that gonorrheal conjunctivitis in adults is often an exception is but another proof of the assertion, for the patient has reached the age of discretion, and has been instructed how to take precautions to prevent infection of the fellow eye. Inversely, all cases of bilateral conjunctivitis are not of necessity contagious.

**False Membranes.**—In view of the frequency of this symptom and the great variety of affections of the conjunctiva in which it may, at some stage, occur, its mere presence has hardly more significance than its absence. But it is not to be regarded lightly, for there is something to be gained from careful study of the character of the membrane itself and its relation to collateral symptoms and to the clinical history. We are wont, of course, to associate a pseudo-membrane in this connection with diphtheritic conjunctivitis, a disease so grave that wholesome fear of it in the mind of the clinician and prompt action when it threatens are most commendable things. As the diagnosis of this disease will be fully discussed further on, only a few points will be mentioned here. The false membrane of **diphtheria**, as compared with others, is distinguished by greater whiteness—or, what is better termed, *canescence*—by greater denseness, thickness and toughness, especially in the early stages. Later it becomes “dirtier” and bloody. Besides, it covers *both* tarsi, and patches of it are often seen on the *ocular* conjunctiva. It may be detached wholly or in part by means of forceps, but is likely to be so tightly adherent in places as to cause bleeding when it comes away. It quickly re-forms and will keep on doing so for a week. The affection is, as a rule, bilateral. The swelling of the lids is excessive and hard. Corneal complications are very frequent. Although false membranes are not uncommon in other conjunctival diseases of infancy and childhood, there is always the possibility of a diphtheritic infection having been grafted upon one that in all other respects save as to the membrane looks plain enough.



The membrane often present with the **purulent conjunctivitis of the new-born** may be the result of the disease or of the previous application of a **chemic agent**, such as nitrate of silver. Most ophthalmologists are familiar with the appearance of pseudo-membranes caused by applications like strong solutions of bichloride of mercury, jequirity, etc. Most all other than the diphtheritic membranes are thin, soft, not detachable wholly or in large plaques by means of the forceps, and have a translucent, gelatinous, yellowish appearance. Sometimes they are mere films of thick, abundant secretion that may be easily wiped off with a cotton swab. They are seen in phlyctenular ophthalmia, but this affection is mostly monolateral, and the other impetiginous eruptions are in evidence also. These membranes are common in the **acute contagious conjunctivitis** caused by the Weeks bacillus, but only during the first three days. Of all the forms of conjunctivitis with false membranes, those that most nearly resemble the diphtheritic variety are sometimes seen in the course or decline of **scarlatina** and **measles**, the result of an attendant **streptococcus infection** of the conjunctiva. There is the same hard, phlegmonous swelling of the lids, and the same frequency of corneal involvement. But the membrane is grayer, more tender, and less tenacious than that of diphtheria. The temperature is high, as opposed to that of the Klebs-Loeffler infection.

**Significance of Pre-auricular and Submaxillary Adenopathy in Affections of the Lids and Conjunctiva.**—Tumefaction of the lymphatic glands corresponding to the territories comprised by the lids and the conjunctiva has a certain interest and importance to the ophthalmologist. It is well known that this enlargement is not a common reaction and that it appears always as an outpost of the defense of the organism against general infection. It ranges from a tiny swelling, sensitive to the touch, to a large suppurating bubo. In most instances the adenopathy is slight and corresponds oftenest to acute affections of the lids, conjunctiva and



lacrimal apparatus. Ordinarily, it is the pre-auricular gland alone that is tumefied. By some this was considered a characteristic sign of gonorrheal conjunctivitis, but in truth it is one often lacking in this disease, and one that is often observed in connection with acute conjunctivitis of different origin. It comes in many cases of **acute contagious conjunctivitis** (Koch-Weeks) and lasts about one week. It is almost constant in **hordeolum**, lasting only two or three days. In **lacrimal pericystitis**, with or without **streptococcus infection**, adenopathy is constant and early. It is the rule in all **streptococcic affections**. In **phlyctenular conjunctivitis** and that of **Morax-Axenfeld** it is exceptional.

Pre-auricular adenopathy warrants the assumption in certain cases that the conjunctiva has been the port of entry of the infection, even in the absence of lesions of that membrane. This has been observed in human glanders. More manifest is the adenopathy of **Parinaud's conjunctivitis**. This lasts from two to five months. It sometimes terminates by suppuration and spontaneous rupture. **Chancre** of the lids or conjunctiva is always accompanied by adenopathy which is apparent the week following the appearance of the chancre. It is an adenopathy that is voluminous, hard, indolent and always affecting several glands. In **tuberculosis** of the lids, conjunctiva or lacrimal apparatus—except in certain cases in which the tuberculous infection assumes the lupus type—adenopathy is always present. Tubercular adenopathy is sometimes indolent, terminating by resolution in several weeks or months, but it is the form that most frequently ends in suppuration of the glands involved. **Streptococcic** infections of the eye may also give rise to acute suppurative pre-auricular or submaxillary adenopathy. Keloid or operative scars in the region of the glands in question have a limited "*anamnestic*" value (Morax).

**Semiology of the Secretions.**—By **conjunctival secretions** one is to understand that, as here discussed, they include all of those that habitually come in contact with that membrane, namely, those of the lacrimal glands, the acinous



conjunctival glands of Krause and Ciaccio, the sebaceous glands of Meibomius, and of the caruncle, and the sudoriparous glands of Moll. Mention has already been made of the functions of the accessory lacrimal glands, including those of Krause. The sebaceous glands around the free border—those of Zeiss—not only serve to oil the cilia, but also to form a greasy film around the palpebral fissure, which acts as a barrier to the too ready overflow of the tears and to the outward flow of the Meibomian secretion. Those of the caruncle, together with the mass of that body itself, block the escape of the tears at the inner angle. The secretion of the Meibomian glands is not normally so oily as that of those of Zeiss, and it is of a more viscous nature. It is being constantly smeared over the cornea and the exposed parts of the bulbar conjunctiva by the nictitation of the lids, where it mingles with the lacrimal secretions to form a protective coating for those delicate and sensitive parts. By its non-conducting properties this film serves to shield the eye from extremes of heat and cold. It also serves to make perfect the occlusion of the palpebral fissure in sleep.

All these secretions are subject to changes of both quality and quantity, some of which are deleterious to the integrity of the conjunctiva. Their quantity is increased in all acute diseases and irritations of the conjunctiva and lids. It is probable that at such times the tears contain a greater percentage of albuminoid material, and that the Meibomian secretion is more viscid. These mix and dry between the edges of the lids when the latter are kept long closed and cause **agglutination** which, of itself, is a symptom of some significance. When, for example, there is *injection of the conjunctiva without agglutination of the edges of the lids it is a sure indication that conjunctivitis is not present*, whereas agglutination with or without other symptoms is indicative of inflammation somewhere, though not necessarily of the conjunctiva. It may be of the lids. The Meibomian secretion not infrequently becomes too oily, and by the winking of the lids a fatty film is spread over cornea and



bulbar conjunctiva, from which the tears slip like water off the feathers of a duck. The altered secretion collects along the free borders in a frothy, chalky streak. A form of xerosis results which is limited at first to the cornea and to that portion of the bulbar conjunctiva which is included within the palpebral fissure, and gradually thickening of the membrane here and other pathological changes ensue. These conditions are peculiar to persons past middle age, and are, in a measure, perhaps, due to retention and bacterial changes in the Meibomian secretion while it is still within its long ducts. That which is expressed from these ducts preparatory to an operation for cataract often has the appearances above described. **Acidity** of the tears is worthy of mention. Their normal reaction is alkaline, hence, grateful to the eye, while an acid condition is irritating. It is well, therefore, on occasion, to make tests with litmus, and if acidity is found, to try to overcome it.

**Purulence** of the secretions of the eye is a symptom that is significant not because of any pathognostic importance it possesses, but because it deserves to be considered with the view to ascertaining its cause. The conjunctival diseases in which the attendant discharge is purulent, either **primarily** or **consecutively**, are numerous and varied. In childhood, aside from those of the new-born, purulency is common in conjunction with **acute contagious conjunctivitis** and with the **impetiginous** forms, not to mention the **diphtheritic** and the **streptococcic**. In conjunctival inflammations of adults, besides that from the gonococcus it is frequent with the **subacute conjunctivitis** from the **diplobacillus of Morax-Axenfeld** and that from the **pneumococcus**. Formerly it was supposed that much was to be learned by the **macroscopic study** of conjunctival discharges, their color, consistence, etc.; and, while such study is not without its rewards, and not to be wholly discouraged, it is, in the present light of Science, no longer deemed of great moment. The presence of pus is, withal, under all circumstances, a sign that the integrity of the eye is in danger.



## CHAPTER V.

### THE GLOBE.

The differential semiology of the ocular globe, taken as a whole, relates to 1. its abnormal positions of rest in its socket, or **deviations**; 2. to its abnormal situations in the orbit, or **displacements**; 3. to disturbances of its mobility, or **nystagmus**; 4. to variations in its tension, or **tonicity**; and 5. to modifications in its size, or **volume**.<sup>1</sup>

1. **Deviations of the Globe.**—When the individual is conscious and the eyes are in a state of comparative repose, or fixed upon some distant object, we assume that the optic axes are directed straight ahead and relatively parallel. During unconsciousness from natural sleep, or other causes, the globes turn so as to cause the optic axes to leave their horizontal position, and probably also that of parallelism. We further assume that this position of the eyes in conscious repose is the normal one. By position of repose we do not mean that of Von Graefe, in which, by the interposition of a prism and a colored glass, or of a colored cylinder, the eyes are, for the time, deprived of the power of binocular single vision. The point toward which the eyes are looking constitutes their **direction**. If the direction of both eyes is not the same or, more correctly speaking, if the lines of vision (**visual axes**) of the two eyes do not meet at the point looked at, the eye whose visual axis misses that point **deviates** and binocular vision is not possible. The eyes are then in a state of **squint** or **strabismus**.

<sup>1</sup> For our present purposes it suffices to consider only those forms of deviation that are apparently objectively to the sight of the examiner. For discussions of all other phases of the subject of the ocular muscles and the movements of the eyes the reader is referred to two other volumes of this system, one by Doctor Pyle, of Philadelphia, the other by Doctor Knapp, of New York.



The word squint includes nearly all the deviations of the globe with which we are here concerned. There are instances in which the squint is not apparent excepting when the individual is asked to look in a direction different from that assumed when in the state of conscious repose, or at an object situated quite near to the eyes. Slight loss of function or power to contract, in certain of the six muscles that control the movements of the globe may exist without producing manifest squint in any line of regard, owing to compensating combined action of certain others. Thus the combined action of the superior and inferior rectus is that of an adductor, and the two oblique contracting together cause abduction. Decided impairment of power, however, in any one of the six will produce squint in some part of the field of fixation. Ordinarily the deviation of an eye is detected objectively by noting the position of the cornea, iris and pupil as compared with that of the same parts of the other eye, and its degree roughly estimated by observing the position of the iris with respect to the canthus toward which it deviates while making the other eye fix in such a manner as to place the center of the pupil at the center of the palpebral opening. To discover very slight degrees of deviation and for the accurate measurement of any degree, methods of examination are required that do not come within the sphere of this work.

A fair idea as to the state of the ocular muscles is obtained first by inspection, as mentioned, then by having the patient fix with both eyes a pencil or other small object, and carrying it in every direction so as to note the associated movements. Next by covering each eye separately with a piece of cardboard while the other eye is made to fix in every part of the field, to note the individual movements. To try the **cover test**, a piece of glass lightly ground is substituted for the cardboard. The grinding on its surface is such that the observer can see the examined eye, yet so opaque as to prevent binocular vision on the part of the patient. The deviating eye is covered by the glass while the other is made to look



straight ahead at the test-object. The deviation of the affected eye as seen through the glass is called the **primary deviation**. The glass is then held before the unaffected eye while the other fixes the object in the same way. The deviation now seen through the glass is called the **secondary deviation**.

Deviations of the globe are usually referred to as **concomitant**, or **non-paralytic** squint, and as **paralytic squint**. It were better to drop the term concomitant as the meaning given to it in this connection is inapplicable and misleading. Without exception the text-books affirm that in this form of squint the eyes maintain their same relative position in every part of the field of fixation. Such is rarely if ever the case save in degrees of deviation that are too slight to appreciate with the eye alone. In *all* marked cases of squint there is limitation of motility in the direction of the faulty muscle. The muscle at fault is usually that away from which the eye deviates; this is always true, of course, in paralytic squint. The Germans denominate the muscle toward which the eye turns in squint as the "Schielmuskel." It is only in cases of spasmodic deviations of the globe, and in those caused by abnormal shortening of a rectus muscle that the deviation is toward the affected muscle. For practical purposes, then, it will serve to divide the deviations of the globe into **functional** or **non-paralytic** and **paralytic**, though it is highly probable that, in many instances, a squint has been classed among the non-paralytic that had its origin in impaired function in the motor nerve of the faulty muscle, dating possibly from infancy. Congenital strabismus is rather paralytic than functional.

**Causes of Non-paralytic Squint.**—Congenital shortening of a muscle is perhaps oftener responsible for deviation of the globe than is generally believed. Such deviations, even when the subject is a child, will not yield to strong traction, or attempts at counter-rotation with forceps, although the subject be in profound narcosis. Indeed, this constitutes the test for this variety of squint. Squint is



rarely due to causes located in the muscles themselves. In those countries where **trichinosis** is most often seen there occurs occasionally squint from degeneration of the fibres of an ocular muscle, the result of the ravages of the trichinæ. The ocular, the masticator and glottis muscles are often affected early in this disease. It may depend upon lack of development in the muscle or faulty insertion of its tendon. A certain few cases are dependent upon purely mechanical conditions, as, for example, adhesive scars from injuries such as burns and traumatisms, or from local diseases, such as ulcerations. From these ensue **cicatricial shortening of the cheek ligaments, contraction of the conjunctiva near the inner commissure,** and many kinds of **symblepharon**. All these may cause deviations of the globe, yet the squint therefrom may be apparent only in certain lines of regard. Amblyopia or anything that interferes with the incentive to binocular single vision, and unawakened fusion-sense and undeveloped fusion-centres, are often cited as causes of squint.

**Disturbances of innervation**, as a prime factor in the etiology of non-paralytic squint, is insisted upon by many. The form of squint most often met with is the convergent (**Esotropia**). As it is, in the vast majority of cases, associated with hyperopia, ophthalmologists early thought to attribute the deviation of the eye in this form to exaggeration of the relation naturally existing between efforts at convergence and those of accommodation. Conversely, in the frequent association of myopia and divergent squint was found corroboration of the theory that these refractive errors are the chief and primary causes of non-paralytic squint. Beyond question these are often the determining causes, but when one considers how relatively few are the cases of squint as compared to the vast number of those in which there is amblyopia—or complete blindness in one eye, or as compared to the still vaster number of hyperopes and myopes who do not squint, these explanations as to the primary causes of such deviations of the globe are unsatis-



factory. Just what is meant by disturbances of innervation of an ocular muscle, or by undeveloped and unawakened fusion-faculties and senses has, as yet, not been made absolutely clear. In this department of ophthalmology so much is obscure, and so much based on theory—so little known, and based on established fact, that in our own study of the differential semiology of non-paralytic deviations of the globe we must content ourselves to take a great deal for granted, particularly as to the causes of non-paralytic squint.

**Paralytic Deviations of the Globe.**—The squint in this form is due to an alteration, congenital or acquired, of the nervous apparatus that pertains to the ocular muscles and their movements, or to a lesion in close propinquity to some part of the apparatus. The degree of loss of function in the muscle is designated as **incipient**, **limited**, or **partial** when the muscle is **paretic**, and as **complete** or **total** when it is **paralyzed**. Among the principal causes of paralytic deviations of the globe, syphilis stands first. It may affect any part of the motor-nervous system of the ocular muscles, and at any period of its progress. Prominent among the causes of the peripheral paralyses are punctured wounds, fractures at the apex of the orbit or at the base of the cranium, hemorrhages, traumatic and idiopathic, localized neuritis from exposure to extreme cold, and inflammations, infections and morbid growths of the surrounding tissues. The ocular palsies of central origin are largely the result of degenerative changes in the nerve-cells and in the blood-vessels of the brain and spinal cord, and for most of these syphilis is directly or indirectly responsible. This is also true as to most of the inflammations and neoplasms that figure in the etiology of deviations of the globe.

Generally speaking, the degree of deviation of the eye in repose corresponds with the extent of the paralysis. It often happens, however, that the deviation of a total paralysis is only manifest in efforts to turn the eye in the direction of the affected muscle. Ocular paralyses are **monolateral** and **bilateral**. Binocular paralysis that concerns the correspond-



ing muscles of both eyes or the corresponding movements of both eyes is designated as **associate**, or **conjugate** ocular paralysis. Parinaud has established two classes of associate movements of the globe, viz., the **parallel** and the **non-parallel**. In the parallel movements the eyes turn in the same sense with respect to the axis of the body. These associate movements are very numerous and complex, but may be relegated to two classes: those that are parallel and horizontal, or to **left or right**; those that are parallel and vertical, or **up and down**. The non-parallel associate movements have for object the maintenance of binocular single vision at varying distances—they are movements of **convergence** and movements of **divergence**. Lack of movement from side to side is **conjugate lateral paralysis**; of that up and down, **conjugate vertical paralysis**. Inability of eyes to effect the non-parallel movements constitutes **paralysis of convergence** and **paralysis of divergence**.

**Location of Lesion.**—The paralysis is **peripheral** when the lesion is situated anywhere along the course of the nerve between its nucleus and its termination in the muscle. Peripheral paralysis is divided into **extra-cranial** and **intra-cranial**. When the lesion is of the nucleus or of any part of the corresponding structures, as of the cortico-bulbar fibres of association farther removed in the brain, the paralysis is called **central**. In order to decide as to whether a given case is of peripheral or central origin, it is necessary that one bear in mind the anatomy of the nerve-supply of the ocular muscles. The superior oblique and the external rectus each has its special motor nerve; that of the first being the fourth, or **trochlear nerve**, and that of the second the sixth, or **abducens nerve**. All the rest, including the levator and the intrinsic muscles of the eye are supplied by the third, or **oculo-motor nerve**. Hence, paralysis of the ocular muscles occurs singly or in groups. Paralysis strictly limited to one of the muscles having its own individual nerve, warrants the supposition that the lesion is peripheral, and one looks carefully into the history for some such cause as syphilis or



fracture of the base of the brain, infection of the middle ear, or extra-cranial lesion or traumatism. When all the muscles innervated by the third nerve are paralyzed it is equally probable that the lesion is peripheral. If the inferior oblique, the sphincter of the pupil and the ciliary muscle are alone paralyzed, one may conclude that the lesion is **orbital**. Paralysis of all the muscles of the eye, both external and internal, constituting **total ophthalmoplegia**, may be set down as peripheral, *i.e.*, either **orbital** or **basilar**. Associated symptoms of the fifth nerve, like **neuralgias** and **neuro-paralytic keratitis**, and of the optic nerve, such as **papillitis** or **optic atrophy** are frequently present. If there are no associated symptoms and if one notes, besides, that there are other symptoms pointing to trouble of the oblongata, the lesion may be called **nuclear**. This is particularly true when the syndrome assumes the type known as **ophthalmoplegia externus**; that is, when the external ocular muscles only are involved, while those of the pupil and ciliary body still retain their function.

The preceding deductions have reference to **unilateral ocular paralyses**. Let us also review briefly a few binocular groupings.

**Conjugate Paralyses.**—Paralysis of the associated movements of the eyes is frequently observed in connection with **disseminated sclerosis**. In conjugate lateral paralysis, the eyes deviate toward the same side, and the deviation persists whatever movement is solicited. The seat of the lesion is around the **eminencia teres**, and involves the nucleus of the sixth nerve. There is, at the same time, facial paralysis (seventh nerve) on the side of that of the externus. All these associate paralyses and spasms are, in short, of central origin; *i.e.*, the lesion is in the cerebrum, the cerebellum or the pons. If it is a **destructive lesion** there is paralysis; if an **irritative lesion**, there is spasmodic contracture of the muscles concerned in the corresponding movements of the two eyes. They are habitually accompanied by such symptoms as hemiplegia, convulsions and torsion of the neck.



# DISTINGUISHING FEATURES OF FUNCTIONAL AND PARALYTIC DEVIATION OF THE GLOBE.

## Functional.

Makes its appearance in infancy and childhood.

Either present in infancy or characterized by gradual and insidious development in after years.

Rarely disappears spontaneously, never as a result of internal medication.

Always confined to one eye.

Only one muscle concerned in its production.

Absence of associated symptoms.

Limitation of movement in the direction of the affected muscle not complete.

Often intermittent.

Disappears in natural sleep and in narcosis.

Diplopia rarely at any time.

Binocular vision not possible in any part of the field of fixation.

Primary and secondary deviations equal.

Never inclination of the head in the direction of action of the faulty muscle.

No vertigo.

Projection perfect.

Most often marked amblyopia in the deviating eye without corresponding ocular lesion.

Deviation is rarely vertical.

## Paralytic.

Comparatively rare prior to adult life.

Prone to sudden appearance and rapid increase in degree.

Often disappears spontaneously, and also, in a large percentage of cases, as a result of medical treatment.

Frequently bilateral.

Often a set of muscles.

Often presence also of other paralytic symptoms.

Very marked—most often loss of motility in that direction.

Constant.

Does not disappear under these conditions.

Diplopia the rule in the early history.

Possible in that part of field in which the affected musculature is not in requisition.

Secondary deviation far exceeds the primary.

Habitually present.

Often—both reflex and from involvement of auditory nerve.

Faulty.

No amblyopia without cause being apparent.

It is in many instances.

In paralytic squint the deviation is apparent in *all* the movements of the globe that are participated in by the affected muscle. If it is the internus, for example, both the lateral movements and those of convergence show the devia-



tion. Functional convergent squint is sometimes intermittent, but this is a phase peculiar to children and young adults. A recurrent convergent squint in adult life is often an early sign of tabes. Hysterical convergent squint is peculiar in that it is a **bilateral functional deviation**, and due to spastic contraction of both internal rectus muscles.

**Displacement of the Globe.**—By this is meant that the globe occupies an abnormal position in the orbit. A displacement forward is called **exophthalmos**, one backward **enophthalmos**, and one sidewise is referred to as a **lateral displacement**.

**Exophthalmos.**—Forward displacement of the globe is by far the most frequent kind, and it is a symptom that is common to a number of disorders. It is rare, however, that the dislocation is strictly **axial**, that is, straight forward. It is usually also lateral to a certain degree. From the semiologic standpoint it is important to observe and to measure the **extent** of the displacement, both forward and lateral, and the **direction** of the lateral deviation; to determine the **progress** of the forward movement of the globe—whether **rapid** or **slow**, to ascertain whether or not the exophthalmos is reducible, and take into consideration other features such as the motility of the affected globe and its lids—the presence of signs of inflammation of the tissues of the orbit, or of those that might be considered as associated symptoms. The sudden appearance of an exophthalmos—in a few hours or a few days—is indicative of an acute inflammation of the tissues of the orbit, or the infiltration of these tissues with blood from a local hemorrhage, or their invasion by pus as from suppuration of one of the accessory sinuses.

**Rapid Exophthalmos.**—The form of exophthalmos characterized by the most rapid development is that from **hemorrhage into the orbit**. It may be the result of a **penetrating wound** that involves a blood-vessel or, in subjects the walls of whose veins and arteries are weak and brittle from **degenerative changes**, of a **severe concussion** as from a **blow** or **fall**. A number of cases of spontaneous hemorrhage



have been recorded—no history of traumatism or strain having been obtainable. Acute inflammation of the orbital tissues—**orbital cellulitis** and infections, **phlegmon** and abscess—constitute the most frequent causes of rapid exophthalmos. **Suppurative periostitis of the orbit** is not rare, especially as regards young children who are recovering from scarlatina and measles; and syphilitic **osteo-periostitis** is equally common in adult life. Both give rise to exophthalmos, and the globe usually deviates to the side opposite the seat of the lesion. Exophthalmos from infection of the contents of the orbit from empyema of one of the accessory sinuses may be rapid or slow. If phlegmonous inflammation ensues the displacement of the globe will be rapid, and its direction laterally will indicate the particular sinus, or group of sinuses, responsible for the infection. Abscess of the orbit from a penetrating wound in which a foreign body is left in the tissues, often causes rapid pushing forward of the globe. The same is true of metastatic abscess of the orbit from such general infections, as those of the streptococcus, staphylococcus—operative or idiopathic—pneumococcus, and typhoid. Most all the forms of exophthalmos just mentioned are accompanied by signs of inflammation of the lids—as swelling, redness and edema. There is deviation of the globe and they are not reducible; that is, the eye cannot be pushed back into position by pressure over it with the index fingers applied upon the upper lid. They are, for the most part, unilateral.

A form of bilateral, rapid, irreducible exophthalmos, with great chemosis of the conjunctiva is often seen in conjunction with septic phlebitis of the sinuses of the dura, especially that from suppuration of the middle ear. Yet another form of rapid exophthalmos may accompany **emphysema** of the orbital tissues. This occurs in certain fractures that involve the inner wall of the orbit, together with forcible expiratory efforts to free the nares of blood; also from violent fits of sneezing or of blowing the nose, especially when there is frailty of the lacrimal canal and fossa. The



air is forced into the cellular tissue of the orbit, producing enormous puffiness of the upper lid. The diagnosis in the early stages is made conclusive by **crepitation** upon pressure, and the fact that the exophthalmos is **reducible**. As septic material often enters the tissues with the air suppurative cellulitis may intervene later when these distinguishing signs fail. Those unfortunate cases of orbital cellulitis that are consecutive to the making of **false passages** in attempts to probe or to syringe the **lacrimal canal** should not be omitted from the causes of rapid exophthalmos. Exceptionally, the exophthalmos from the relaxation of the paralyzed muscles in **exophthalmoplegia totalis** and **externa**, as, also, that from Basedow's disease, are of rapid development. The last two varieties are both axial and **reducible**, and the last is **bilateral**. The characteristic associated symptoms facilitate the diagnosis.

**Slow Exophthalmos.**—Under this head comes the type of exophthalmos most usually met with in **Basedow's disease**. **Aneurysm of the basilar artery** produces bilateral exophthalmos very like that of exophthalmic goitre, but the associated symptoms are those relative to the nerve centers. **Cavernous angioma** causes exophthalmos that is neither pronounced nor wholly reducible, while **arterio-venous aneurysm** involving the **cavernous sinus**—or, as it is commonly called—**pulsating exophthalmos**, is characterized by considerable protrusion of the globe, by enlargement and tortuosity of the vessels of the conjunctiva and lids, and, most pathognostic of all, by visible and palpable pulsation of the orbital tissues, and by a *bruit* that is distinctly audible on placing the ear on the adjacent temple or forehead. In this variety of exophthalmos the globe recedes when pressure is made on the common or the internal carotid of that or both sides; it is reducible, and there is, in nearly every instance, the history of a traumatism of the head to account for the conditions.

Among the forms of exophthalmos of moderately slow development are those consequent upon deep inflammations



of the orbit, as, for instance, extensions and infections around the apex from **disease of the sphenoid sinus**, those resulting from syphilitic and tubercular **osteoperiostitis** of the orbital walls, and those from rapidly growing **sarcomata of the orbit** or of the sinuses or of the temporal fossa. They are all non-reducible. All save the last are attended by pain, and tenderness, and most all by inflammatory symptoms in the tissues interested—such as edema and swelling of the lids. The true sarcomata arise from the bones of the orbit or neighboring cavities, and are of relatively rapid growth and extreme malignancy. They are not painful. The epitheliomata spring from the mucous membrane of the nose, or that of the maxillary or the sphenoidal sinus, or of the ethmoid cells. Part of the tumor is often accessible in the nasal cavity for purposes of investigation. These malignant tumors when arising from the nasal or other median cavities of the skull are not infrequently observed to invade both orbits, thus producing bilateral exophthalmos. The exophthalmos from the rather rare growth known as **chloroma**, or, more properly, as **chlorosarcoma**, from the green color of the new tissue, is usually bilateral, and often of rapid development.

Fluctuation is not present, even in the suppurative kinds of exophthalmos until the later stages, and exploratory incisions must not be resorted to hastily. Whenever there is doubt as to the nature of the agent that causes the exophthalmos, and there is a possibility of its being syphilitic, it is well to make the Wassermann blood test, and if this is positive, to try vigorous antiluetic treatment, whether or not there are other signs of the disease.

Slowly developing exophthalmos is produced, in the vast majority of cases by the growth of tumors that are primarily of the orbit or extend into this cavity from those adjacent. The tumors that originate in the orbit are primarily of the optic nerve, of the bony structures, of the soft parts, or they are cysts. Palpation alone will suffice to give an idea as to the character of most of these growths. Those of the optic



nerve cause exophthalmos that is axial, and pressure over the protruding eye gives rise to pain and a sudden backward movement of the patient, and the exophthalmos in the early stages is partly reducible. The globe is quite movable while the tumor is small. These tumors are peculiar to young subjects and are rare after the age of 30. Loss of sight is likely to occur with or even before the appearance of the exophthalmos. Those of the soft parts are **fibromas** or **fibro-sarcomas**, arising from the connective tissue of the orbit, such as Tenon's capsule, the aponeurosis of the muscles, and the periosteum. They are benign, not painful nor sensitive, are likely to occur in healthy individuals, and often send out finger-like processes that can be readily detected by palpation. Exostoses are not infrequently back of an exophthalmos. When the tumor is where it can be palpated, it is easily recognized by its extreme hardness and its racemose outline. It is of very slow growth, and is primarily of the orbit, or extends into it from the frontal or the maxillary sinus. Neither the fibroma nor the exostosis is characterized by pain unless the former be a neurofibroma, in which cases there may be painful or sensitive nodules present in the skin supplied by the ophthalmic divisions of the fifth nerve. The **cysts** of the orbit that cause protrusion of the globe are principally the **dermoid** and the **hydatid**. They are occasionally multilocular, and may often be recognized by their fluctuation. Their contents are available for examination through aspiration by means of a Pravaz or other syringe.

**Enophthalmos.**—True recession of the globe in the orbit, excepting when the result of emaciation, is a very rare affection. There are many instances of seeming or **false enophthalmos**. If the palpebral fissure of one eye opens less widely than that of the other, it gives the impression of a drawing backward of the globe. Congenital ptosis with microphthalmos and paralysis of the cervical sympathetic nerve are examples of false enophthalmos.

**True Enophthalmos.**—Actual depression of the globe to a position in the orbit deeper than the one formerly occupied,



or deeper than that occupied by its fellow constitutes true enophthalmos. The most frequent cause of this depression is a traumatism—one that results from a crushing blow, such as miners are subject to in blasting, by which some part of the wall or rim of the orbit is fractured. The backward displacement of the globe is primarily affected by the force of the blow, and the enophthalmos is made permanent by the cicatricial contraction that ensues. *Absorption of the orbital fat* in the extreme emaciation of old age and that of certain diseases, like cholera and dysentery, that rapidly deplete the blood, are examples of true enophthalmos. After advancement-operations for squint particularly when no setting back of the opposing muscle has been made a part of the procedure, and the advancement has been considerable, enophthalmia is perceptible.

**Nystagmus** is a term designating an abnormal mobility of the globe. It is a disturbance of ocular stasis—in reality, **tremor of the eyes**. The movements of the globe are involuntary, associated, synchronous, rhythmic and usually more or less jerky. Nystagmus is designated according to the direction of the movement as **horizontal, rotary, vertical, oblique** and **mixed**. **Horizontal** nystagmus is also commonly called **oscillating**, a term not well chosen, for the globe may be said to oscillate in nearly all kinds of nystagmus. In **true** horizontal nystagmus the oscillations are precisely like those of a pendulum, *i.e.*, they are to and fro past a central point, in contradistinction to what is called **pseudo-nystagmus**, wherein the oscillations are more jerky and begin at an eccentric point, move farther away from this point, then more slowly back to it again, and never **pass** it. The direction of the jerky movement in pseudonystagmus is the same as that in which the eyes were being carried when oscillation started.

Horizontal nystagmus is the most common form, and next is the rotary, in which the oscillation of the globe is back and forth on its antero-posterior axis. Vertical nystagmus, or straight up and down movements on the



transverse axis, is rare. Still more rare is **unilateral nystagmus**, though it is less rare in the vertical form than in the others. The peculiar slow, and far-reaching movement of blind eyes, or those deprived of central vision has been called **searching nystagmus**. It is less rhythmical than nystagmus and the direction of the movements may vary anywhere between the horizontal and the vertical. In no form of nystagmus is the movement of the eyes constant in degree and rapidity, and in most instances it is intermittent. The oscillations vary in rapidity and extent under divers influences. Their rate is anywhere between 50 and 200 per minute, and their length of excursion from 1 to 5 mm. The amplitude of the movement stands in an inverse ratio to its rapidity. It is at its maximum in one individual when the accommodation is relaxed, and at its minimum in another under similar conditions. It varies with the particular point of regard and with the physical and mental state, and is absent during sleep.

Nystagmus is further classified as **congenital** and **acquired**. As it is probable, however, that few if any cases of nystagmus are present at birth, a more rational classification, perhaps, would be that of the nystagmus of **infancy** and **childhood**, and that of **adolescence** and **adult life**.

**Nystagmus of Infancy and Childhood.**—This may for convenience be subdivided into (a) that of **peripheral origin**, dependent upon visual troubles, and (b) that of **central origin**, dependent upon disturbances of the motor apparatus of the globe. In the first category are all cases of nystagmus that are the result of bilateral amblyopia dating from infancy. Chief among the causes of this amblyopia are the corneal scars consequent upon **ophthalmia neonatorum**. It is by no means rare in conjunction with **congenital cataract**, **retinitis pigmentosa**, the posterior polar atrophy of **central choroiditis**, and in **physiologic anomalies** of the **choroid** and **retina** such as **coloboma** and **albinism**. It has often occurred to the writer that in some of these instances the nystagmus is perhaps a purely compensatory phenomenon,



and is for enhancement of the visual acuity. Here the rate of the oscillations is at the maximum and their excursion at the minimum in attempts at distinct vision. The principle involved seems to be similar to that by which a landscape is invisible through a paling fence, to a passenger on a railway train, so long as the train is stationary, but more and more distinctly visible as the train gets under way. Or, perhaps, a better illustration is furnished by a moderately transparent sheet of tracing paper when laid over a picture or a printed page. The details of the picture or the print may be indistinguishable while the tracing paper lies still, but becomes clearly manifest if the overlying paper is moved rapidly back and forth. It is as if, by this short and rapid oscillation of the eyes, a relatively greater number of precipient elements are brought into requisition, at the center of the retina, to compensate for the lack or sparseness in development of these elements in the eyes under discussion. Or, as if by spreading the retinal image over a greater area, the number of cells under excitation, and the resultant number of visual impulses generated, is so augmented as more nearly to approach the normal.

In the second category, or nystagmus acquired in infancy due to disease or disturbance of the nervous apparatus pertaining to the ocular movements, the principal cause, and the one that comes first to mind is **tubercular meningitis**, resulting in **juvenile optic atrophy**. It is probable that **hereditary syphilis** is responsible for similar conditions, and it is impossible definitely to classify this disease according to the seat of the lesion. Nystagmus developing in infancy and in eyes in which exist no apparent lesions is thought to be in some instances the result of intra-ocular hemorrhages at birth. **Unilateral amblyopia** seems more likely to produce **squint** than nystagmus.

**The Nystagmus of Adolescence and Adult Life.**—This group is subdivided into (a) **Nystagmus from diseases of the nervous system**, (b) **Occupation Nystagmus**, (c) **Otic Nystagmus**, and (d) **Reflex Nystagmus**.



**Nystagmus from Disease of the Nervous System.**—Easily first as a cause in this class is **multiple sclerosis**. This is peculiarly a disease of adolescence and the early adult years. While nystagmus is not a constant symptom in the disease, according to Charcot and Parinaud, it is present in at least 50 per cent. of the cases. It is of the horizontal variety and is continuous or intermittent. Uhtoff found it continuous in 12 per cent. of the patients, and pseudo-nystagmus, or nystagmic oscillations at the limitation of the lateral movements, in 46 per cent. Nystagmus is very frequent in **syringomyelia**, and sometimes in **diffuse myelitis**. It is exceedingly rare in tabes, while **pseudo-nystagmus**, or jerkiness near the limits of excursion of the lateral movements, is frequent in this affection, and is usually accompanied by motor paresis in general.

**Occupational Nystagmus.**—Under this head comes a form of nystagmus peculiar to those who work in dark places and with eyes and body for long periods in constrained positions. Although it goes by the general name of **miner's nystagmus** it applies only to the diggers of coal in the leads or veins, who usually recline while working, and with eyes directed upward, backward and to one side. The nystagmus comes on after several years of such work. It is of the rotary kind, and sometimes the antero-posterior axes of the globe also describe tiny ellipses. Unlike most other varieties of nystagmus, it is a source of great inconvenience to the subject, as all objects in the field of vision seem to dance in unison with the movements of the globes. Fortunately, the movement is not persistent, as it soon ceases after the individual works for a time in bright light; but it will recur when the eyes are subject to the same conditions that originally produced it. **Vertical occupational nystagmus** has been observed in the case of an accountant who worked day after day adding long columns of figures in a book of folio size, but in perfect light (**Percival**). Sulzer reported a similar case relative to a compositor in a printing establishment.

**Otic or Auricular Nystagmus.**—It has long been known



that nystagmus is a symptom in certain inflammations of the middle and inner ears, but the interpretation of the phenomenon has rested obscure. In its simplest form otic nystagmus does not materially differ from that sometimes occasioned by forcibly syringing the external auditory canal with hot water, or by sending a swift current of air into the middle ear through the eustachian catheter, and with or without dizziness and fainting. The symptom depends upon a modification of function in the labyrinth from excess of pressure. It is similar to the nystagmus observed in operative and idiopathic lesions of the labyrinth, and in acute inflammations and suppurations of the tympanum, and is due to irritations of the vestibular portion of the auditory nerve. It is supposed that the **direction** of the nystagmus is in accordance with that of the particular semicircular canal whose ampulla is implicated. This form of nystagmus is not to be confounded with that which occasionally develops in connection with the intra-cranial complications of suppuration of the middle ear, as in **brain-abscess** and **sinus-thrombosis**. The nystagmus here is a symptom of the secondary involvement of the central nervous system, and not of the otitis which caused the brain-complication.

**Reflex Nystagmus.**—To this class are relegated the nystagmus sometimes seen in severe irritation of the eye, as from a foreign body on the cornea, acute inflammations of the conjunctiva, etc. Kraus has published two cases of unilateral nystagmus, one occurring after an operation for cataract, the other after injury which destroyed the sight in a myopic eye. The nystagmus affected only the operated and the injured eye, respectively. Pyle has reported a case of unilateral nystagmus which could be produced and checked at will. In a case of bilateral vertical nystagmus with vertigo occurring in the fifth month of pregnancy in a colored multipara, 36 years of age, reported by Malone, objects danced up and down before her view, a phenomenon rare even in the acquired form of nystagmus, save in that of miners and in the acquired unilateral form.



**The Tension or Tonus of the Globe.**—The normal intra-ocular tension is represented by the resistance necessary to be overcome by the fingers or by an instrument to cause indentation of the average healthy globe. Practice teaches us how great is this resistance to the fingers, and there are several mechanical contrivances for taking and accurately recording the tension. (**Tonometry.**) The degree of pressure exerted upon the outer walls of the globe by the fluid contents varies within certain physiologic limits. In the direction of **excess** this physiologic limit is soon passed, and the more delicate tissues begin at once to suffer injury. Whereas in the direction of **deficiency** there is more latitude. Just how low the pressure may fall without the mere relaxation of itself becoming deleterious cannot be definitely stated. Certain it is that the eye may be unresisting under palpation to the extent of feeling moderately soft and the organ be in perfect health. By the tonometer of Schiotz or that of Nicati the limit of safety upward is 25 grams of mercury, but may fall as low as 14 grams without detriment. Between these physiologic extremes the degree of tension as ascertained by palpation is called zero, or normal; slight hypertension is called  $T+1$ ; moderate hardness,  $T+2$ ; and extreme hardness,  $T+3$ . The corresponding degrees of hypotension are similarly denominated, the plus being replaced by the minus sign. An approved tonometer is essential for precise data relative to the intra-ocular tension. An increase in the tension above the normal is known as **hypertonus**, and a diminution as **hypotonus**.

**Increased Tension or Hypertonus.**—It is assumed that the reader is familiar with the other objective and subjective signs of glaucoma with which we are not at present directly concerned, but with its principal symptom. Since, however, hypertension always means glaucoma, we must consider it semiologically under that name.

**Glaucoma** is divided into **primary**, or **idiopathic**, and **secondary**, or **consecutive**. Primary glaucoma is again divided into **inflammatory**, or **irritative**, and **simple**, or **chronic**.



**Primary Inflammatory Glaucoma.**—The violence of the symptoms in this disease and its duration are in direct proportion to the degree of augmentation in the tension and to the length of time it is excessive. The chief associated symptoms are trigeminal neuralgia, nausea, vomiting, nervous excitement, violet redness of the conjunctiva and sclera, edema of the upper lid, chemosis, steaminess and anesthesia of the cornea, ovoid dilation and fixedness of the pupil, murkiness of the iris and loss of sight. Less constant symptoms are shallowed anterior chamber and retinal arterial pulse. When these are exaggerated and appear suddenly the glaucoma is **acute** or **fulminating**. The hypertension may disappear in a few days or a week and most all the symptoms with it, or it may persist for months or for years, constituting **sub-acute** or **chronic** inflammatory, and **absolute** glaucoma respectively. It is rarely simultaneously bilateral.

**Primary Chronic Glaucoma, or Simple Glaucoma.**—In these cases the associated symptoms go to the other extreme and are characterized by the greatest slowness of development and insidiousness. The excess of tension is rarely so great as to be perceptible by palpation, and the disease is recognized by the syndrome **chromatic halos** about lights, contraction of the **nasal fields of vision**, **excavation of the optic discs** and shallowness of the anterior chambers. The disease is usually bilateral.

**Secondary or Consecutive Glaucoma.**—There are many reasons that lead to the belief that glaucomatous tension is **always secondary**; but for convenience this term is applied to all those cases wherein the hypertension is palpably dependent upon a traumatism or of a morbid condition somewhere in the globe. The traumatisms that cause hypertension are either the results of direct or indirect violence, such as contusions, penetrating wounds, and of surgical operations. **Post-operative glaucoma** is not infrequent after operations upon the crystalline lens. After cataract extraction it is usually consecutive to some iris-



complication; and after **discission** of the cataractous or the transparent lens, it is secondary to the swelling of the cortex. Among the principal morbid conditions in which hypertension occurs are **adherent leucoma**, **posterior synechia**, **acute cyclitis**, **partial or complete luxation of the lens**, **diffuse syphilis of the choroid**, or **conglomerate tubercular choroiditis**, **hemorrhagic retinitis**, and any of the diseases of the choroidal and retinal blood-vessels. Hypertension is often of special semiologic value in forming an idea as to whether or not a tumor primarily intra-ocular has become extra-ocular. The tension is likely to be about normal so long as the growth is strictly within the globe or sclera. Hypertension and **detachment of the retina** are suggestive of an intra-ocular tumor. **Mydriasis from drugs** tends to cause **hypertension** of the globe by the mechanical effects of the bunching of the body of the iris at the base of the cornea, and the blocking of the venous and lymphatic circulation in the anterior portions of the globe. This form of secondary glaucoma is sometimes observed in interstitial keratitis when the mydriasis is prolonged, and in the eyes of elderly subjects with degenerated vascular systems, when the stasis consequent upon the mydriasis leads to accidents of the choroidal and retinal circulations.

**Decreased Tension or Hypotonus.**—In proportion as the pressure within the globe lessens, the organ not only becomes softer to the touch, but its volume is slightly diminished, the palpebral opening somewhat narrowed, and the pupil perceptibly myotic. In consequence of hypotonicity of the globe caused by certain drugs and concussions, the refraction of the eye is lessened. A myopic eye may become more nearly normal as to its refraction, and a normal one hyperopic. Instances are on record in which the difference due to the lowered tension was as much as 6 to 8 diopters. Hypotension is, *as a rule*, **secondary** or **consecutive**. The nearest approach to **primary hypotonus** is a rare and obscure disease known as **essential ophthalmomacia**, or **transitory phthisis bulbi**. In addition to the syndrome of hypotension,



there is present in this affection phenomena indicative of **ciliary irritation**.

Reduced tension is an important diagnostic sign in **penetrating wounds and ruptures of the sclera** that are invisible. Even the smaller wounds serve greatly to diminish the tension, but the symptom is available only so long as the wound remains unhealed, unless it be of the ciliary zone, in which event minus tension may persist after healing. Hypotension is habitually present in the graver forms of uveitis, whether of spontaneous or traumatic origin, as in **syphilitic iridocyclitis**, in **sympathetic ophthalmia**, and in **iron or steel in the uvea or vitreous**, with or without **siderosis**. In the process of evolution of neoplasms within the globe, hypotonus succeeds hypertonus; and this is, in a measure indicative of the **invasion** or the **passing of the sclera** by the cells of the growth. In **uncomplicated detachment of the retina**, decreased tension is a constant symptom; this is also true of **atrophy of the globe in any stage**. **Repeated instillations of cocain-solution** reduce the tonus of the globe, and this organ becomes soft after it is enucleated and after death.

**Volume of the Globe.**—In estimating the volume or size of the globe by ocular inspection there are certain errors to be guarded against. For example, an abnormally wide palpebral fissure, especially in comparison with that of a normal fellow eye, gives the impression of greater size. The eye opens wider under the primary effect of cocain, in **excitation of the sympathetic**, in paralysis of the third nerve, or **lagophthalmos**, and in **exophthalmos**. On the other hand, a narrower palpebral fissure in one eye may mislead one into believing that the globe is reduced in volume. The lids come closer together in **enophthalmos**, in **cicatricial contraction of the conjunctiva**, in paralysis of the sympathetic, from occlusion by bandaging, and long habit of closing the lids, to exclude strong light or to prevent diplopia.

The volume of the globe may be roughly estimated by holding back the upper lids and having the subject look sharply downward. Its greater lateral diameter may be approxi-



mately measured with blunt calipers, the eye being under cocain. The only precise way in which to measure the antero-posterior diameter of the living eye is by means of its refraction. The average diameter of the normal eye is about 24 millimeters, from which we subtract 1 millimeter for each three diopters of hyperopia, or add the same if the eye is myopic. The volume of the globe is **abnormally great** in **myopia**, in **advanced hydrophthalmos**, in general **ectasia of the sclera**, as after **ophthalmia neonatorum**, in certain kinds and stages of **intra-ocular tumor**, and in suppurative panophthalmitis.

**Abnormal lack of volume** is either congenital or the result of disease. That which is congenital when it is from pronounced underdevelopment of the globe is known as **microphthalmos**. In this case not only is the size of the globe much less than normal, but also that of the cornea. In addition, there are usually present other congenital defects, such as epicanthus, ptosis, squint, and coloboma of the iris. Hyperopia of high degree is but another form of incomplete development of the globe. Diminished volume indirectly due to disease or traumatism we constantly note under the names **atrophy of the globe** and **phthisis bulbi**. The size of the eyeball in atrophy varies from just slightly below its normal size, when the shrinkage is first made apparent by hypotonus, and a slight flattening over the insertion of the inferior rectus, through that intermediate size, when the globe assumes a quadrangular shape, from pressure upon it of the four recti muscles, to a size so diminutive that, save for the tiny remnant of the cornea which is usually still visible, all semblance of a globe is lost.



## CHAPTER VI.

### THE CORNEA.

Because of its transparency in its normal state, the cornea is invisible in ordinary ocular inspection. It comprises a little more than one-sixth of the surface of the outer wall of the globe, but as its curvature is greater than that of the sclera, which forms the remainder of the outer wall, seen in profile, it seems to stand out like the crystal on a watch. Just as with the cornea, when one looks at a watch it is not the crystal that is seen, but it is the dial and hands; so, in looking at the eye it is not the cornea, but the iris and the pupil beyond it that are visible. The cornea manifests itself only by the reflections upon its surface, of the source of light. For any portion to be visible, therefore, may be taken as an indication that something abnormal is present. The base of the cornea, *i.e.*, where it joins the sclera, represents an ellipsoid whose vertical diameter is about 10.5 mm., and whose horizontal diameter is about 11.5 mm. Its radius of curvature is between 7 and 8 mm.

**Modifications in Dimension and Form.**—The size and shape of the cornea are not constant nor uniform even in a state of health, and are liable to a diversity of alterations through the influence of morbid processes. In **highly hyperopic** eyes, and in **microphthalmia** the cornea is relatively smaller than normal, and, while bearing the same relation to the size of the globe, its curvature is proportionately less, until, in extreme cases, its curvature is not greater than that of the surrounding sclera. In some of these stunted eyes the dimensions of the cornea are reduced to one-third of the normal, and useful vision is possessed by some whose measurements are half, or even less than half, those of the fully developed eye. Reduced size of the cornea is not



always congenital. In rare instances it is the result of a perforating ulcer in infancy, and the healing has been such as to obliterate practically all trace of the disease. The facts are brought out only by the anemnesis.

Symmetrical augmentation in the volume of the cornea with conservation of form and transparency is a common affection and is variously known as **hydrophthalmus**, **keratoglobus**, and **keratomegaly**. It occurs in all degrees, from the slighter enlargements that are consistent with normal vision to those that are enormous, and entail blindness more or less complete. This condition was formerly called **buphthalmia** or **ox-eye**. Hydrophthalmus is due to congenital anomalies in the iridociliary regions, among which is absence of the canal of Schlemm. It is really **infantile glaucoma**, and is usually a manifestation of hereditary syphilis.

Irregular augmentation of volume of the cornea as a whole or in part, with partial or complete opacity, is known as **opaque staphyloma**, and as **anterior staphyloma** and **cicatricial staphyloma**. The staphyloma or **ectasia** is called **total** or **partial (circumscribed ectasia)** owing to the extent of involvement. Total staphyloma in its worst state is gigantic in its dimensions, and entails a corresponding immensity in the rest of the globe. Partial staphyloma varies in size from a tiny bead to that of a coffee-grain. The enlargement is white or yellowish-white in the thicker places, and blue-white, from uveal pigment, in the thinner ones. The iris is always implicated in such formation. It is often important to determine the cause of the staphyloma and, as this bears a certain relation to the period of life, the first requisite is to ascertain the age at which the expansion of the cornea was first noticed. If it dates from infancy, it is likely that one may obtain a history of eye-trouble with discharge of matter during the first few weeks after birth. If the onset of the preceding local disease can be pretty definitely fixed at a time a few months subsequently, and the child was in very poor general condition, **keratomalacia** is suggested. The



staphyloma in these two counts is bilateral or else there is something in the other eye or in the history to indicate that the primary disease was bilateral.

If the eyes had been normal up to the age of one year, or from that anywhere up to the fifth or sixth year, and the anemnesis points to a long siege of sore eyes with intense photophobia and eruptions in the face and head, and, possibly, lumps on the neck, one thinks at once of extensive ulceration of the cornea from **phlyctenular ophthalmia**. Bilaterality is not so common in staphyloma from this cause. A peculiarity of staphyloma from keratomalacia is that it is likely to be partial and confined to the lower segment of the cornea. Then, there are instances in which secondary infections such as diphtheria, scarlatina and measles are to be considered in relation to the staphyloma. In others, traumatism, like punctures from scissors or knives, and explosions of powder, have led the way to the infection that resulted in the staphyloma. In adult life, the most frequent causes of staphyloma of the cornea are traumatism, common among which are foreign bodies in the cornea, punctured wounds, abrasions and burns; in short, occupational injuries chiefly. Among the diseases acquired, gonorrhea with local infection is the one most often responsible. Staphylomas from burns and explosions are frequently bilateral. Gonorrheal staphyloma is not so frequently bilateral in the adult as is that of the infant, for two reasons—in the adult the infection is likely to be unilateral and precautions taken to prevent involvement of the other eye.

The **curvature** of the cornea is subject to modifications, which may be from pathologic or physiologic processes. The most frequent is **astigmatism**, regular and irregular. Astigmatism is **regular** when the curvature of one principal meridian of the cornea is greater than that of the opposite meridian, and it is corrigible by glasses. Astigmatism is said to be **with the rule**, when the meridian of greater refraction is vertical, and **inverse**, or **against the rule**, when this meridian is horizontal. Regular astigmatism is congenital



or acquired. Congenital astigmatism remains indefinitely of the same degree or tends to increase. Acquired astigmatism is the result of thinning of the cornea from keratitis in some form, together with the pressure—probably spastic—of the lids and from contraction in the cicatrices of traumas and operative wounds, especially those of linear form. This last form is always present after operations for the extraction of cataract, and those for iridectomy, and is proportionate to the extent to which the keratotomy encroaches upon the area of clear cornea. Unlike congenital astigmatism, that from wounds tends to decrease with time, and sometimes disappears entirely: nor is it so habitually bilateral. In **irregular** astigmatism, the curvature of the cornea does not conform to any geometric rule, and is always acquired. It is observed in connection with leucoma and macula of the cornea from the lesions of childhood, such as suppurative, phlyctenular and interstitial keratitis, or it may be consecutive to most any form of corneal lesion occurring at any period of life. It is unilateral or bilateral, without any regard for rule.

**Keratoconus** or **conical cornea**, also variously designated as **hyperkeratosis** and **staphyloma pellucidum**, is essentially a deformity of curvature in the central and median portions of the cornea. True keratoconus is, in a sense, idiopathic. It begins about the age of puberty, it may be in a perfectly robust subject, without a history of a previous inflammatory process or any apparent cause, and much like regular astigmatism, and its refractive error is sometimes amenable to artificial correction. It is more frequent in the female. Later the acuity of vision is lowered to the degree of causing great inconvenience. It is then that the aspect of the affected eyes—for the affection is commonly bilateral—becomes diagnostic; that is, they are characterized by *twinkle* or *unnatural lustre*. By skiascopy one sees, in the centre of the pupil, and concentric with it, a circular shadow of shifting irregularity, similar to that described under "lenticular myopia," or sclerosis of the nucleus of



the lens. As in certain forms of cataract—the zonular and the nuclear, for example—the acuity of vision in conical cornea is sometimes enhanced, or the regular astigmatism rendered more corrigible by mydriasis. Looked at in profile, the conicity of the cornea is plainly visible, and the summit is so thin as to be easily indented. Still later the apex of the cone, from pressure of the upper lid, assisted by gravity, takes a position somewhat below the centre of the cornea and becomes opaque. In other instances keratoconus is consecutive to degenerating diseases of the cornea, such as **prolonged superficial ulcerative inflammation**, or the **vascular keratitis of trachoma**, always abetted by the attendant blepharospasm, and by the normal intra-ocular tension.

**Opacities of the Cornea.**—As stated at the beginning of this section, as one looks ordinarily into the space occupied by the normal cornea, nothing is visible there save the reflected image of the source of illumination, as a window by day, and a lamp-flame by night. If anything else is seen it means an opacity either *on* or *in* the cornea. True, when viewed by strong oblique focal illumination, which is absolutely essential to the study of these affections, the corneal tissue shows as a faint bluish haze, on top of which one can, by moving the beam of light, detect an appearance as of almost microscopic dust. If there exists a lesion, be it ever so small, it can be readily seen and located, whether in the superficial, parenchymatous or profound layers of the cornea. The investigation is often vastly aided by the instillation of a drop of **fluorescin** solution. Thorough scrutiny of the cornea is further facilitated by recourse to the transmitted light of an ophthalmoscopic mirror reflected from the fundus. Here dilation of the pupil is desirable.

Strictly speaking, a **foreign body** in or upon the **cornea** would constitute an opacity, and its presence and its location must often be sought by the means just alluded to. By usage the term opacity of the cornea has reference to a mark or a scar of something, usually a wound or an inflammatory



or ulcerative process that has occupied the same site, but in which active changes have ceased; at any rate, active pathologic changes have ceased. Certain degenerative changes may be still in progress, or absorbent ones, whereby often an opacity of considerable extent is eventually removed. No great importance attaches to fine hair-splitting distinctions as to whether a given corneal mark or spot is a **macula** or a **nebula** or a **leucoma**, but it is often of considerable moment to be able to distinguish a **true cicatricial lesion**, for instance, from an actively inflammatory one. Opacities of the cornea are described and classified for obvious reasons, as **diffuse** and **circumscribed**, and as **superficial** and **deep**.

**Diffuse superficial corneal opacities** may affect the whole, or varying portions, of the membrane. Those injuries and morbid processes that involve only the epithelial layers of the cornea, or, at most, go no deeper than Bowman's membrane, such as the milder and uncomplicated cases of **phlyctenular**, **vesicular**, **filamentous** and **dendritic keratitis**, and the slighter **chemic** and **thermic** cauterizations, like those from **acids**, **lime** and **molten solder**, either leave no trace or else a diffuse superficial opacity.

**Diffuse Deep Corneal Opacities.**—The most frequent cause of these is **ophthalmia neonatorum** complicated with extensive breaking down of corneal tissue. The lesion is most often present in both eyes. The opacity is dense white or is crossed by a straggling corneal blood-vessel or two. It is rare that the entire cornea is interested unless it is also staphylomatous. The same may be said of the leucomas that succeed the infected and neglected **phlyctenular ulcers** of the cornea, though these are generally smaller, and less likely to be bilateral. Associated with them one often finds cicatrices about the neck pointing to an accompanying adenopathy. The next in frequency are the deep, diffuse opacities left by **interstitial keratitis**. These are less often bilateral than those of the ophthalmia of the new-born and are more symmetrical; that is, they are rather prone to be



centrally located and to be densest at the centre and thinner toward the periphery. Their area is large in proportion to the gravity and duration of the disease. Other signs of hereditary syphilis are seldom lacking. A rarer and highly individualized kind of deep diffuse corneal opacity, and one also significant of inherited syphilis, is that which is the alternative of transparent keratoglobus in **infantile glaucoma** or **hydrophthalmus**. It is, in most instances, bilateral. It is observed at birth or within the first few weeks of infant life. What gives the opacity its peculiar distinction is the resemblance of the cornea to a white opal, or a bead of thin porcelain. So delicate is the opacification that one can almost make out the details of the iris beneath.

The foregoing affections enumerated as terminating in deep, diffuse opacities of the cornea are more often identified with infancy and childhood, though most of them are occasionally seen in later life. In still older subjects, sub-acute or chronic inflammatory glaucoma sometimes causes this kind of opacity.

A highly characteristic form of deep, rather diffuse corneal opacity, and one that has some semiologic value, is that seen occasionally after severe acute general uveitis, in which that feature known as **punctate keratitis** or **decemetitis** has been specially pronounced. The opacities, which are frequently bilateral, are about one-third the area of the cornea, or what might be termed its *lowest* third. Here an infiltration that began with the posterior layers of the cornea, extended through the interlamellar portion, and finally involved the whole thickness, but never got beyond the triangular area originally covered by the dots. It is strongly identified with syphilis, both hereditary and acquired. Permanent dots are left in the posterior layers in other instances, consecutive to decemetitis.

**Circumscribed Corneal Opacities.**—It is impossible to make a broad distinction between the circumscribed corneal opacities that are superficial and those that are more pro-



found. This is particularly true of those that are smaller and more sharply defined. The tiny dots, grayish-white, and only seen by focal illumination, that result from the lodgment of minute foreign bodies, such as particles of emery and iron, in the anterior layers, are superficial. The denser, white, more sharply defined spots, or **albugoes**, however, go deeper. They are such as are caused by the penetration of grains of powder, cinders that have entered hot, bits of iron that have struck with considerable force, small, central phlyctenular ulcers that have reached Desce-met's membrane—or even perforated it. Then there are the linear opacities of varying lengths and configuration that bespeak a wound of the same form, and it is often by one of these that diagnosis of traumatic cataract or foreign body in the vitreous or choroid is made possible.

Of the opacities that involve the entire thickness of the cornea the greatest number, by far, are those consecutive to **ophthalmia neonatorum** and **phlyctenular keratitis**. In consequence of the secondary infection and ulceration of the cornea there occurs a perforation of greater or lesser extent. More often the iris is caught in the resulting cicatrix, thus constituting **adherent leucoma**. Often, when the ulcer is quite central and the perforation is not too extensive, a corresponding opacity is left on the anterior capsule of the lens at the point where it lay in contact with the corneal opening while the anterior chamber and posterior chambers were drained, or obliterated. This is **pyramidal cataract** or **anterior polar cataract**, and it is sometimes present without there being any sign of central corneal opacity. In this event either the perforation of the cornea so healed as to leave no trace, or the pyramidal cataract was of fetal origin.

A most striking species of circumscribed superficial corneal opacity is that which results from **ribbon**, or **band-like keratitis**—both **primary** and **secondary**. The primary variety of this keratitis occurs in the eyes of subjects past middle age, and without any special irritative or inflammatory associated symptoms. The other is secondary to a low



grade of uveitis, and is peculiar to younger persons. The opacity presents essentially the same features in both instances. It appears as a broken or a continuous band of glistening white lying horizontally across the centre of the cornea. In primary ribbon keratitis the band is composed of splotches and dots that, on cursory inspection, closely resemble the unctuous froth that is sometimes squeezed from the Meibomian ducts of these elderly individuals. Calcareous degeneration of Bowman's membrane gives rise to these interesting opacities.

**Annular Opacities.**—The most familiar of these is the **arcus senilis**. This is rare before the age of fifty and exceedingly common thereafter. The opacity begins, *bilaterally*, as a delicate arc near the upper limbus. It may extend so as to form a complete circle, or it may become stationary when of only slight extent. In exceptional cases it attains some width, but there is always a ring of clear cornea between it and the limbus. It is free from attendant inflammatory phenomena, is never stainable by fluorescein, that is, it never ulcerates. In this way it differs from the primary **annular ulcer** of the same class of subjects, and from **annular abscess** of the **cornea**, which is either **traumatic** or **metastatic** and occurs at different periods of life. The last two are frequently unilateral. Another confusing variety of annular opacity, usually bilateral, is rather peculiar to young females between the ages of 20 and 30, and known as **sclerosing keratitis**. It, too, respects the confines of the vascular zone, that is, it does not extend to the periphery, and is made up of a number of coalescent spots of interlamellar degeneration or degenerative inflammation. The progress of the disease is very slow, and very active reactionary features are seldom apparent. There is some deep, circum-corneal injection at the start, and a fine vascularity peripherally in the *substantia propria* a little later on, but there is little inconvenience occasioned, and the *epithelium remains intact* throughout.

There remains yet one kind of annular opacity which is unique, in that it is not separated from the limbus by a ring



of transparency. This is the irregular opacity due to severe and prolonged attacks of **ocular spring catarrh**. The palpebral conjunctiva may also show signs of the disease.

**Pigmentation or Melanosis.**—This is an unusual condition and is either congenital or acquired. Less than a dozen cases of **congenital** pigmentation appear in ophthalmic literature. The pigment is deeply situated, near the centre of the cornea, and appears as a small, brown, ellipsoid, or as a spindle, long axis vertical, and it is composed of numerous tiny brown dots. It is symmetrically bilateral and, as a rule, is associated with myopia. It is probably related in some way to the pupillary membrane of fetal life. **Acquired** pigmentation of the cornea is much less rare, is mostly of traumatic origin, and is due to the presence of brown hemoglobin left from a former hemorrhage. The color is even, not dotted. It occurs in irregular plaques in the depths of the cornea, more often in the lower half, where gravity has sent the blood from which it is derived, though it may be located above the horizontal meridian. It has been known to occupy the entire area of the cornea. It is unilateral, and is more often seen in the eyes of middle aged or elderly subjects than in the young.

**Vascularity.**—It is well known that the transparent cornea does not, in its normal state, contain any blood-vessels, hence, to their development here under different pathologic conditions considerable etiologic significance is attached. The blood-vessels of this provisional circulation so vary in size and number as to be on the one hand very difficult to see, and on the other so much in evidence as to impart to the cornea a deep red color. The vascularity is said to be **superficial** when it is of the **anterior layers**, and **deep** when it is of the **parenchyma**. Most all forms of keratitis are more or less characterized by the formation of new blood-vessels, though in many, close scrutiny with strong illumination and convex lenses are needed to reveal them. The commonest example of manifest superficial vascularity is found in **trachomatous pannus**. It affects, more especially, the



superior segment, and even, as occasionally happens, where the whole cornea becomes cherry red, the lower segment is not so much altered as the upper. The general course of the vessels is from above downward. A certain amount of vascularity often persists after the trachomatous process has long since ceased.

But little less common is the superficial vascularity that attends **phlyctenular keratitis**. It appears with the ulcerative stage as a band or sheaf of vessels that traverses the shortest space between the ulcer and the limbus. This condition is not to be confounded with **pterygium**, which is a process of the conjunctiva itself encroaching on the cornea, nor with **secondary ribbon keratitis** recently alluded to. The seat of a foreign body that has long been imbedded in the cornea may also be distinguished by a track of superficial blood-vessels. **Deep vascularity** of the cornea pertains almost exclusively to syphilitic lesions. It is a prominent, if not always a conspicuous symptom in the various phases of **interstitial keratitis**. In severer types it is sufficiently developed to give to the parenchyma the diagnostic splotchy coloration known as **salmon patches**. After all vascularity has long since disappeared, the courses of the larger vessels can still be traced, by proper means of examination, as dark lines traversing the outer zone of the cornea. Deep vascularity is also present in the secondary involvement of the cornea in tuberculosis of the iris.

**Disturbances of sensation** in the cornea do not manifest themselves by any special objective or subjective phenomena, and, because of their semiologic value in certain local and general affections, they must be sought after. The cornea is not sensitive to pressure; it is sensitive to heat only in the peripheral portions and not at the centre; while its entire surface is uniformly sensitive to pain. From the diagnostic standpoint, phases of **anesthesia** or hypoaesthesia of the cornea are of greater importance than are those of **hyperaesthesia**. Among the local affections that modify its sensitiveness it may be stated in a general way that all the ulcera-



tions tend to produce hypoesthesia in their immediate vicinity while the neighboring regions remain normally sensitive or are hyperesthetic. In **zona** and in **neuroparalytic keratitis**, on the contrary, the anesthesia is complete, and includes the whole extent of the cornea. This is more invariable as regards the last named, and explains the absence of pain at any time during the attendant infection and ulceration. This form of keratitis indicates a lesion of the trigeminal nerve, and one nearly always located around the Gasserian ganglion. The sensibility of the cornea is more or less deadened in all forms of **glaucoma** except the simple, that is, in all cases that are characterized by any active inflammatory conditions, and is often one of the first diagnostic signs of hypertension. It is especially valuable in wounds of the transparent lens, whether accidental or operative, as indicating **secondary glaucoma**.

Anesthesia of the cornea is sometimes present in **tabes**, but always accompanied by cutaneous numbness over the regions supplied by the trigeminal. It is frequent in leprosy from infiltration of the ophthalmic division of the fifth nerve (nerve of Willis) and of the cornea. Hysterical anesthesia of the cornea is usually attended by a form of hemianesthesia. Occasionally there also coexists hysteric amblyopia or amaurosis. Corneal hypoesthesia is part of the syndrome **sensitivo-sensorial hemianesthesia by organic lesion**—lesion of the posterior part of the capsule, or extensive cortical lesion of the parieto-occipital region. In narcosis from ether and chloroform, anesthesia of the cornea indicates the moment when the operation may begin. It is well to bear in mind that anesthesia of the cornea may be produced by certain alkaloids. The one most employed is cocaine, or the aqueous solution of its hydrochlorate. One drop on the cornea will cause superficial anesthesia in thirty seconds, which will last for about a quarter of an hour. Eucain, holocain and stovain act similarly, but the anesthesia is of shorter duration and mydriasis is not so pronounced.



## CHAPTER VII.

### IRIS.

As regards their general semiologic import there are only two features in connection with affections of the iris—that is, of the features having reference to the aspect of the membrane itself—that call for consideration, and they are **design** and **color**. In order to appreciate the slight alterations in the color of a diseased iris not only is it necessary to study the different tints that are physiologically present, but especially, if one wishes to describe them, a certain degree of chromatic education is also requisite. Many otherwise excellent clinicians are deficient as concerns training of the color-sense.

**The Design** (Fig. 2).—It is important that the clinician acquaint himself thoroughly with the topography of the front surface of the iris. The markings here, taken as a whole, constitute the **design** of the iris. The markings are properly designated **details**. The details that make up the designs are regularly present in the normal eye, though they vary considerably in appearance with the density and distribution of the pigment and the arrangement of the bundles of fibres constituting the stroma.

**The details** from within outward are:

1. The pigment ring.
2. The pupillary zone.
3. The corona.
4. The ciliary zone { Contracting folds.  
Radiating streaks.  
Crypts.
5. Peripheral zone.

**The pigment ring** is a portion of the uveal lining that is



turned outward to *bind* the pupillary border, as it were. It shows more plainly in a light iris, in contraction of the pupil and when the pupil is white from cataract. When, by means of the light of the ophthalmoscopic mirror reflected from the fundus the pupil becomes a luminous disc, it is seen

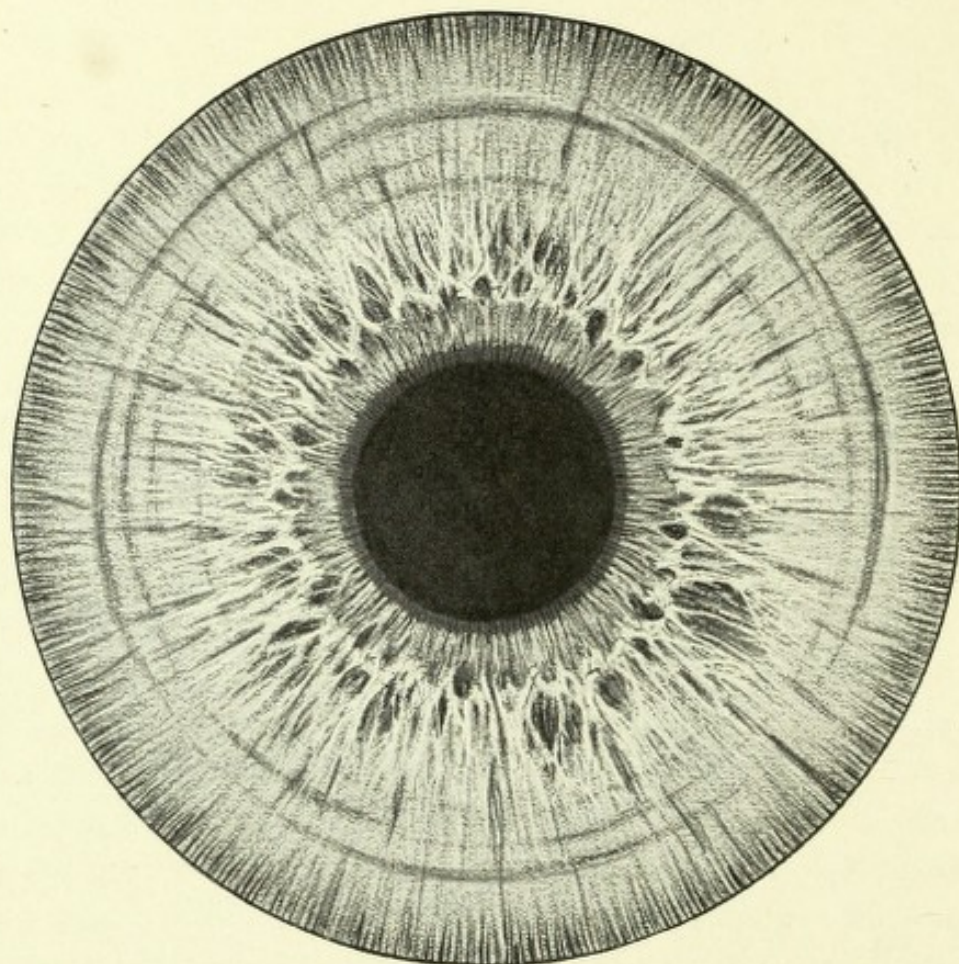


FIG. 2.—The iris. Immediately surrounding the pupil is the *uveal ring*, made up of dark brown pigment, and broken into many radiating folds. Just outside of that is the *pupillary zone*, also made up of fine radiating folds. Next comes an elevated, serrated network, well named the *corona*, for it sits like a crown on a maze of crypts. The network of the corona is more circumscribed inward, but it extends well over the next zone outward, which is the *ciliary zone*. In the latter zone are usually irregular cavities, or crypts, that are really spaces in the vascular layer. Often many of the radiating folds, or pleats, from the pupillary zone are carried across this zone. In addition it also contains the concentric, or contracting folds. The outer belt, which is mostly hidden by the opaque base of the cornea, is called the *peripheral zone*, and is composed of radiating folds coarser than those of the pupillary zone.

that the circumference of this disc is not an even circular line, but that it is nicked because of tiny irregularities in the pigment ring.



**The pupillary zone** is a relatively narrow portion, coextensive with the sphincter muscle, distinguished by tiny radiating folds and often by the sharp serration of its external outline. These deep folds and the sphincter are to the varying sizes of the pupil what the puckerings and the draw-string are to the mouth of a purse.

**The corona** is a circular elevation between the pupillary and the ciliary zones. The higher portions are the processes where the pupillary membrane of fetal life were attached; indeed, one can usually see here loose ends or tags, the remains of that membrane. The processes enclose deep, irregular crypts that are often richly pigmented.

**The ciliary zone** comprises the greater part of the visible iris. A number of the radiating folds extend into it from the pupillary zone. Toward its outer limits it is marked by folds parallel with the circumference of the iris—the **contraction folds**. These folds open out when the pupil contracts *ad minimum*. Other markings on the ciliary zone are the lighter radiating streaks that follow the trunks of the blood-vessels that lie beneath the anterior endothelium. They are not regularly radiate, but are more or less devious and often form fantastic figures. Openings, or **crypts**, of different forms and dimensions, in the more anterior layers, are almost constant in the ciliary zone. These are rendered more or less conspicuous by their depth and width, and the shadows cast in consequence, or by the manner of the distribution of pigment in or around them. Most often they present the general appearance of irregular blotches or mottlings. Exceptionally they individually, or in combination with the intervening lighter markings, assume the forms of letters or numerals.

**The peripheral zone** is mostly invisible in straight front view, owing to the opaque portion of the cornea that overlies it. It is somewhat darker than the ciliary zone, and is composed of fine radiate folds that are thought to owe their origin to contraction of the circular fibres of the ciliary muscle.

Morbid processes in the iris interfere with the whole design.



of the iris, or with its details in accordance with their extent and location.

**Physiologic Modifications in the Color of the Iris.**—Until recently it was taught that the color of the iris depended upon the amount or thickness of the pigment that constituted its uveal lining. This is true with respect to the eyes of infants, and, in a measure, of the different shades of blue and gray irides in adults, but not at all of the hazel, the brown and the black. The pigment contained in the uveal and retinal cells constituting the posterior stratum of the iris is nearly or quite black. In the iris of most infants the color is dark blue, or slate. Here the black pigment is seen through a thin, primitive white stroma, and, by interference, appears bluish. In proportion as the cells of the stroma pile up, the blue is lightened or clarified. If the stroma does not further change the iris remains blue, in other words, its permanent color is the result of interference purely. In a majority of instances, however, when the child nears the age of 12 to 18 months the iris begins to take on *positive* color. This is the result of the formation of pigment-granules within the protoplasm of the cells of the stroma—particularly those situated anteriorly—the round and the stellate cells. These granules vary in color from yellow through intermediate shades of yellow-ochre and brown-ochre to deep brown, and all shades are derived in some subtle manner from the hemoglobin. All iris-pigment is of hematic origin. The shades of gray, hazel, brown and black depend upon the tint, the number and the segregation of the pigment-granules in the anterior portions of the stroma. Their arrangement varies as greatly as their hues and their quantity, thus giving rise to diverse mottled effects, and, in rare instances, to an actual difference in the color of the eyes of the same individual—piebald eyes. It would be unfortunate, however, to mistake a beginning melanosarcoma of the iris for a spot of congenital pigmentation.

**Heterochromia** is a term that refers to unequal coloring of the iris, one sector, for example, being light—blue or gray—



and another dark—hazel or brown—without any departure from the normal in other respects. **Heterochromia** may be confined to one eye or it may be bilateral. When there is a difference in the color of the two irises the condition is called **heterophthalmia** or **dicoria**. According to some observers the lighter eye in dicoria is lacking in vigor, and is prone to certain affections, such as cataract. The lining pigment of the iris is nearly always visible over and about the sphincter, and sometimes, as has already been remarked, forms nodules or processes, or indentations that are plainly visible. Hirschberg called those excrescences of pigment which turn over upon the anterior surface of the sphincter zone **ectropion of the pupil**. They have no special significance.

**Pathologic Color.**—The principal affections in which the study of changes in the color of the iris is a valuable aid to diagnosis are iritis, diffuse intra-ocular hemorrhages, and iron or steel in the uveal tract or in the vitreous. The detection of slight modifications is greatly facilitated if the affection is unilateral, or, at least, if the iris of one eye retains its normal coloring, thus affording the advantage of **contrast**. The modifications in the color of the inflamed iris depend upon the kind and the severity of the iritis. In true, simple, serous iritis, in which the disease is superficial, discoloration of any kind seldom occurs. A great deal has been said about the discoloration of the iris from **hyperemia** and **congestion**. In reality the capillaries of the stroma are so sparse that it is only those blue and blue-gray eyes, that get their color from the uveal pigment by interference, and in the shades of hazel and light brown, that hyperemia can cause any alteration in the tint, and then the effect of the layer of red blood is to merely cause darkening of the original blue—not to produce green. Hence, in the rare cases of uveitis in infants, as for example, in connection with certain infections of the uvea, and in iritis from intra-ocular growths, the iris of the affected eye is notably **darker** than that of its fellow. The red of the injected subendothelial capillaries shows more plainly in the hazel and the brown than in blue eyes, because



the superposed colors are not complimentary. For a greenish hue to be imparted by interference it is necessary that the stroma of the iris be the seat of serous and cellular infiltration, thus overlaying the blue with a yellowish instead of a whitish translucent medium.

For the iris itself to be discolored in eyes of any shade **the inflammation must be parenchymatous**. Such coloration is **not muddy** and it is more pronounced in the thinner, or middle zone of the iris, least so at the thickest, or corona. The discolorations of the iris described as **dirty, muddy**, etc., are due not so much to alterations in the hue of the iris *per se* as to overlying fibrinous exudates and gelatinous deposits on the iris and on the endothelial lining of the cornea, and often also, in great measure, to turbidity of the aqueous. The blue iris then turns a muddy green, and the color of the brown one becomes what Panas fittingly termed "**faded chamois**." In suppurative iritis the color is often designated "brassy" and "dirty straw" irrespective of the natural color in the case. After a long siege of chronic iritis, one often finds large blood-vessels, mostly veins, running over the anterior surface of the iris, or neoform capillaries in the ciliary zone, and so numerous sometimes are these last as to impart a reddish tinge to that part of the membrane.

**Siderosis**, or the impregnation and staining of the iris by the oxid of iron, is often the decisive symptom in the diagnosis of iron or steel within the eye-ball. The rusty color of the iris is highly characteristic, and is of itself sufficient to determine the presence of the foreign body. Siderosis of the iris is not infrequently simulated by the yellow-brown discoloration seen in conjunction with recurrent intra-ocular hemorrhage. The products of old hemoglobin tend to impart hues of yellow and brown to the tissues.

**Atrophy of the Iris.**—The principal causes of degeneration of the iris, as tabulated by Venneman, are:

1. Chronic, spontaneous inflammation of the membrane, leading to sclerosis.
2. Exaggeration of the intra-ocular tension, by which the



entire iris is compressed, or its peripheral zone is brought and held in forcible contact with the base of the cornea.

3. Stretching forward of the membrane, from pressure of the aqueous in the posterior chamber, when the pupillary border is adherent to the capsule of the lens.

4. Long contact of an exudate, or other inflammatory product, or of a fragment of lens cortex, causing a chronic irritative inflammation at that spot.

5. Senile arterio-sclerosis.

6. Toxi-infective arterio-sclerosis. As from gout and rheumatism.

As a general rule sclerosis of the ciliary body complicates atrophy of the iris.

Atrophy of the iris may be roughly classed as that **from inflammation**, and that **from compression**. It is never primary.

*From Inflammation.*—In no organ of the body is inflammation more promptly followed by degeneration than in the iris. Sometimes a single attack is sufficient to induce a very considerable degree of atrophy; and the chronic or the recurrent forms of iritis always produce this result, either *partial* or *general*. The partial phases affect the iris as a whole or only portions of it. That is, the degeneration consists in a slight depigmentation of the cells over the entire surface, imparting to it a general dull, slightly yellow or brownish tint—spoken of as *washed-out look* and *dead-leaf aspect*. When this partial degeneration is confined to certain areas only, the effect is a mottling of the dull yellow and brown—*aspect tigré*. Again, to the general, partial degeneration there is added a deeper one in plaques, when the affected spots assume a dull gray, or slate color. All depends upon the extent of obliteration in the lumen of the vessels from the endovasculitis that characterizes the disease. In syphilitic iritis, wherein endarteritis constitutes the dominating feature, many of the arteries are obliterated wholly or in part, through sclerosis, and, in consequence, the membrane is



proportionately thinned. The thinning is evinced by the disappearing of the *design* and the *topography* of the iris. Instead of the normal "reliefs," the iris becomes flat and monotonous. It is in these elevations that lie the small endothelial capillaries of the anterior face of the iris, whose obstruction leads to the flattening, by sclerosis, of the embryonary connective tissue. In advanced cases crypts and elevations alike disappear. Now the sclerosed radiating vessels appear with abnormal distinctness, by reason of their thickened white walls, and the thinning of the iris. Sclerosis of the vessels entrains atrophy of the stroma. In its place there is often formed a thin layer of fibrillary connective tissue, so veiling the black uveal pigment as to impart to the whole iris a slaty tint. Sometimes one sees, meandering anomalously over the surface of the flattened membrane, a large blood-vessel. It is most often a new formed vein, returning the blood to the last of the ciliary processes that have been spared by the accompanying sclerosis of the ciliary body. At its point of origin there is a plexus of microscopic veinules, so numerous as to simulate an interstitial hemorrhage. A profoundly degenerated iris is prone to become extremely fragile, and friable. In attempting iridectomy upon it, for example, it is difficult to seize with the forceps. When caught, and traction is made to withdraw it, one may only succeed in breaking off an amorphous fragment. This is particularly true of degeneration from senile arteriosclerosis.

*From Compression.*—When long subjected to pressure greater than that which is normal to the globe the iris atrophies because of the mechanical and paralyzing effect upon its nourishing vessels. Hence, chronic glaucoma is always attended by more or less atrophy of this membrane. True, here the two causes, sclerosis and pressure are usually coöperative. This is, in many instances, also the case in the condition known as *iris bombé*. Whether the intra-ocular tension is raised or normal, or even subnormal, the



pressure of the fluid encapsuled behind the iris has the same effect as compression. The attenuation of the iris is, in a measure, contingent upon the stretching effect of the force behind it, but, in a much greater measure, upon the sclerosis, or disappearance of the stroma. Consequently, when the degeneration is partial and in plaques, the sclerotic portions are indicated by their greater distention. While those parts in which the blood-vessels have better preserved their calibre are less prominent, though still of a convexity somewhat accentuated. Interspersed with the different elevations there may be certain areas that are yet lower, that appear sunken, and are perfectly fixed. At such points the membrane is adherent to the lens. These varying mounds of ectasia, with their alternating hollows, impart a sort of knobbiness to the iris. By oblique illumination one may often locate the adherent portions, distinguishing them by their depth from those that are free, or less depressed. Occasionally this becomes an important question, for instance, in choosing the site for an iridectomy, as in recurrent iritis. Again, the stroma is absent over a large segment of the iris, leaving only the uveal layer, which bulges forward, giving the semblance of a melanotic sarcoma—*strophyloma of the uvea*.

From the combined effects of inflammation and pressure, as in senile arterio-sclerosis with prolonged hypertension of the globe, the wasting of the iris often becomes so extreme as to reduce it to a mere frame-work of fibrillary connective tissue. Both stroma and uveal pigment cells having so completely disappeared, that, by ophthalmoscopic examination, through the larger interstices of the net-like remains there shines the scarlet fundus reflex; or, if the lens be opaque, by oblique illumination the white of the cataract may be seen through the openings. In certain rare cases these holes in the iris are so clean cut as to resemble accessory pupils, or *polycoria*, and to cause monocular diplopia, or *polyopia*. Such conditions are particularly apt to obtain after the absorption of condylomata in syphilitic iritis.



Exceptionally the uveal epithelium undergoes a peculiar kind of degeneration while yet the stroma is, to all appearance, fairly intact. The cells grow softer and but slightly cohesive. On the slightest contusion, as in handling with the iris forceps, they are dislodged in great numbers, and their pigment, mingling with the aqueous, looks like a weak suspension of India ink. In diabetes these cells are subject to a glycogenous degeneration, when the same discoloration of the aqueous is likely to occur in the progress of an iridectomy or an extraction.

It becomes a matter of great moment at times to distinguish apparent changes in the aspect of the iris from real ones, or those occasioned by changes that have occurred in cornea, which is the medium through which we see the iris, from those in the membrane itself. Owing to the peculiar character of the corneal trouble the iris may seem dull, lacking in detail, or even discolored. In the early stages of interstitial keratitis and of inflammatory glaucoma the steaminess and opalescent ground-glass effects of the cornea are apt to cause erroneous ideas relative to the appearance of the iris. So, too, in certain low grades of **uveitis** and **iritis serosa**, opacities of the posterior layers of the cornea may give to the iris a tarnished or discolored look. Careful scrutiny with the aid of oblique focal illumination, and, if need be, with that of the stereoscopic loupe, will clear all doubt.

**Iridodonesis, trembling or shaking of the iris**, is never a purely physiologic phenomenon. Under normal conditions the inner zone of the iris is held firmly against the anterior surface of the lens—that is, it does not float in the aqueous, and the latter is rendered incapable of displacement *en masse* in any direction by the rigid surroundings. It is like the contents of a liquid leather bottle, or a wine-skin, that is tightly filled and securely closed, *unshakable*. Now let out enough of the liquid to relax the leather walls of the bag without leaving an actual empty space, and one can agitate the contents by shaking. Although there is no



empty space the walls yield to the weight or inertia of the liquid. It is precisely the same with the eye. So long as the intra-ocular tension is normal or raised, there is no tremulousness of the iris. But let the tension be reduced, and added to this absence or luxation of the lens, or liquid degeneration of the vitreous, and the iris shakes with the movements of the globe. This is **iridodonesis**. Neither aphakia, nor fluid vitreous alone, nor both together, however, can produce it save to an insignificant degree while there is normal or elevated tension. Liquid degeneration of the vitreous, and shrinking or crumpling of the lens, without dislocation, may give rise to iridodonesis. Provided the inner zone of the iris is still supported by the lens only the peripheral zone can shake. Indeed, the movement is usually more pronounced in the lower portion of the iris. Contrary to what might be suspected, mydriasis does not favor the movement, nor does total annular posterior synechia prevent it.

Iridodonesis in a highly myopic eye is a sure sign of hypotension, also of fluid vitreous. Here the osmotic tension of the vitreous has been reduced to the point where it no longer counterbalances that of the aqueous. According to Fuchs if the instillation of eserine into the apparently normal eye can give rise to iridodonesis it is because this myotic is capable of making subnormal the intra-ocular tension, or of destroying the perfect equilibrium that had previously existed between the osmotic tension of the aqueous and that of the vitreous.



## CHAPTER VIII.

### THE ANTERIOR CHAMBER.

**Dimensions.**—The measurements of the anterior chamber vary physiologically with age, with the size of the entire globe, and also, within certain limits, with individual peculiarities. The dimension which most concerns us from the semiologic standpoint, is the sagittal diameter, or depth, though the opposite measurements are not to be ignored. The physiologic depth depends largely upon the quantity of aqueous humor that is being formed. The average depth of the chamber in the adult is 2.5 to 3 mm. It is normally shallower in both extremes of life. In looking for signs of an intra-ocular growth in infants one could mistake the normal shallowness for that caused by a tumor, and in the aged it could be taken for that of simple glaucoma. Increased depth of the anterior chamber is normal, one might say, after removal of the lens by operative means; the same is true in myopia.

**Abnormal Measurements.**—The anterior chamber becomes effaced in proportion as the iris approaches the posterior surface of the cornea. This effacement is brought about mechanically, as when a neoplasm in the vitreous chamber pushes the lens and iris forward, or it is the result of peripheral adhesions between iris and cornea, as in glaucoma. In the latter case *all* dimensions are decreased. The chief causes of partial or complete obliteration of the anterior chamber are injuries, operative wounds, crowding forward of the contents of vitreous chamber, and local pathologic processes within the chamber itself. Injuries operate directly and indirectly; that is, one that results in an opening through the cornea operates directly by draining off the aqueous, and one that causes a hemorrhage into the vitreous operates



indirectly by crowding, and one that causes a forward luxation of the lens operates indirectly by pressure from behind. It sometimes happens that a traumatic perforation of the cornea is so tiny, and so situated, as to be invisible in ordinary inspection, and obliteration of the anterior chamber is the only evidence of it. To discover obliteration in conjunction with corneal ulcer means that the latter has perforated, and is not an uncommon incident even as regards certain very small, but surprisingly deep ulcers not accompanied by inflammatory reaction. After extractions and iridectomies for the anterior chamber to remain empty for more than a few hours constitutes **delayed union**, and this is usually due to the presence in the corneal incision of a portion of the iris or shreds of capsule or fibrin, etc. Re-efacement after the incision has once closed means an accident either from without or within. The first might be a blow or undue pressure over the globe, the second expulsive hemorrhage of the choroid. Perceptible shallowness of the anterior chamber after extractions and iridectomies, without any leakage of the wound, points to detachment of the choroid. Crowding of the contents of the vitreous is consequent upon exudations, as in **metastatic exudative choroiditis**, in **profuse hemorrhages** and the **growth of tumors in the vitreous**.

Of the local pathologic processes that affect the depth of the anterior chamber the principal are those that are consecutive to iritis and to chronic glaucoma, either simple or inflammatory, and these, in turn, have reference mainly to changes in the floor of the chamber. The floor is constituted by the whole of the iris together with so much of the anterior capsule of the lens as is enclosed by the pupillary circle. The inner zone of the posterior portion of the iris rests not lightly but, owing to tenseness of the musculature of the iris, and to the fact that the plane of the iris is posterior to that of the anterior summit of the lens, with a certain degree of force, directly upon the anterior capsule so long as the pupil is at its minimum width. It thus acts like



a valve between the aqueous in the posterior and that in the anterior chamber, and interchange between the contents of the two chambers is only intermittent, that is, in moments of partial dilatation of the pupil. From either serous or parenchymatous iritis union readily occurs between the parts of capsule and iris that are in contact. If this union is complete there is what is known as **exclusion** or **seclusion of the pupil**, consequently the floor of the anterior chamber becomes one piece, and any interchange of fluid between the two chambers must be through osmosis. The secretion of aqueous from the ciliary processes is more rapid than that from the anterior surface of the iris, hence, in exclusion of the pupil this tends to raise the floor of the anterior chamber—crater-shaped iris, or **iris bombé**. In exclusion of the pupil, fluid may also be poured into one or the other of the front chambers of the eye from the blood in the capillaries of the iris by exosmosis if the solid constituents of the aqueous in either become deficient. This may have the effect either to increase or to decrease the depth of the chamber, owing to the direction of the flow. Reference has already been made to effacement of the anterior chamber at the periphery. The study of this narrowing of the confines of the cavity by adhesions between the iris and the cornea, or **ankylosis of the irian angle**, is an interesting and important feature of ocular semiology. Just why the anterior chamber is made so shallow in glaucoma *without* these adhesions, as in the acute inflammatory form, is not quite clear, but it is a constant and valuable sign, nevertheless.

**Depression of the floor** of the anterior chamber is usually what constitutes **increased depth** of the cavity. Exception to this is to be found in **hydrophthalmos** or **infantile glaucoma**, in which it is accomplished by elevation of the roof or dome, or, literally, by expansion and bulging of the cornea, as the iris does not alter its plane in this affection. The anterior chamber in this disease may reach a depth of from 15 to 18 mm. Depression of the floor is brought about by luxation or subluxation of the lens backward, and in iritis serosa,



by a sort of hydrops of the two aqueous chambers, and in exclusion of the pupil by excess of fluid in the anterior chamber. With the last two conditions there is presented the unique association of increased depth of the anterior chamber and increased tension of the eye-ball. Inversely, there is often hypotension in exclusion of the pupil in conjunction with marked effacement of the anterior chamber. Abnormal depth is, moreover, a symptom of aphakia, as after extraction; and, when the iris is intact, it is sometimes the only evidence, in cursory inspection, that a cataract has been removed.

**Visible Contents.**—Modifications in the appearance of the contents of the anterior chamber, as clues to troubles going on within the eye, are well worth considering. Common causes of such modifications are **hypopyon** and **hyphema**. The first is a general term for accumulation, to a greater or lesser extent, of whitish or yellowish material in the lowest part of the anterior chamber. It is called **true** when it is composed partly of pus-cells—cells that have migrated from the capillaries of the iris, mixed with fibrin, and **false** when it is principally made up of young round endothelial cells, or other superficial cellular elements. It appears under seemingly very diverse conditions, yet, after all, in most cases it harks back to similar pathologic processes. In traumas, such as foreign bodies in the cornea and within the globe, it is significant of infection. The same is true of that seen after operations that consist in incising or puncturing the cornea. It is common in connection with ulcerations of the cornea, but the amount of the hypopyon is not necessarily in accordance with the extent and gravity of the ulceration. It is also frequent with iritis. Here it may serve, on occasion, to aid the differentiation between serous and parenchymatous iritis. The false hypopyon of serous iritis is more fluid, and follows more promptly the movements of the head in seeking, by gravity, the lowest portion of the anterior chamber. If the head of the subject be shaken rapidly, false hypopyon can often be made tempo-



rarily to disappear, as thus the cells composing it are mingled throughout with the aqueous. It is habitually less, and of lighter color, *i.e.*, not so yellow, in the serous form, nor is it so apt to coagulate as the true hypopyon of the parenchymatous.

Fluid hypopyon is distinguished by the straight level it maintains on top. If it coagulates, its upper surface curves **convexity upward**, or it becomes irregular, so long as the hypopyon is on the increase, and regularly or irregularly **concave** when on the wane, or being absorbed. The property of hypopyon to move about in obedience to the laws of gravity may be utilized at times to decide whether the appearance at the bottom of the anterior chamber is really an exudate or a beginning neoplasm at that point. Also, in the event of a foreign body in the chamber, which may become enveloped in whitish material and simulate hypopyon, the head may be shaken and turned about so as to scatter the envelope and reveal the substance. Slight or moderate hypopyon is sometimes an early sign of **metastatic uveitis** (**cyclitis** or **choroiditis**.)

**Hyphema.**—Blood in the anterior chamber is often more an evidence of traumatism than of disease. It may be from an actual perforating wound that involves the conjunctiva or the iris at the base of the cornea, or it may be from a concussion, by which an intra-ocular vessel is ruptured. Its presence or absence after ocular injuries is worth noting. It is frequently observed after operations for cataract, and after iridectomy from wounding the conjunctiva or the iris. Occurring in the course of the after-treatment relative to these operations it usually indicates that the eye has suffered violence in some manner, and the blood, of itself, is of little consequence. If, however, there is persistent pain and redness or other symptoms of irritation of the eye, it is probably significant of uveitis. Recurrent hyphema after operations that involve opening of the globe, and in connection with uveitis, is a sign of gravity. Luetic, diabetic, and arterio-sclerotic subjects are specially prone to it. It often



appears with the hyperemia which precedes an attack of iritis. Blood sometimes appears in the anterior chamber in connection with sarcoma of the iris or ciliary body, and spontaneously in purpura and leukemia. Like hypopyon it may serve to hide a foreign body and necessitate paracentesis with or without lavage to clear up matters—figuratively and literally.

Among the occasional **anomalous contents** of the anterior chamber are **transparent** or **implantation cyst of the iris**, and **anterior dislocation of the transparent lens**, and they are both of peculiar aspect. The body of the first overspreads the iris with a faint tinge of yellow that has a thin, dark outline. There is generally a history of a traumatism or of a surgical operation to account for it. When the clear crystalline is luxated so as to pass forward through the pupil and lie on the iris it is recognized by its bright, glassy outline, by the magnification of the iris and pupil beneath and by the depressed plane of the iris. There is often secondary glaucoma and the acknowledgment of an injury with it. If the dislocated lens is cataractous there can be little doubt as to its identity.

*Particles of Pigment Free in the Anterior Chamber.*—As will be seen on referring to the section on persistent pupillary membrane these remains are subject to breakage and absorption. We shall also see that pigment is sometimes adherent to the centre of the anterior capsule, and that so lightly as to cause no trace of anything abnormal in the endothelial lining of the capsule directly beneath it. This anomaly, too, is supposed to be a relic of the fetal capsulo-pupillary membrane. Tags and shreds of this membrane are often observable clinging to the highest parts of the **corona iridis**. The masses or granules of pigment sometimes mingled with degenerated epithelium and fibrillary connective tissue, which have occasionally been discovered (for instance, by Bock, Businelli and Apetz) lying loose in the anterior chamber are doubtless but the disintegrated fragments of the pupillary membrane.



## CHAPTER IX.

### PUPIL.

**Physiologic Modifications of Diameter.**—The analogy between the iris and pupil and the diaphragm and stops of a camera is as true as its citation is trite, and, with regard to the later models of the camera, it is truer still, for here the adjustable stop fulfills precisely the same office for the camera that the pupil does for the eye. Yet, with its two purely mechanical functions to perform, viz. (a) the regulation of the intensity of light that enters the eye, and (b) the sharpening of the retinal image by shutting off circles of diffusion of the dioptric system, the physiology and the pathology of the pupil are most complex and its symptomatology is most eloquent. Be it understood that the two functions under discussion are those of the **pupil**, or aperture of the iris. If the entire office fulfilled by the **iris** were under consideration one should not omit the important part it plays in supplying the aqueous humor and in maintaining the normal intra-ocular tension.

The study of the diameter and the reaction of the pupil is of little greater consequence to the ophthalmologist than to the neurologist, or in fact to physicians in general. It is true that our pathologic conceptions, especially those which concern diseases of the nervous system, have been greatly modified relative to the character of the information furnished by the semiology of the pupil. The result has been, however, more to alter former ideas as to the value of the pupillary symptoms in question than to actually lessen that value, which is, and must always remain, inestimable. Between maximum and minimum accommodation and convergence under steady illumination there is a variation in



the pupillary diameter from  $2\frac{3}{4}$  to  $4\frac{3}{4}$  mm. The diameter is increased from  $1\frac{1}{4}$  to  $3\frac{1}{4}$  of a mm. by covering the fellow eye. Neither the different states of the refraction nor the degree of pigmentation of the iris affect the **relative** diameter of the pupil. Let the size of the eye-ball be great or small, the dimensions of the pupil are maintained in exact proportion.

The **average diameter** of the normal adult pupil in a globe of average volume, seen by diffuse daylight, with the eyes in repose and facing the light, if in a room, is about four millimeters, but through different influences it may range anywhere between one and ten millimeters. Its outline is circular and about concentric with that of the iris. It is somewhat above the average diameter in childhood and adolescence and below it in the extremes of age and infancy. In all, however, it fluctuates incessantly during all periods of consciousness and throughout life. In natural sleep only is the pupil in repose and it is then strongly contracted. In **rigor mortis**, through rigidity of the sphincter, the pupil becomes smaller, immediately after that larger, and eventually, when the globe has lost all tone, again contracted. When the blood-pressure is suddenly stayed, as in the face of immediate death and at the moment when the narcosis of an anesthetic becomes dangerous, the pupil dilates mechanically from the force of the intra-ocular tension; in consequence of the sudden stoppage of the heart the arterial pressure is so lowered that no more blood enters the globe to counterbalance the tenseness there.

Aside from individual variations the pupillary diameter is influenced by so great a number of factors that, in the study of the semiology of the pupil, one can take into account only those modifications that are extreme, or at least quite manifest; and, in addition, inequalities in the size of the two pupils is to be reckoned with. An obvious diminution in the diameter is designated as (1) **myosis**, obvious excess as (2) **mydriasis**, and obvious difference in the relative diameter of the two pupils as (3) **anisocoria**.



**Myosis**, or abnormal contraction of the pupil, is the result either of excitation of the third nerve, **spasm of the sphincter muscle**, or of enervation or paralysis of the sympathetic—**paralysis of the dilator**—or it may result from purely mechanical causes. Common causes of spasm of the sphincter are superficial irritations of the cornea, such as foreign bodies and abrasions of that membrane, hyperesthesia of the cornea and retina, eye-strain, the local effect of certain drugs, intoxications like those of morphin and urea, and the presence of any intracranial affection that stimulates or irritates the nerves of the sphincter without destroying the central cells with which they communicate. The light-reflex and response to efforts of accommodation and convergence are, in a measure, retained in most of the forms. Given unilateral myosis, especially if there is irritation of the eye, one looks first for a source of corneal irritation and next for the **mechanical myosis** that comes with hyperemia and inflammation of the iris. Spasm of the sphincter from corneal stimulation is well demonstrated by squirting a stream of boric acid solution upon the cornea from an eye-dropper. A 2 per cent. solution of **pilocarpin** dropped into the eye will provoke a myosis that attains its maximum in thirty minutes, and that disappears after three or four hours. **Eserin**, as a myotic, is more energetic, as a 1 per cent. solution will, in the same length of time, produce a myosis lasting from thirty-six to forty-eight hours. These computations have reference, of course, to the normal eye.

Among the intoxicants that cause myosis are **opium** (or morphin), **nicotine**, **chloral** and **urea**. The first two enter the system by the alimentary canal or hypodermically and cause moderate contraction of both pupils which lasts several hours. In profound coma, therefore, if instead of the usual mydriasis there is myosis, poisoning by morphin or opium is suggested. In early or moderate coma the pupils are contracted. At the onset of **tubercular** or other **meningitis**, in **hemorrhagic encephalitis**, in **tumor and abscess of the brain** there is, sometimes, a corresponding or a bilateral myosis.



If the lesion extends to the nucleus of the third nerve the myosis is succeeded by mydriasis.

**Paralysis of the cervical sympathetic** is always accompanied by myosis. This myosis has been often demonstrated in the human subject by Jonnesco's operation. The pupil in paralytic myosis still retracts to light and to eserin, and may be dilated by atropin. Associated symptoms are ptosis and anesthesia of the fifth nerve. In extirpation of the superior cervical ganglion the pupil is made more readily dilatable by **adrenalin**, whereas normally it is but little affected by that drug. Its mydriatic action is inhibited by its power to lower the intra-ocular tension. Paralytic myosis is also observed in traumatism of the cervical sympathetic, sometimes in neuritis involving the brachial plexus of that side, in **tubercular cervical pachymeningitis** and in **syringomyelia**. A number of affections that produce lesions in the vicinity of the cervical sympathetic have been known to cause a corresponding myosis. Among them are **adenopathies**, phlegmonous angina, goitre, tumors of the mediastinum, etc. But no general disease is so frequently the cause of myosis as **syphilis**, notably when the disease in question manifests itself as a **cerebro-spinal localization**, that is as **tabes**, and as **general paresis**. It occurs in the first period of tabes, and **even to a high degree, after complete tabetic optic atrophy**, whereas in other forms of optic atrophy there is ordinarily mydriasis; and it may persist for an indefinite time. Hence, **myosis and acquired blindness** becomes a syndrome of tabes or general paresis. Tabetic myosis resists the action of cocain and atropin. Myosis is now and then seen in **hysteric amaurosis**, especially when accompanied by photophobia, spasm of accommodation and blepharospasm. In **natural sleep** the myosis is said to be in accordance with the profoundness of the act, and the light-reflex remains undiminished.

Coppey has tabulated spasmodic and paralytic myosis with reference to the effects of myotics and mydriatics, as follows:



### Spasmodic Myosis.

Atropin dilates the pupil as under normal conditions.

Cocain is inert, as spasm of the dilator cannot overcome that of the sphincter.

Eserin produces no effect. (Others have stated that other spastic myoses are not so great as that caused by the local action of eserin.)

### Paralytic Myosis.

Atropin produces moderate dilation, as the two paralyses are exactly opposite.

Cocain dilates the pupil if the nerve terminations of the dilator are intact.

Eserin produces maximum myosis.

Both forms of myosis may reach such an extreme as to rob the symptom of its clinical value. The sensible pupil, for instance, may be reduced to a diameter of one millimeter (**micoria**) and yet be wholly without pathologic significance.

Diminution of the intra-ocular tension entrains infallibly contraction of the pupil, at least, to a degree; for this form of myosis is never extreme like that from spasm of the sphincter. This is illustrated constantly in making the corneal section for an extraction or iridectomy. The pupil also contracts, and is extremely difficult of dilation by atropin, when there is hyperemia of the iris. These are instances of **mechanical myosis**.

**Mydriasis.**—Relative dilation of the pupil, like myosis, may be functional, *i.e.*, due to **excitation** or **irritation** of the **cervical sympathetic** by an intra-cranial or a cervical lesion, or through the influence of psychic and sensory phenomena to **paralysis of the sphincter**. In functional mydriasis the light- and convergence-reactions are present. The mydriasis of cocain is an example of the functional kind. That from the instillation of adrenalin solution is an instance of what has been called **angio-spastic mydriasis**,—*i.e.*, from ischemia of the iris—**mechanical mydriasis**. Spastic mydriasis is



often observed in young subjects, particularly when mentally excited, and is not to be confounded with that sort of paresis from fatigue of the accommodative apparatus, seen in the same class of subjects from imprudent use of the eyes in near vision and which is also characterized by mydriasis. The mydriasis of **anemia**, either local or general, is indirectly of the functional variety, though directly it is **mechanical**, and is due to attenuation of the vascular portion of the iris. Ischemic mydriasis always precedes syncope, and it is present with the Cheyne-Stokes type of respiration during the period of acceleration in the respiratory movements. It is the antithesis of myosis from hyperemia of the iris.

**Paralytic mydriasis** is really paralysis of the sphincter of the pupil, and is the effect of a drug or of a disease. In paralytic mydriasis the dilation of the pupil is never extreme, like it often is when produced by drugs.

Following the classification of Venneman, a distinction will be made between (a) bilateral mydriasis with or without loss of the visual function, and (b) monolateral mydriasis not accompanied by blindness.

**Bilateral Mydriasis with Blindness.**—The entire suppression of the visual faculty caused by a lesion of the retina, of the optic nerves, of the chiasm or of the optic tracts—in other words paralysis of the centripetal pupillary fibres—has for result mydriasis. Exception must be made as regards a certain number of cases in which blindness from optic atrophy is in relation to tabes or to general paresis. In discussing myosis, we have already seen that, notwithstanding complete blindness, the contraction of the pupil in tabes may persist for a long time. All lesions, then, that involve the entire retina do *not* entail mydriasis. Among those that *do* are **obliteration of the central artery of the retina**, **section of both optic nerves by a gun-shot wound**, and, generally speaking, the optic atrophy referable to **intra-ocular tumor**, to **tubercular or syphilitic meningitis** and to **fracture of the base of the cranium**. The mydriasis in these instances seems to be due to relaxation of the sphincter



consequent upon the absence of any excitation of the centripetal pupillary fibres. It is not due to paralysis in the true and strict sense of the word.

It may be said that all blindness resulting from a lesion **not cortical** has for effect mydriasis with absence of the light-reflex. Such is not the case in **cortical blindness** from circulatory or other lesions of the occipital lobes, for here the pupils retain their normal diameter and their reaction to light. Exceptions are found in the mydriasis of coma—other than that of opium or morphin before referred to. In this state there is marked mydriasis with loss of reaction to light; but, in these cases, it would seem that the mydriasis is ascribable to want of photo-motor excitation.

**Bilateral Mydriasis with Conservation of at Least Partial Vision.**—In the presence of such mydriasis the first thought is that it is medicamentous—*i.e.*, caused by atropin or some substance of similar properties. It is an old story, how the mother or the father wished to show the child for whom they were prescribed, that the eye-drops would not hurt and put them in their own eyes, or how the druggist instilled a couple of drops into his customer's eyes to cure a slight inflammation and to save for him a doctor's bill. They become alarmed at the symptoms and consult, not the one who prescribed the drops, but another. The parent is ashamed to confess because of the stupidity of the act and the other is afraid to disclose the illegal act of the druggist, hence the examiner must determine the nature of the mydriasis by the symptoms. **Atropin** and **duboisin** both produce a maximum mydriasis, always accompanied by paralysis of the accommodation. A single drop of a 1 per cent. solution of the atropin sulphate causes dilation of the pupil in fourteen minutes that will last for two or three days, provided the eye is quiet. Solutions of duboisin sulphate need to be only half the strength of those of atropin, and the effect is more prompt to appear and also to disappear. Solution of eresin will substitute myosis for the last mentioned two forms of mydriasis, but unless the latter is



nearing the end of its period it will reassert itself after a few hours. The hypodermatic injection of preparations of belladonna, sufficient to produce the same effects upon the pupil and the accommodation, will also give rise to the systemic phenomena of the intoxication. Certain preparations of **pilocarpin** contain what are known as **isomeres** of pilocarpin, or **isopilocarpin**, which have the power of **dilating** the **pupil** but not of paralyzing the accommodation. They present the same chemic reactions as ordinary pilocarpin. The mydriasis which is present, and more or less pronounced, in most all cases of acute and chronic inflammatory glaucoma with preservation of useful vision may be reduced by myotics and increased by mydriatics, and the reactions to light and accommodation are obtainable, though it may be only in slight degree. Bilateral mydriasis from paralysis of the third pair consecutive to syphilis, is by no means uncommon. It relates to meningitis or periostitis at the base, or to a neuritic process when the syphilis affects the tabetic type. It would also seem that syphilis may also attack directly the nuclear centres. It is admitted that this occurs in **bilateral internal ophthalmoplegia**, which is characterized by mydriasis and paralysis of the accommodation. This syndrome is considered grave, as it indicates that the causative process threatens to involve other nuclear centres—superior and inferior polyencephalitis—but such extension does not necessarily occur.

**Post-diphtheritic ocular paralysis** furnishes a fine example of the **indirect**, or **functional** (central or cortical) paralysis as opposed to the **direct**, **true**, or **muscular**, as in the traumatic form, wherein there is an actual lesion of the muscle or of its nerve-fibres. It supervenes during the period of convalescence, or within the first two months that follow the appearance of the false membranes in the nose or throat. While it is, in most instances, confined to the ciliary muscle it often involves also the sphincter of the pupil. The paralysis is usually incomplete, as atropin instilled will bring out latent hyperopia, and eserin will, for the time, restore



the function both of the ciliary and the sphincter muscles, thus proving that they are of themselves intact. Post-diphtheritic paralysis follows only the general diphtheritic process, never the local conjunctival infection, and but rarely the croupal exudation on the mucosa of the vagina, or on the surface of a superficial wound. It occurs, of course, mostly in young subjects. Associate paralysis of the palate, of the extremities and of the bladder are frequently present. More rarely certain of the external ocular muscles participate in the loss of function—notably the external recti, causing convergent squint.

In **botulism** or **ptomain poisoning**, an alimentary intoxication from the ingestion of food, such as canned meat, and fish-products contaminated by the development of an anaerobic microbe, the **bacillus botulinus**, bilateral paralysis referable to the oculomotor nerve is an almost constant symptom. It affects, by preference, the sphincter and the ciliary muscle, though not always in equal degree; for example, there may be preservation of the light-reflex and total absence of accommodation, and *vice versa*. Sometimes the external ocular musculature is involved. The mydriasis of this affection appears about ten days after the first symptom of the poisoning. Similar in nature is the ocular palsy due to poisonous mushrooms. With the exception of that from **agaricus muscarius**, in which there is myosis and spasm of the accommodation, this form affects the sphincter and ciliary muscle. Like the post-diphtheritic variety these other toxic ocular palsies disappear spontaneously. With **chloroform narcosis** there is mydriasis in the first stage, that of excitement, while during the remainder of the anesthesia the pupil is narrow. If it suddenly dilates syncope is imminent. If, however, the mydriasis comes slowly and gradually it indicates either an awakening, with emesis, or an excess of absorption of the chloroform with complications more serious.

In **acute meningitis**, **septic sinus-thrombosis**, **brain-abscess**, and **meningeal apoplexy**, bilateral mydriasis is not rare. It



often succeeds spasmodic myosis, and alternates with it, or persists throughout the course of the disease; but neither its presence nor its absence is significant as to the seat and the nature of the lesion.

The bilateral mydriasis that often accompanies hysteria and epilepsy is characterized by immobility to the light-impulse, and there is nothing about it to distinguish that which is hysteric from that which is epileptic.

**Unilateral Mydriasis.**—Some of the affections which cause bilateral mydriasis may also sustain the same relation to the unilateral form, and have been already mentioned. They are, for example, mydriatics when the drug has come in contact with only one eye, traumatisms and, most frequently, syphilis. This is particularly true of the luetic infection when it exerts its action upon the centrifugal pupillary fibres, in the orbit, in the cranium or in the vicinity of the nuclear centres. Simple unilateral mydriasis without interference with the light-reflex or accommodation and **unilateral internal ophthalmoplegia** are both frequent in syphilis, hereditary or acquired.

The causative agent, or lesion, in unilateral mydriasis may be **intra-bulbar** or **extra-bulbar**. To the first belong **contusions** and other traumatisms of the globe, with or without wounds of the iris or dislocation of the lens. With the last named complication the dilation is apt to be irregular and the iris tremulous. Blows from the balls used in tennis and other games are common factors in such mydriasis. The pupillary reactions to light and convergence are seldom affected.

**Traumatic paralysis of the sphincter of the pupil** is accompanied by palsy of the other intra-ocular muscles. It lasts for weeks or months but disappears in time. The mydriasis is never so great as that from atropin, as it is augmented by the instillation of a solution of this drug, while it is easily overcome by eserine. The accommodation is restored in a comparatively short time after the injury. If the contusion is recent, or the globe is still soft, there will be myosis and



immobility of the pupil—lack of accommodation alone indicating the ophthalmoplegia interna. The pupil will not respond to atropin so long as there is extreme hypotension. When the tension rises a single drop of atropin solution may cause a dilatation that will last for a considerable period. Myopia may suddenly develop in an otherwise emmetropic or slightly hyperopic eye after a traumatism, associated with myosis and minus tension, and be mistaken for a sign of spasm of the accommodation, and the ophthalmoplegia interna be thus overlooked. It will be found, however, that the eye is incapable of seeing with any stronger concave glass than the one which just measures the error. The probable explanation of the myopia is that the lens, through the concussion and the reduced tension, has assumed a more spherical form. This myopia is not nullified by atropin. Later, when the tension rises, the total ophthalmoplegia interna becomes apparent. The iris may disappear entirely, from retroversion, as a result of trauma of the globe, leaving a condition that simulates aniridia.

All traumatisms, neoplasms and inflammations capable of implicating the third nerve anywhere along its course from the nucleus to the globe may be an extra-bulbar cause of mydriasis. Contrary to former conceptions which were to the effect that the lesions in question must produce complete paralysis of the motor-oculi, they may be limited to the pupillary and accommodative fibres, *i.e.*, the dissociation of the paralysis does not necessarily imply a nuclear lesion. The extent of the mydriasis is variable, and the reactions of the pupil are preserved or lost according to the case.

The monolateral mydriasis of glaucoma is rare unless it is in conjunction with that which is consecutive to injuries, neoplasms, etc.; or, in other words, with secondary glaucoma. Glaucomatous mydriasis is peculiar in that it is accentuated in an upward direction.

With regard to **hysteric** mydriasis, since the pupillary centres are not subject to the authority of the will it would seem that they should not be deemed accessory to the



manifestations of this neurosis. Yet so numerous have been the observations tending to prove that hysteria may disturb the function of the iris-muscles that one is justified, after having exhausted all other means of interpretation in a given case of mydriasis, in naming it **hysteric**.

**Anisocoria.**—Inequality in the diameter of the two pupils is either **congenital** or **acquired**. It has been denied, but much oftener affirmed, that there is such a thing as physiologic anisocoria. Those who deny its existence explain the allegations of those who do *not* by attributing them to errors of examination, and by declaring them to be cloaks under which to hide ignorance as to the true pathologic causes that are always responsible for the condition whether said causes can be discovered or not.

**Congenital Anisocoria.**—That an inequality in the size of the pupils is often present at birth, or is discovered during the first months of infancy or first years of childhood, is incontestable. It is equally certain that, of the great number so characterized, the vast majority go through life without it being possible for any other than a physiologic cause to be definitely assigned for the peculiarity. In many instances it is so pronounced that the individual possessing it is aware of its presence, as also are all the near kindred and intimate acquaintances. In many others it is so slight that it requires trained powers of observation to detect it. It is more than probable that **anisometropia** is responsible for its existence. What more probable than that extreme differences in the refraction of the eyes—as in **antimetropia**—the myopic eye which is unquestionably larger than its hyperopic congener should also be possessed of a larger cornea, a larger iris and a larger pupil? Indeed, statistics rather favor the theory that physiologic anisocoria is most frequently associated with anisometropia. There still remains a large number of cases in which there is no difference in the refraction, or any other apparent condition to account for the anisocoria. The fact that there is often a certain degree of incorrigible amblyopia in one or the other eye—



though most often, perhaps, in that with the larger pupil and greater refractive power—lends weight to the opinion that back of the inequality of the pupils there may sometimes be an error in development, or an intra-uterine affection of a pathological nature.

**Acquired Anisocoria.**—It is often impossible from the evidence furnished by the relative diameter of the pupils alone in a case of anisocoria to exclude cases such as have been already alluded to in discussing the various kinds of myosis and mydriasis. For there is no sharp line dividing bilateral myosis and mydriasis from the unilateral forms, nor is there such a line dividing anisocoria from the different varieties of myosis and mydriasis. In those bilateral forms there are often differences in the degree of the dilation or the contraction in the two eyes, and there are many stages in their development and decline in which there must of necessity be over-lappings, and in which mere pupillary measurements are of no diagnostic value. Cases of anisocoria, then, are likely, on cursory examination, to be misleading, and it behooves one to study them with the utmost care. We will assume that anisocoria resulting from **unequal** bilateral myosis and mydriasis, and that in which neither pupil can be said to be myotic or mydriatic, have not been touched upon. **Persistent anisocoria** is a designation that has been applied to the less mutable and less transient types of acquired anisocoria. Since Baillarger, in 1850, discovered the frequency of anisocoria in general paresis, many observations have substantiated his assertions.

As has been said, **syphilis** plays the principal rôle in the production of anisocoria, with or without modifications of the reflexes. The inequality of the pupils may be unattended by any other ocular or general symptoms, and at all stages of the disease. When the luetic process takes the form of general paresis, anisocoria becomes extremely frequent—close to **50 per cent.** The **tabetic** type furnishes a smaller percentage of anisocoria—possibly not over **25 per cent.** The anisocoria observed in cerebral hemor-



rhages and softenings doubtless has the same ultimate origin, since the vascular lesions to which such cases are directly due are, as a rule, consecutive to syphilis. **Intracranial tumors**, notably those arising about the quadrigemina, the peduncle and the cerebellum may cause inequality of the pupils. Anisocoria from unilateral sympathetic myosis associated with **aneurysm** of the aorta has been already referred to.

**Anisocoria in Pulmonary Tuberculosis.**—Pupillary inequality, as a phenomenon accompanying unilateral pulmonary tuberculosis, has for some time been recognized, and was first described by Geza Fodor. In a large majority of cases in which the sign occurs, the pupil is wider on the side of the pulmonary involvement. This formerly led to the belief that the reaction is due to a stimulation of the sympathetic nerve-fibres, causing, on that side, a spastic mydriasis. Nevertheless, in many instances, the opposite is true, namely, that the pupil of the unaffected side is the wider, a fact which led many observers to discard the symptom as having no practical value. It remained for Leo Wolfer to determine the real cause of the pupillary difference, which is an excitation of the sympathetic fibres due to pressure exerted by enlarged bronchial glands of the affected side. For this reason it is not necessarily observed in the pupil corresponding to where our objective findings lead us to locate the pulmonary process, in so far as the glands on this side are not invariably and of necessity sufficiently enlarged to stimulate the corresponding sympathetic fibres.

After observing a large series of cases, Tuechter has been able to substantiate these latter findings, and he is satisfied that a comparative dilation of one pupil signifies an enlargement of the bronchial lymph-glands of the corresponding side. As such glandular involvement is usually tuberculous in character and takes place at a time when the lung itself has not as yet shown destructive changes, he feels that this sign is most valuable in the early diagnosis of pulmonary



tuberculosis. Before drawing a definite conclusion as to the significance of this phenomenon, it is of course necessary to exclude affections of the eye which may cause pupillary differences, as well as certain conditions of the thorax, such as tumor or aneurysm. This phenomenon, if we look for it, can very frequently be found in tuberculosis of the bronchial lymph-glands in children. It forms a valuable point in diagnosis in that type of the disease in which the onset is insidious, and in which the child has not as yet acquired the characteristic paroxysmal attacks of cough.<sup>1</sup>

**Alternating Anisocoria, or Anisocoria à Bascule.**—Sometimes the pupils in anisocoria alternate as to which is of the greater diameter, at one time it is the right, at another the left, and it is this "see-saw" peculiarity that gives the name. It is the *Springende Myrdriasis* of the German authors. It has been observed in the affections in which anisocoria is frequent, such as general paralysis, and tabes, and has also been noted in isolated cases of **multiple sclerosis, chronic cervical myelitis** and **infantile cervical paralysis**. Frankel admits its possibility in **exophthalmic goitre, neurasthenia** and **hysteria**, and believes it to be spasmodic mydriasis from organic lesions that cause excitation, direct or reflex, of the ocular sympathetic. This *spontaneous* alternating mydriasis is not to be confounded with that which is *provoked*. In order that this may be brought about there must be an inequality in the reflex excitability of the pupils—photo-motor, accommodative, palpebral. Take, for example, a case of general paralysis in which the pupils are, as usual, dilated, and in which the pupillary light-reflex is very active on the left side and very feeble on the right. In moderate light the right pupil is the smaller, whereas by intense illumination the reverse is true.

**Transitory anisocoria** is considered a symptom somewhat diagnostic of **neurasthenia**, or, at least it is assumed that permanent anisocoria warrants the exclusion of neurasthenia.

<sup>1</sup> Abstracted from Article by Tuechter, of Cincinnati, in the *Jour. of the American Med. Assn.*, Feb. 24, 1912.



Inequalities of short duration have also been observed in connection with certain forms of acute meningitis—particularly at the beginning. On the other hand, persistent anisocoria has been reported in cases of neurasthenia, but Morax believes that in the latter instances there may be presumption of an organic (syphilitic) lesion.

**The Pupillary Reactions.**—The iris is not loosely suspended in the aqueous but is held taut by the tonicity, or more correctly speaking, perhaps, by a sort of simultaneous, tonic contraction of the two opposing muscles—the contractor and the dilator (if the last can properly be called a muscle). It is thus in the best possible position to respond promptly and instantly to the demands of the retina, whether it be to modify the quantity of light admitted to it, or to qualify the netness of the image upon it—or both. The result of these impulses is to cause constant alternations in the diameters of the pupils, or oscillations, that are called **reflex movements**. Any excess in the tone of the sphincter, be it much or little, results in a corresponding contraction without in the slightest degree affecting the tone of the opposing force. The converse is true of the dilator. Hence, like reflex movements in general which are never so precise as voluntary movements, the oscillations of the pupils, owing to the varying degrees of illumination and to the varying distances of the objects fixed in the act of vision, are literally incessant. In reflex contraction of the pupil, for instance, from superabundance of light, the sphincter acts with too much energy and makes too small the orifice by which the rays of light enter, and a reflex dilation instantly follows to correct the exaggerated contraction. This also resulting in **over**-correction, requires a second correction, and so on, till a new change is demanded. These oscillations are plainly visible when the changes in the intensity of the light are decided. Disturbance in the physiologic *intention* of such pupillary oscillations, and exaggeration of their *extent*, constitutes **hippus**, or **iridokinesis**. This is an abnormal, slight or pronounced, more or less rapid, con-



tinuous mobility of the pupils irrespective of any change in the surrounding illumination. It has been frequently observed in pathologic conditions, especially in the **tubercular meningitis** of infants. It has also been noted in the reparatory stage, or that of recovery, of oculo-motor paralysis, and is likened by Heddaeus to the nystagmiform oscillations caused by the external muscles supplied by the third under similar conditions. This would give to the phenomenon signification quite different from that it has in meningitis. The movement ceases in sleep. On the whole, it does not seem to be of great diagnostic value, but it is not to be confused with the physiologic oscillations just described.

**Light-reflex or photo-motor reflex** is the name given to the dilation and contraction of the pupil due to the influence of the light. The nervous impulses or incitations to these pupillary movements originate in certain cells of the retina, travel by way of the sensory fibrillary extensions or neurons of these cells to the inferior or cerebro-spinal centres of the brain (**centripetal route**). From there, after reflexion, the incitations descend by the motor neurons of the third nerve to the muscle-fibres of the **sphincter pupillæ** (**centrifugal route**). The whole circuit traversed by the neurons from the retina and back to the iris is called the **light-reflex arc**. Whatever is the nature of the retinal elements that receive the excitation that culminates in the light-reflex, it is certain that they are most numerous in the macular zone of the retina, becoming gradually less so as the periphery is reached, and that their fibres, known as **centripetal pupillary fibres**, mingle in the optic nerve with the visual fibres from which they are distinguished by their larger size. Like the visual fibres, they undergo partial decussation in the chiasm, so that each optic tract contains crossed and direct centripetal pupillary fibres in addition to the visual fibres.

Before arriving at the external geniculate body the pupillary fibres separate from the optic fibres, pass inward round the external geniculate body, and into or through the substance of the anterior corpora quadrigemina, just beyond



the aqueduct of Sylvius, to terminate in the nucleus of the third nerve. In the nucleus the extremities of the pupillary fibres connect with the small cells of the sphincter nucleus—the centre of the light-reflex—each sphincter nucleus receiving both crossed and direct fibres. From here depart the **centrifugal** or **sphincter fibres**, which reach the iris by way of the ophthalmic ganglion and the ciliary plexus. As stated places of the light-reflex arc, at the ganglions and even in the retina itself, secondary or **homolateral association fibres** are intercalated. Hence luminous excitation of any part of the retina results in contraction of the pupil. The rapidity and the scope of the reaction depends upon the intensity of the excitation and its locality in the retina.

Besides the homolateral association fibres relative to the reflex arc of one side of the body, there exist heterolateral fibres, or those connecting the arc of one side with that of the other. It is possible that these association fibres of the reflex arc are supplemented by others connecting the centres still higher up. All the heterolateral association fibres and the decussation of the centripetal pupillary fibres at the chiasm constitute the apparatus that presides over the **consensual pupillary reaction**. To this is due the fact that when the right pupil, for instance, contracts to luminous excitation of the retina of the same eye the left pupil also contracts, and in the same proportion. Owing to the presence of the association fibres just alluded to one of the optic tracts may be completely destroyed yet the pupils both respond to light.

**Reaction to Accommodation and Convergence.**—In that act by which the eyes are adjusted for distinct vision at different distances, in proportion as the visual axes are made more or less convergent, the pupils become more or less contracted. These associated movements have been called the **convergence pupillary reflex**. The phenomena in connection with these movements, and the apparatus by which they are produced are subject to the will, and not purely reflex like those relative to the photo-motor movements, hence the



wording that heads this section. For convenience, however, we may continue the designation "**convergence-reflex.**" Like other voluntary movements those of convergence and accommodation are accomplished by muscles that act in opposing sets or pairs. The two circular muscles, sphincter of the pupil and the circular ciliary muscle of Mueller are the accommodators for objects approaching the eyes. In unison with these, act the abaiso-convergers of the globe (*interni* and *inferlori*). The dilator of the pupil and the longitudinal ciliary fibres of Bruecke are the accommodators for objects receding from the eyes, and in conjunction with them act the elevato-divergers of the globe (*externi*, *superiori* and *inferior obliques*). Venneman distinguishes the union of these two pupillary movements as **associated voluntary pupillary reaction**. The slower and more precise contraction of the pupil in accommodation is cited as among the features that distinguish it from a reflex-contraction. The contraction seems to be more nearly related to the act of convergence. In bilateral post-diphtheritic paralysis of accommodation, for example, the pupils respond to the voluntary convergence—but do not contract if one eye is covered and accommodation for a near object only is attempted.

**Dilation Reflex.**—As before stated, the pupils in sleep are contracted. On awakening they instantly dilate by a reflex that has nothing in common with the light-reflex. This is the **dilation reflex** which occurs in response to energetic excitation of most any part of the sensorium, as sudden pain or fright. Having dilated, the pupils at once return to the size imposed by the light-reflex. This reflex is of **cortical** origin or **psychic**, as opposed to the light-reflex, which is of **cerebr, -spinal, or axial** origin. Its arc is more complicated than that of the latter, and hardly concerns us here. This reflex is responsible for the painful dazzling sensation experienced on awakening from profound sleep, and in the face of strong light. It is capable of giving material aid in diagnosis, as its absence indicates either spastic myosis or



sympathetic paralysfs. On waking a child or a young adult in bright light if the pupils do not at once dilate they are not normal. In an otherwise healthy child absence of the reflex could be due to toxemia, as from opium or morphin.

There are several other pupil-reflexes of somewhat lesser importance. The **peripheric** reflex is the result of mechanical excitation of the terminations of the sensitive nerves of the iris and the reversal of the nerve-current takes place in the ganglionic nerve plexus of the ciliary region. Here the centripetal impulse is the inverse of that in the light-reflex in that it is *from* the iris. This contraction of the pupil is taken advantage of in replacing prolapse of the iris that occurs as an immediate accident in the extraction of cataract. The light crushing of the iris through the lids or the trituration of it with the spatula serves as the excitation. The iris of the freshly enucleated eye of an animal is still sufficiently sensitive to the chemic or caloric action of light to cause this reflex.

**Inverse or paradoxical pupil reflex** is also called **orbicularis** and **palpebral reaction**. The names Graefe, Galassi, Westphal, Piltz and Gifford have all been used to designate it. This phenomenon, first mentioned by von Graefe, in 1854, consists in a contraction of the pupil coincident with an energetic contraction of the orbicularis of the lids; it matters not if the contraction of the lid muscle fails in its effort to close the lids, or that the examiner interferes by holding the lid back with his finger. If the forcible effort to close the lids succeeds, and the upper lid is lifted quickly by the finger as soon as the strain of the orbicularis begins to relax, the pupil is found still retracted and it at once dilates, in spite of the entrance of the light, *i.e.*, it is a truly paradoxical or inverse pupillary reflex. It has been usually explained as a simple associate or synergic contraction with the contraction of the orbicularis, though some have supposed it to be due to intra-ocular congestion consequent upon stasis of the orbital circulation from pressure of the orbicularis in the forcible closure of the lids. Morax considers it a sort of antagonist to the dilator or psychic reflex. At the com-



mand to close the lids the sensorium experiences a premonitory sense of the sudden suppression of the important function of vision. The consciousness of the imminent disappearance of a stimulation of the first order permits the pupil to contract by the same reflex action that reduces its size in sleep. On the instant the lids are reopened the psychic reflex, suspended for a moment, causes dilation until the light-reflex readjusts the pupillary diameter to the surrounding illumination.

Gifford believes it to be an associate contraction with that of the palpebral portion of the orbicularis, and that if the motor impulses to this part of the muscle come from the posterior portion of the third nucleus, as declared by Mendel, its influence on the sphincter is not difficult to understand. Gifford thinks the sign may be of some value in determining whether the sphincter is paralyzed in cases of tumors and diseases in the vicinity of the nucleus of the third nerve. The reaction is difficult to see in any save eyes in which the light and convergence reflexes are feeble, like certain diseases of the optic nerve and retina. In subjects with tabes and those with general paresis, in whom the pupil does not respond to light, this reaction is most plainly seen.

Pupillary reflexes of minor importance are **Haab's cortical reflex**, or **attention reflex**, the **oculo-pupillary reflex** or **cutaneous reflex**, and others. Haab found that, by placing a person in a dark room and calling his attention to a lighted lamp, or other bright object situated in his field of vision, on the instant that any movement of head or eyes was made that caused a variation of either convergence or accommodation, the pupil contracted. Others discovered that the lamp was unnecessary, as it was sufficient to **pretend** that it was there. Piltz obtained the same reaction in the pupils of subjects totally blind from optic atrophy by representing the presence of a bright light. The reaction is not of semiologic interest. The production of the sign is proof positive that there is no organic anesthesia. The exaggeration of the reflex is sug-



gestive of hyperesthesia. It may exist in tabes in spite of the absence of the light- and convergence-reflexes.

**The cutaneous reaction** is a sympathetic phenomenon that consists in reflex dilation of the pupils through the influence of tactile or thermic or algesic excitation of the skin, the conjunctiva or the cornea. Varady, by pricking the skin of the malar region for thirty seconds obtained slow and complete dilation of the pupil. This was followed by sudden contraction which was greater than that present when the experiment began. The slowness of this reaction differentiates it from the light and accommodative-reflexes.

Venneman tabulates the three pupillary reactions as follows:

1. A pupillary reflex with peripheric center in the ganglionic nervous plexus of the ciliary body: Reflex contraction of the pupil to direct excitation of the sensory fibres of the iris.

2. A reaction of the pupil with inferior centre in the cerebro-spinal axis. This reaction is double: Contraction of the pupil to excessive luminous excitation of the retina and dilation under insufficient luminous excitation. The whole constitutes the **pupillary light-reflex**.

3. A pupillary reaction with superior or cortical centre. This is also a double reaction: A dilation of the pupil under energetic excitation of the cerebral cortex by way of any nerves either of sense or sensation, including the retina; a contraction of the pupil through insufficiency of cortical excitation, or inattention of the sensorium to the existing irritations.

Save the first, they may all be bilateral for a unilateral excitation, consensual or symmetric.

**The hemianopic pupil reflex** is identical with the light-reflex but it is elicited under peculiar circumstances. In 1883, Wernicke, on purely theoretic grounds, advanced the hypothesis that in cases in which hemianopia was caused by a lesion of an optic tract, and situated in front of the external geniculate body, contraction of the pupil to light could only



be obtained by excitation of the normal half of the retina; and that in hemianopia produced by a lesion situated between the external geniculate body and the occipital cortex, the hemianopic pupillary reflex would be lacking. That is, the pupil would contract, though the excitations were confined to the blind or anesthetic portion of the retina. Theorizing along the same lines, Swarz conceived that the hemianopic pupillary reflex could be obtained without hemianopsia when the lesion was situated between the geniculate body and the sphincter nucleus. The truth of Wernicke's theory was first verified by Leyden, and since by a number of observers. On the other hand, many trained and astute observers actually deny that there is any such thing as a hemianopic pupil reflex. In view of the numerous cases wherein, notwithstanding the actual existence of lesions of the chiasm and tracts, the reaction failed to appear, its semiologic value has been reduced. Examples of such failures are: blindness of the right eye and temporal hemianopsia of the left; bitemporal hemianopsia from antero-posterior bisection of the chiasm by an aneurysm of an anomalous artery; temporal hemianopsia from gumma at the chiasm; temporal hemianopsia in a case of acromegaly. Its value is partly redeemed, however, by other instances wherein the presence of the sign verified the diagnosis in those lesions.

**Modifications of the Light-reflex.**—For an adequate appreciation of slight departures from the normal reaction of the pupil to light, and in order that they may have their true value, it is necessary studiously to observe the phenomena it presents in healthy subjects of the different ages and under varying intensity of illumination. In general terms, the effect that morbid processes have upon this reflex is to either diminish or to abolish it.

**Diminished reaction, or sluggishness of the pupil** is obviously of little significance unless it is *relatively* considered or unless the slowness is quite pronounced. The investigation is best made by artificial light. It is apparent



in some diseases at the posterior pole, like central chorio-retinitis and in the toxic amblyopias of nicotin and alcohol. It is well to bear in mind that nicotin-alcohol amblyopia and syphilis are often found in the same individual, and their pupillary symptoms may be superposed. The sign often precedes complete loss of the light-reflex in the syphilitic; again, it may be encountered in the reparatory stage after paralysis of the sphincter. In acute and subacute glaucoma there is frequently sluggishness or even immobility of the pupil to light stimulus.

**Absence of the Light-reflex with Preservation of the Convergence-reflex. Argyll-Robertson Pupil.**—The distinguished Scotch ophthalmologist who first described this symptom and whose name it bears, at first called attention to the myosis which is coexistent in so many instances, as part of the syndrome; but the two essential features may be present with pupils of normal diameter or even with marked mydriasis. The lesion producing it may be of the optic nerve, of the nucleus of the third nerve, or of the points of distribution between them, viz., the extern algeniculate body and the anterior corpora quadrigemina. Having noted its existence in a given case the first thought is of syphilis, hereditary or acquired. It is particularly likely to appear in the course of those distinct clinical types of syphilis of the nervous system, **tabes** and **general paresis**. Following the classification of Morax the discussion of the Argyll-Robertson pupil may be classified, according to the conditions under which it is found, as follows:

- a. As an isolated symptom in syphilis.
- b. In tabes.
- c. In general paresis.
- d. In cerebro-spinal syphilis.
- e. In cerebro-spinal affections not luetic.
- f. In insanity.

**As an Isolated Symptom in Syphilis.**—In consequence of many observations to that effect, the fact is established that this sign may be present for periods ranging from 5 to



8 years unaccompanied by any other objective symptom of the disease. If this symptom exists, few other symptoms are needed to make conclusive diagnosis of syphilis.

**In Tabes.**—It is extremely frequent in established tabes according to statistics in from 70 to 90 per cent. of cases. In 25 to 35 per cent. there is also anisocoria. It may be bilateral or unilateral. Once developed, it persists indefinitely, or, at least till loss of the convergence-reflex. A certain number of cases of tabes develop without showing this or any other disturbance of the pupillary movements. Hence the absence of the sign does not eliminate this affection in the diagnosis. Figures compiled by Vincent give four cases with normal pupils out of five tabetics, seven with pupils immovable to both light and convergence, and forty with the Argyll-Robertson pupil. Of these 40, there was myosis in 23, mydriasis in 6, and normal diameter in 11.

**In General Paresis.**—According to statistics of Moeli, the Argyll-Robertson pupil was found in 74 per cent.; and in other respects the pupil signs here have about the same numerical ratio that they do in tabes.

**In Cerebro-spinal Syphilis.**—The manifestations of this clinical type are of infinite variety, since the lesions of the nerve-centres may be in relation with those of neighboring **bone**, affections of the **meninges**, circumscribed and diffuse, and with divers **vascular lesions**, such as obliterating endarteritis, arterial and venous thrombosis, hemorrhages, etc. Other consecutive cerebro-spinal affections in which the sign may occur are cerebral hemiplegia, infantile hereditary luetic encephalopathies, progressive meningo-myelitis, lateral sclerosis, chronic poliomyelitis, Friedreich's disease and hemato-myelia (all **syphilitic**).

**In Non-specific Cerebro-spinal Affections.**—The Argyll-Robertson pupil is so rare in multiple sclerosis that one is warranted in asking if syphilis was not coincident in the few instances noted. It has been found in several cases of syringomyelia. It is declared impossible as a result of parenchymatous lesions comprising the eighth cervical



root, these disturbances giving rise simply to myosis without loss of the light-reflex. In the reported cases of the peculiar form of muscular atrophy known as hypertrophic interstitial neuritis, which is accompanied by pronounced tabetiform troubles, the Argyll-Robertson pupil was constantly present and other symptoms of syphilis, hereditary or acquired, were absent.

**In Insanity** the ratio of the sign to the number of cases varies enormously with the different authors, partly, perhaps, because of the difficulties encountered in examining the eyes of this class of subjects, and to the fact that the etiology of the insanities, and particularly the rôle of syphilis in their production, is still very obscure. It is reasonably certain that, aside from the cases of insanity associated with general paresis, and those in which some other type of syphilis cannot be definitely excluded, it can be said that the Argyll-Robertson pupil is extremely rare in conjunction with insanity. From a semiologic standpoint about as much importance can be attached to the phenomena when incomplete, that is, when there is simple diminution of the pupillary reaction and conservation of the convergence reaction. Rochon-Duvigneaud and Heitz have shown that after keeping some of these patients with Argyll-Robertson pupils in the dark for a few minutes, then throwing a strong light into the pupil there was perceptible retraction to light and a slight dilatation to obscure illumination. This has been called also Sanger's reaction.

**Consensual Reaction** has also been designated as the **indirect reflex**, and it presents certain combinations from which one may draw conclusions pertinent to diagnosis:

1. *If the pupils contract equally when only one is illuminated the consensual reaction is normal.* To be sure that both eyes are alike the right and the left pupils are successively illuminated. We may assume that the apparatus of peripheral, sensorial perception is not seriously altered, not more than the nervous apparatus that transmits the impulses concerned. In case of **monocular amaurosis**, we may con-



clude that the visual trouble is of cortical origin—organic or functional (hysteric)—but we would not be justified in suspecting simulation without there were other strong reasons for the presumption.

2. *The pupil on the side exposed to the light does not contract; but the other contracts normally. On changing the light to the other side the consensual reaction is lacking.* This indicates a peripheral lesion in the nervous apparatus for innervation of the pupil on the side first examined. Such is the case in paralytic mydriasis—it may be of syphilitic origin (cerebral, tabetic, genero-paralytic) or caused by peripheral section or lesion of the oculo-motor or one of its pupillo-motor branches. A phenomenon analogous to this is seen in tabes when the Argyll-Robertson pupil is unilateral, and when the other pupil retains its normal reactions. Babinsky has demonstrated that in this case the affected pupil rests immobile to the light, not only in attempts at direct excitation but also when the opposite eye is illuminated. The healthy pupil, on the contrary, reacts both directly and consensually.

3. *The pupil on the side exposed to the light does not contract, nor does its fellow, but changing the light to the other side causes contraction of both pupils.* This modification of the consensual reaction indicates a profound trouble either of the apparatus concerned in peripheral perception or transmission. Such conditions obtain in lesions of the eye or of the optic nerve that produce total monocular blindness as, for example, in embolism of the central artery of the retina, in unilateral optic atrophy, except it be tabetic, for then the pupillary reactions are most often altered.

4. *The consensual reaction is lacking in both eyes only in lesions of the peripheral or the central nervous system, most probably syphilitic.*

**Modification of the Convergence-reaction.**—This reaction has less semiologic importance than that of the photo-motor reflex. The dissociation of the reflexes, the inverse of that observed in the Argyll-Robertson pupil, does not ex-



ist at least not for eyes possessed of luminous perception; there is always abolition of the light-reflex when there is absence of light-perception. On the contrary, as regards subjects who are totally blind, whose pupils do not respond to light, one may observe contraction of the pupils when they try to converge with energy while they hold the finger on the tip of the nose. Immobility of the pupil to light and convergence, like the Argyll-Robertson type is seen in cerebro-spinal syphilis (**tabes and general paresis**). In these cases the pupils are either in a state of myosis or of normal diameter. It goes without saying that in cases of **paralytic mydriasis** (paralysis of the third pair or from the instillation of cycloplegic solutions) loss of the convergence-reflex is inevitable.

**Myotonic Contraction of the Pupil.**—There occurs occasionally a peculiar modification of the reaction to convergence that consists in the following: A pupil immobile to the light contracts more slowly than in the normal state under the influence of convergence. The consecutive relaxation takes place still more slowly, and may require 10 to 20 seconds, whereas it would only require 2 or 3 seconds in the normal state. Piltz and Sanger have observed this myotonic contraction after the orbicular contraction of the pupil. In one instance it took 10 minutes for the pupil to regain its normal diameter. It has been observed in **general paresis** and in **tabes**. It exists only on one side. Noume remarked it in a case of a diabetic and in an alcoholic. Its presence does not warrant any particular conclusion.

### EXPLANATION OF THE SCHEME OF THE PUPILLARY FIBRES.

BY M. V. MORAX.

The scheme of the paths followed by the fibres of the pupillary centres on page 124, Fig. 3, is a partial reproduction of that of Bach and Meyer (*Arch. f. Oph.*, 1903, Vol. LV, p. 484) which will enable the reader to form a more precise



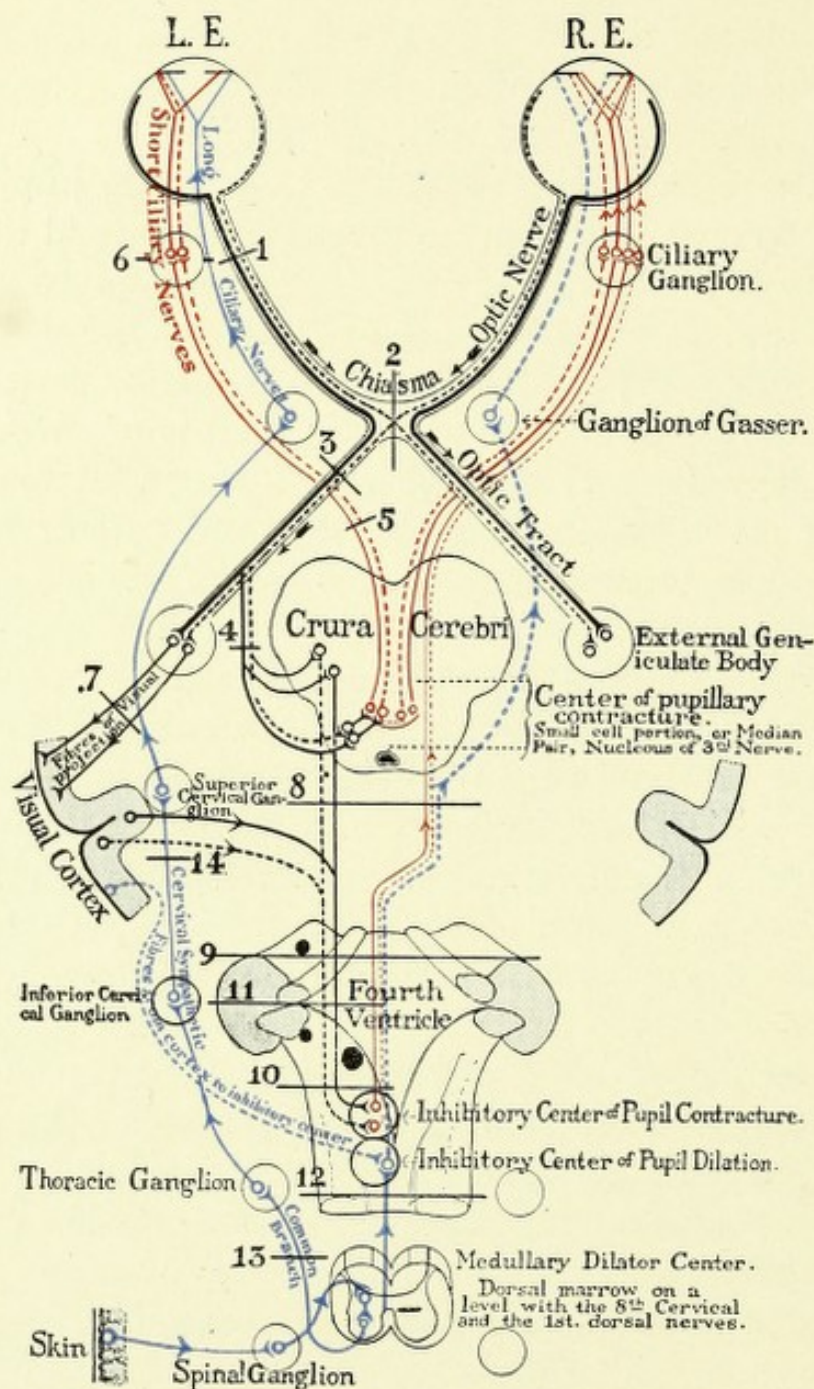


FIG. 3.—Scheme of paths taken by the centrifugal and centripetal pupillary fibres. Showing also the effects upon the eye of certain sections or lesions. (Adaptation after Morax.)

- Dilator fibres.
- Direct centripetal pupillary fibres.
- - - - Crossed centripetal pupillary fibres.
- Direct constrictor centrifugal fibres.
- - - - Crossed constrictor centrifugal fibres.
- - - - Inhibitory fibres of pupil dilatation.
- - - - Inhibitory fibres of pupil contraction.



idea, not of their real courses and seats, but of the hypotheses formed on the subject. Certain facts have, nevertheless, been deduced in this connection, not only by experimentation, but also by rigorous clinical observation. Such is the case, for example, relative to the centripetal pupillary fibres in the optic nerve, the chiasm and the optic tracts. The difficulties begin when it is a question of establishing the relations between the centripetal and the centrifugal motor fibres. Morax has shown the connections assumed by Bernheimer, who admits the existence of cells intermediate between the terminations of the centripetal fibres and the median, or small cell nuclei of the oculo-motor nerve (nucleus of Edinger-Westphal), which he makes the centre of innervation for the pupillary sphincter. Each of the nuclei is in touch with the nucleus of the opposite side, which explains the consensual reaction. Bach contests the rôle of the nucleus of Edinger-Westphal as to the innervation of the sphincter. He places the centre of reflexion about the corpora quadrigemini, but without definitely locating it, and admits that the semi-decussation of the pupillary fibres at the chiasm is sufficient to account for the consensual reaction. Finally, as to the centrifugal fibres, Bach, contrary to Bernheimer, cites a semi-decussation in the crura in front of the oculo-motor nuclei.

These centrifugal fibres pass through the ciliary ganglion, and they arrive at the sphincter of the iris by the short ciliary nerves. The course of the centrifugal sympathetic fibres is confirmed experimentally and clinically. The centre is situated in the dorsal marrow, at the level of the eighth cervical and the first dorsal nerves; this is a dilator centre whose paralysis produces myosis of the corresponding side. The fibres pass the anterior horns in the thoracic ganglion by the communicating branch of the sympathetic; they attain the inferior cervical ganglion, then the superior, by the cervical sympathetic, and pass from this ganglion into the cranium, with the carotidian branches, to reach the Gasserian ganglion. From there, they pass by the



ophthalmic nerve of Willis and the long ciliary nerves, without any connection with the ciliary ganglion, and terminate in the dilator muscle of the pupil.

Recent experiments of Bach and Meyer have led these authors to assume the existence at the bulb of two inhibitory centres, one for contraction of the pupil, the other for dilatation. Their conclusions have yet to be verified. Be this as it may, it is believed that the inclusion of these centres, in the scheme here reproduced, will render comprehension more easy than would a long description. The connections of these centres with the cortex are purely hypothetical, and only answer to the desire to give an explanation of the cortical reflexes.

In the figure are placed numbered lines of sections, or destructive lesions, whose effects are indicated by the following table:

**Lesion 1.—Section of the left optic nerve.** Photomotor reflex lacking in the left eye, and consensual reaction lacking in the right. Illumination of the right eye causes contraction of both pupils.

**Lesion 2.—Section of the chiasm.** Pupils equal. The illumination of each pupil separately causes reaction of the corresponding pupil and that of its fellow. Bitemporal hemianopsia. Hemiotic pupillary reaction (?).

**Lesion 3.—Section of the optic tract.** Hemianopsia and hemiotic pupillary reaction(?).

**Lesion 4.—Section of the centrifugal fibres on the proximal side of the geniculate body.** Hemiotic pupillary reaction without hemianopsia(?). Bilaterality of this lesion could result in the Argyll-Robertson sign(?).

**Lesions 5 and 6.—Section of the common motor-oculi, or section of the ciliary ganglion.** Accommodative paralysis, and moderate mydriasis. The left pupil reacts only to sympathetic excitations. This experiment proves that, after section of the motor-oculi, excitation of the fifth still gives rise to a pupillary contraction.

**Lesion 7.—Hemianopsia without modification of the**



pupillary reactions. If the lesion is bilateral—blindness with preservation of the pupillary reactions.

**Lesion 8.**—Light-reflex very rapid in both eyes because of the suppression of the inhibitive pupillary centre in the medulla oblongata. An exciting action will, on the contrary, produce myosis.

**Lesion 9.**—Laying bare the medulla oblongata entrains diminution or suppression of the photo-motor reflex. The pupillary immobility may last for an hour. Complete section at No. 9, or even demi-section at 11, will cause the reappearance of the pupillary reactions, and render them normal in both eyes. It is upon these experiments that Bach and Meyer base their hypothesis concerning the existence of one inhibitory centre for photo-motor or pupillary contraction and another for the dilatation of the pupil.

**Lesion 10.**—According to Bach and Meyer bulbar semi-section in this location produces pupillary immobility to light of the opposite side, while the reaction of the corresponding side is conserved. This section would have the effect of suppressing the action of the inhibitory centres of the right eye.

**Lesion 11.**—Suppression of the sympathetic reaction. Mydriasis on pinching the skin of the cheek.

**Lesion 12.**—Section of the bulb at this point causes only a transient dilatation, and does not modify the pupillary reactions.

**Lesion 13.**—Section of the communicating branch of the first dorsal nerve. Paralysis of the sympathetic fibres. Myosis of the left eye by paralysis of the dilator.

**Lesion 14.**—Section of the cervical sympathetic. Myosis, same as from Lesion 13.

**Anomalies of configuration** relate to the form or outline of the pupil and are **congenital** or **acquired**. We have already discussed matters pertaining to the diameter of the pupil. Among the commoner congenital anomalies of form is that caused by a break in the continuity of the iris, called **coloboma** of the iris. This produces an elongation of the



pupil, usually downward and slightly inward. It is **partial** or **complete** in that it extends part way or all the way through the iris. It differs from the **operative** coloboma by its more oval outline—suggesting the form of a Gothic window—its smaller size and its constancy of location. It may be bilateral or unilateral. In rare instances there occurs merely an outward prolongation of the uveal pigment—**ectropion of the uvea** (Axenfeld) more often in dark eyes, that gives the appearance of an elongation of the pupil. This constitutes **false coloboma**, and like those cases in which the stroma of the iris has been stripped off, leaving the uveal portion adherent to the lens, it may require close scrutiny to disclose its true nature. Absence of the iris, **aniridia**, means also absence of the pupil. It is usually congenital, although it may also be acquired as, for example, the result of the tearing out of the iris in an attempt to perform an iridectomy in case of a refractory patient. The congenital form is frequently associated with cataract. It is often hereditary to a remarkable degree, and is most always bilateral. In the congenital form the aniridia is occasionally only partial. All phases of absence of the iris are found, from the incipient coloboma, through all forms of incomplete aniridia, to apparently total absence of the membrane, although it has been denied by a number of investigators that congenital absence of the iris is ever absolute.

**Dyscoria** is the term given to irregularity of pupillary outline. The type most frequently observed is that which is the result of adhesions between the iris and the anterior capsule of the lens consecutive to iritis. The area of the pupil is usually small as well as irregular. Depending upon the situation of the adhesions the pupil may not show any irregularity excepting where it dilates or contracts, as, for example, when influenced by atropin or eserine. Thus evidence of a former iritis is sometimes obtained only in this manner. Opacity of the anterior portions of the lens are often associated with this form of



dyscoria. If any part of the iris is fast in a corneal cicatrix, distortion of the pupil, or elongation in the direction of the scar, is the result. This form is less irregular in outline than the foregoing, and is constant in **partial leucoma adherens** and almost so in **cystoid cicatrix**.

**Ovoid deformities** of the pupil are extremely frequent. They are from both local and remote or systemic causes. Familiar examples of those from local causes are to be observed after recent **wounds and contusions of the globe**, with or without direct implication of the iris; those dependent upon **partial anterior dislocation of the lens**; and those that are due to **limited posterior synechia**. A slight flattening in the contour of the pupil at a given point is often significant of posterior synechia at that point. As syphilis is indirectly answerable for most of the local causes of dyscoria, so it is also for the vast majority of the systemic causes. Both the ovoid and the less regular deformities of the pupil are of great frequency in connection with tabes and general paresis. Indeed, as regards tabes, dyscoria is a cardinal sign. Varying degrees of myosis, mydriasis and anisocoria are often present also in these affections.

A slight departure from a truly circular outline is often physiologic. In fact, an almost imperceptible ellipsity is characteristic of the normal pupil—long axis near the vertical. The great test by which to differentiate the various types of pupillary distortion that are the result of local or congenital causes from those that are due to diseases of the nervous system is the **light-reflex**. In the vast majority of instances those of the type last mentioned are in conjunction with the Argyll-Robertson pupil, and irresponsive to light.

There are **other anomalies** of the pupil that have no special reference to contour and that deserve mention here.

**Acoria**, absence of the pupil, is usually due to closure of the pupil in consequence of an inflammatory process, which follows an accidental traumatism or a surgical operation that implicates the iris. The last is the most frequent



cause. **Atresia of the pupil** is a commoner term for the condition.

**Corectopia, or abnormal situation of the pupil**, is usually but an exaggeration of the normal disposition, *i.e.*, the pupil is situated a little more inward and downward from the centre of the iris than is habitually the case. In rare instances the eccentricity is so great as to place the orifice near the inner and upper limbus. Race is thought by some to affect the position of the pupil; to some of the black races, for example, there is attributed the peculiarity of an *upward* and inward displacement. The more pronounced the corectopia the more the form of the pupil is altered (*dyscoria*). The corectopia may be **simple**, *i.e.*, not attended by other ocular abnormalities, when it is usually monolateral and reacts to light. Or it may be **complicated** by the remains or scars of an intra-uterine inflammation, such as atrophy, discoloration and irregular pigmentation of the iris. The latter reacts feebly or not at all to light. In such instances, the lens is often cataractous, but not in subluxation. It may be of one or both eyes. The most frequent ocular anomaly seen in combination with congenital corectopia is ectopia of the lens. This form is usually bilateral and the symptoms are described under ectopia of the lens. The foregoing refers mainly to **congenital** corectopia, which is often found in conjunction with other connate defects, such as ptosis and microphthalmos. Misplacement of the pupil is also often **acquired**. For instance, an injury, or anything that will cripple a segment of the sphincter will cause displacement of the pupil to that side. It is present to some extent after most all simple extractions of cataract—the displacement being toward the corneal incision.

**Polycoria** consists in existence of multiple pupils. In addition to a fairly regular central pupil there may be one or more smaller and less regular openings in other parts of the iris. Again, the membrane presents two or three apertures none of which is centrally located; or, there may be a coloboma of the iris which is separated from the pupil by a



narrow bridge (diplocoria). Diplocoria is probably the most frequent form of polycoria. The last term is probably applicable to those cases in which, with the existence or the absence of a central pupil are found more than one accessory opening in the tissue of the iris. The number of these has been known to be as high as eleven. Next to diplocoria the most frequent form of polycoria is that in which the extra openings are in what should be the zone of the sphincter, or encroaching on the ciliary zone. In rare instances, as concerns congenital polycoria, the opening is at the periphery, and simulates traumatic dialysis. These supernumerary apertures are smaller and less regular in outline than the main pupil, and do not react to light. The **congenital** forms of polycoria do not interfere with the vision. **Acquired** forms, however, such as traumatic perforations of the iris that remain patent, sometimes produce **monocular polyopia**. Unilateral diplopia is not uncommon with iridodialysis, but the latter cannot be properly called polycoria. An example of operative polycoria is found in Pope's peripheral iridectomy.

**Persistence of the Pupillary Membrane. Membrana Pupillaris Perseverans.**—These terms, habitually employed to designate the anomaly in question, are somewhat misleading, for, as we find it, it is seldom what could properly be called a membrane, being usually but an insignificant remnant, in the pupil, of the vascular network that was there in fetal life. Until 1861, when Adolf Weber, of Darmstadt, described the differences between them, the synechias and exudative pseudo-membranes consequent upon intra-uterine iritis were confounded with persistent pupillary membrane. To many ophthalmologists it would doubtless be surprising to know how frequently one can, by careful scrutiny, find certain remains of this membrane; especially in the eyes of infants and young children. For instance, of 300 newly born children, Koenigstein found the fetal remains in twenty-one, and of 150 examined by Schleich thirteen showed them. Often the relic consists of but a



single filament, hardly larger than a strand of spider's web, stretching across some part of the pupil, and difficult to see with the unaided eye. Doubtless in later years many of these disappear by breakage and absorption.

The chief characteristics which distinguish the true remnants from those that are the result of inflammatory products are their manner of attachment and their elasticity. The filaments are always attached to the front surface of the iris and never, as are the false kind, to the pupillary border. For the most part they rise from the corona, or just over the *circulous arterious iridis minor*, but in many instances they are found adherent to portions of the ciliary or median zone of the iris. They are somewhat elastic, stretching across even the widely dilated pupil, but bulging forward in curves when that orifice contracts. These filaments may be moderately multiplied, or to so high a degree that, by their branching and anastomosing, they constitute a veritable membrane; but this form is extremely rare, as ordinarily the affair is so slight as not to interfere with vision nor to be noticed by the intimates of the possessor. Indeed, it is remarkable how little amblyopia is caused by even the more pronounced forms. They are sometimes attached to the anterior capsule, and, more rarely, to the cornea.

The writer has recently seen one attached to the anterior capsule, in a man of forty-five. The adherent part was oval and the filaments, some six or seven in number, arose therefrom and were inserted in the corona of the iris. They were brownish, corresponding to the color of the iris, and the whole presented precisely the appearance of a diminutive spider, the resemblance being enhanced by the movements of the filaments (legs) in obedience to the varying sizes of the pupil. The man's attention had never before been called to any peculiarity of the eye and his vision was but slightly affected, being 20/20 in that eye. It is probable that many of the instances of pigment at the centre of the anterior capsule, or in the deeper layer of the cornea, and even



of free pigment in the aqueous, and where no remains of a membrane are visible, have had their origin in connection with the fetal pupillary circulation. Concomitant anomalies such as aniridia, coloboma of the iris and choroid, corectopia, polycoria, zonular cataract, etc., have been observed. Remains of pupillary membrane are usually monolateral.



## CHAPTER X.

### THE CRYSTALLINE LENS.

Affections of the crystalline lens are not numerous, and their semiology refers mainly to displacements of that organ from its normal or well-centred site in the dioptric apparatus or **luxation**, and to **cataract**—or to its principal symptom, namely, loss of transparency—either in the substance of the lens or in its envelope.

**Luxation of the Lens.**—Luxation, strictly speaking, means a dislocation. Luxation of the lens, generally speaking, includes all grades and degrees of dislocation, whether partial or complete, but it is proper and more discriminating to refer to the slighter dislocations, when the lens is more or less fixed and not wholly removed from its normal fastenings, as **subluxations**, and to bodily displacement, with complete severance of its zonular detachments, as luxations. Usage, however, seems to warrant great latitude in the use of these terms, for our best authorities are continually confusing them. For convenience, this subject may be divided into three heads:

1. Ectopia or congenital subluxation.
2. Spontaneous luxation; displacement.
3. Traumatic luxation.

**Ectopia Lentis.**—It was Stellwag v. Carion, who, in 1856, first employed the term ectopia in this connection; but it was Sippel, in 1859, who, in a thesis that has remained a classic clearly established the distinction which should be made between ectopia and congenital luxation of the lens. By **ectopia** one understands that anomaly of the crystalline in which that organ is *developed* outside of its normal seat, and is primarily decentred, as it were, with the dioptric system in which it is the most important factor. By **luxation** are



designated those abnormalities in which the lens, though accurately centred in the beginning, has become misplaced and ultimately occupies an unfavorable or even a vicious position. Ectopia and subluxation may both become complete luxations, but ectopia cannot become subluxation. Ectopia is characterized, uniformly, by a certain degree of atrophy, or lack of development of the lens not found in subluxation.

According to most observers congenital displacement of the lens is more common in persons of Anglo-Saxon descent than in those of the Latin races. The subjects of the affection are nearly always intelligent, and otherwise well developed, though there may be present other ocular anomalies, such as aniridia, coloboma of the uveal tract, nystagmus, and, most frequent of all, displacement of the pupil. This **corectopia** is usually in the opposite direction to that of the misplacement of the lens. Ectopia is hereditary and familial to a remarkable degree. This fact was first cited by von Graefe, in 1855. It affects parents and their offspring throughout as many as five and six generations, and extends to the nephews and nieces. It has been observed in no fewer than ten children of a father who had ectopia lentis. In this instance the mother afterward remarried and bore children with normal lenses (Stanford Morton).

The diagnosis of ectopia of the crystalline is seldom made before the child is sent to school, when the want of good vision becomes apparent, and the matter is brought to the notice of a specialist. This is not true of many instances in which the affection has already been noted in a member of the same family, for the professional advisers of those concerned may be on the lookout for it. The subjects are always more or less amblyopic, and hold objects very close to the eyes, after the manner of myopes. Indeed, the non-aphakic portion of the eye in these cases is frequently myopic. Attempts to improve the vision by correcting lenses is likely to be disappointing. It is only when the displacement is considerable, *i.e.*, where the non-aphakic



portion of the pupil is large, that they are susceptible of such improvement. In the vast majority the direction of the misplacement of the lens is upward, or upward and outward. Next in frequency is upward and inward displacement; while few are downward, and still fewer inward or outward. The affection is habitually bilateral, and the direction of the displacement is, as a rule, symmetrical in the two eyes. Moreover, the particular type or order of the ectopia in the hereditary cases is common to both parent and child. Exceptionally there is a tilting of the crystalline, which does not facilitate the diagnosis. In the uncomplicated cases the lens usually retains its transparency. An important and almost constant sign of ectopia of the lens is tremulousness of the iris, or **iridodonesis**. The anterior chamber is often somewhat deeper on the side from which the lens is displaced.

By oblique illumination, and with the pupil dilated, one can sometimes see a difference between the two portions of the pupil that are occupied and unoccupied by the lens. The first gives back certain reflections, the second is totally black. But it is by the light from the mirror of an ophthalmoscope, or that of a skiascope, reflected from the fundus of the eye that the picture of ectopia of the lens is most striking. What constitutes its characteristic feature is the dark shaded arc that crosses some part of the bright scarlet disc of the pupil. This is the equatorial border of the crystalline, and the crescent between its convexity and the opposing concavity of the pupillary border indicates the extent of the displacement.

On the concavity of this arc, the darkness fades gradually into the scarlet light from the fundus, while the convexity is composed of a sharp, crisp black line, the line of total refraction or diffraction, marking the outer limits of the crystalline. In favored cases, one may at will get two images of the eye-ground, a larger one through the lens and a smaller through that part of the pupil where the lens is missing. The degree or extent of the displacements varies



greatly in different cases, or even, occasionally, in the same case. It may be so slight as to require the pupil to be dilated *ax maximum* in order to detect the typical chink between lens and pupillary borders, or it may be so great as to render this visible with the pupil of normal diameter. Ectopia constitutes a **predisposition to luxation**.

The case of congenital, bilateral ectopia referred to in the following paragraph is decidedly out of the ordinary. The child, a girl of seven years, was brought to me in December, 1905. She was the fourth in age of six children. None of the rest, and neither parent, had any ocular anomaly. There was lateral nystagmus, which is common with con-

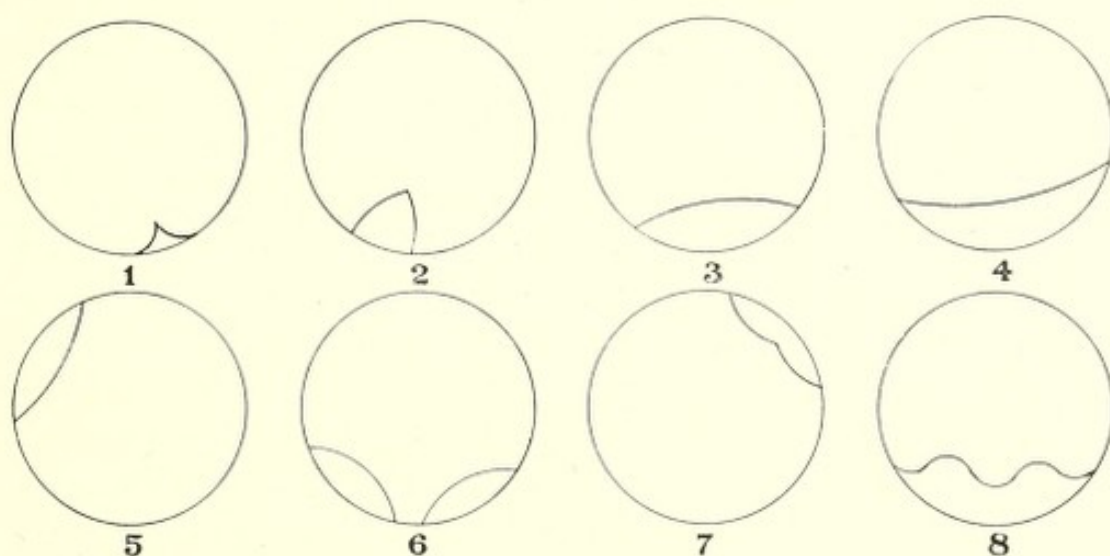


FIG. 4.—Varieties of lens coloboma. (After Dor.) 1. Notch; 2. triangle; 3. ellipse; 4. crescent; 5. external ellipse; 6. double ellipse; 7. double overlapping crescent; 8. accolade.

genital ectopia. The peculiar features have reference to the pupils, and to the direction and the degree of the displacement of the lenses. There was downward corectopia in both eyes, and the pupils were of quadrilateral, or rectangular form—long axis vertical. The right lens was misplaced upward and the left downward, and to an unusual extent. So that the arc of neither lens encroached upon the pupillary area except when there was marked mydriasis. Ordinarily, in order to see the lens with the ophthalmoscope, it was necessary that the eye be rotated in the



direction of the displacement. The child looked *over* the right lens, and *beneath* the left. The lenses were transparent. The choroids were poorly developed, but there was no coloboma of any part of the uveal tracts, nor of the lenses. There was iridodonesis. With a +12 lens the vision of either eye was 20/100. The girl was brought again to me on May 3, 1912. The conditions were then the same as they had been six and one-half years previously save that *the lenses had become opaque*. This was rather to be expected, for with a displacement so extreme there could have been but a slight portion of the zonule intact, therefore but scant nutrition.

**Ectopia or Coloboma?**—These two conditions are, at times, to be differentiated (Fig. 4). Certain types of coloboma of the lens are readily distinguishable. Such, for example, are the notch-form coloboma and those that consist in concavities of varying dimensions in the outline of the crystalline. These were first described by von Arlt, in 1849, and are often found in conjunction with coloboma of the uveal tract. It is only the cases in which the coloboma shows merely as a flattening of the equatorial border, or as the section of a very long ellipse, that offer difficulties in this line.

In these forms of coloboma the dark outline of the lens in the defect is of a greater radius of curvature than that of the normal or the ectopic lens. Coloboma of the lens is usually situated downward, which only adds to the confusion, since ectopia is most often in an upward direction.

Toldt has recently (1899) tabulated points that distinguish ectopia from coloboma of the lens as follows:

- a. The previous occurrence of a traumatism.
- b. Heredity.
- c. Bilaterality.
- d. Unequal depth of the anterior chamber.
- e. The coexistence of corectopia.
- f. The absence of ocular malformations other than the corectopia.



g. Tremulousness of the iris.

h. The presence of broken zonular fibres when, on the contrary, folding or irregularity of the border of the capsular sac is suggestive of coloboma.

i. Regular astigmatism, where irregular astigmatism bespeaks rather a coloboma.

Coloboma of the lens is almost as constantly unilateral as ectopia is bilateral.

There is yet another type of anomaly of the crystalline that resembles, in some respects, both ectopia and coloboma; this is **pseudo-coloboma**, and it might almost as correctly be called **pseudo-ectopia**. It consists in lessened convexity of a part of the border of the lens due to primitive malformation or defect in the zonule at that place. Indeed, the two conditions of ectopia and coloboma are here sometimes so nearly identical as to be inevitably confounded. True coloboma is a deformity in which a sector of the lens is actually lacking, and is consequent upon the obliteration, or lack, of a branch of the vascular system of the primary crystalline capsule (hyaloid system) which may or may not have reference to the non-closure of the fetal fistula or cleft. The result is a notching, or indentation, of greater or lesser depth or extent in the border of the lens. When the border is merely retracted, from a lack of continuity in the zonule at that point, without other signs pointing to a positive decentring of the crystalline as a whole, it is sufficient to denominate the condition as a pseudo-coloboma. This may occur after fetal life from a circumscribed rupture of the zonule. It is to be remembered that in ectopia there is incomplete development of the entire crystalline, and that it is smaller than is the space normally reserved for it.

**Spontaneous Displacement of the Lens.**—It is difficult to definitely classify many of the cases of apparently spontaneous displacements of the crystalline. Doubtless some of them are further luxations where had existed congenital subluxations, and others the result of traumatism, such as from sneezing, coughing, or from concussions that have passed



unnoticed or have been forgotten. But certainly there are instances of subluxation and luxation without congenital malformation of the lens or of its zonule, and in which are either no history of trauma, or are present abnormal conditions in other parts of the globe sufficient to account for the defect. Conditions which predispose to luxation are myopia, degeneration of the choroid and of the vitreous body, or any cause that interferes with the nutrition or with the tonus of the zonule.

Doctor H. Dor, of Lyons, recognizes three types of spontaneous luxation of the lens, namely:

1. Luxations that succeed congenital subluxations.
2. Luxations that succeed elongation of the globe (Myopia).
3. Luxations that succeed degenerations of the zonule of unknown origin.

From the clinical point of view there is a reason to distinguish subluxations and complete luxations, and in each of these forms also a variety in which the crystalline retains its transparency and another variety in which it is opaque. In subluxation with transparency, the subjective symptoms are much the same as those described under ectopia. The same is true of certain of the objective symptoms. The vagaries of complete dislocations of the feebly suspended lenses are numerous and interesting. Now the dilation of the pupil, as from atropin, will cause the transparent lens to enter the anterior chamber. Again, an individual is able to displace the lens into the anterior chamber or to make it return to the posterior chamber, or, at least, behind the iris, at will.

The spontaneous luxation of a cataractous lens, whether complete or partial has at times been hailed as a great boon, in that it restored sight to an eye that had been blind. A subluxated lens, or a cataractous lens with an exceedingly frail zonule, is prone to further luxation to any degree and from causes that are often imperceptible. Small wonder, then, that shocks and jars either of the head alone, or of the body as a whole, which are out of the ordi-



nary, should be attended by complete dislocation of such lenses. Hence, numerous instances have been recorded of the sudden restoration of sight to eyes that were previously blind from cataract, some of which contain an element of the ludicrous. For example, Janin relates the case of a lad of fourteen, blind from cataract in both eyes since infancy, who was by some younger companions taken for a walk in the fields. His comrades discovered a bird's nest high up in a tree and persuaded him, as being the largest boy of the party, to climb up, under their guidance, and try to obtain the nest. He undertook to do so, but in attempting to reach the prize, lost his balance and fell, feet foremost, to the ground, then measured his length on the grass. When he recovered his senses he was able to tell his frightened auditors the glad and startling news that he could see. He was borne in triumph to his home, where his delighted parents verified the information by examining the boy's eyes and ascertaining that the cataracts had disappeared. Burekhardt, of Basil, tells of an incident that is analogous, namely, how an Alsatian peasant woman, with double senile cataract, in whose case he had advised extraction, while consenting to the operation, but being a fervent Catholic, decided first to make a pilgrimage to a certain shrine and ask the Holy Virgin to bless the hands of the operator. On her way to the church she fell down a flight of steps into a cellar and dislocated her lenses, so that when she returned to her medical adviser he had only to fit her with glasses.

The extent of the displacement of the subluxated lens may be partial or complete. If partial, the direction is likely to be as in ectopia—*i.e.*, oftener upward, or upward and outward. When complete, the number of cases wherein the vitreous on the one hand, or the anterior chamber on the other, is the ultimate lodging place of the lens, is about equal, with perhaps a slightly greater tendency forward than backward. While the anterior chamber is less tolerant of the presence of the dislocated lens than is that of the



vitreous, instances are not wanting to prove that either may be the seat of the vagrant for an indefinite period without the eye suffering serious consequences. The disastrous symptoms of a forward complete luxation, however, are oftener those of a gradually increasing secondary glaucoma, and an ectasia of the anterior portion of the globe.

**Traumatic Displacement of the Lens.**—This form is due to rupture of the zonule through violence, and results from severe concussions of the entire body, as in falls from a height, and those concussions that are more local, as from blows upon the head, without rupture of the other parts of the globe. These constitute the **simple traumatic luxations**. Then there are the **complicated traumatic dislocations**, as from penetrating wounds, and from outward violence of such a nature as to cause rupture of the globe. In some cases of contusion of the eye, without other traumatism, there has been noted the sudden—perhaps only transitory—appearance of a myopia. Some authors have explained this as a relaxation of the zonule—others by irritation and spasm of the ciliary muscle. Becker attributes it to paralysis of the ciliary muscle. But seeing that with such paralysis there is inevitably paralysis of the accommodation, thus removing further from the eye the **punctum proximum**, and producing the opposite condition from that of myopia, Dor considers the myopia in point as consequent upon a forward displacement of the crystalline, and with it the principal focus of the dioptric system.

A partial tearing away of the zonule may occur, without subluxation of the crystalline and give rise to myopia, hyperopia or lenticular astigmatism. Myopia, with shallowing of the anterior chamber, if the displacement is forward, hyperopia, with increased depth of the anterior chamber if backward, and astigmatism, from relaxation of the zonule which affects only a certain portion of a meridian of the lens. The diagnosis of this variety of astigmatism is made by means of a skiascope, or of the ophthalmoscope, after having excluded the presence of a corresponding corneal astigmatism.



The astigmatism is, as a rule, not sufficiently regular to admit of correction by means of glasses.

With traumatic myopia and shallow anterior chamber, there may be hypertension of the globe, while with the hyperopic form, and increased depth of the anterior chamber, hypotonus has been observed. In the last two the vision is more susceptible of improvement by appropriate glasses than is that from traumatic astigmatism. In some instances the refractive error thus incurred remains permanent, in others it disappears with recovery from the accident that engendered it. These traumatic alterations in the refraction are by some deemed serious in proportion to the length of time they persist after the accident, and prognostications as to the outcome, and considerations as to any indemnity to be accorded the victim, are best withheld for a time, biding the ultimate issue.

Again, there may be partial giving way of the zonule with subluxation of the lens in the hyaloid fossa. Here the symptoms are identical with those in spontaneous subluxation, that is to say, the appearance of a myopia with astigmatism, and paralysis of the accommodation. The diagnosis is ordinarily quite easy: After dilatation of the pupil the border of the lens, as it traverses the pupil, may often be seen. In the more favorable cases, that is, when the subluxation is slight, correcting glasses may be of advantage. In others the lens may become cataractous, or the luxation increase, or iridocyclitis and a state of glaucoma supervene.

**Total Rupture of the Zonule. Luxation of the Crystalline without Rupture of the Globe.**—When the rupture of the zonule is complete, the luxation is most often equally so, and the lens passes either into the anterior chamber or into the vitreous. Occasionally, however, it remains for an indefinite time behind the pupil, though more or less decentred, and more or less forward or backward as regards its normal position. In some cases it is so mobile that the patient can, at will, cause it to enter the anterior chamber or into the vitreous. The traumatism is sometimes a direct



wound of the eye, as from a blow of the fist, or of a whip, or a stone, or branch of a tree. In those countries where stock raising is a prominent industry, as in Switzerland, the injuries that are most frequently the cause of luxation of the lens are those due to blows on or about the eye from the horns of cattle, or from the heads and hoofs of horses given by these animals either accidentally in their attempts to rid themselves of flies, or with vicious intent. Other common causes are striking the eye against blunt projecting objects, and against the corners of tables and the uprights of chairs while walking or stooping in the dark. It is true that many of these cases are also complicated by rupture of the globe.

In most instances the crystalline sooner or later becomes opaque, though in a few it retains its transparency for a remarkably long time. In one cited by von Graefe it remained transparent for twenty-five years. As before stated, the forward displacements of the transparent lens carry with them the advent of myopia, and those backward that of hyperopia. Among complete luxations may be classed those in which there still exists a narrow bridge of the zonule connecting the lens and the ciliary processes. This remnant of the zonule is oftenest, perhaps, situated downward, and the lens then falls backward and downward, with its greater (formerly backward) convexity in contact with the lower segment of the ciliary body. In other cases the bridge is situated laterally, and acts as a hinge, allowing the lens to swing like a door, now into something like its normal position—or shut, as it were, and again to swing in the opposite direction, partially or wholly opening the pupillary area. The appearance of complete luxation of the lens into the anterior chamber varies with its size, color and state of transparency. A full-sized transparent lens may be detected by the pushing backward of the iris and by the glistening circular reflex that marks its equatorial border, which reaches to the visible limits of the cornea. The position and extent of this reflex vary with the character and situation of the source of the illumination. In pro-



portion as the tint of the luxated lens is yellowish or amber, or as it has lost its transparency, the diagnosis becomes easier. The perfectly transparent crystalline in this location looks like a large bubble of air, while one of yellow or amber hue closely simulates in appearance a globule of oil of corresponding dimensions.

Recently in consultation with a colleague the writer has

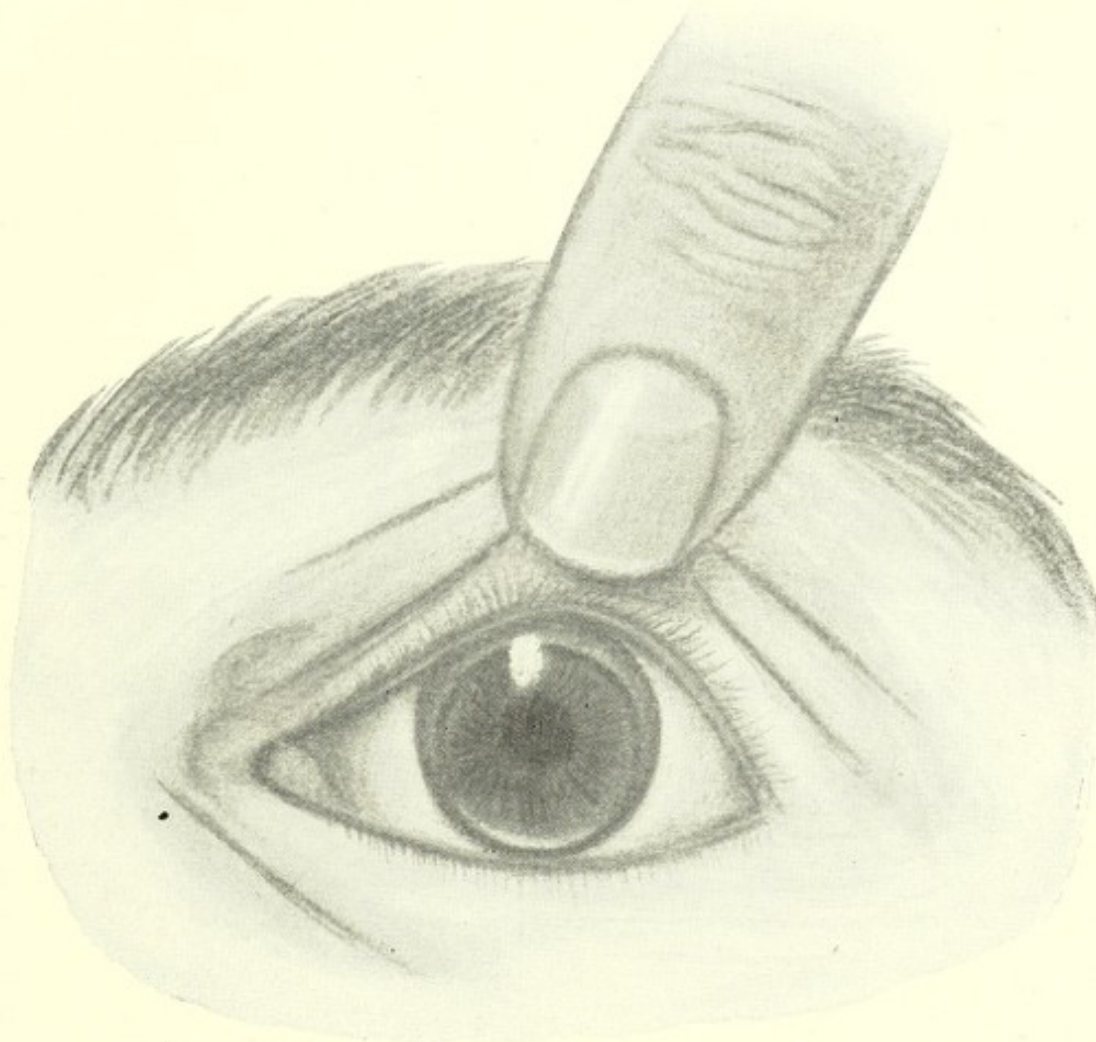


FIG. 5.—Discolored lens in anterior chamber.

seen a unique example of complete luxation into the anterior chamber. The subject was a girl nine years of age. Five years ago, while playing near a railway line, in the construction of which dynamite was being used, a blast went off near by, and in consequence she received a wound in the vicinity of the left eye from a flying stone. From that time



the vision was much reduced on that side. Two weeks previous to the consultation, while romping with a sister, their heads collided, and it was at once noticed that something else had happened to the defective eye. It was after the second injury that the child was brought to my colleague. On first looking at the eye the appearances were those of blood, or the remains of blood, in the anterior chamber—nearly filling the cavity. Upon closer scrutiny it was observed that the upper limits of the translucent brownish-red mass showed a clean dark convex outline above against the light blue-gray of the iris. A crescent of the iris was visible still above the mass. The entire circle of the dilated pupil could be seen through the mass (Fig. 5). Slight jarring of the head caused perceptible oscillations of the upper border of the mass. The upper segment of the pupillary border seemed to turn forward, or toward the observer, and the whole of the covered portion of the iris to appear as if nearer than the uncovered crescent. These effects are explained by the refraction and slight tilting of the luxated lens. At the lowest portion of the anterior chamber was seen a small bright crescentic reflex of a yellowish-pink tint, marking the lower segment of the equatorial border of the lens.

Again, the small and shrunken lens, or a hard nucleus from around which the softer lens-substance has been removed by absorption, settles to the lowest part of the anterior chamber, and can be made to change its position with the movements of the head and in obedience to the laws of gravity. Here, too, the appearances may be those of a bubble of air, or a globule of oil, owing to whether the body in question is transparent or yellow.

In complete backward luxations into the vitreous there is total blackness of the pupil by oblique illumination, and absence of the images of Purkinje. By examination with the ophthalmoscope the body of the crystalline, usually opaque, may be seen lying at the bottom of the vitreous chamber, and more or less mobile in response to the move-



ments of the globe, and still possessing some semblance to its normal form. Sometimes its recognition is difficult, or even impossible, in consequence of its altered form, or of surrounding vitreous opacities which tend more or less to hide it from view. When the traumatism which produced the luxation is recent the lens may be so enveloped in blood as to render it invisible.

The principal **complications** that often accompany traumatic luxations of the lens are mydriasis and distortion of the pupil, dialysis, ruptures and inversions of the iris, ruptures of the retina and choroid, intra-ocular hemorrhages, degenerations and opacities of the vitreous, detachment of the retina, and vicious alterations of the intra-ocular tension.

**Complete Luxation of the Crystalline with Rupture of the Sclera or of the Cornea.**—This form is the result of direct violence to the globe, though the rupture in its walls is not necessarily at the point of contact with the injurious agent, being probably more often elsewhere. This is explained not as due to wound by *contra coup*, but as a sudden irresistible rise of the intra-ocular tension consequent upon the great force of the blow, and the yielding of the globe at the point of least resistance. If it is the sclera that sustains the rupture, the wound is ordinarily concentric with, and 2 or 3 mm. from, the corneal limbus; and its situation is superonasally, or somewhere in the space between the insertions of the superior and internal rectus muscles, and behind the iris and in front of the ciliary body. The thinnest portions of the sclera are in the vicinity of the insertions of the recti muscles, where it measures as little as 0.3 mm. in thickness, and the most vulnerable locality is where the tendinous insertions are furthest removed from the cornea. As the globe is least protected by surrounding bone at the temporal side, it follows that the point of attack is most frequent from that direction. Hence, in thirty cases of subconjunctival luxations of the lens published since 1875, Massie found that the displacement was inward in ten, upward and inward



in four, outward in seven and downward in none. The rupture is of the cornea only in young subjects, though in these too it is sometimes of the sclera. The tear in the ocular wall is in most instances accompanied by dialysis of the iris.

In most instances the dislocated lens either escapes from the globe through the rupture, or is found beneath the conjunctiva. Exceptionally it enters the vitreous chamber, or is caught in the wound itself, but almost never is found in the anterior chamber. It is rare that the capsule of the lens is torn. The same causes prevail in this complicated species of dislocation as in those without rupture of the globe, namely, injuries from the horns of cattle, from striking the eye on the corners of tables, the uprights of chairs and beds, from blows of fists, gouging by fingers, from flying projectiles, like the balls of various games, stones, billets of wood, from kicks of horses, rebounding branches of trees and lashes of whips, and the relative frequency of these causes is about in the order here given. It is impossible to state even approximately the ratio of complete luxation of the lens to the total number of ruptures of the sclera and cornea.

If the lens lies beneath the conjunctiva the diagnosis is made easy by the characteristic tumor it occasions. If the case is of long standing, little or nothing of it may remain, though instances are not wanting in which its volume has not been sensibly diminished after lying for years beneath the conjunctiva. But in the great majority of instances it escapes entirely from the eye and is never found. Here the diagnosis of aphakia must be made in the usual manner, that is, by the absence of lenticular reflexes, the high hyperopia, etc. After all, luxation of the lens of any form is a relatively rare occurrence.



## CHAPTER XI.

### CATARACT.

The word cataract was first employed by Matteus Platearius, of Salernum, in the year 1150, though the older Arabic term for an opacity in the pupil meant "the water that falls in the eye." From this time until the last of the seventeenth century, and the beginning of the eighteenth, all visible opacities of the optic media were designated as cataract. It is true that Kepler, as early as 1611, demonstrated the physiologic and optic properties of the crystalline lens, but it was nearly a hundred years later, chiefly through the work of Maitre-Jean and Brisseau, that cataract and opacification of the lens were found to be identical. However, it was well into the eighteenth century before the fact was generally recognized.

Cataract is supposed to be derived from the Greek *καταραχτης*, "a waterfall," or from *καταρρηγνμι*, "to rush down." It is also the *Γλαύχωμα* (*hypochyme*) of Hippocrates, the *Υπόχυμα*, or *σπόχσις*, of Galen, the *suffusio* of Celsus. Other terms are *gutta opaca*; *aqua*; *aqua descendens in oculo*; *aquæ descensus vel cataracta*; *der graue Staar* (or *Star*) of the Germans; *la cataracte* of the French; *la cataratta* of the Italians, and *la catarata* of the Spanish.

By the term cataract is now understood any opacity situated between the vitreous body and the pupil, or between the anterior hyaloid membrane and the bottom of the anterior chamber. To put it differently, it is an apparent loss of transparency in the lens itself, or in its capsule, or in both.

**Classification.**—Cataracts are separated into two great divisions, viz., **congenital** and **acquired**. Following the example of M. Louis Dor, of Lyons, cataract is clinically divided into three varieties: **Congenital**, **traumatic** and



**dystrophic.** The congenital and the traumatic will be considered separately. The dystrophic, then, will include all the rest, that is, the senile, the symptomatic, the chemic, etc.

We may study and classify cataracts according to diverse points of view, as in the following table:

1. According to the **age** at which it appears:

Congenital

Juvenile

Adult

Senile

2. According to the **cause**:

Spontaneous

Traumatic

Symptomatic:

Albuminuric

Arteriosclerotic

Chemic:

Thermic:

Heat

Cold

Electric

Diabetic

Glaucomatous

Malarial

Phosphaturic

Naphthalinic

Spasmodic:

Ergotinic

Ciliary cramp

Tetanic

Thyroidismic

Uveitic

3. According to **consistency**:

Liquid

Soft

Semi-hard



Hard  
Ossific  
Calcific

4. According to the **color** :

White  
Gray  
Greenish  
Amber  
Black  
Blue

5. According to **extent** :

(a) Total  
(b) Partial:  
    Nuclear  
    Perinuclear  
    Cortical  
    Capsular

6. According to the **seat and disposition** of the opacities :

Central:  
    Nuclear  
    Perinuclear  
Anterior cortical  
Posterior cortical  
Anterior polar  
Posterior polar  
Equatorial  
Disseminated  
Punctate  
Zonular

7. According to the presence or absence of **complications** :

Simple  
Complicated

8. According to the **period of development** :

Incipient or commencing  
Immature  
Mature  
Hyper-mature or regressive



**Anatomically**, cataract is classified as **lenticular**, as **capsular** and as **capsulo-lenticular**. Going from front to back, one encounters, first, the anterior leaf of the lens-capsule, with its endothelial lining; second, the lens-substance, consisting of anterior cortex, nucleus and posterior cortex; third, the posterior capsule. Each of these portions may lose its transparency, thus giving rise to (a) anterior capsular cataract; (b) anterior cortical cataract; (c) perinuclear cataract; (d) nuclear cataract; (e) posterior cortical cataract, and (f) posterior capsular cataract. True capsular cataract is extremely rare, what is mistaken for it being most often certain opaque deposits on the front of a normal anterior capsule, or a seeming opacity of this membrane, due to folding or deformity of a really transparent capsule.

Normally, the internal surface of the anterior capsule adheres to the anterior face of the lens-substance through the intermediary of the intra-capsular cells of Werneck. This adherence is sometimes destroyed in consequence of a morbid condition whereby an opaque liquid forms between the anterior capsule and the cortex. This constitutes the beginning of what is known as **Morgagnian cataract**.

Any deposit in the pupillary area, or between the iris and the anterior capsule, whether of fibrin or coagulated lymph, pus, blood, or uveal pigment, forms an opacity described as **false cataract** (*cataracta spuria*). Its seat is outside the capsule, or *upon it*. True cataract, then, unless otherwise denominated, means an opacity of the lense itself.

### **Senile Cataract.**

Loss of transparency in the crystalline lense is not the inevitable result of senility. The oft asserted declaration that we would all have cataract if our lives were sufficiently prolonged is not supported by the facts. Cataract is a disease and not a phase of senile evolution. Its frequency increases from birth to the age of seventy, then decreases.



As Dor succinctly remarks: "There are cataracts among the aged, but there is no such thing as senile cataract." Really, only a small minority of old people develop cataract. The term "senile cataract" has, however, become so thoroughly established by usage that we are justified in applying it, particularly to the most common variety that is associated with advanced years, viz., the **subcapsular cortical**. Hence, unless otherwise specified, this is the kind that is treated of in these pages.

**Pathologic Anatomy.**—This is important to us in the present work, mainly because it enables us better to appreciate and to understand the objective symptoms, or better to interpret the clinical picture presented by the diseased lens. To go to any considerable extent into the literature of the subject would require a large volume by itself. Beginning as late as 1883, with the monumental work of Otto Becker, and coming down through the researches of Magnus, Schoen, Priestley Smith, Hess, Schirmer, Ginsberg, Roemer and L. Dor, one has about all that is known relative to the genesis of what is called senile cataract.

**Histology.**—In the study of cataract we distinguish only three layers of lens-tissue, and not seven, as formerly taught. They are (1) the epithelial, or subcapsular layer; (2) layer of cortical fibres; (3) layer of nuclear fibres. In the vast majority of cases cataract is a lesion of the cortical layer and of the subcapsular portion, the fibres of the nucleus remaining unaffected.

According to Otto Becker, most of whose assertions on this subject still hold good, the first signs of cataract do not occur as opacity of the fibres themselves, but in the appearance of vacuoles or lacunæ, of oval or fusiform shape, and of varying dimensions, situated *between* the fibres. Their contents also vary, in some being composed of the pale rounded globules or "balls of Morgagni," in others of homogeneous or granular masses. In either case one has to do with coagulated albuminous products. Later the



fibres themselves lose their transparency—their protoplasm becomes granular—and next a state of hydrops ensues which completely deforms them. Vacuoles appear in the fibres, especially at or near their free extremities, in the vicinity of the star. Their nuclei become elongated and thinned, lose their chromatin, then disappear, and their places are taken by vacuoles. Calcareous, colloid and cholesterin deposits make their advent in the degenerated fibres. Meanwhile, similar degenerative processes have been occurring in the subcapsular cells, and, in addition, certain proliferative processes, and the formation of peculiar vesicles.

It is possible for the entire cellular layer to disappear, and thus the way is prepared for the complete **absorption of the cataract** which is now and then observed. A peculiarity of the proliferative process is that which concerns the equatorial cells, by which, through their pushing backward, the posterior capsule becomes lined with a layer of extremely large and irregular cells, to which has been given the name of **pseudo-epithelium**. All these epithelial cells, whether true or false, may lose their nuclei, and become vesicular. Again, more recent researches as to the beginning of cataract seem to warrant the conclusion that the first morbid phenomena have their seat in the subcapsular cells—that these are *primary*, and not, as supposed by Magnus, secondary. Salfner, in his studies of the experimental changes in the lens due to the influence of naphtha, and Leitner, in seeking the cause of diabetic cataract, both found the subcapsular cells to be the initial location of the lesion. Leitner declared that so long as these cells remained normal in diabetic subjects, no opacity appeared in the lens, even though the vitreous might be saturated with sugar. These findings are in harmony with all the facts so far deduced in this connection, and enable one equally to comprehend the nature of cataracts from mechanical agents and those from chemical and physical ones.



As a result of the experiments of Widmark, relative to the effect of light upon the lens of the rabbit, the same conclusions were reached. He employed a 4000 candle-power electric light. In some instances the eye was protected from the ultra-violet rays by means of a screen composed of a solution of quinine; in the others there was no such protection. Wherever the lens was unprotected either by the iris or by the screen, lesions were produced which began as a karyokinesis of the subcapsular cells; then globules appeared in the nuclei of the cells, which gradually replaced these bodies, then filled the entire cells. The process extended deeper and deeper, causing opacities more or less dense.

In over 90 per cent. of beginning cataract the zones of opacification progress inward from points near the equator, occupying the anterior and posterior zones, but avoiding the equator itself, which is thus left as a transparent ring. In the rest, or a little over 7 per cent., the opacity shows first about the equator of the nucleus, none being visible in the superficial portions of the cortex.

**Macroscopic Description of Cataract.**—Foerster examined 72 cataracts taken from the eyes of the fresh cadaver, the ages of the subjects ranging from 54 to 87 years. He placed them in watch-crystals filled with vitreous humor, and put over them covers of thin glass made fast with sealing-wax. What struck Foerster most forcibly in these examinations was that *the nucleus of the lens was never opaque*. It was always the cortex. This has since been abundantly verified. Foerster concluded that the opacification began first, not in the endothelial layer, but in a zone situated at a little distance from the capsule. It was Malgaigne who, after having carefully examined 60 cataracts, discovered that the layer of subcapsular cells was the primary seat of the opacifying process.

According to Foerster, the most frequent forms of senile cataract are those in which the opacity appears as small white clouds extending partly or entirely across the width



of the nucleus. The front face of these opacities presents concentric elevations, giving the semblance of waves. Or they appear as white bands coincident with the meridians of the crystalline, broadest at the equator, and terminating in points as they approach the poles. These are often referred to as **radiating spicules**. They vary greatly in length and breadth in the same lens. Sometimes they unite to form continuous bands across the pupillary field. Those nearer the capsule are convex, or bowed, while those that lie deeper are less curved. These forms comprised about one-half the cataracts that Foerster examined.

In about one-fourth of his 72 cataracts, the opacities consisted in nebulous masses without definite form. A yellowish-gray girdle surrounded the equator of the nucleus. Upon examining it under a magnification of 60 diameters, this girdle was found to be composed of globules resembling minute drops of oil. These globules were larger and more densely congregated about the equator than nearer the poles. Those cataracts that were in a state of extreme incipency showed only minute, straight striæ, pointing inwardly around the equator.

The opacities, then, follow concentric layers, or zones, and hence, in a sense, all cataracts are zonular. The chief differences consist in the volume of the nucleus and in the fact that in congenital zonular cataract the opacified layers are sharply defined, and there is not that tendency to total cataract that usually characterizes the acquired forms. Upon the thickness of the affected layer of cortex, and upon the density of the opacity, depends the aspect of the cataract. If these be thin, the cataract will appear gray when within the eye, and of an amber hue when removed therefrom. If the layer is thick, and the opacity dense, the lens will appear almost white whether *in situ* or after extraction.

**Relation of Cataract to Alterations in the Ciliary Processes.**—The fact that certain lesions of the intra-capsular cells produce nutritive disturbances of the lens fibres,



eventuating in cataract, does not necessarily assign to those lesions the *primary* rôle in the process. Many believe that the *initial* changes occur in the cells of the ciliary processes, and class the lesions of the intra-capsular cells as *secondary*. However this may be, it is certain that morbid conditions and processes existing in the ciliary body are largely concerned in the origin of cataract. Congestion, tumefaction and hyperplasia of the ciliary processes, and trophic disturbances in their epithelium, as also in that of the posterior layer of the iris, are almost constant in all forms of acquired cataract.

**Hydration or Dehydration?**—The normal, or non-cataractous lens dehydrates with age, and this loss of water was formerly taught to constitute the cause of cataract. It has been more recently ascertained that a cataractous senile lens is much richer in water than a normally transparent one. The error arose from the fact that, as demonstrated by Priestley Smith and Treacher Collins, the lense increases in weight with age, the rate being about 4.5 milligrams for each succeeding decade—but that *cataractous lenses are smaller and weigh less*.

The following *résumé* by L. Dor constitutes the gist of what we have ascertained relative to the pathogenesis of cataract:

1. The opacification of the crystalline is due to the disorganization of the transparent albumins that are contained in the lens-fibres in consequence of the double process by which the albumins are hydrated, and their soluble portions are carried away.

2. This double process is not simultaneous. One is subordinate to the other. In certain cases (*e.g.*, glaucomatous cataract) the aqueous is forced into the interior of the lens-capsule, and it is secondary to this phenomenon that are produced the lesions of the subcapsular cells that set free and permit the absorption of the soluble albumins.

3. In a great number of cases it is found at the same time that in addition to the lesions of the cells of the lens-



capsule there exist also lesions of the cells that cover the ciliary processes, and likewise an intense congestion of the ciliary processes themselves.

4. The last findings allow the supposition that in these cases the pathologic processes which engender cataract, exert, beforehand, their action upon the ciliary body; the liquid filtered through this region acquires enormous proportions, and it probably is that which causes the alterations in the cells of the lens-capsule.

5. Further progress in the solution of the problem is retarded by the question as to what the causes are that determine the congestion of the ciliary processes and the alterations in their epithelial secretory cells.

6. Cataracts produced experimentally by means of salt or sugar are not true cataracts, seeing that they are the result of dehydration of the crystalline, and that lenses thus opacified regain their transparency when placed in water; while true cataract is the result of hydration, and does not clear up under water.

In view of the foregoing, Dor concludes that:

1. Acquired cataract is produced by a hydration of the crystalline lens, and the consequent disappearance of its soluble albumins.

2. This double process, while it is not attributable to a modification of the normal physical conditions, such as heat, light and electricity, is secondary to penetration into the lens of certain hydrating or cytolytic ferments contained in the serum.

3. The penetration of these ferments into the crystalline is rendered possible by alterations in the subcapsular cells, which alterations are very often secondary to those occurring in the epithelial cells of the ciliary body.

4. The alterations of the cells in question are most often provoked by vaso-constrictor poisons.

5. Amber and black cataracts owe their existence to phenomena of oxidation and not to phenomena of hydration.

6. As regards those forms of senile cataract in which the



nucleus is yellow, brown or black, and the cortical layers are gray or white, there is simultaneously oxidation and hydration.

7. Cataracts artificially produced by salt or sugar are the result of dehydration and cannot be classed as true cataracts.

**Subjective Symptoms.**—Prior to the discovery of the ophthalmoscope, by Helmholtz, in 1852, these symptoms were of vastly more importance than they are at the present day. They have a certain value, especially in reference to senile cataract, and are useful now mainly in explaining to us the complaints of our patients. These will vary considerably with the kind of cataract. Among the earliest symptoms is **fatigue** upon slight use in sharp or close vision. This may be referred as much to the eye that shows no sign of cataract as to the one in which such signs are manifest. The same has been observed in connection with a central opacity of the cornea. One who is accustomed to equal and normal vision of the two eyes, when one lens begins to opacify, perceives that reading is less easy, that the eyes tire more quickly, and that the letters are edged with colors, and that they dance. Binocular vision is disturbed by the diverse images to be fused—one sharp, the other blurred. The condition is such that, while not actually one of diplopia, is equally trying, and the more intelligent subjects are apt to find that they are more comfortable if they close or otherwise exclude the eye with the affected lens, and to wish for the time when the cataract may be extracted. Then comes **diminished visual acuity**; there are mists or clouds and motes or spots before the eyes (*muscæ volitantes*). The *muscæ* from incipient cataract, however, do not float, nor wave, nor settle down from gravity, like those from opacities in a fluid vitreous, but maintain their same relative position in the visual field—they are *fixed*. Both kinds may be present in the same eye, for degeneration of the vitreous, giving origin to specks and shreds that cause moving shadows, is common in conjunction with



senile cataract. There is **distortion of bright objects**, particularly those at a distance. A lamp-flame is drawn out into queer processes; the moon seems to have horns. In one instance the failing vision is first manifest in near work, such as reading and sewing; in another the first evidence of it is inability to see objects at a distance.

The character of the subjective symptoms depends upon the nature and seat of the lenticular opacities, and upon the alterations in the refraction of the crystalline as a whole. The **physical laws** that here find their application are formulated by Dor as follows:

1. Very small, punctiform opacities, it matters not where they may be situated in the lens, not only do not cast any shadow upon the retina, but the number of luminous rays which they arrest or irregularly refract is too small to have any effect upon their normal reunion at the principal focus, and consequently upon the vision.

2. When the opacities are more extended, encroaching upon or near the axis of the lens, the decreased illumination and lack of sharpness of the image are most apparent when the pupil is contracted, especially when the opacity is in the anterior portion of the lens. The stronger refraction of the peripheral layers of the crystalline, due to aberration of sphericity, causes these eyes to become slightly myopic, and explains why the vision is first rendered poor for distance.

3. Opacities confined to the periphery of the lens— anterior or posterior cortical cataract at the equator— exert no influence upon the vision so long as they do not fall within the pupillary field.

4. Certain striæ that traverse the pupil, whether of cortical or of secondary cataract, sometimes produce monocular diplopia or polyopia, particularly for bright objects, like the flame of a lamp or the moon.

In addition, **subjective examination of the normal human crystalline** gives interesting data relative to its structure. Persons of average intelligence can perceive the forms of



opacities or slight irregularities of refraction in their own lenses by looking at a bright light through a stenopæic opening. The opacity or the irregularity projects its outline upon the retina. Thus it is shown that, no matter how young or vigorous the subject, the lens is never of that perfect and even transparency exhibited by an artificial lens made from flawless glass. There are always radiating lines, dividing it into sectors, while throughout the sectors themselves there appear dots, streaks and spectral shadows in greater or lesser profusion, as illustrated by the spectrum of Donders. This was first demonstrated by Listing, who obtained an entoptic image of his lens by looking at a bright sky through a pin-hole in a bit of cardboard. A clearer image, as discovered by Donders, is furnished by admitting to the eye a homocentric beam through a half-millimetre aperture in a metal plate upon which a brilliant light is concentrated by a condensing lens. It was on this principle that Professor Barrett, of London, planned his **entoptoscope**, by which he was enabled to follow and record the process of development of cataract. Owing to contraction of the pupil, the field will be very restricted unless mydriasis is first induced. This inconvenience may be avoided by resorting to the method of Norris, whereby the lens spectrum can be more readily observed by taking as the source of light the images reflected from the posterior and anterior surfaces of a convex lens of 20 D.

Small opacities, even when centrally located, as pyramidal cataract or larger ones that are confined to the periphery, have little or no influence upon the vision, and are only discovered incidentally, as in seeking suitable glasses. Opacities situated in or near the axis of the lens may diminish the retinal illumination without being capable of appreciably lowering the vision. This last occurs only when they are of poorly defined outline, or when they are semi-transparent and permit only diffuse light to reach the retina, or when they are so extensive as



to occlude a large part of the pupil. The same is true of small anterior or posterior polar cataract if the surrounding capsule is irregularly elevated or corrugated. Peripheral opacities, when sufficiently extensive, often produce a lenticular astigmatism accompanied by monocular polyopia. This form of polyopia was first noted by Thomas Young, in 1801, and was further studied by Helmholtz, who demonstrated that it could be elicited in the normal eye, where it is due to a difference in refraction of the various sectors of the lens. This fact is less difficult of verification in older persons. It is, nevertheless, often an early sign of cataract. Not infrequently the polyopia exceeds diplopia, the image being more than once repeated, and in exceptional cases, particularly with respect to a point of light, the repetition runs into the hundreds. We have already seen that bands of opacity which cross the pupillary area may also cause monocular diplopia. To distinguish monocular diplopia from that caused by deviation of the globe, one needs, of course, only to cover the fellow eye.

**Lenticular Myopia.**—In stratified opacities of the lens we encounter myopia with diminution of the visual acuity. This myopia is *real* in certain cases, since one can often materially improve the distant vision by means of concave glasses, which does not hold as regards the myopia from spherical aberration, before alluded to. Often this form is only *apparent*, or, like that from amblyopia, or of defective media in general. Objects are held close to the eyes, not because of a shortened dioptric focus, but in order that the image, none the less blurred, may be the better perceived because of its greater size. Transient periods of myopia have, in a number of instances, been observed as a forerunner of tetanic cataract. Myopia is not an infrequent sign of beginning diabetic cataract, and is due to increased refraction of the lens, not to elongation of the globe. Swelling of the lense may result in shortening of its focus either by (a) inhibition of liquid by the cortical layers, which, although diminishing their particular index



of refraction, increases the total index of the crystalline, or (b) the convexity of the lens possibly is augmented. Sclerosis of the nucleus in elderly subjects unquestionably causes lenticular myopia. Hence, the rather sudden appearance of a myopia in persons who are no longer young suggests that it is lenticular, although it may or may not be accompanied by any actual opacities of the lens.

When the pupil is dilated focal illumination may show a distinct grayness, or a yellowish-gray opacity, of the nucleus of the lens, and that by transmitted light the nucleus appears as a dark disc in the centre of a bright zone of scarlet. This is **true nuclear cataract**, and is relatively rare. It is supposed to occur at a somewhat earlier period of life than the subcapsular cortical, yet the two kinds have been known to be associated in the same lens. Roemer says it usually appears about the age of forty. At that time the opacity is small and faint, and is less distinctly visible with the ophthalmoscope than by oblique illumination. In the course of years the intra-nuclear opacities gradually increase until, on close examination, punctate spots can be seen. The opacity is, at first, diffuse, and, in the unmixed form, there are no radiating streaks. What one more frequently finds in these cases of senile lenticular myopia is what has been called **pseudo-nuclear cataract**. That is, there is present no demonstrable opacity in the lens—both nucleus and cortex are transparent.

The phenomena as observed with the ophthalmoscope, the pupil being dilated, are, nevertheless, quite striking and characteristic. The central portion of the pupillary reflex seems somehow distinct from the peripheral, and when the mirror is rotated, as in a retinoscopic examination, a circular shadow comes and goes in the centre of the pupil. On actual skiascopic test of the refraction it is often ascertained that the centre of the illuminated field is myopic, and the peripheral zone is emmetropic or even hyperopic. Or, by moving the head from side to side at the focus of the convergent rays emitted by the central portion, the retinal



vessels seen there move in the *opposite* direction, while those seen through the peripheral field move *with* the motions of the observer. The status of refraction of the lens is, therefore, a duplex one, a condition that has been called "lens with double focus." These phenomena are the result of an abnormally great difference between the index of refraction of the nucleus and that of the cortex. By studying the lenticular light-reflex, as in the experiment of Purkinje, a normal image is seen from the convexity of the cortex, and one of exaggerated distinctness from that of the nucleus. How can these differences in the indices of refraction in the outer and inner portions of the same lens be best explained? Has the nucleus become unnaturally dense, as from dehydration, according to the theory of Guttmann, or has the cortex become less refractive than normal by imbibition, as suggested by Demicheri? It is more probable that the cortex remains the more normal, and that the nucleus takes on an unwonted density, else the phenomena in question would be more frequently attended by positive lenticular opacities. In this connection Hess has called attention to a possible abnormal convexity in the surface of the nucleus as a factor in producing the double focus.

**Second Sight.**—In common with a great many others, the writer has often observed the rapid development of a myopia of as high as four, five, and even six diopters, in the eyes of elderly persons, their previous refraction having been known to be emmetropic or hyperopic, and whose distant vision could be vastly improved by the proper concave lenses, their pride in being able once more to read without their glasses little lessened by the alternative of requiring lenses for objects more remote.

The disturbance of vision for distance is not, however, usually corrigible to a satisfactory degree for an extended period by glasses, but in most instances, sooner or later, the vision is so lowered from the diffraction as to call for extraction of the cataract. The fact that lenticular



myopia may exist for a considerable time before the appearance of any visible opacities, doubtless explains why a greater number of such cases are not found associated with cataract. I believe senile nuclear cataract is more frequent than is commonly supposed. It is quite true, on the other hand, that a small nucleus, close around which is a layer of opaque perinuclear opacity, could be readily mistaken for true nuclear cataract.

The intensity of the subjective phenomena in senile cataract is usually in direct ratio to the extent of the opacity and of the disturbed refraction of the lens. Whether the cataract be central, nuclear or perinuclear, the phenomena in question are about the same. At first the vision fails for distant objects, and later also that for things near at hand. An ever-thickening fog seems to settle on everything around. Strong light is peculiarly blinding and distressing. The patient sees better at twilight than at mid-day, better indoors than out, better with back to light than when facing it, and all for the reason that whatever favors dilatation of the pupil, and thereby the admission of more light to the retina, enhances the vision; while that which entrains contraction of that orifice results in amblyopia, effects the opposite of what is true of eyes with normal media. Hence, weak mydriatics and smoked glasses are often helpful. Subjects with this form of cataract when walking in bright light are characterized by an aspect of "**groping.**" They bow the head, shade the eyes with the hand, draw down the brows, half close the lids, and feel their way carefully along, it may be with the aid of a cane. This is in strong contrast to the erect bearing, wide-open eye, and confident march of the subject of optic atrophy. The eyes tire quickly from incipient cataract, often the one less affected as quickly as the other, just as in cases of slight central opacities of the cornea.

With regard to symptoms such as *muscæ volitantes*, phosphenes, photophobia, pain in the eye, and injection of the bulbar conjunctiva, these are not the result of the cataract,



but of an inflammatory condition of the iris or choroid, of which the cataract itself may also be the result. Exception is to be found in swelling of the lens, as in diabetic cataract, when impingement of the swollen cortex upon the ciliary processes and upon the iris results in photophobia and other signs of irritation. Then, of course, strong light is rendered doubly inconvenient. In the earlier stages, especially if the opacities are developed pretty well alike in both eyes, fine work at close range is often facilitated by the use of convex lenses several diopters stronger than are called for by the age of the patient. Long before all the cortical layers become opaque, ability to read even very large print is lost, and, lastly, that of counting fingers as well—only the movements of the brightly illumined hand are preceptible. Vision for form is now lost, only that for light and that for vivid color near at hand, and in broad patches, remaining. Owing to the yellow hue of the lens, light blue is usually the first color to become unrecognizable. At this period it is customary to pronounce the cataract **ripe, or total.**

**Perception of Light.**—In the most advanced stages, unless in the event of dense calcific deposits, or of an unwonted tint of brown in the cataract, the perception of light—quantitative and qualitative—is preserved, as can be verified by varying the intensity and the color of the test light.

It must be remembered, however, that perception of light does not depend solely upon the state of opacity of the lens, but in great measure upon the sensibility of the retina. The study of this symptom, then, is of the utmost importance in permitting us to determine before an operation the degree of vision we may expect to obtain. It is incumbent upon us to measure the quantity of light perceived. It was to this end that von Graefe constructed his **photometer**. Seeing that this apparatus is somewhat expensive and its management inconvenient, it is rarely employed, resort being had to the simpler and quite effective use of a small flame, or of the reflection from a plane



mirror of a light of feeble intensity. This examination is not indicated in cases in which ability to count fingers is retained. In the harder forms of senile cataract, with rather dark nucleus, with fine straight radiating striæ in the cortex, the light of a tiny candle may be perceived at the distance of eight to ten meters. With gray, pearly cataracts, with large white cortical stripes, it is seen at five to six meters, and with the cystoid or sacculated forms, containing milky fluid, as in the Morgagnian cataract of elderly individuals, the light often disappears at the distance of three to four metres. From oxidation a lens may reach a stage of such ink-like blackness as to interfere with the perception and projection of light, especially if the pupil be small. Calcareous and osseous cataracts may become so impervious to light that all that is perceived reaches the retina through the sclera. These latter forms are generally present only in amaurotic eyes. In hypermature cataracts the cortical masses, at first tumefied, then thickened, become more homogeneous, when it is found that the distance at which the light is perceived increases, or ability to count fingers may be restored; the anterior chamber may become less shallow, and the lenticular myopia diminish—facts that are important in determining the period of retrogression. Certain states of the refraction also have their influence. Graefe ascertained that in cataracts associated with high degrees of myopia or hyperopia the light was seen at a greater distance after correction of the refractive error.

**Projection of the Light.**—This is the power of the subject with cataract not only to perceive the light in any part of the field of vision, but to recognize and approximately to indicate its position. Projection is faulty when the examined is unable to assign the light at least to the quadrant of the field in which it is located. Perception with faulty projection may indicate a relative scotoma in the region upon which the light falls, as in low, partial detachment of the retina, or multiple lesions, as in disseminated choroidi-



tis. Lack of perception and lack of projection at a given point bespeak an absolute scotoma, or a destructive lesion. It is possible at times, in fairly advanced cases of senile cataract, to satisfy one's self as to the integrity of central vision. It is well known that sclerotic subjects of advanced age are prone to degenerations (chorio-retinal), confined to the macular region. That while perception and projection of light may be, on cursory examination, seemingly perfect, a more careful investigation with the aid of a not too bright point of light, projected from a small plane mirror, might elicit the fact that there is absence of central vision. If such be the case, the patient should be warned against expecting too much as the result of a contemplated extraction.

**Objective Symptoms.**—After having ascertained the acuity of vision and the status of the visual fields in a case of supposed senile cataract, before proceeding to an examination of the lens itself it is important to make careful study of the iris and its aperture—the pupil. In the inspection of the **iris** special attention is bestowed in seeking for signs of atrophy in that membrane. Are the color, details of design and topography sufficiently preserved to conform to those normal for the age of the individual? (See section on "Atrophy" in the Chapter on "The Iris.") Marked degeneration of the iris, without a history of previous acute iritis, would lead one to suspect a like sclerosis throughout the entire uveal tract. This, added to the presence of a small pupil, that is but slightly responsive to light and darkness, is yet more strongly suggestive of atrophy of the choroid. A small pupil, and one of but moderate latitude in its range of movement, is to be expected in the old, and, of themselves, these qualities have no special significance. A prompt and full reaction of the pupil to light is one of the surest evidences of the general integrity of retina and choroid as well as of the iris. The presence of posterior synechias would tend to cause fixity of the pupil in proportion to the extent of the adhe-



sions. This complication is often indicated by irregularity of outline in the aperture.

During this part of the examination it is most natural to look for evidences of cataract in the **pupillary area**. The best-known sign of opacity of the lens is the grayness or whiteness which supplants the normal black of this area. It should be remembered, however, that in many individuals old enough to be the subject of the disorder in question there is physiologically a slight gray or yellowish-gray reflex from the pupil, which has no reference to an actual opacity of the lens. The anterior capsule acquires a dusty look, which, mingled with the senile yellow of the lens, gives the reflex.

At this time there should be noted the **depth of the anterior chamber**. Extreme shallowness often denotes a swollen condition of the lens. In the absence of diabetic cataract, however, or of a glaucomatous process, a shallow anterior chamber is frequently the result of senile hypertrophy of the ciliary processes. As recently pointed out by Hess, these enlarged processes crowd forward, obliterating the periphery of the posterior chamber, pushing the iris before them, and drawing the lens after them. They can even tend to wedge in between the lens and the iris, so that the pupillary border no longer rests snugly upon the anterior capsule, but is held up, leaving a space between the two. Abnormal depth of anterior chamber is indicative of a shrunken lens, or it may mean a subluxation, with fluid vitreous. If the latter conditions exist, the iris will most likely be tremulous—in whole or in part. In the event of a lifting up of the iris by the ciliary processes just referred to, a shadow would be cast by the membrane upon the lens, and it could easily be mistaken for the shadow that has been so often referred to as signifying immaturity of the cataract, while the latter might be entirely opaque. It is to be noted that this shadow is, in the first instance, upon the lens, whereas, in the second instance, it is *into* the substance of the lens.

**Examination of the Lens.**—A necessary prelude to a



comprehensive inspection of the crystalline body is the dilatation of the pupil. In these elderly subjects prolonged mydriasis is to be avoided because of possible hypertension of the globe therefrom. A comparatively **safe mydriatic** is to be had in a mixture of equal parts of homatropin and cocain solutions, in the strength of 4 per cent. Two drops, with an interval of five minutes between instillations, will usually suffice. The two chief methods of examining the lens are:

1. By strong **oblique or focal illumination**, with the aid of magnifying lenses for the eyes of the observer.
2. By **direct ophthalmoscopic and skiascopic examination**, with and without auxiliary convex lenses.

**Oblique Illumination.**—A binocular or stereoscopic loup is an invaluable adjunct. By this method one recognizes the form, the extent and the seat of the opacities in most all the stages of partial senile cataract. One sees whether they are subcapsular, cortical, perinuclear or nuclear. If they are cortical the small, radiating, wedge-shaped striæ, grayish and straight, arranged around the periphery of the lens, points directed toward the centre, indicate the beginning of a hard cataract. They commonly make their appearance first in a tiny comb-like arrangement, inferior nasally, and well behind the iris. If, on the contrary, they are wide and white, the cortical layers are softer. If the streaks appear milky, or have a satiny or pearly lustre, their aspect is due to liquid. In cases slightly more advanced the spicular and radiate formation of the opacities is less pronounced, but may be fusiform, rounded, ovoid, linear, irregularly pyramidal, or band-like. They encroach upon and even cross the pupil. They increase in both size and number. Large portions of many strata become opacified. The radiate arrangement grows less and less perceptible. The depth of the anterior chamber may decrease from swelling of the cortex. A while longer and the pupil shows as a grayish-white disc; we can no longer look into the cortical layers; yet, upon close scrutiny,



we can still see thin streaks that outline the sectors of the lens, and there is still a trace of radiation in the sectors themselves.

The foregoing is not true of all such cataracts. Some arrive early at a stage of cessation of progress, while others begin premature regression, or are converted into mixed types.

**Ophthalmoscopic Examination.**—Owing to the fact that in this part of the investigation we use as a background the red reflex from the fundus, the method is adapted only to cases of partial cataract. The ordinary ophthalmoscopic mirror and the direct mode are employed, and convex lenses are used up to a strength of ten or twelve diopters. Instead of looking **upon** the opacities, and viewing them by the light reflected from them, as in the first method, we now see them as silhouettes intercepting the red light transmitted from the fundus. From gray or white they have changed to black. They are much more numerous, especially when magnifying lenses are interposed. Minute dots are now seen, that were before invisible. Moreover, intermingled with the shadowy spots there appear a certain number of bright ones—brighter even than the surrounding tint from the eye-ground—and having the same radiate arrangement as the opacities. From diffraction they seem almost to possess a white luminosity of their own. They represent accumulations of clear fluid between the disintegrating lens fibres. By rocking the mirror, while slightly moving the head from side to side, and to and fro, these bright markings come and go, in accordance with their different indices of refraction, and the incidence of the rays of light. They may occasionally be seen in the pupillary area, when focal illumination had shown nothing there—lying just beneath the anterior capsule, and reminding one of those light, fusiform defects that occur in window glass as the result of elongated bubbles of gas. In form they are rounded, though more or less drawn out. The black spots vary in shape from round, through all grades of oblong, to



mere lines and stripes. Small spines at the periphery become broad wedges, from accretions of smaller dots. The more advanced the case, the broader and longer become the shadows, even extending across the entire field, as fantastic designs or amorphous splotches, until the whole ground is filled in, and all red from the fundus is obliterated.

Since the centre of motion for the globe lies within the lens, it is an easy matter to **locate by their parallax displacement** the variously distributed opacities, while the globe is being made to rotate. Those that move with the cornea are, of course, in the anterior cortex, and *vice versa*. Those that remain stationary, or nearly so, are at or around the nodal point.

**Skiascopic examination** is useful chiefly in the examination of sclerosed lenses, or a form of pseudo-cataract, particularly in the double focus kind, already described.

It must not be understood from the foregoing reviews of symptoms that regular progression, in the order named, is an invariable rule in subcapsular cortical cataract, which is the commonest kind, and that which is under discussion. These cases come to the ophthalmologist in all stages of progression and retrogression. Certain among them, apparently diagnosed correctly as incipient cataract, seem to arrive at a period of cessation. Amelioration or disappearance of the primary cause of the morbid processes in the lens, or something, has occurred, which has served to arrest them, and, to all appearances, the lens remains, for indefinite periods, without further alteration. This is especially true of those irregular forms of anterior and posterior cortical cataract, and those in which the opacities are confined to the lower and inner quadrant of the lens near the periphery. Therefore, *so long as the patient can exercise two-thirds normal vision, it is imprudent, as Dor says, to pronounce the word "cataract" within his hearing.* It is, perhaps, allowable, for the sake of one's professional credit, to report the findings, in strict con-



fidence, to a near relative or friend of the patient. Years ago von Ammon described an affection of the crystalline characterized by peripheral opacities, and called it "geron-toxon lentis," supposing it to be to the lens what "arcus senilis" is to the cornea, and that the opacities had no great tendency to encroach upon the centre. It was about the same condition that Schoen took as his point of departure for his theory of the accommodative origin of cataract. Besides, **the possibilities for the clearing up of cataractous lenses**, with and without local and systemic treatment, are becoming more and more recognized. And these incipient cortical cataracts are precisely those from which most is to be hoped in this respect. Hence, another reason for guarded prognosis in these cases.

Whether or not opacification is progressive in a partial cataract, or what is the approximate rate of progress in a given case, can only be ascertained by keeping it for a sufficient period under careful observation; meanwhile recording the vision, and making successive diagrams of the shadows seen in the pupil by the ophthalmoscopic examination just described. By such means the point may be reached at which we can with impunity use perfect candor in dealing with the patient.

This brings us to the consideration of **what constitutes maturity in senile cataract**. Ophthalmologists are fully in accord upon this question. When a cataract has developed in the regulation way until a fairly consistent white opaque mass replaces the entire normal cortex, when the opacity comes apparently right up to the iris; that is, admitting of no shadow from that membrane, when the intumescence of the lens is at its maximum, and the depth of the anterior chamber at its minimum, when the movements of the iris are most restricted by friction upon the swollen lens, then the cataract is mature, or **ripe**. When it is ripe for extraction is another matter. If the time most propitious for surgical intervention could be chosen, it would be that when the cataract had got actually started upon the stage of



retrogression; when its volume had sufficiently diminished to allow the floor of the anterior chamber to reach its normal level, and the movements of the iris were free again. Unfortunately, this exact period can be seldom chosen.

**Hypermature Cataract.**—For a cataract to be hypermature it must have passed through the stage of maturity. Through one form of degeneration then, maturity is attained; through another, hypermaturity. The death of the crystalline is exemplified in the mature cataract. Just as in case of a perfect apple, for example, having reached its full size and development in a state of greenness, or of living union with its sources of life in the tree, it becomes a thing by itself, and proceeds to *ripen* by changes occurring *within*. Then the stem withers and it falls to the ground. Now other forms of degeneration ensue. By one it hardens and shrinks; by a second it turns black from oxidation; by a third it softens till all but the core becomes a complex fluid. These correspond to phases of hypermaturity in the cataract. By one process, the opaque cortical masses rid themselves of water, and by very slow condensing, hardening and (partially) clearing, it becomes of the consistency and appearance of the nucleus around which it tightly clings, eventually—after years, perhaps—having the semblance of freshly hardened, yellow gelatine. From having been blind the eye may now regain a modicum of vision. In this state it may remain indefinitely. Such is the **sclerosed lens** of aged subjects. In accordance with the second phase we have what is known as **black cataract**. Here the process is almost identical with that of the first phase, save that the color passes on from amber to brown, and, in extreme cases, to that little short of inky blackness. Formerly it was believed that the color was derived in some manner from the blood, but late researches, among them those of Gatti and Hess, have demonstrated that it is due to oxidation of the tyrosin, which is set free by decomposition of the albumins of the crystalline body. At a glance, the pupil in many cases of black cataract seems to



be normal, *i.e.*, there is apparently no reflex from it, but on strong focal illumination there comes to view a dull gray through which shimmers a tinge of dark brown; or the brown alone may be seen. In conformity with the third phase, as illustrated by the apple, the liquid resulting from the decomposition of the cortical fibres, either because of unusually impervious walls, or some other reason, does not escape from the capsule. The fibres undergo at one and the same time all the stages of hyalin, fatty, cholesterin and calcific degeneration. Broken-down remnants of fibres, globules of fat, crystals of cholesterin and granules of lime first compose a mass whose consistency grows less and less until there remains only a bag of liquid, to the bottom of which sinks the nucleus, together with such of the other heavier constituents of the mixture as do not cling to the sides of the sac. This is **hypermature liquid cataract, or Morgagnian cataract**. Here the aspect of the pupil, on cursory examination, differs but little from that of well-advanced cortical cataract, though it is sometimes strikingly white. Focal illumination and the loop, however, fail to show any traces of radiation or other markings in the gray or white pupillary field that are peculiar to the cortical form. Instead, one may find what look like patches of thickened capsule, or glistening white dots (lime granules) lying against the capsule. There is likely to be **iridodonesis**, particularly if part of the Morgagnian liquid has been absorbed. On dilating the pupil, and having the patient make sudden and extended rotations of the globe, glimpses can often be had of the brown nucleus as it comes in contact with the anterior capsule in downward movements of the eyeball. It is often quite deeply oxidized. With a candle an image can sometimes be seen from the convex surface of the nucleus, thereby giving an idea of its size, hence, also, of the necessary extent of the corneal section to be made for its extraction. All three of these forms are well suited to surgical intervention. Yellow sclerosed lenses may be safely extracted when the vision



is 20/200 or 20/100. Both these and the black cataracts are suitable for simple extraction. For the Morgagnian type a small preliminary iridectomy is desirable, as the nucleus is prone to be erratic, and lavage is needed to free the forward chambers of the insoluble contents of the capsule.

**Mixed Types.**—In addition to the several varieties of senile cataract already described, many present themselves that are combinations of these varieties, but their differentiation is often impossible. Cortical cataract may undergo complete dissolution, nucleus and all, without having passed through its successive stages of formation. In this manner it has been known to disappear entirely. Or it may be characterized by a mushy gray cortex with a normal or an oxidized nucleus of varying proportions. Again, the anterior cortex may have departed, leaving a mass consisting of nucleus and posterior cortex. In some a certain degree of vision may be obtained, when, to wait for maturity of the cataract, in the sense of expecting sight to be reduced to mere light-preception, is to wait in vain. As previously stated, this stage may have passed, and a little sight have been restored.

**Acquired Cataract Other than Senile.**—Among the many causes of cataract, aside from those incident to the pathology of age, there are few that possess clinical features sufficiently distinctive to be of semiologic value. True it is that opacity of the lens is often an important associated symptom of a more general disorder, as a disease or a toxemia, but neither the subjective nor the objective local signs are, in the vast majority of instances, notably different from those that have already been described. Worthy of mention, however, are the forms classed as **diabetic, anterior polar, posterior polar, and punctate.**

**Diabetic Cataract.**—Opacification of the crystalline in connection with diabetes mellitus is relatively so frequent as to indicate a positive causal relation. Moreover, the cataract often has special characteristics which enable one



to recognize it regardless of any knowledge of the urinalysis, and without having detected the acetone odor in the breath. This assertion holds, however, only as concerns the younger victims of the disorder. While glycosuria is not peculiar to any definite period of life, it is only when it occurs during the first three or four decades that an accompanying cataract has these distinguishing traits. After the age of forty-five, or, at most, fifty, it does not differ from the ordinary senile form. Indeed, in view of the tremendous gravity of the disease in youth, as compared with that in the old, the question has been asked as regards the latter: "Is there such a thing as diabetic cataract or merely cataract in a diabetic subject?"

The chief characteristic signs are the **rapidity of development** and the **aspect within the pupil**. Corroborative ocular symptoms are **enfeebled accommodation and bilaterality**. In case of a diabetic child, total double cataract has been known to form in a few hours—often in a few days—and at this age it seldom requires more than a few weeks. The appearance in the pupil is that of skimmed milk, or bluish-white, the tint being perfectly even. In adults, and even up to middle age, the development, while it requires more time, is still relatively rapid. A peculiarity of the young adult diabetic cataract is the appearance in the lens of certain radiating streaks of matt white, that have been likened to mineral wool—"asbestos stripes." In 15 to 20 per cent. of young diabetics the accommodation is diminished from two to five diopters. Swelling of the cataractous lens, and suddenly acquired myopia, although common to other forms of cataract, are more frequent in the diabetic type. Neither swelling nor opacity, however, is the result of the imbibition by the lens of sugar, as was formerly supposed. In both young and old, diabetic cataract is liable to a regressive metamorphosis similar to that of senile cataract, but much more rapid. In the younger subjects nucleus and all may liquefy and the residue be absorbed.



### Anterior Polar Cataract.

The most frequent variety is that which is the result of small perforating central ulcers of the cornea, notably those complicating ophthalmia neonatorum. In consequence of the perforation the anterior chamber is evacuated and the lens comes forward, applying its capsule to the opening, when, partly from the mechanical and partly from the inflammatory, irritation thus excited, proliferation takes place in the subcapsular epithelium at the place of contact. The ulcer heals, the anterior chamber is restored, but there remains a permanent central opacity of the lens. In most instances, as a result of the corneal exudations filling the ulcer and lying upon the capsule, the cataract takes the shape of a small eminence, projecting forward into the anterior chamber—**pyramidal cataract**—which is *always* acquired. When there is no elevation the opacity is due to the subcapsular disturbance alone, and lies between the capsule and the cortex. This kind can be differentiated from congenital anterior polar cataract only by the corneal opacity that is usually left by the perforating ulcer. The cornea in early childhood, however, has such powers of recuperation that the opacity is not always present, though the ophthalmometer or Placido's discs may show an irregular astigmatism at the corresponding spot, or the anamnesis may help the diagnosis. On rare occasions the lens, in receding after closure of the perforation, and having a drop of tough plastic exudation on its capsule, spins a thread that becomes organized into a column of connective tissue, uniting cataract and corneal opacity. Exceptionally, a perforating ulcer near the periphery of the cornea may cause anterior polar cataract, nor is it absolutely essential that the ulcer should perforate. Rare causes are phlyctenular and serpent ulcer and iridocyclitis.

### Posterior Polar Cataract.

The importance of diagnosing and differentiating this affection lies in the fact that when it is acquired it is usually



a sign of disease in the fundus. The chief subjective symptoms are, at first, a speck in the field—a fixed musca, a little outward from the centre, that ever flees in that direction in efforts to bring it in the line of vision. If the opacity extends, the paracentral specks increase in number, surrounding the centre, and eventually their place is taken by a black disc. By focal illumination, with dilated pupil, the opacity shows as a dull gray reflex. Its form is habitually stellate. This is best shown by the ophthalmoscope and transmitted light. By this method also, and parallax displacement, its location behind the nodal point is determined. It is likely to occur in high myopia with choroidal lesions and detachment of the retina. It is sometimes seen in conjunction with severe central or macular chorioretinitis, when the initial opacity takes the form of rosettes that tend to spread, and come forward, involving the posterior cortex—sometimes even the anterior—producing total cataract. Beginning posterior polar cataract associated with choroiditis is supposed to be a sign that the vitreous body has become disorganized.

Opacities at the posterior pole are frequently seen in connection with **retinitis pigmentosa**, beginning as a single dot, then sending out a few radiating processes; but here there is not that tendency to extension forward just alluded to, the cataract remaining small and stellate. Posterior polar cataract may also result from injury to the globe, with or without lesion of the capsule, and from prolonged uric-acid poisoning.

The congenital form occurs *upon* and not *within* the posterior capsule, and is most often a relic of the vascular capsular membrane of fetal life; hence, likely to be found in conjunction with remains of the hyaloid artery, or with other congenital anomalies, such as uveal colobomata and microphthalmos. The stellate shape is not characteristic of the congenital variety.



### Congenital Cataract.

It is not always possible to determine in a given case whether the cataract is congenital or whether it was acquired in the earliest years. This is not a matter of great moment, for we may consider as congenital not only all those that existed at birth, but also those that developed later, *provided no cause can be assigned for them other than the causes known to engender the congenital forms.* Accordingly, cataracts found in children up to the ages of fourteen or fifteen may be pronounced congenital. In older subjects the diagnosis becomes more doubtful, or critical, as then they are more likely to be confounded with acquired cataract.

Congenital cataract may, for convenience, be divided into two great classes: First, those resulting from anomalies in the fetal circulation, that is, of the hyaloid artery and of the anterior vascular membrane. Second, those that are not of such origin. To the **first** belong the **total soft, the membranous or siliquose, the calcareous, the cystic, the posterior and anterior polar, the central punctiform, and the fusiform.** To the second belong the **zonular, the nuclear, and the total hard.**

Operations for congenital cataract are liable to be disappointing in their results because of connate defects in the fundus.

**Total Juvenile Soft Cataract.**—This is by far the most frequent form of the total variety, and is said to constitute one-half to one-third of all the cases of congenital cataract. It owes its origin to the fact that at a given time the vascular capsule is attacked by perivasculitis. After the disappearance of the vascular capsule the opacified lens is left as a manifestation of the inflammatory process. It has a marked tendency to liquefaction and absorption, by which it is transformed into either siliquose or membranous cataract. By focal illumination one is enabled to look into the substance of the lens, and if one discerns certain



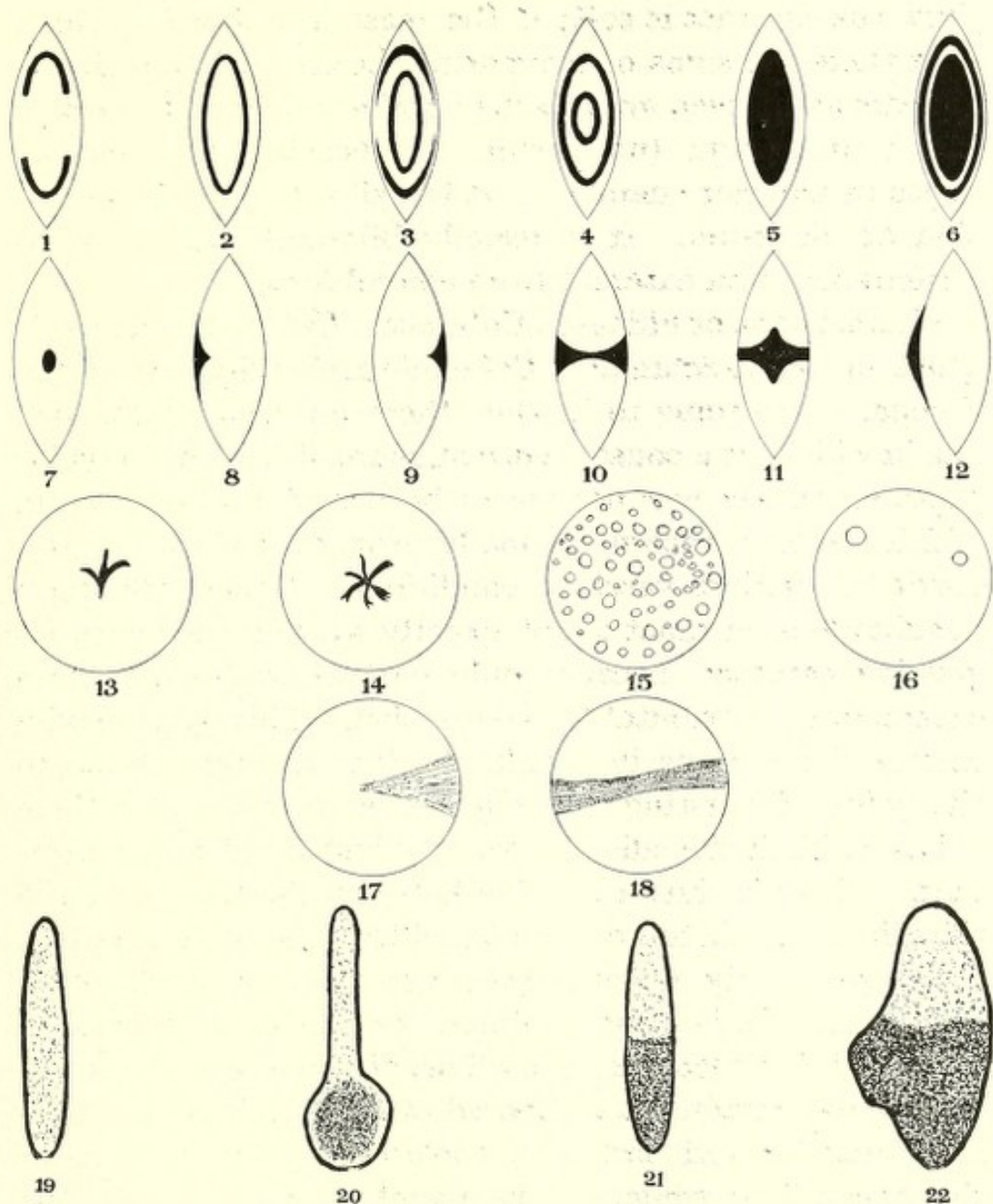


FIG. 6.—Forms of congenital cataract. (*After Dor*). 1. Partial zonular; 2. complete zonular; 3. partial double zonular; 4. complete double zonular; 5. zonular with opaque hard nucleus; 6. double zonular with total opacity; 7. central punctiform; 8. anterior polar; 9. posterior polar; 10. bipolar; 11. fusiform; 12. pyramidal; 13. 14. stellate; 15. punctate; 16. vesicular; 17. cuneiform; 18. bandlike; 19. siliquose; 20. siliquose with fallen nucleus; 21. siliquose with gravitated debris; 22. purse-like, fetid.



radiating striæ, or certain mottlings of gray that are *fixed*, the cataract is **soft**; if the mass is uniformly white, or if there are signs of movement therein, it is **liquid**. It appears as an even gray in the pupillary field, with normal iris, round pupil (no posterior synechiæ) and normal depth of anterior chamber. It is well suited to the operation of discission. It is usually bilateral, and may be confounded with cataract from convulsions.

**Membranous or Siliquose Cataract.** *Cataracte arida siliqauta*, of Adam-Schmidt. *Cataracte aride siliquouse*, of the French.—According to L. Dor, there are two types, viz., one in which, as a consequence of anomalies of the vascular capsule, the lens was not normally formed; and another, in which the lens, though normally formed, is absorbed as a result of morbid vascular conditions. Hence, the term membranous cataract is not exactly synonymous with regressive cataract, because only part of the membranous cataracts are attributable to regression. This part includes merely those forms in which, the lens having swollen to the point of bursting, the nucleus is absorbed and there exists at birth the siliquose variety instead of a soft cataract. There is thus a relation between the total soft and the siliquose. In this connection **siliquose** and **membranous** are more nearly synonymous, yet there is a shade of difference. In botany a silique (or silicle) is a flattish, oval, pod, between the two valves of which are the seeds. A siliquose cataract is composed of the two leaves of capsule (anterior and posterior) enclosing certain remains of the crystalline; while, in the membranous cataract, the two leaves are in contact throughout. In order to prevent confusion with secondary membranous cataract it were better, perhaps, to denominate as siliquose all those now under discussion, whether or not the lens residue has been wholly absorbed.

Clinically, there is a close analogy between the siliquose and the secondary cataract, for the latter may be entirely membranous, or it may contain encapsulated lens-remains.



In both the aspect is that of a white membrane stretched across the pupillary field. But by the ophthalmoscopic examination, with pupil dilated, a ring of red is seen around the opacity, suggestive of the appearances in zonular cataract. The red ring, or reflex from the fundus, in the zonular cataract, however, is seen through the transparent cortex, whereas here it is seen through the zonule of Zinn (Fig. 7). The opaque portion is less even and less round than in the zonular, and seems sunken. The membranous cataract is usually adherent to the iris, because of the vascular disturbances at the time of the regression, and remains of the pupillary membrane are often present. The anterior chamber is likely to be deeper than normal, and its floor tremulous (iridodonesis). The operation of discission is not suited to this kind of cataract, though Agnew's blunt hook operation gives admirable results.

**Calcareous Cataract.**—As found in young children this is but a siliquose cataract in which the nucleus has become incrustated with salts of lime. Sometimes the capsule, too, is calcareous. Again, only the capsule is calcific, and contains fluid (*cataracta lactea*). At other times the entire nucleus is calcified (*cataracta gypsea*). According to Beer, the diagnosis of *cataracta lactea*, or **milky cataract**, may be made by allowing the patient to hold the head for a time in one position, so as to permit the free lime granules to gravitate to the bottom of the calcified sac. It will then be observed that the lower portion is white, representing the deposit, and the upper yellow, representing the fluid, and that there is a sharp horizontal line of demarcation between the portions.

**Cystic Cataract. Cataracte Burseolée (Purse-Like). Fetid Cataract.**—Here, after the period of regression, some of the liquefied relics of the crystalline have remained in the capsule, have undergone a special kind of decomposition, and the mass is often putrid. Upon opening the capsule an odor of gangrene escapes. *In situ*, this cataract has the appearance of a deflated balloon, a part of which pro-



jects into the anterior chamber, like a hernia. The contents are generally yellow, and the condition might be mistaken for a combination of **ectopia lentis**, and complete detachment of the retina. If the bag, or puse, is lax, the bulging into the anterior chamber is disposed to be greater below than above, and it may oscillate slightly with movements of the head. Cystic cataract is very rare.

**Anterior Polar and Posterior Polar Cataract.**—The congenital forms of these have, with one exception, been sufficiently mentioned in connection with their acquired forms.

**Stellate Anterior Polar Cataract.**—This seems to be one of the strongly hereditary or familial types of congenital cataract. It occupies the exact centre of the pupil and is flush with the anterior capsule. It is usually of small dimensions, being seldom large enough to interfere seriously with vision. The star may be six-pointed or some of the points may be lacking. It exhibits no tendency to progression. It is not to be confounded with those stellate opacities that are sometimes seen deep in the cortex, anterior and posterior. In these latter the main rays of the stellate figure follow the lines between the sectors, sometimes giving off smaller branches, the whole extending across the entire field of the crystalline. On close inspection the star is seen to be composed of minute dots, like a sort of stipple.

**Central Punctiform Cataract.**—In the central punctiform variety the opacity appears as a single white dot, or tiny ball, clean-cut, and regular of contour, situated at the exact centre of the lens. Like the stellate anterior polar type, it is familial in a high degree, does not progress, nor does it induce profound amblyopia.

**Axial or Fusiform Cataract. Spindle Cataract. (Knies.)**—So named because of a fusiform or spindle-shaped opacity that extends perpendicularly through the crystalline, from pole to pole, its greatest diameter being where it engages the nucleus. Occasionally the opacity does not



reach all the way through the lens. According to Bach, who was able to tabulate only ten cases (one experimental) in the literature prior to 1897, the anterior and posterior portions of the opacity owe their origin to fetal circulatory disturbances, and the central portion to the same causes that produce central punctate cataract.

**Zonular Cataract. Lamellar, or Stratified Cataract. Schichtstaar.**—The popular designation "zonular" was first employed by MacKenzie, because of the zone that surrounds the opacity in this species of cataract. It is the commonest, in occurrence, of all the congenital cataracts. Its great distinguishing feature is a central circular opacity encompassed by a ring of greater (often perfect) transparency. The opacity is sometimes so slight as to be invisible in ordinary observation, *i.e.*, to cause no decided diminution in the normal blackness of the pupil. When visible, its aspect is peculiar in that it seems *back of* or *deep in* the pupil, and its surface presents a uniform, pearly gray. By oblique illumination, and with pupil dilated, the opacity appears as a regular disc, which, in different cases, may vary in diameter from three to seven millimetres. The size of the disc depends upon the number of lamina that are opacified.

The make-up of zonular cataract is to be borne in mind. This consists in a relatively clear nucleus enveloped by a thin layer of opaque cortex. There may be one or two opacified layers, occasionally an imperfect third one, separated each from the other by a somewhat thicker stratum of transparent cortex. If only a single layer exists, it closely envelops the nucleus, causing an opacity that about coincides in diameter with the average pupil. It is sometimes possible to get one Purkinje image from the front convexity of the cataract and another from its posterior concavity. By very intense focal light and the use of magnifying lenses, it is often found that the opaque disc is covered over with numerous points and dots. Moreover, the surrounding cortical zone is frequently crossed by



radiating streaks, like those seen in beginning subcapsular cortical cataract. A peculiarity of certain of these streaks is that they overlap the borders of the opacified disc, while others are seen to pass behind it, the two sets converging to unite further outward. These are called "riders," because they straddle the equator of the opacity. They are thought to constitute but an early stage in the formation of an additional cataractous lamella. Just in proportion as the outer zone is obstructed by riders and other opacities, in that proportion the cataract tends to progress. When this zone is transparent, or very nearly so, the cataract may be considered as stationary.

By ophthalmoscopic examination there appears the circular silhouette of the opacity, surrounded by a girdle of scarlet light transmitted from the fundus. If the rays from the mirror are perpendicular to the opacity, the latter appears blacker around its periphery, and gradually less so toward the centre, where it becomes somewhat diaphanous. Cases have been reported in which there is actual transparency at the axis. This thinning of the shadow toward the centre is explained by the convergence of the anterior and posterior leaves of the opacity. In this respect zonular cataract differs from the nuclear form, wherein the shadow is densest at the centre. If the observer's head is moved to one side, and the mirror is tilted, the cataract appears as a uniform black. The entoptic figure in this form of cataract, as described by subjects of sufficient age and intelligence, exactly corresponds with that shown by the ophthalmoscope. It is that of a round, dark spot with a lightish centre, the whole surrounded by a circle of bright light.

The degree of visual acuity in zonular cataract depends mainly upon the degree of saturation in the opacity, and but in slight measure upon its diameter. For the cataract is nearly always wide enough to more than coincide with the pupil, excepting when this aperture is dilated. If the affected strata are but feebly saturated and the nucleus



s clear and not sclerosed, the sight is likely to be surprisingly good. In cases in which there is little vision through the average pupil, dilatation by a mydriatic, or exposure to obscure light, will often vastly improve it. In others a wider pupil makes no difference. This is the test for susceptibility of improvement to an **optic iridectomy**.

Myopia, either simulated or real, is the rule with zonular cataract. Objects are held very close up, and the line of vision is directed obliquely. A very few of these patients are benefited by concave glasses. Surgical treatment gives the best results: discission in the young, extraction in the old, or optic iridectomy at any age.

The affection is generally bilateral, and nystagmus is often associated with it, as it is with most forms of congenital cataract. It is occasionally combined with axial or with subcapsular cortical cataract.

Many believe that zonular cataract is not always present at birth, for the reason that competent observers have looked for it then in vain, but observed it later in the same subject. Whether it is always *apparent* at birth or not, certainly the cause is there, nevertheless. In other words, "if not *actually* congenital, it is, at least, of congenital origin" (L. Dor). It is well established that these cataracts grow more opaque during the early years, but later most of them remain stationary.

Verification of a prenatal cause is found in the fact that zonular cataract is pre-eminently hereditary and familial, and that it is frequently associated with defective bone-formation, infantile convulsions, and with certain malformations, such as those peculiar to the thyroid gland, etc.

Zonular cataract is to be differentiated from other forms, notably from the membranous and siliquose. In these there is often a central, round capacity, surrounded by a more or less transparent zone. It can be demonstrated, however, that the outer zone of transmitted light from the fundus in the zonular variety passes through the cortex of the lens, while in the membranous it is through the zonule



of Zinn (Fig. 7). In zonular cataract the pupil is round, freely movable, and the anterior chamber is of normal depth. In membranous cataract the pupil is most often irregular, its borders adherent to the cataract or to the zonule, and the anterior chamber abnormally deep. The diameter of the lens is usually less than normal in zonular cataract.

Another characteristic of zonular cataract is its liability to sclerosis of the nucleus and fluid degeneration of the cortex—processes by which it is converted into Morgagnian cataract. Hence, all Morgagnian cataracts cannot prop-

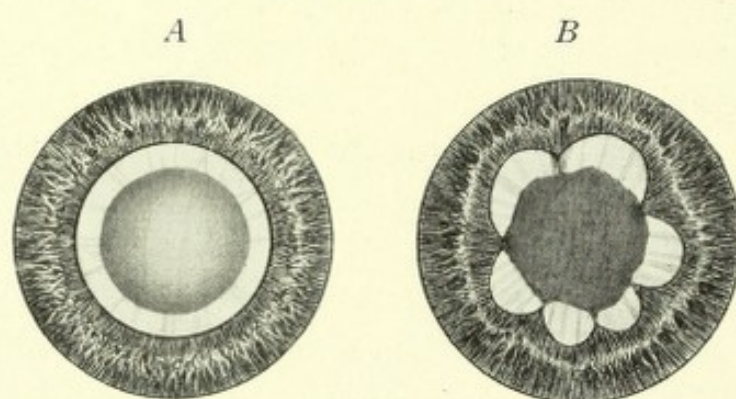


FIG. 7.—*A*. Zonular cataract, and *B*. membranous or siliquose cataract, as viewed by light reflected from the fundus of the eye. In *A* the rays of the bright zone pass through transparent cortex. In *B* the rays of the bright openings pass through an abnormally broad zonula of Zinn.

erly be called **hypermature**. For further differentiations, see "Morgagnian," and "hypermature."

### Errors of Diagnosis.

To the well-informed and experienced ophthalmologist the recognition of cataract is not difficult, but to the young or the untutored one, and to practitioners in other branches of medicine, the diagnosis is not always as easy. Among the things most often mistaken for cataract are central opacities of the cornea, the products of exudations upon the anterior capsule, the lens-reflex in glaucoma, and whitish reflexes in the vitreous, such as glioma, pseudo-glioma and total detachment of the retina. Then, too, amaurosis



from optic atrophy and hemorrhage in the vitreous have sometimes been confounded with black cataract.

**Central opacity of the cornea** is differentiated by its position at the very anterior pole of the eyeball, and by the fact that the pupil and the iris can usually be seen behind it.

**Inflammatory exudations** from the iris and their products, known as **false cataract**, have already been referred to, and are distinguished by reason of their situation *upon* the anterior capsule instead of *beneath* it. They often are of a yellowish tint, and overlie the pupillary border and encroach upon the lesser circle, or sphincter portion, of the iris.

The **greenish reflex** from the pupil in **glaucoma** is attended by sluggishness or absence of the light-reflex, or, in the more advanced and in the inflammatory forms, by dilatation of and immobility of the pupil, pericorneal and conjunctival injection, and by hypertension of the globe.

In **glioma**, **pseudo-glioma** and **total detachment of the retina**, with a transparent crystalline, the opacity may be distinguished by its great depth within the eye, by the presence of all three images of Purkinje, and often by blood-vessels overlying it.

In **amaurosis** from optic atrophy the fundus lights up brightly to rays thrown through the pupil by the ophthalmoscopic mirror. In **black cataract** the third image of Purkinje is lacking, while in **hemorrhage in the vitreous** it is present. Moreover, in black cataract strong focal illumination will reveal a brown reflex coming well up to the pupil.

### Traumatic Cataract.

Cataract from injury is classified as **direct** and **indirect**. Direct when the lens is actually pierced or penetrated by the wounding agent, as when a pointed instrument enters and is again withdrawn, or when a foreign body lodges within or passes through it. Indirect, when the lens does



not come in contact with the wounding agent, as when the injury consists in contusion of the crystalline, with or without rupture of its capsule or of the zonule of Zinn, or of two or of all three at once, through concussion. It may be a blow upon the globe or in its vicinity, or through a jar that implicates the head or the entire body. Traumatic cataracts are also divided into **simple** and **complicated**. Simple when the lens alone is implicated; compound, when other structures of the globe are involved either primarily or secondarily.

**Direct Simple Traumatic Cataract.**—The aspect of the cataract varies according to the nature of the injury, the length of time that has elapsed since it was inflicted, and that of the object that produced it. If the wound is from something very small or slender, as a sewing needle, or a small particle of flying steel, one may find only a small scar in the cornea, perhaps also in the anterior capsule, and extending backward from the latter an opacity of the lens. It is well-known, however, that a small foreign body may penetrate the lens without producing an opacity.

If the wound of entrance into the globe is at the periphery of the cornea or in the sclera, no trace of it may be visible, the diagnosis resting solely on the character of the lenticular opacity and the anamnesis. When the iris has been pierced, the site is usually indicated by a hole in that membrane, or by an adhesion to the underlying capsule. It is not to be inferred that cataract from slight punctures or wounds from tiny pointed objects are always partial; a large percentage of them, particularly as concerns the younger subjects, are total. All depends upon whether or not the wound in the capsule cicatrizes promptly. If the healing is not delayed, the opacification of the cortex is arrested, or limited, and the cataract either remains partial or there is restoration of transparency more or less complete. Numerous examples of such reparation are not wanting in the literature. Wounds several millimetres or more in extent do not heal, but rather enlarge, by rolling or furl-



ing back of the capsule, and through swelling and extrusion of the cortex from contact with the aqueous. Extended opening of the capsule occurs in a goodly number of traumatisms of the lens, whether direct or indirect. In this event the resulting cataract often presents itself as **consecutive**, either membranous, or siliquose, the substance of the crystalline having wholly or partially disappeared by absorption. In wounds of the posterior capsule the vitreous plays the same rôle in producing opacity of the lens, but the process is much slower. Posterior wounds have a far greater tendency to cicatrize than the anterior and equatorial ones. When the wound is near the equator, in addition to the primary or local opacity, there is often a secondary, stellate one at the posterior pole.

In the young, traumatic cataract not only forms more quickly, but is often absorbed with an equal rapidity. As regards young children, and in the simpler cases, *i.e.*, when not complicated by iritis or iridocyclitis, complete disappearance may occur in two weeks. The average duration of the process in children of from six to ten years, is two or three months, and six to nine months in older children and young adults. The younger the person the less the likelihood of an incomplete absorption, and the ultimate formation of a more or less dense secondary cataract. Complete absorption has often been reported in individuals up to fifty years of age, and exceptionally in those up to sixty-nine. The length of time that elapses between the injury and the appearance of the resultant cataract is extremely variable, being anywhere from a few hours to a few years. When very slow in developing, the cataract is termed *tardy*.

**Complicated Traumatic Cataract.**—Most all traumatic cataracts are, at the start, accompanied by signs of irritation, pericorneal injection, hyperemia of the iris and conjunctival injection. But so long as the symptoms do not pass a certain degree, as the pupil responds to a mydriatic, and as no evidences of pernicious infection are manifest, the cataract may be considered as uncomplicated. The ir-



ritation generally subsides with the healing of the capsular wound, provided no foreign body remains in the eye. Iritis is, as a rule, the first complication observed, as it is also the most frequent. The severity of the inflammation may be of any degree between that so slight as to leave only an isolated, small posterior synechia or two, through all grades of adhesion, leading to complete and permanent union of the contiguous portions of the iris and anterior capsule. In the still graver inflammations, the different grades are characterized by exudations into the anterior chamber, and blocking the pupillary area, extension of the uveal inflammation so as to become *general*, on to the septic forms, wherein only certain tissues of the globe are involved, or a true panophthalmitis ensues, ending with **phthisis hulbi**.

**Secondary glaucoma** is of great frequency in connection with traumatic cataract. The hypertension is brought about in two ways: (a) By the rapid intumescence and bursting out of the cortex into the anterior chamber, the purely mechanical pressure forcing the iris into close and total contact with the cornea, and obliterating the filtration angle—**mechanical secondary glaucoma**. (b) By inflammatory adhesions between the iris and capsule, cutting off communication of the posterior with the anterior chamber, or between the root of the iris and the corneal base, and the isolation and obliteration of the important channels there for the egress of the intra-ocular fluids—**inflammatory secondary glaucoma**.

The two influences are often associated in the same eye, as when a portion of the iris, the capsule or the vitreous is incorporated with the wound. The mechanical variety is peculiar to the younger subjects and supervenes more quickly. An otherwise uncomplicated traumatic cataract, caused by a clean puncture of the crystalline, can swell to such proportions as to cause tremendous hypertension in a day or two. The inflammatory variety is relatively slow to occur, and is more often seen in older persons, the tunics of whose globes are less permeable and elastic.



**Cataract from a Foreign Body in the Lens.**—The diagnosis is not difficult if the opacification around the offending substance is but slight. Hence, the necessity of making examination by oblique illumination and by the ophthalmoscope at the earliest possible moment. If it can be only surmised that the foreign body is in the lens, the next recourse is to be had to the *x*-rays, provided the substance is opaque to them. If iron or steel, siderosis, beginning within the capsule, is fairly indicative that the crystalline holds the particle, though this discoloration may first show in the lens when the iron is immediately behind the posterior capsule, or in the vicinity of the ciliary processes. The sideroscope will occasionally aid in the localization of a magnetic foreign body in the lens.

Small foreign bodies that lodge well within the lens, if they are aseptic and not of a nature to undergo chemical disintegration and decomposition, are well tolerated. Even such metals as iron, copper and lead may become so encapsuled as to be rendered innocuous and remain there for an indefinite period. If a foreign body is situated in the lens, even though the capsular wound may have healed immediately, and the particle be very minute, there is a strong tendency to total cataract. A bit of iron or steel so small as to make no shadow in an *x*-ray exposure, or as to remain motionless to the traction of the most powerful magnet, and to cause no deflection in the needle of the sideroscope, may cause complete opacification of the lens, and be itself invisible. I have recently observed such a case. A young workman, while turning brass on a lathe, felt something fly into his left eye. He turned to a fellow-workman at a neighboring lathe, who claimed to have removed a sliver of brass from the injured eye. There was little or no reaction, but, in the course of six weeks or two months, a complete cataract developed. Then it was that the patient came into my service at the Illinois Eye Infirmary. In the absence of any sign of a foreign body other than the cataract itself, a discission was performed.



In consequence, the lens broke up rather freely, and the greater portion of it was absorbed. Then was brought to view a tiny piece of steel, its longitudinal dimension not exceeding one-half millimetre, and its lateral, one-third. It had a thin envelope of connective tissue, which gave evidence of slight siderosis. By putting the giant magnet in contact with the cornea, the steel, together with its capsule, was loosened and drawn into the lower iridic angle, whence, after corneal section, it was extracted with the hand magnet.

Direct traumatic cataract is, as a rule, unilateral.

**Indirect Traumatic Cataract.**—It is well known that cataract may result from concussion of the body, as in jumping or falling from a height and landing on the feet, or otherwise, in sitting down violently through slipping off the feet, by a blow upon the head, by commotion in blows upon the eye, as the result of convulsions, and from a stroke of lightning. Like the direct, this form is divided into **simple and complicated indirect traumatic cataract**. The complications consist in dislocations of the crystalline, rupture of the capsule, rupture of the sclera, rupture of the retina and of the choroid, iridodialysis, intra-ocular hemorrhage, and detachment of the retina; being all traumas by *contrecoup*. Doubtless invisible rupture of the zonule often exists in what is classed as simple.

As regards their clinical aspect, the indirect simple variety usually begins as a stellate opacity of the posterior or of the anterior cortex, or of both simultaneously. If it remains partial, this configuration is retained. The rays in the last or combined form follow the sectors and extend to the equator, growing broader from centre to periphery. The opacification thus begun is most likely to become total. When only one pole is the initial seat, it is most often the posterior. The arrangement or design of the opacities is here either quite regularly star-like, or it is an intricate leaf-like radiation. These have less tendency to become total than the combined. Indeed, many instances of



stellar traumatic cataract have disappeared by regeneration. The accident that engenders the cataract is often so slight or of a nature so insignificant as to be forgotten before the trouble with the lens is discovered, or it is met with at an age so tender as to be unnoticed.

**Cataract from convulsions** constitutes the simplest of all traumatic cataracts. It is, of course, peculiar to children, hence, also, most often total and soft. It may, however, occur in connection with violent spasm of the ciliary muscle whatever the age of the subject, as, for example, in **tetany and tetanus**. Bilaterality is more habitual in this than in any other kind of traumatic cataract.

**Cataract from Electric Shocks.**—Any severe discharge of electricity, be it natural or artificial, that traverses the head or body in such a manner as to include the eyes, may cause opacification of the lenses, as attested by some dozens of recorded observations. This form of traumatic cataract is closely allied to that from convulsions with spasm of the ciliary muscle. It is to be distinguished from cataracts produced by **electric flashes** of tremendous intensity. These belong to the same class as those from violet or ultra-violet light, *x*-rays, etc., which are *dystrophic*, and not *traumatic*. The kind under discussion is known also as **cataract from fulguration**. As in the convulsive form, the initial disturbance is in the ciliary body—violent spasm of the muscle, congestion and lesions of the processes, leading to alterations in the subcapsular lenticular epithelium. Like the convulsive, too, the opacification of the lens from fulguration is prone to both totality and bilaterality. Yet, unlike the former, *cataract from electric shock is rarely uncomplicated*. Chief among the complications are inequality of the pupils, uveitis more or less extensive, rupture of the choroid, and rupture or detachment of the retina.







## PART II.

# THE FUNDUS OCULI.

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## CHAPTER XII.

### THE PUPILLARY REFLEX AND THE PAPILLARY REGION.

It may be confidently asserted that the average ophthalmologist will more readily avow his limitations in knowledge relative to the things he sees at the fundus of the eye than that pertaining to any other part of his specialty. This is doubtless, in great measure, because of the fact that our impressions of those things are obtained in a manner altogether special and peculiar. Men look into the eye with the ophthalmoscope much after the manner in which they look at a painting; in either instance deriving from the act ideas and opinions that are curiously varying and often at variance. Indeed, the requirements for obtaining correct notions concerning the phenomena of the eye-grounds and those for the proper appreciation of a painting are not dissimilar. It is not alone an aptitude for the interpretation of color, design and perspective, though these are the chief features of both. It is something else—something subtle and inexplicable. Then, too, slight variations of these features, as regards the eye, are often enormously different as to their significance. Here everything depends upon *seeing* that at which we *look*. To this end let us first study the various normal appearances of the fundus, and then consider them in relation to those that are anomalous and pathologic. *It is of the utmost importance to always examine both eyes.*



**The Pupillary Reflex.**—Considerable information as to the fundus is acquired by careful observation as to the lighting up, as it were, of the pupillary area when the light from the ophthalmoscopic mirror is first thrown into the eye. Normally this is glowing yellowish-white or slightly salmon-pink if the light is reflected from the very small area of the optic-nerve entrance, and glowing fiery red when from the rest of the fundus. In blonde and lighter subjects the tint of this glow is bright scarlet, and of deeper, or vermilion tints in darker individuals. These differences in the intensity of the red reflex are due to corresponding differences in the general tone of the choroid as the color of the optic nerve and retina are too slight, under physiologic conditions, to cause any decided modifications. As a rule, and as regards the white race, the darker the subject, the deeper the red glow of the pupil. This rule, however, is far from being invariable, as many dark brunettes, likewise many individuals among the Mongolian and Malay races, have eye-grounds but slightly pigmented, while those of many blondes are rich in pigment. The fundus of the pure negro is very dark, or, as is often the case, gives back a tawny tint, like that from the *tapetum choroidea* of certain lower animals. The color here, however, is from the inner surface of the choroid, or, rather, from the lamina vitrea just external to the hexagonal pigment, and not, as in the animal, from a highly reflecting layer of peculiar structure *in the choroid*, and located between the choriocapilaris and the layer of larger blood-vessels.

**The quality of light** used for the ophthalmoscopic examination imparts something of its own tint to the pupillary reflex, as also to that of the fundus details. By ordinary artificial light they are more yellow than normal, while only daylight can show them in their true colors. If there is present a brilliant white area of some extent it means something abnormal. It may indicate one of many things, among which are optic atrophy, atrophy of the choroid, coloboma of the choroid, or white infiltration of the retina; as from



embolism or thrombosis or opaque nerve fibres, proliferating retinitis, the ganglion-cell degeneration of amaurotic family idiocy, and certain stages of the effusions of metastatic choroiditis; or it may mean true glioma of the retina. When they occupy a large part of the vitreous chamber, the last two cause ordinary daylight to be reflected from them so as to render them visible, or even conspicuous, to the naked eye, and sometimes tinted slightly with yellow, red or green, giving rise to the characteristic glare known as **amaurotic cat's eye**. A soft grayish or greenish-white reflex filling all or part of the pupil, especially if it undulates with the movements of the globe, points to **detachment of the retina**. A dull white or slightly grayish reflex, that comes from a surface that is obviously quite mobile, is suggestive of the denser membranous bodies that float in the vitreous, and that are consequent upon **chorio-retinitis**.

**The Papillary Region.**—The papilla is that short section of the optic nerve beginning at the lamina cribrosa—or where the supporting connective-tissue framework of the nerve-fibre bundles (neuroglia) and the myelin sheaths of the fibres themselves are left behind—and terminating with the summit of the slight elevation, composed of nerve-fibres and blood-vessels, which is continuous with the inner surface of the retina. On looking into the depths of the normal eye the most conspicuous object to be seen is the papilla, or, as it is called often in ophthalmoscopic parlance, the **optic disc** (see frontispiece). It is situated decidedly inward and slightly upward from the posterior pole of the globe. It is circular, or slightly ellipsoid vertically, and measures somewhat less than two millimeters in actual diameter. In all eyes save those of albinos, or others devoid of pigment, it is of lighter hue than are its surroundings. It is subject to great variations in coloring both in health and in disease. At the centre, where the nerve-fibres are sparse or wanting, and there is only a pit, one sees only the white of the lamina cribrosa. The color of the papilla refers to



that of the zone next to the central white, made up of optic nerve-fibres and occupying the greater part of the area of the disc. This is pink for the temporal half, and of deeper tone, including more to rose, for the inner or nasal half. The color is supposed to be derived from that of the capillaries of the nerves-fibres, qualified by the white of the lamina cribrosa and supporting connective tissue, and is deepest where these fibres are most numerous or piled the highest.

Surrounding this pink zone is a narrow ring of white, the **connective-tissue** or **scleral ring**, and external to that a more or less complete, narrow, irregular circle of a pale chocolate color—the **pigment ring**. When lacking it is usually so at the nasal side. Within physiologic limits the pink of the disc may be pale almost to whiteness, especially at the temporal side, or it may deepen to old rose or light rose-madder. The red, particularly in hyperopes, is often nearly as deep as that of the surrounding fundus. Its depth is greatest in youth, gradually fading with advancing years and almost lacking in extreme old age. It varies considerably in width, encroaching upon the scleral ring or upon the central white area, till often the scleral ring is almost lost, or, in extremely rare instances, it reaches almost to the centre of the disc. The fact must not be overlooked that no small amount of color is given to the papilla by the large trunks of the retinal vessels which pass through its centre and over its surface. In the young, whose optic discs always appear smaller, and whose retinal vessels are of larger calibre, a goodly portion of this area of the disc is covered by the scarlet and crimson bands of the arteries and veins of the retina. In advanced years when the disc appears larger from senile degeneration of the papilla, and the calibre of the retinal vessels is lessened—for it is the size of the blood-column one sees and not the external extent of the vessel wall—the coloring lent by them is relatively slight. These vessels serve also to give information as to the topography of the papilla. Although the whole papillary region seems



to present a uniform flat surface, the optic nerve-fibres here form an elevated ring, or hollowed mound, which rises abruptly to its greatest height over the intermediate zone of the disc and gradually descends to the general level just outside of the pigment ring. Since the retinal vessels cling to the sides of the physiologic excavation within this elevated ring, and since they keep to the surface of the mound, they show by their arrangement, their curvatures, and by their

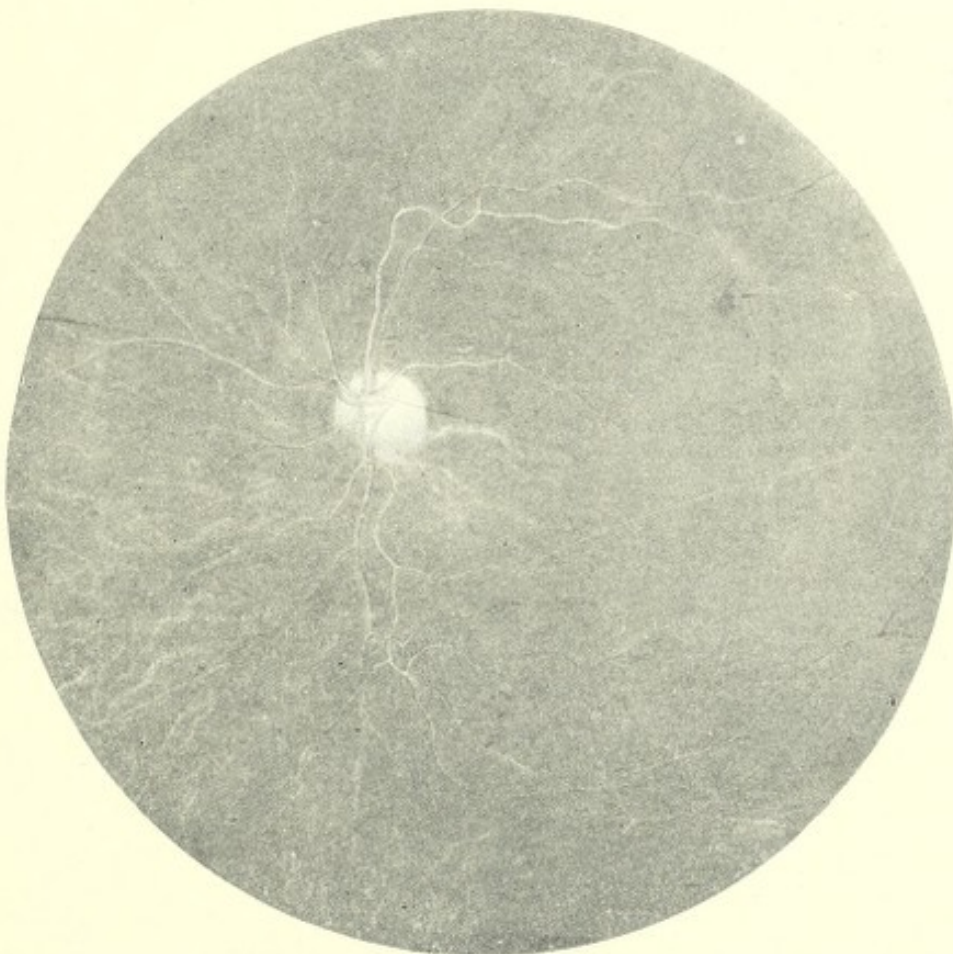


FIG. 8.

paralaxes the size and shape of the entire papilla. The relative altitude of the mound can also be estimated by measuring with the ophthalmoscope the difference in the refraction of the papillary and peripapillary regions. In general hyperemia of the head, the papillæ become redder; and in cyanosis, as in "blue" children, wherein there is



patency of the cardiac foramen ovale, they look as if they were stained purple.

The pigment, or choroidal ring, is subject to modifications in extent, though to a limited degree. After certain traumatism—like ruptures at the fundus from *contra coup*, and after some forms of chorio-retinitis (specific), there is pronounced massing of pigment about the borders of the disc, but in these instances it has not the veiled chocolate tint of the normal state, but appears glaringly black. Small splotches of pigment are not rare inside the border of the disc, but extensive pigmentation in this situation is extremely unusual. Like that of the choroidal ring, pigmentation of the disc has been known to follow deep traumatism of the globe. Strange to say, among the few observations that have been published relative to immoderate pigmentation in and around the disc, several have had reference to the eyes of albinos. Fig. 8 shows unique encroachment of pigment upon the disc. The subject was an intensely black negro girl. The disc, of deep scarlet pink, but appearing very light by contrast with the dark brown of the general fundus, was nearly obscured by pigment. The whole bore a striking resemblance to the full moon in partial eclipse. The defect was bilateral. In all other respects the patient was normal.

**Pathologic Modifications in Color.**—In the active stages of all varieties of **papillitis**, or **optic neuritis**, the color of the disc is nearly always much accentuated. The ophthalmoscope reveals the morbid features of this condition, such as dilation and tortuosity of the smallest visible vessels and other things, of which more will be said further on.

**Optic Pallor and Atrophy.**—These conditions are characterized by a deprivation of the papilla of its normal tint, and the substitution of mere whiteness, or of a different tint, and they are sequels of inflammatory or degenerative processes of the optic nerve itself or of the retina, or they are secondary to affections of the central vessels of the nerve. Both pallor and atrophy may affect only a sector of the disc—usually



the lower temporal portion—or they may extend to the entire area. Temporal blanching of the nerve-head is an important auxiliary symptom of the neuritis of **alcohol and tobacco poisoning**, though it does not necessarily give any indication as to the gravity of the disease. It may precede any degenerative changes in the nerve and is always bilateral. The whitened segment may comprise only one-third the area of the disc, or as much as two-thirds, but even in the more advanced cases, there is usually some color left at the nasal side.

Temporal pallor is also observed in **multiple sclerosis**, where it is most often unilateral, and the borders of the disc remain sharply defined. It has little tendency to extend to other portions of the papilla, often remaining stationary for a long period. In this respect it is unlike that of tabes, which begins as temporal pallor but rapidly becomes an atrophy of the entire disc. **Thrombosis** of a branch of the central retinal artery, **macular chorio-retinitis**, and **gumma** of part of the papilla sometimes result in circumscribed decoloration of this region. Partial blanching from the causes mentioned is not to be confounded with certain congenital anomalies, such as small plaques of opaque nerve-fibres which do not extend beyond the margin of the disc (as are discussed later) or connective-tissue prolongations of the lamina cribrosa.

**Optic Atrophy.**—In order the better to appreciate the conditions under which atrophy of the optic nerve originates and progresses, it is necessary to remember that the optic nerve-fibres, or conductors of luminous impulses, *are the axis-cylinders of the ganglion cells of the retina*; cells and cylinders constitute the third set of retinal neurons, which extends from the retina to the external geniculate body at the base of the brain. When the ganglion cell is destroyed its axis-cylinder degenerates throughout its whole length, from eye to brain. This is **ascending**, or **centripetal atrophy**. When the fibre loses its continuity or its integrity at any part of its course, degeneration occurs in both directions—



ascending from the lesion to the brain, and **descending**, or **centrifugal atrophy**, less rapid, from the lesion to the retina.

**The causes of optic atrophy** are:

- a. Traumatic section or rupture of the fibres.
- b. Direct action of a poison on the axis-cylinders or ganglion cells.

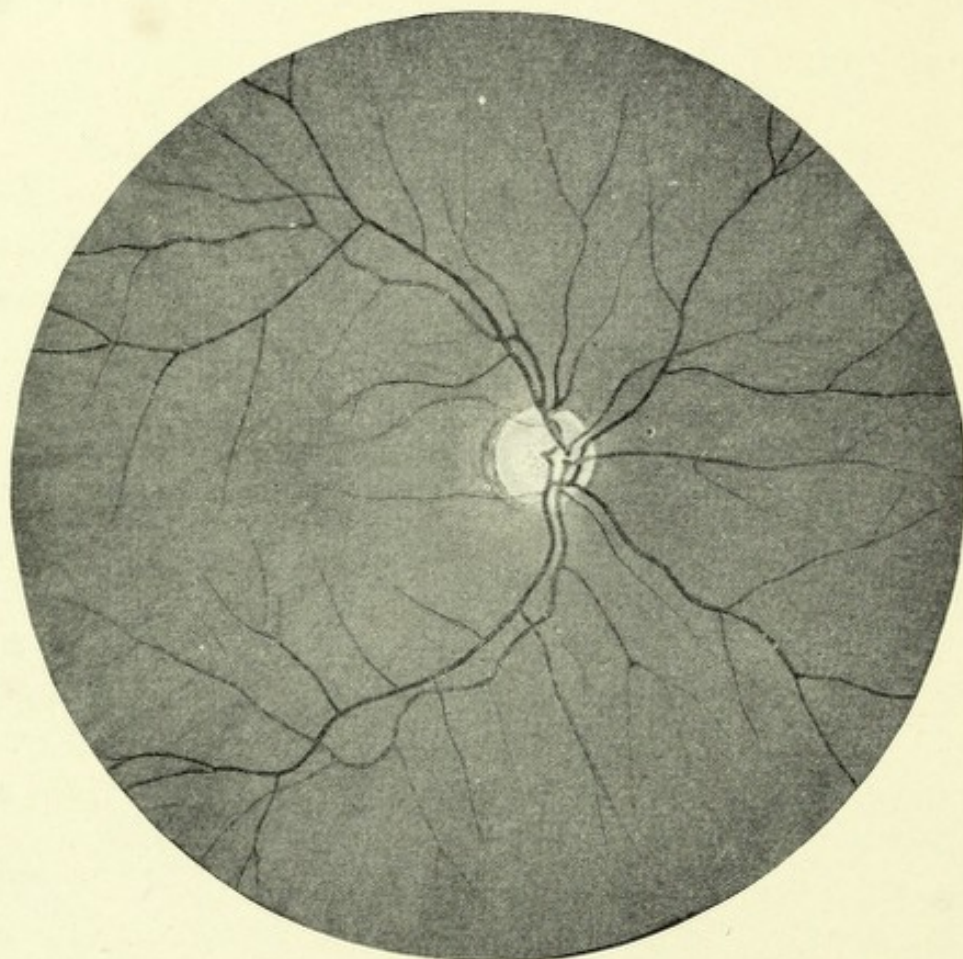


FIG. 9.—Simple or primary optic atrophy.

c. Complete arrest or too prolonged insufficiency of the nourishing blood-supply.

d. Prolonged mechanical compression of the retina or the optic nerve.

e. Inflammation of the interstitial tissue leading to secondary degeneration of the nerve-tissue.

It is probable also that atrophy may result from non-use, without any direct lesion of the optico-retinal neurons.

Clinically sharp distinctions between these different forms



from any standpoint is impossible, hence their classification is difficult and unsatisfactory. For instance, some would make but two kinds of optic atrophy—the one from **local**, or **peripheric**, the other from **central cerebral** or **spinal** causes. This is simple but unstable, for the reason that what one authority will class as local will by another—or even by the same at another time—be considered as central. The atrophy of **tabes**, for example, which has commonly been termed **spinal**, according to recent researches, begins in the cells of the retina. Again, others would call the ascending atrophy from destruction of the ganglion cells **secondary**, and that from papillitis **consecutive**, to distinguish them from the varieties of **descending** or **simple** atrophy; and would regard as **primary** those cases of **simple** atrophy wherein loss of function is in direct proportion to the apparent progress of the disease; and as **secondary simple** atrophy, those from loss of continuity by section, rupture, or erosion, and when the change in the papilla is slow to appear. It is impossible with the ophthalmoscope to distinguish any difference between the so-called primary, and other forms of simple atrophy.

It seems most convenient to classify optic atrophy as **simple**, and **secondary** or **neuritic**. In either case there is **decoloration** of the papilla as a whole. In the simple form (Fig. 9) the disc retains its normal form and size, while its borders are more sharply defined than normal, owing to the thinning of the nerve-fibre layer above it. The blood-vessels of the retina remain, for a time at least, of nearly normal size and appearance, the openings of the lamina cribrosa distinctly visible, and through contraction of the changed neural tissue in the nerve-head and that of the adjacent elastic tissue, with the possible addition of a chronic glaucomatous tension, there ensues a light cupping of the entire disc known as the **saucer-shaped excavation of optic atrophy**. On the other hand, if the symptoms have reference to secondary atrophy, or one preceded by papillitis and with edema, the contour of the disc will have lost its sharpness and its regularity and will have shrunk some-



what in size (Fig. 10). Instead of being circular in outline it may have become irregular, or polygonal. Instead of a **translucent white** like that of **pressed snow**, as in the primary form, this white is **opaque, mat or chalky**. Instead of a gentle depression of the nerve-head, with the lamina cribrosa showing plainly at its bottom, the whole area is crammed full of tissue as of cotton-wool, and devoid of detail. There often remains a degree of elevation of the

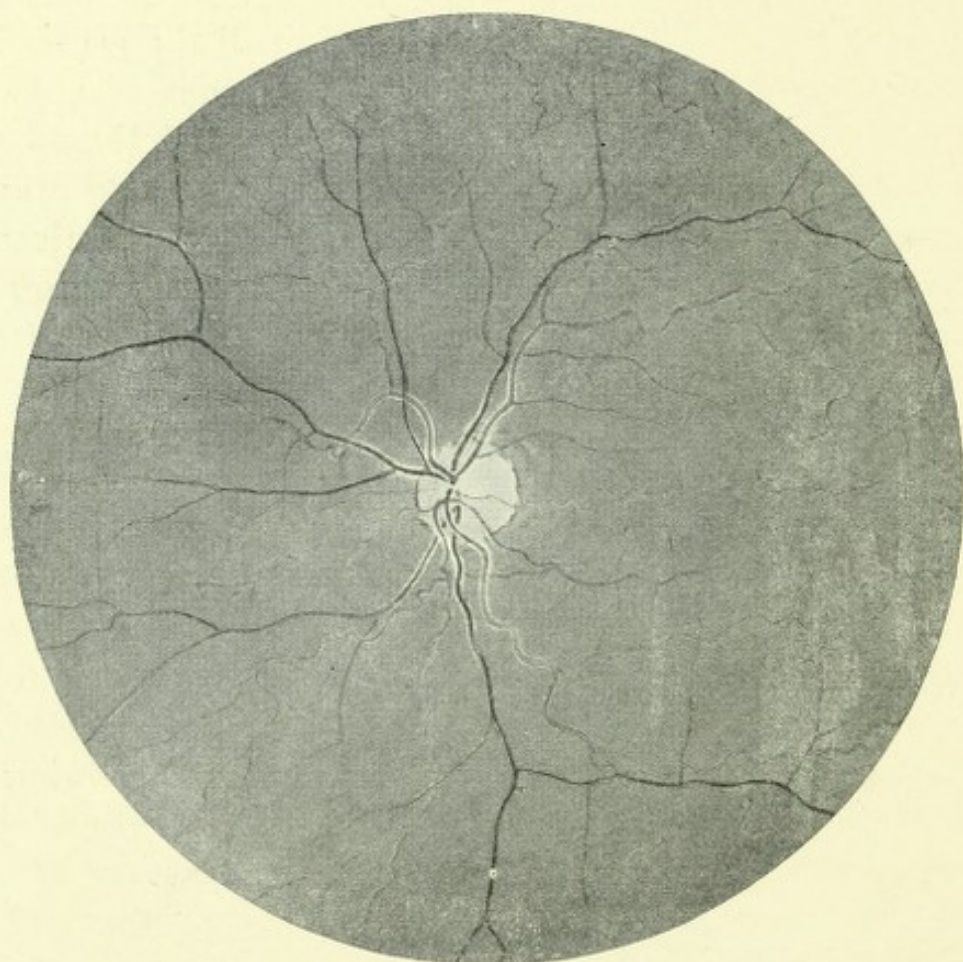


FIG. 10.—Secondary optic atrophy.

nerve-head long after the inflammation has subsided, and the retinal blood-vessels plainly show, for an indefinite time, how fully they shared in the process. They are, as a rule, greatly reduced or actually obliterated as to calibre, are tortuous, and for varying distances from the centre of the papilla their courses can best be traced by the white lines made by their thickened walls.



By far the most frequent cause of primary atrophy of the optic nerve is **syphilis**, and particularly syphilis in the guise of the two well-known neuropathic clinical modalities, **tabes** and **general paralysis**. The atrophy secondary to syphilitic lesions of the orbital or intra-cranial portions of the optic nerve may be unilateral, but that from tabes and general paralysis is always bilateral. In tabetic atrophy, one eye often remains apparently unaffected for months after the other has become profoundly involved, but it is doomed to the same fate if time be allowed. The simple atrophy of **multiple sclerosis** is similar in many respects to that of tabes, but in the greater number of instances the blanching does not extend to the whole papilla, and the degree of discoloration may be greater along the *nasal* margin of the disc, contrary to what is true of the tabetic kind. It is far from being uniformly bilateral, nor is ultimate blindness inevitable. Accompanying subjective symptoms that aid the differentiation are: For tabes, concentric narrowing of the fields of vision and absence of scotomata; for multiple sclerosis, sector-like defects of the fields and, frequently, central scotoma. Then, too, there are the objective symptoms referable to the pupils, the reflexes, the coordination, etc.

There are forms of descending atrophy from lesions of the optic nerve behind the entrance of the central retinal artery, and as far back as the chiasm, like those from section or erosion, in which loss of function precedes the atrophy by a long time. Even after the papilla has become completely blanched, the retinal vessels, in these cases, often remain practically unchanged in appearance for several years. Atrophy consecutive to **retro-bulbar** neuritis is frequently characterized by moderate dilation and tortuosity of the retinal veins.

The causes of secondary optic atrophy vary in frequency with the age of the subjects. In childhood it is due chiefly to neuro-retinitis secondary to **meningeal infections**, the result of cerebro-spinal meningitis, pneumococcus



infection, and suppuration of the middle ear. These cases are often bilateral. Many others, both bilateral and unilateral, in young subjects are consecutive to papillitis in conjunction with infectious diseases, such as hereditary syphilis, sphenoidal sinusitis, influenza, rheumatism, malaria, typhoid, measles, diphtheria, polyneuritis, variola, erysipelas, scarlatina, tuberculosis, chlorosis, and typhus. It is probable that a large percentage of these, too, are complicated by meningitis.

In the adult, syphilis stands first in the causation of secondary atrophy—the original inflammatory process reaching the papilla in various ways, as from the orbit, the sinuses and the meninges. The choked disc of intra-cranial tumor is, after all, but a papillitis, and leads frequently to optic atrophy. Then there is the large class that results from morbid processes of the vascular system, like the so-called **embolism of the central artery of the retina**—which is in reality, as a rule, thrombosis from endarteritis—and from the thrombosis of endophlebitis; to the same class properly belongs the **neuro-retinitis of albuminuria**. Atrophy resulting from **intoxicants** is, in a large proportion of cases, secondary to optic neuritis. This is especially true of the acute, fulminant intoxications, like that from ingesting or inhaling large quantities of alcohol—methyl or ethyl—or that from over-doses of quinine. The slower toxemias lead to atrophy that is not distinguishable from the simple form. The same is true occasionally of anemic atrophy from continued loss or impoverishment of blood. When the vascular supply is suddenly shut off from the retina, however, or the loss of blood from hemorrhage is enormous, visible traces are likely to remain permanently in the retina and papilla.

**Coloration in Optic Atrophy.**—It is very common to observe a decided tint in the atrophied nerve, variously described by the observer as gray, blue, green, yellow, dirty, smoky, etc. Formerly, optic atrophy was actually classified according to the color of the affected papilla. For instance, of the simple forms, white atrophy was classed as



cerebral and blue as **spinal**; the dirty gray atrophy as **consecutive**, and the dirty and smoky disc as **secondary**. This method of grouping naturally proved most elusive, for while these different tints depend, in a measure, upon the cause of the atrophy, they have greater reference to the severity and rapidity of the initial process, to its stage, or period, and to the concomitant alterations of the tissues, like edema, hemorrhages, leukocytic infiltration, etc. A soiled yellow tint, like that of old wax or sperm has, probably, the most constant significance, as it is almost pathognomonic of **congenital pigment degeneration of the retina**, and bespeaks the presence or the advent of the typic fantastic black markings in the retina (Fig. 11). The aspect of the papilla in **retinitis pigmentosa** is often so characteristic that by it alone one might make the diagnosis. While its tint is yellowish like old white wax, it has also a tinge of rose, and, taken as a whole, the color while much too pale and uniform for that of a normal papilla could never be mistaken for that of ordinary atrophy. Withal, the contrast with the tawny, washed-out appearance of the peripapillary region is but slight.

A solid looking, ivory-white papilla is suggestive of the rapid degeneration consequent upon ischemia by obstruction of the central artery, or upon violent intoxications like that of methyl alcohol. With ischemic atrophy of the kind just mentioned, evidences of degeneration are often conspicuous in the retina, such as white opacity remaining around the posterior pole, and white streaks marking the courses of former blood-vessels. Grayish tints are common with tabetic atrophy *in the early stages*—the same with regard to bluish or greenish, though these are more rare. If the case can be followed long enough, the tint will be found to have given away to white. Recent secondary atrophy often presents pronounced hues of gray and blue, but these colors do not last.

**Modifications in the Form and Aspect of the Papilla.**—The chief variations to be observed in the size and configuration



DESCRIPTION OF FIG. 11.

Advanced stage of retinitis pigmentosa in an elderly subject.



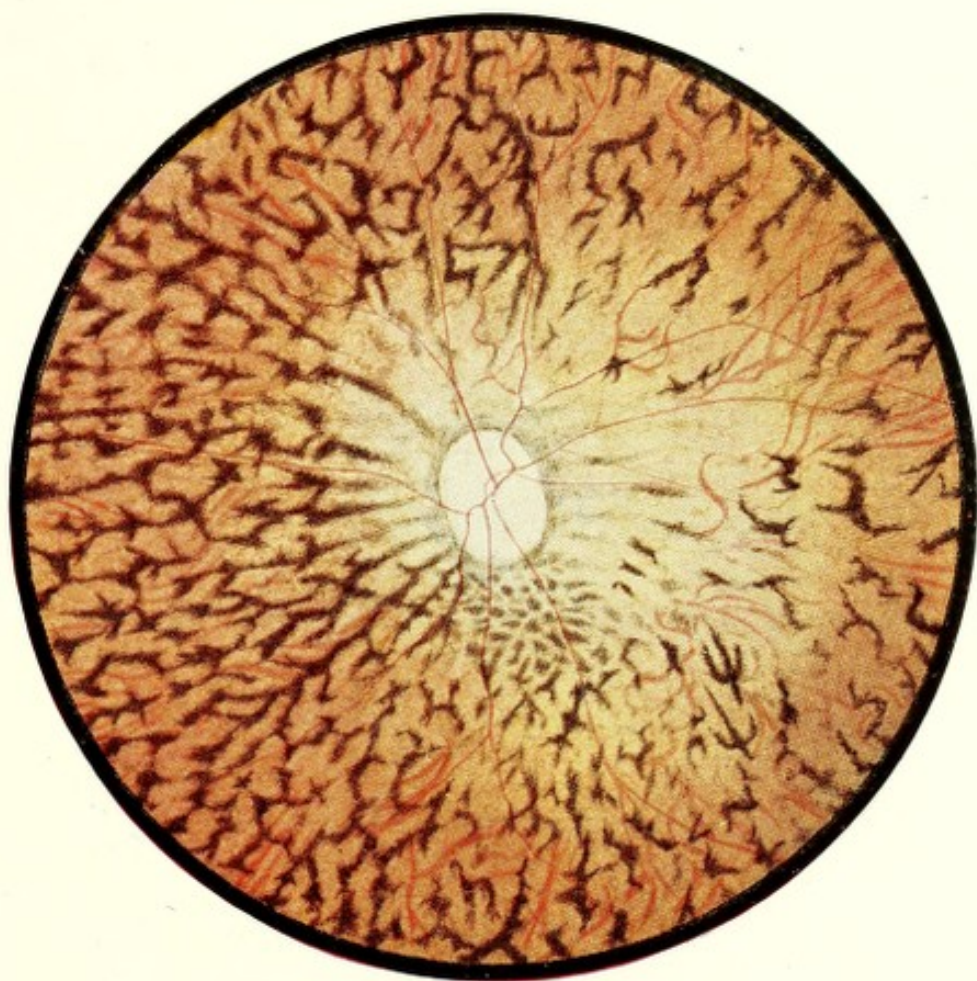


FIG. 11.







of the papilla are of a **congenital** nature, and, for the most part, are bilateral and symmetric. The acquired variations that are of semiologic value are far less numerous. It is not always easy, however, to differentiate the congenital from the acquired. **Obscuration of the border**, for instance, is sometimes physiologic, as in certain young hyperopes, and in the cases of **pseudo-papillitis**, to be described later. It is often only *apparent*, as in the ophthalmoscopic examination of high astigmatism. It is of great clinical importance to distinguish true acquired obscuration, which means an inflammatory process, from the false and seeming. All three latter are, as a rule, bilateral. Astigmatism can be at once excluded by looking first by one ophthalmoscopic method and then by the other. By the indirect method the long axis of the papillary ellipse will lie in the opposite direction to that it assumes in the direct, and the blur of its elongated borders, and that of the vessels running in a direction parallel with these borders is not at all, or only slightly, apparent. By varying the focus, in the direct method, blurred features may be rendered distinct, and *vice versa*. Besides, the amblyopia which might have been thought due to a pathologic condition, is relieved by lenses that correct the astigmatism. In beginning papillitis the obscuration usually affects only one side of the disc, most often the nasal. The adjacent cortical portion of the nerve-head broadens toward the centre, narrowing the physiologic excavation, becomes redder than normal, and running to it are tiny, tortuous vessels that were capillaries and are now made visible by their engorgement. Not infrequently a whitish infiltration of the parts, or a small patch of exudation, mat, zinc-white in appearance, or a minute hemorrhage along the course of one of the enlarged capillaries, is noted thus early. The advent of these phenomena at this stage is especially characteristic of **albuminuric neuro-retinitis**.

Rarely is the involvement of the papilla partial, save when it is the result of an extension of an affection of the retina, or retina and choroid. All the details of the optic



disc are obliterated, the place it occupied is of a uniform tint with the rest of the fundus, and can be located only by noting where the main stems of the retinal vessels centre and disappear. The vessels themselves all become more or less sinuous; the veins more so than the arteries; the veins are also larger than normal, and their natural crimson has given way to a veiled purplish tint. The width of the arteries is decreased, and their red has an extra admixture of orange.

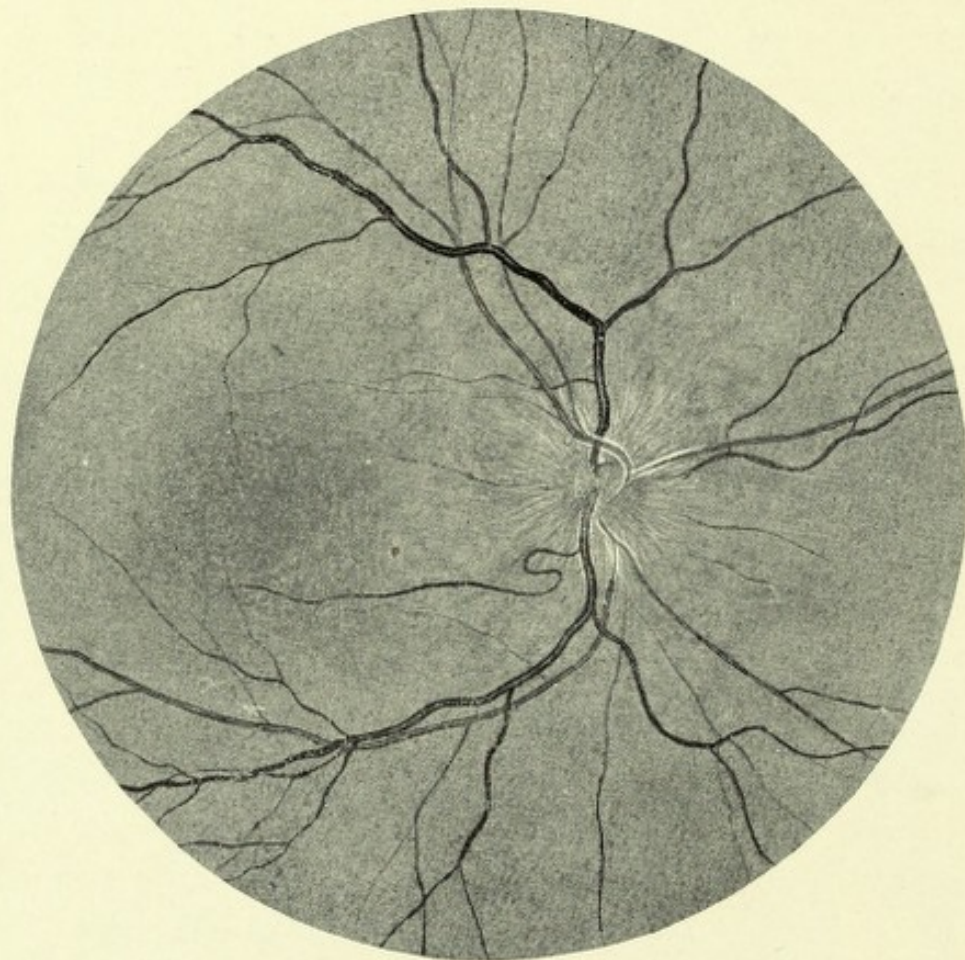


FIG. 12.—Papillitis.

The as yet normal, and ordinarily invisible walls of the vessel-trunks now show plainly as white streaks, because of the even background of color that is present. The papillary and peripapillary regions present a curious, dusky, opalescence that contrasts with the unchanged parts around. There is moderate edema and swelling of the papilla, which may extend to the macular region, and show



as a very faint opacity in which can be traced the extremely delicate gray striation which marks the distribution of the macular bundle of optic nerve-fibres. The foveola appears as a roundish spot of deeper pink, proving that only the nerve-fibre layer is interested. This is **papillitis** (Fig. 12).

**Choked Disc.**—We will now assume that no other signs of disease are to be seen in the fundus. The diagnosis of beginning papillitis is clear if, in addition, there are present signs of acute retinitis or chorio-retinitis. Often the hyperemia extends to the entire papilla, the physiologic cup is

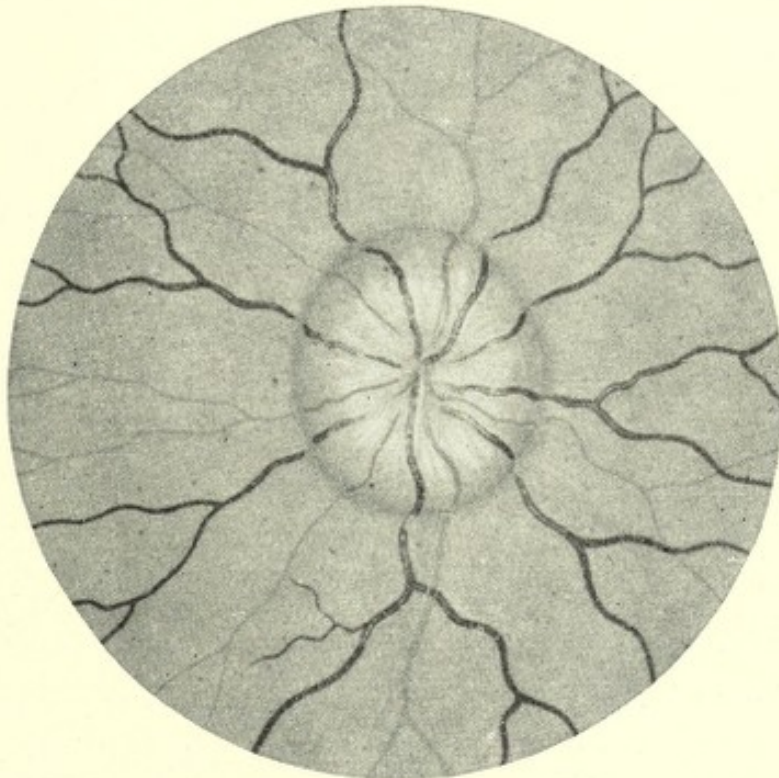


FIG. 13.—Choked disc.

obliterated, the borders of the disc disappear—both as to its scleral and its pigment rings—and the papilla from having been about on a level with the rest of the fundus, becomes devoid of all signs of its normal transparency and is a solid mound (Fig. 13, after Haab). This is evinced by the disposition of the trunks of the retinal vessels, and may be verified by ophthalmoscopic measurements. The swollen condition of the papilla is also made obvious by fine, grayish-white, radiating striæ, that are usually present, covering the



papilla and extending for a short distance from it. These are the optic nerve-fibres rendered less transparent and more refracting than normal. Both the striæ and the trunks of the retinal vessels that emerge at the centre of the papilla are seen to *bend away from the observer*. The veins, as they course over the papilla, seem somewhat turgescient and of darker color than normal, and the arteries of slightly reduced calibre and lighter, but a short distance away they may regain their wonted appearance. If there is a seeming break in the continuity of the vessels as they pass from the base of the declivity onto the surrounding eye-ground, it indicates that the tumefaction of the papilla is exceptionally precipitous and circumscribed. With the pupil widely dilated, and by moving as far as possible to one side, one can succeed in looking at the side of the swelling, and see the vessels disappear over the opposite side somewhat after the manner in which they are lost to view over the rim of a glaucomatous excavation. Still there are no other ophthalmoscopic evidences of an inflammatory process in the papilla, and *none* of such a process in the retina.

The foregoing constitutes a partial description of what is commonly termed **choked disc** or **papilledema**, called **Stauungspapille** (of von Graefe) by the Germans and **papille étranglée** by the French. It is in reality a form of papillitis characterized by great papillary stasis and edema. It has been proposed to call it by the more appropriate name of **edematous papillitis**. To continue the description: The hyperemia has not increased; on the contrary, the color of the nerve-head instead of being redder, is now grayish, as if the mass were lardaceous. If the case is an extreme one the height of the mound may be as great as 3 mm.—*i.e.*, about nine dioptries by the ophthalmoscope. Every movement of the observer's head causes paralactic movement of the vessels at the summit of the mound, and the light from the mirror can be made to fall from different directions, thus producing a play of the shadow of the elevation, and accentuating its relief. The button-like eminence, in-



stead of being regularly convex, has a dimple at the centre, marking the site of the **porous opticus**, or **physiologic cup**. The diameter of the papilla may have become twice or three times that of the normal. In most instances the other eye is similarly affected. This is choked disc pure and simple, and it announces in no uncertain terms that there is cerebral compression.

Seldom is the average clinician permitted to see this typical condition. It is an ideal, as it were, and to hope to find it in every detail as just described is seldom realizable. The same is true, in great measure, of the other form of papillitis mentioned. These cases come under observation in all stages, and with all manner of variations in the ophthalmoscopic picture. One is more likely to find additional or atypical features—chiefly, retinal hemorrhages, white exudations and areas of degeneration, or a number of the typical signs may be lacking. Again, the presentation may be as much, or more, that of the stage of atrophy or **degeneration** as that of inflammation or **progression**. Or there is an accompanying involvement of the median layers of the retina—for it is well known that neuro-retinitis or choked disc caused by compression from intra-cranial syphilis may readily be complicated by **albuminuric retinitis**.

As before stated, choked disc is most always bilateral, though seldom developed alike in both eyes. A certain localizing value has been ascribed to the condition when occurring first in one eye, as indicating that the neoplasm was on the corresponding side. Bilaterality is more strictly the rule if brain-tumor is the cause—less so if due to syphilis of the brain. True, simple, inflammatory or infectious papillitis is often monolateral. When caused by a lesion of the orbit, as from traumatism, septic cellulitis, syphilitic osteo-periostitis, extension to the orbit of disease of the accessory sinuses, of tumors and hemorrhages, it is usually monolateral. When the cause is intra-cranial, as from meningitis, septic phlebitis of the sinuses, it is bilateral; if from a general infectious disease, most frequently mono-



lateral. When from an intra-ocular affection, it is usually only one eye; when from intoxications, both eyes, and when from general vascular disease, about equally divided between monolateral and bilateral.

"Choked disc, when present, is doubtless one of the most valuable signs of tumor, but it must constantly be borne in mind that it is absent in all cases at an early period, in most cases until a late period, and that many tumors have a long life. The discussion as to the percentage of cases in which choked disc occurs seems therefore to be beside the mark; for, in measure as the diagnosis is made earlier, doubtless it will be absent in an increasingly greater proportion. Tumors allowed, uninterrupted, to run their full course will all probably show a certain stage of choked disc before the end" (Cushing). According to rather extensive statistics, in 90 per cent. of cases of choked disc, as found clinically, a tumor is responsible for the compression, and at least 80 per cent. of brain-tumors produce choked disc. Its advent is generally supposed to be earlier with the tumors involving the cerebellum than with those of the cerebrum. This may be because it has so often been the appearance of the choked disc alone that decided the diagnosis, even though that phenomenon had been long delayed. If the papilledema is of an intensity that is out of proportion to the other symptoms, it is suggestive of an intra-cerebellar growth.

**The degree of visual acuity** that accompanies choked disc is most variable. The sight often remains good even into the advanced stages; or it may be profoundly affected almost with the first signs of the changes in the papilla. In these cases, normal central visual acuity may be associated with great narrowing of the peripheral fields. Rapid and early loss of sight calls urgently for palliative measures. (*Decompressive surgery.*)

Bright curved lines, concentric with the papilla, are occasionally seen about the peripapillary region in conjunction with severe papillitis, and indicate thin folds of **retinal detachment**.



**Pseudo-choked Disc.**—In recent years, a number of cases have been recorded of an anomalous physiologic condition which resembles choked disc to an extraordinary degree. So strong has been the resemblance in some instances that the diagnosis has, for a time, been left in abeyance. Fig. 14 is a drawing of one of these cases published by the writer about twelve years ago. The chief points of differentiation are that there is not the lardaceous enlargement of the

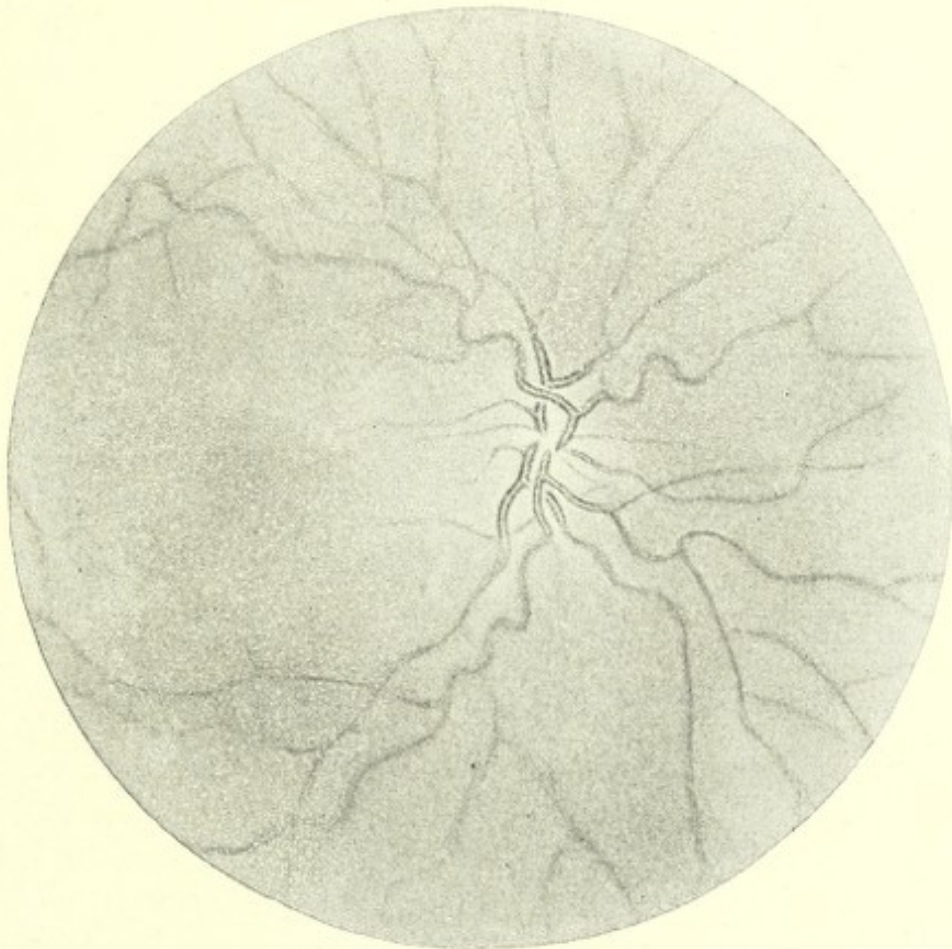


FIG. 14.—Pseudo-choked disc.

papilla; that, aside from their tortuosity, there is absolutely no change in the appearance of the trunks of the retinal veins; that there is no enlargement of Mariotte's blind spot, which is characteristic of choked disc; the visibility of the optic nerve-fibres is not enhanced; and there is absence of any associated symptoms, such as intermittent severe headaches, sudden vomiting, vertigo, cramps, etc., though



enough of these may be present merely to involve the diagnosis. The hyperopia at the summit of the papilla in the case from which this drawing was made was thirteen dioptries, while that of the macula was only four. Vision with the correcting glass was subnormal. There was frequent severe headache and vertigo. These were relieved by glasses that corrected the hyperopia. It may be here remarked that many patients with choked disc do not consult the physician because of visual troubles, but to obtain relief from violent headache and other symptoms. The patient alluded to, a female, has been under observation for more than twenty years and the papillæ remain unchanged. The vision now, with the same glasses, is more nearly normal.

Loring and Graefe attribute the indistinctness of the borders of the disc in these cases to a superabundance of connective tissue at the papilla. With this view Uthoff virtually agrees, his explanation being that it is due to a want of diaphaneity in the nerve-fibre layer.

**Crescents, or Cones, and Colobomas in the Papillary Region. Congenital Crescents.**—Congenital defects in the formation and the arrangement of the tunics of the globe at and around the papilla are exceedingly common, but they are usually inconsiderable and unimportant. From a semiologic point of view, however, their aspect can be invested with a significance of great moment, as we shall attempt to show in the course of this chapter. The eyes in which ophthalmoscopic inspection does not reveal some such irregularity in this vicinity are vastly in the minority. They usually appear as a small crescentic spot in the choroid at the border of the disc, which looks as if the greater part of the pigment ring had pulled slightly away from the scleral ring, leaving a thin stratum of the choroid still covering the sclera in the gap and a mere remnant of the pigment ring at its original site (Fig. 15). Their location is most often either outward or inward—*i.e.*, temporally or nasally—less often downward, very rarely upward. They vary greatly in dimensions, by far the greater number being



of insignificant width, yet few of them extend around less than one-fourth to one-third of the circumference of the disc. The larger ones are situated outward and slightly downward or downward, but as a rule they maintain their crescenting outline. Occasionally one is found at the temporal side of the papilla that is larger and of semilunar shape, but this is likely to be one that has been stretched since birth. More exceptionally they are annular—entirely surrounding the

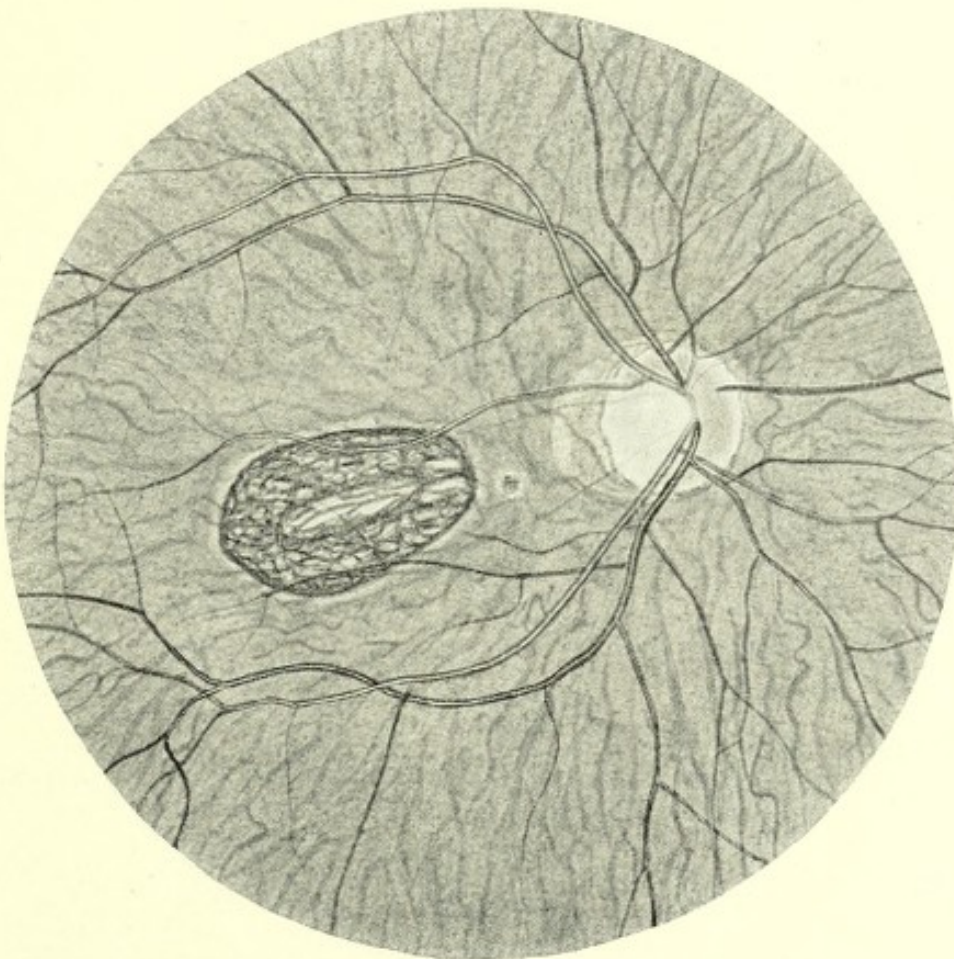


FIG. 15.—Cleft of nerve-head, and conus outward. Central coloboma of choroid.

disc (Fig. 16). The color of the space intervening between the convex and the concave borders of the smaller crescents is a uniform brownish-yellow, like ochre or chamois. In proportion as they are larger and the enclosed area is greater, they are characterized by peculiar vague details. Faint checkerings of pigment with interspaces of the tawny yellow, and meandering stripes of pale red occupy the



space—never any white, and always veiled, as if behind a layer of thick tracing paper, thus presenting a decided contrast by its lack of color to the brighter, more brilliant choroid on the one side, and by its somewhat more sombre tone, to the color of the disc upon the other. In brief, these crescents are but attenuated, or partially developed bits of the choroid, the dark mottling is pigment, the tawny, thinner pigment and capillaries, and the red streaks are

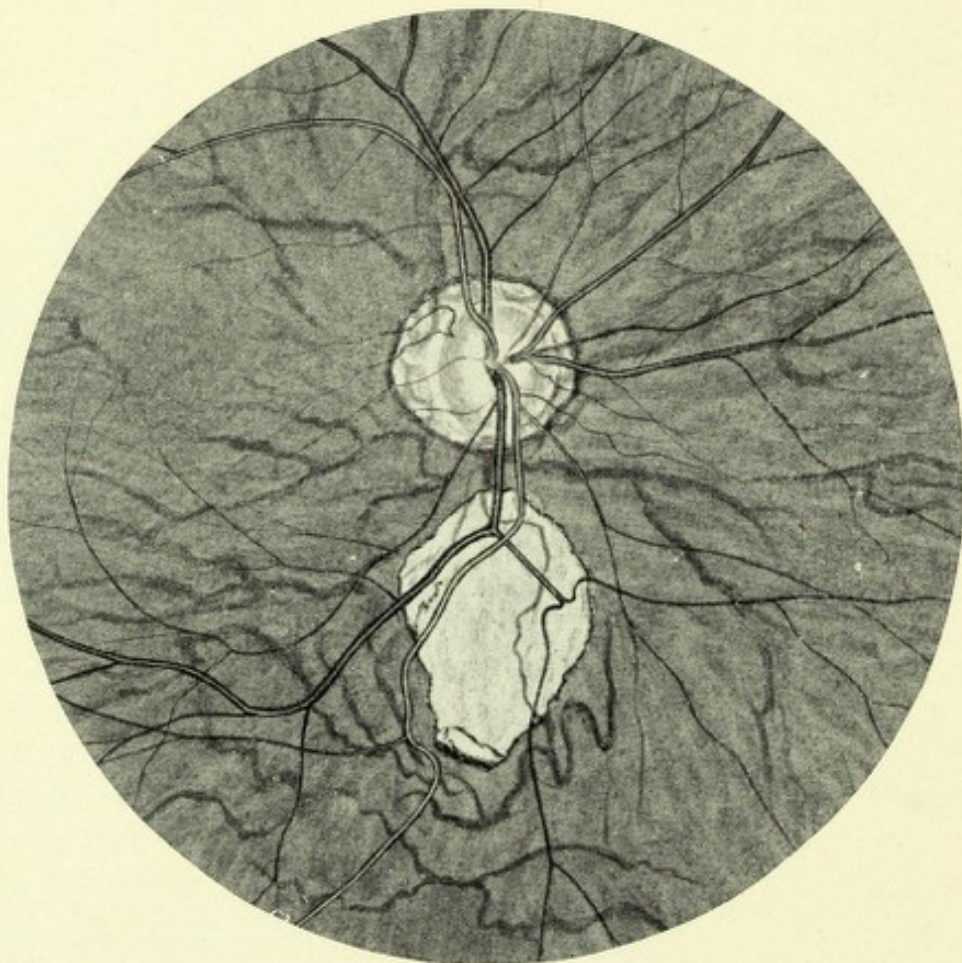


FIG. 16.—Annular conus. Small coloboma of choroid downward.

blood-vessels—all abnormally thin, and made to appear more so by a thick covering of retinal membrane. These are **congenital choroidal crescents**, also called **cones** (**conus**, plural **coni**).

Most of the discussion relative to the origin and nature of the congenital crescent has referred to those situated beneath the papilla, though the same applies whatever the location.



Liebreich (1859) thought that this conus was identical with posterior staphyloma. Jaeger, in 1861, pronounced it an anomaly in the closure of the fetal cleft. Schnabel, in 1874, made a distinction between congenital and acquired conus, and sclerectasia posterior. He noted that the congenital crescent was much oftener at the inferior border of the papilla than was the acquired form. This doubtless had led to the wide-spread belief that congenital crescent is habitually in that location. According to Schnabel, congenital conus is **partial coloboma of the choroid**. When a congenital defect of the choroid occurs in any direction and shows **bare sclera** it is a **complete coloboma** of the choroid, unless, perchance, it be one that is changed by subsequent ectasia. Fuchs and Szili admit that inferior conus is analogous to choroidal coloboma. Schnabel, the first to examine anatomically such a crescent—situation infero-external—demonstrated the absence of the internal layers of the choroid, together with the pigment epithelium. The retina he found doubled upon itself over the area, forming a fold whose free border rested against the optic nerve, and whose inner leaf was joined normally with the optic fibres. This folding gives three thick layers of retina overlying the defect instead of the one normally present, and, together with the thinness of the choroid in the defect, explains the veiling of the details in the crescent.

Elschnig makes a distinction between cone and coloboma, in that the cone is on the same level with its surroundings while the coloboma is ectasic, *i.e.*, bulging backward. It is not always possible to make this distinction by the ophthalmoscope. Elschnig's researches concerning **temporal conus at the papilla** have shown that in many cases of high and hereditary myopia, accompanied by ophthalmoscopic modifications of this region, that the ectasic crescent here answers precisely to a true coloboma.

**Acquired or Myopic Crescents.**—What has just been said relative to congenital crescents was mainly because of the bearing it has upon the differentiation between the **progres-**



sive and the **stationary** crescent. Through heed, on the part of adviser and patient, of the warning given by the fundus signs, doubtless many of the evils consequent upon that insidious pathologic process in the tunics of the globe, by which they become attenuated and stretched, might be prevented or relieved. Chief among these evils is **progressive or malignant myopia**, with its train of distressing visual phenomena, among which are disorganization of the vitreous, detachment of the retina, luxation of the lens, cataract, and glaucoma.

To qualify acquired crescent as myopic is, in a sense, misleading and inappropriate, inasmuch as the presence of this form of crescent is not necessarily a sign of myopia. It is, however, a sign of **abnormal elongation of the antero-posterior axis of the globe**. We know that the eye at birth is characterized by hyperopia and that through enlargement, in the natural course of growth, it becomes less hyperopic, emmetropic or slightly myopic, in accordance with the degree of original hyperopia. This means increase in the length of the antero-posterior axis **as a whole**, without elongation, or **physiologic**. Elongation of the globe is the result of changes—chiefly inflammatory and degenerative—in the posterior two-thirds of the globe, by which the sclera is thinned and drawn out—particularly around the posterior pole. The posterior two-thirds of the antero-posterior axis only—if one may be allowed the expression—are elongated.

**Pathologic lengthening of the antero-posterior axis** is the same process, whether seen in a hyperopic, an emmetropic or a myopic eye. Elongation wherein the axial increase is somewhat in excess of the normal often occurs without ophthalmoscopic signs, and without reference to any morbid conditions. When it is sufficient, however, to cause myopia of more than three dioptries if there are no visible signs of changes in the papillary region it is a source of surprise, or one suspects spastic myopia. The most conspicuous and distinctive of these signs is the crescent of lighter tint than either disc or choroid and situated at the temporal side, its



concave edge joined to a segment of the circumference of the papilla, its convex edge turned toward the macula. The dimensions of the crescent increase in the same ratio as the elongation of the globe, or as the activity of the changes which are responsible for its existence. Sometimes instead of being a crescent it is, primarily, or through extension, a ring around the papilla. In rare instances the defect sends out wing-like projections. As an instance of how relatively

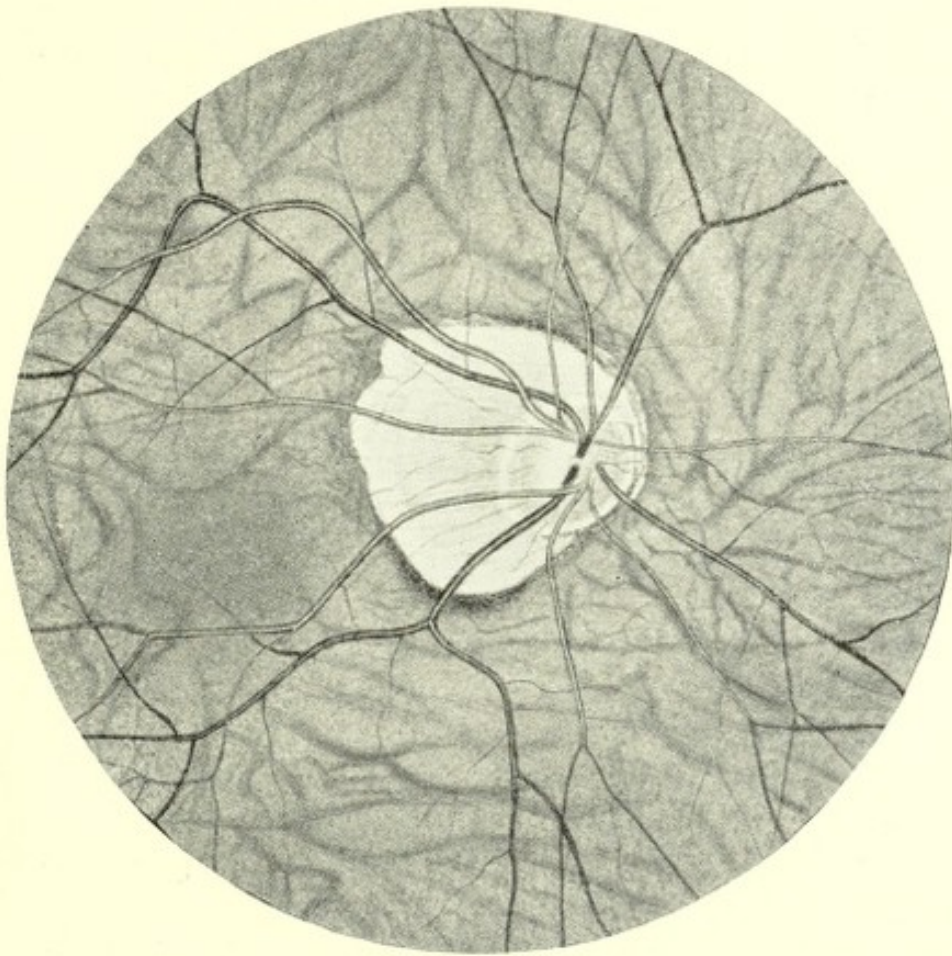


FIG. 17.—Acquired, or myopic crescent.

often myopic crescent is acquired, it will suffice to cite the fact that it is found more than four times as frequently after the age of twenty as it is before. It is doubtless present at birth in exceptional cases, but it is especially peculiar to acquired myopia. True congenital crescent, as we have seen, exists in most eyes, whatever the state of the refraction, and of itself means nothing.



**Acquired crescent**, while occasionally seen in conjunction with hyperopia or emmetropia, *is always indicative of elongation of the antero-posterior axis of the globe*, and this usually signifies myopia. The myopic crescent differs from the congenital in being whiter, that is, in reflecting more light. The sclera is more thoroughly denuded. The area of the crescent slants away from the observer, and the disc is turned in the same direction, making it appear as an

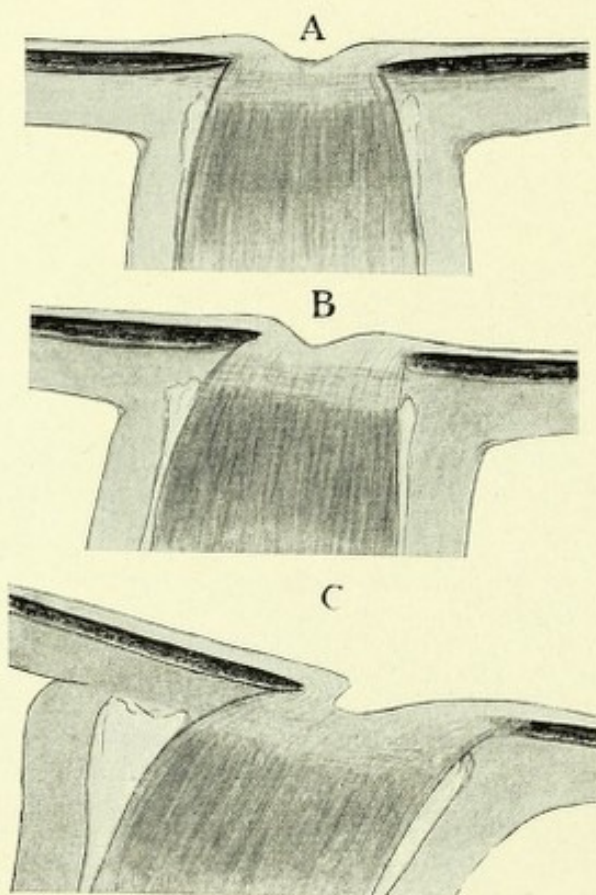


FIG. 18.—Horizontal sections. Right eye. A, Normal; B, beginning distortion of nerve-head; C, more advanced stage of distortion.

ellipse—or foreshortened. It has not the soft, vague, velvety detail of the congenital kind—is rather devoid of special detail of any description (Fig. 17). In the larger, semilunar, fan-like or wing-like or annular varieties, a straggling choroidal vessel or two may cross the area. It is now known that the congenital conus or crescent, especially when situated at the temporal side of the papilla, is often converted into the myopic variety. It is predisposed,



by its physical properties precisely to the conversion, constituting, as it does, a weak spot in that location.

The enlargement of the crescent is accomplished partly through mechanical drawing away of the choroid consequent upon the stretching backward of the sclera, and partly through thinning and atrophy supervening from the sclero-choroiditis. The retina usually remains over the crescent, as evinced by its blood-vessels crossing the area. Enlargement of Mariotte's blind spot, however, indicates lack of function of the retina at this point. Another trait that gives individuality to the myopic crescent as seen with the ophthalmoscope was first cited by Jaeger. He said, in reference to it: "It looks as if some force were attempting to slide the choroidal (pigment) ring over the disc toward the macula." And that is precisely the situation. (Fig. 18, B and C.) By the creeping backward of the choroid the concavity of the pigment ring is crowded upon and piled up against the nasal border of the papilla; at the same time its temporal concavity is pulled further and further away from the papilla. To put it differently, the nasal portion of the choroidal aperture encroaches upon the scleral aperture while the temporal portion withdraws. So strong is this push upon the part of the retina and choroid that the sharp, overhanging edge, or spur, of the scleral opening at the inner side is shoved into the lamina cribrosa, thus becoming more shelving, causing the nerve-fibre bundles to fold upon themselves, as it were, or to drop under a shelf, and favoring the enlargement of the cul-de-sac of the intervaginal space of that side. This distortion is made apparent ophthalmoscopically by the manner in which the trunks of the retinal vessels curve and disappear over the displaced rosy zone of the papilla into the narrow physiologic excavation—**real** distortion as distinguished from the **apparent** distortion from fore-shortening.

**Evidences of Progression as seen in the Myopic Crescent.**—The majority of myopes have the good fortune to have no further increase in their refraction after the age of twenty



years, and their eyes remain healthy. The myopia inclines to the malignant type only when there is a smouldering, lingering inflammation which involves all the tunics of the posterior portion of the globe and the papilla. An old, quiescent myopic crescent presents a uniform glistening white or faintly yellowish surface, bounded temporally by a deep, ragged outline of pigment. There may be an irregular splotch or two of black pigment within its boundaries, or, as before stated, a few attenuated choroidal veins. If, on the other hand, the surface (or concavity) of the crescent is of a bluish or slaty hue, especially if the tint is crossed horizontally by the fine striæ of the optic nerve-fibres, there is activity in the disease. The same is true when, instead of a uniform white area, or one with a splotch or two of pigment within its confines, one sees there a few rows or dots or broken stripes of pigment.

Other signs of activity and progression are to be observed in the papilla. Its tint, unlike the pink or roseate one of health, is dusky and indefinite, as if a thin coating of opaque neutral-tint or slate-color had been painted over both the disc and its borders. This bespeaks mild and chronic congestion, and is identified with the evolution of the myopia and likely to be conspicuous in proportion to the rapidity of the process. Sometimes the signs appear before the myopic crescent, or any distortion of the nerve-head. They are then to be regarded as evidences of **beginning myopia**. As a result of this slow optic neuritis many myopes of advanced years exhibit a blanching of the optic discs, particularly of the temporal halves. Occasionally the retina is involved, its veins become darker and more undulating and, if the subject be young, the surface of that membrane loses its peculiar lustre, as of watered silk, and the fundus assumes an appearance like that of middle life. The peripheral light-sense is diminished in these neural complications.

Still another sign of the developmental stage of myopia is the over-riding and piling up of the pigment ring at the nasal side of the papilla, referred to elsewhere. After the



elongation of the globe ceases the pigment here undergoes rarification and its appearance is no longer pathognomonic.

**False Crescents.**—There are other crescents that seem by their solid whiteness and their topography, as well as by the otherwise perfectly normal and healthy condition of the eyes in which they are found, to be merely variations in the contour of the scleral ring (Fig. 19). Many times they seem to rise above the level of the adjacent eye-ground, and the

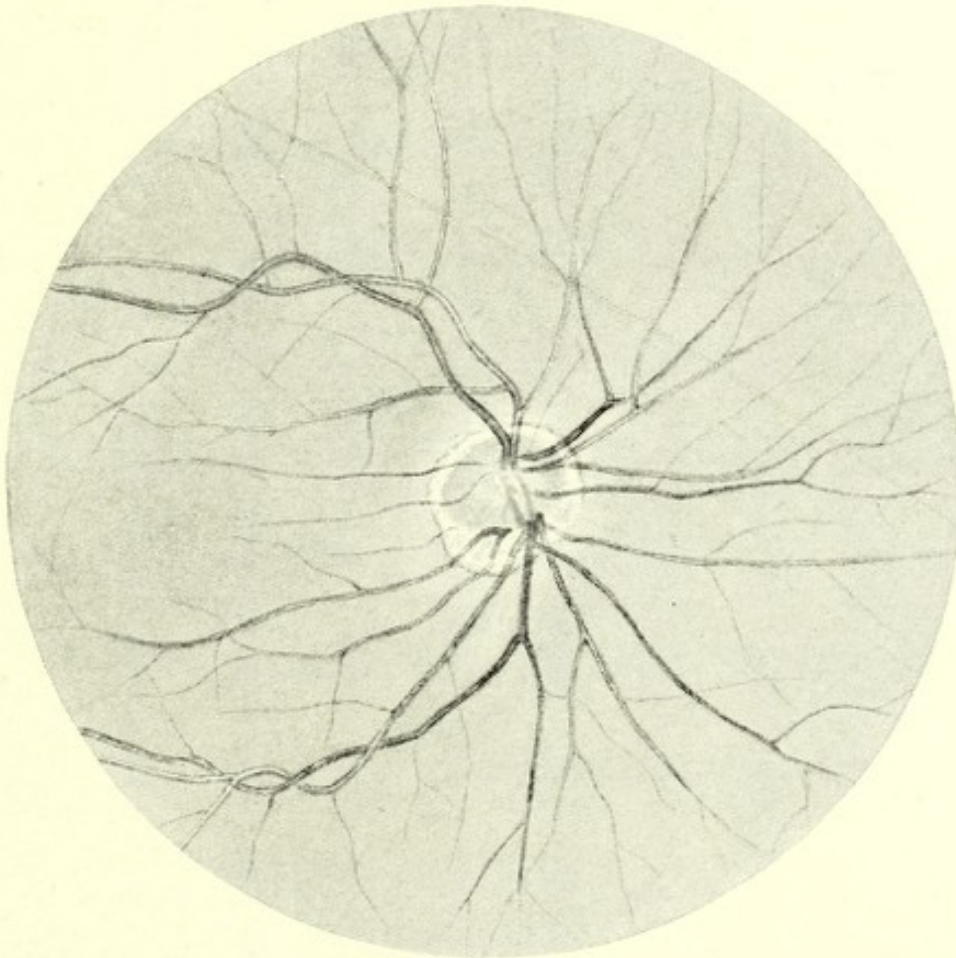


FIG. 19.—False crescent, or Fuchs' coloboma.

retinal vessels appear to rise in curves when they run over them. Still other featureless white crescents infringe on the papilla so as to cause flattening of its border, and, unlike those just alluded to, are on a plane further back than that of the surrounding fundus. These have been ascribed to imperforate areas of the sclera; that one here looks upon a crescentic area of sclera lying within the space allotted to the



papilla, yet containing no openings for the passage of the nerve-fibre bundles. These two varieties of crescent ought not to be difficult of differentiation. As a rule, all crescents are bilateral, yet there are many exceptions.

**Temporal Cleft of the Nerve-head.**—Under this name some years ago, the writer described certain anomalous appearances at the papilla which, up to that time, he had

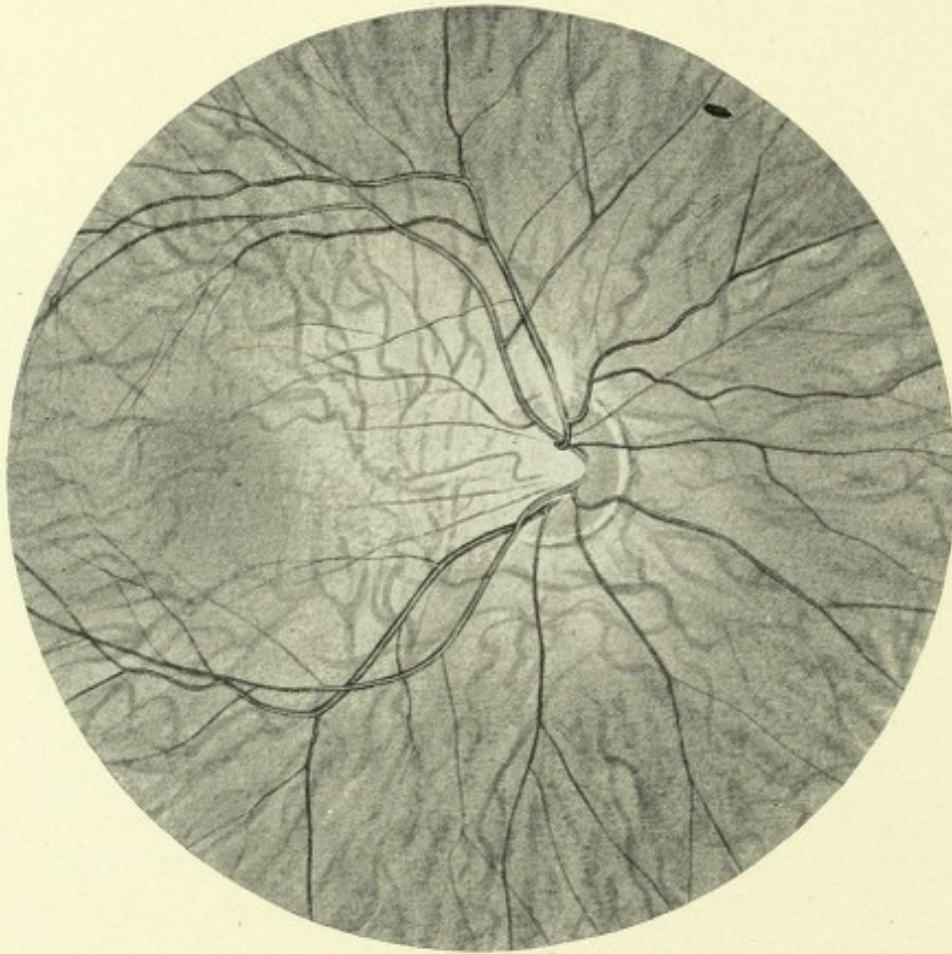


FIG. 20.—Temporal cleft of the nerve-head in which are seen two posterior ciliary arteries.

never seen any reference to by word or picture. The exact conditions figure in several of Frost's beautiful colored drawings, and are well reproduced in his atlas, published in 1905. Frost calls it "a large physiologic cup," and "physiologic cup opening outward." The pictures, in this instance, are not illustrative of the so-called physiologic cup, but of the congenital crescent, or conus, which accompanies it.



The paper that furnished the published article just referred to was read before the Ophthalmic Section of the American Medical Association at Atlantic City in 1905, and was illustrated by six or eight colored drawings shown by means of the opaque projector. Since then I have observed many more, and yet I have not seen any other reference to this particular anomaly. Fig. 20 is a reproduction of a drawing

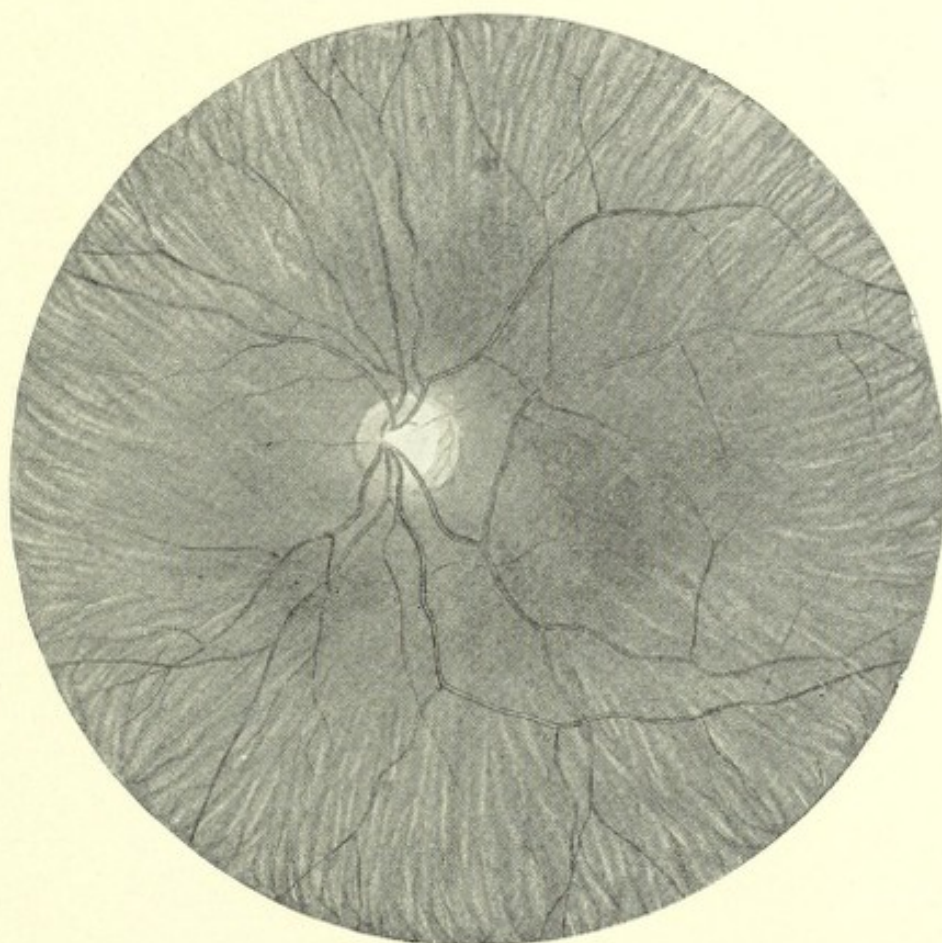


FIG. 21.—Outward (temporal) cleft of the nerve-head, and outward conus. Thin choroid.

depicting it. As nearly as the anatomic arrangement can be told by the ophthalmoscopic examination the defect seems to consist in absence infero-temporally of the scleral ring—or, rather, of the constricting spur of the scleral opening for the nerve-head, for an extent corresponding to one-third to two-fifths of the circumference. Its position is either straight outward, or outward and downward. In



one instance only have I observed it straight downward. I once thought that the lamina cribrosa might be lacking in the area of the cleft. This may be true in a certain number, yet it is no less true that its presence there is often obvious.

The nerve-fibres are all crowded into a thick rosy crescent at the nasal side, and all the rest of the papilla consists of a deep white excavation. In many instances the papilla seems to be turned outward as in posterior staphyloma, so

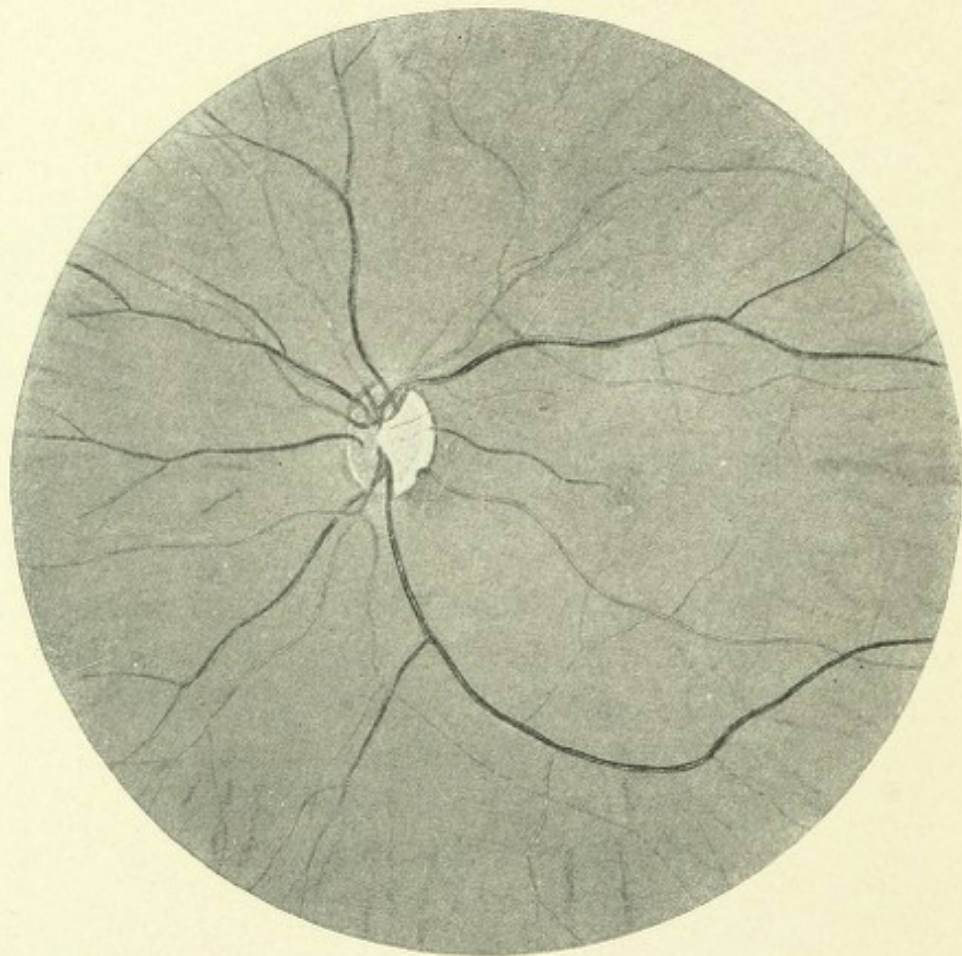


FIG. 22.—Outward cleft of the nerve-head without a conus. Large cilio-retinal artery.

that the retinal vessels disappear abruptly over the rosy crescent. There seems to be no optic nerve-fibres in the cleft, which appears to be deep in the nerve-head. Mapping of Mariotte's blind spot shows it elongated in the direction of the fovea, as if indicating that the axis cylinders in flowing around from the pink crescent above and below, failed to unite near the outer margin of the disc owing to their



anomalous arrangement in the lamina cribrosa. The foveola, in many instances, is situated abnormally low down in the fundus, as if there were torsion of the globe toward the temple. Associated with the condition is usually astigmatism—most often myopic—subnormal vision, thin choroids (Fig. 21), powder-grain distribution of the pigment epithelium, congenital conus in the same direction as the cleft (Fig. 22), and anomalies of the retinal blood-vessels.

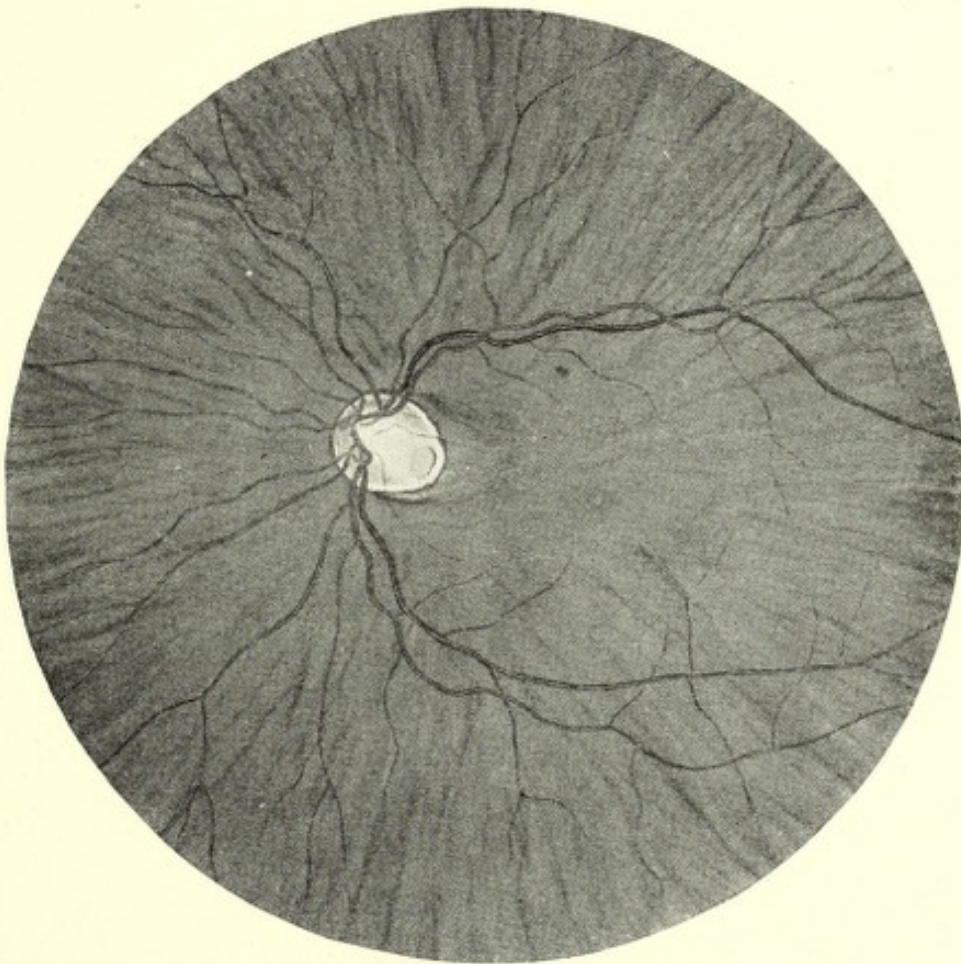


FIG. 23.—Coloboma of the papilla.

This anomaly, like the crescent, is usually bilateral.

**Coloboma of the Papilla.** (Fig. 23).—Coloboma of the papilla when not associated with coloboma of the choroid downward, is rare, the number of cases thus far reported probably not exceeding sixty. It is of comparatively little semiologic import, though it has been confounded with optic atrophy and with annular posterior staphyloma (progressive



myopia). According to VanDuyse, who is an authority on colobomas of the eye, to colobomas of the optic nerve, while their ophthalmoscopic images differ greatly as to minor detail, may be ascribed the following common characters:

*a.* Enlargement of the papillary area with irregularity of form.

*b.* Partial or total excavation, the greatest depth being in the lower part.

*c.* Glistening white surface alternating with shades of gray in the excavated portions.

*d.* Special disposition of the vessels.

The general outline is usually ovoid, and the area, always abnormally great, may be, according to von Hippel, twenty times that of the normal disc. Often the excavated portion is surrounded by what seem to be exaggerations of the pigment and scleral rings. When the whole area within the scleral ring is depressed the aspect closely resembles a magnified glaucomatous excavation. The depth of the depressions within these rings is subject to enormous differences, as can be roughly read in their shading and in the tint and density of the gloom with which they are invested. The shallower ones are white with bluish line or light shadow marking rim or concavity, while the deepest are of one even tint of blue-gray or dark slate. Not infrequently there can be seen, most often in the upper part, the rose color that indicates the presence of optic nerve-fibres. The cavities are cystoid deformities of the bulbar wall, or of that and the dural sheath of the optic nerve where it joins the sclera. They are single, or multiple, and differ in size and shape as they differ in their manner of reflecting or absorbing the light. Sometimes they are half as large as the globe—or even larger. The cavity of the cystoid expansion only shows, of course, when its mouth is turned toward the front. If the cyst is closed, or its aperture is turned away, the examiner will not be aware of its presence. Their situation in the papilla, like that of the cleft, is down-



ward, or downward and outward. The retinal vessels rise from the excavated portions of the coloboma—in a passably regular manner when the excavation is complete and not over-deep; that is, they emerge near the centre and pass over the scleral ring. In the irregular and cavernous kinds, the vessels appear first at the extreme periphery, or climb up the walls of the deeper cavities and come out of their mouths. Anatomic examinations have demonstrated that in many colobomas of the optic-nerve entrance, the central retinal vessels do not enter the substance of the nerve. The defect is, as a rule, bilateral.

**Opaque nerve-fibres constitute** an anomaly of the papillary region that has often been mistaken for white inflammatory exudation of the retina or of the choroid, or a dense infiltration of these tissues. This is not to be wondered at in an observer not habituated to the use of the ophthalmoscope, for the white often goes out from the retina like horns that follow the trunks of the retinal vessels, the general effect being not unlike that of thrombosis or embolism of the central artery or the earlier stages of syphilitic chorio-retinitis—or the connective-tissue proliferation of the degenerative period of the last named disease.

As is well known, the glistening, snowy-white of this anomaly is due to the fact that the axis-cylinders of the retina, throughout the affected space, are not naked, as is their natural state, but that each is enveloped in a myelin sheath, such as it always wears in its course along the optic nerve. It was formerly taught that these fibres simply failed to leave off their covering at the lamina cribrosa as normally. VanHippel and others, who have investigated the matter, assert that opaque nerve-fibres are never present at birth, but that they are acquired in infancy. In certain cases the ophthalmoscopic picture seems to uphold this assertion, for it shows a distinct break between the lamina cribrosa and the beginning of the plaque of white—making it certain that the myelin sheaths were not *carried on* from the optic nerve entrance but that they were *assumed* just



outside of the borders of the disc, the papilla itself being of perfectly normal appearance.

Minute tufts of myelin fibres are often observable at the upper and lower borders of the disc and attract no special attention. It is only when their width exceeds that of the aggregate of the vessel-stems at those points that they become conspicuous. Those of any considerable size are remarkably striking and distinctive. Their aspect is

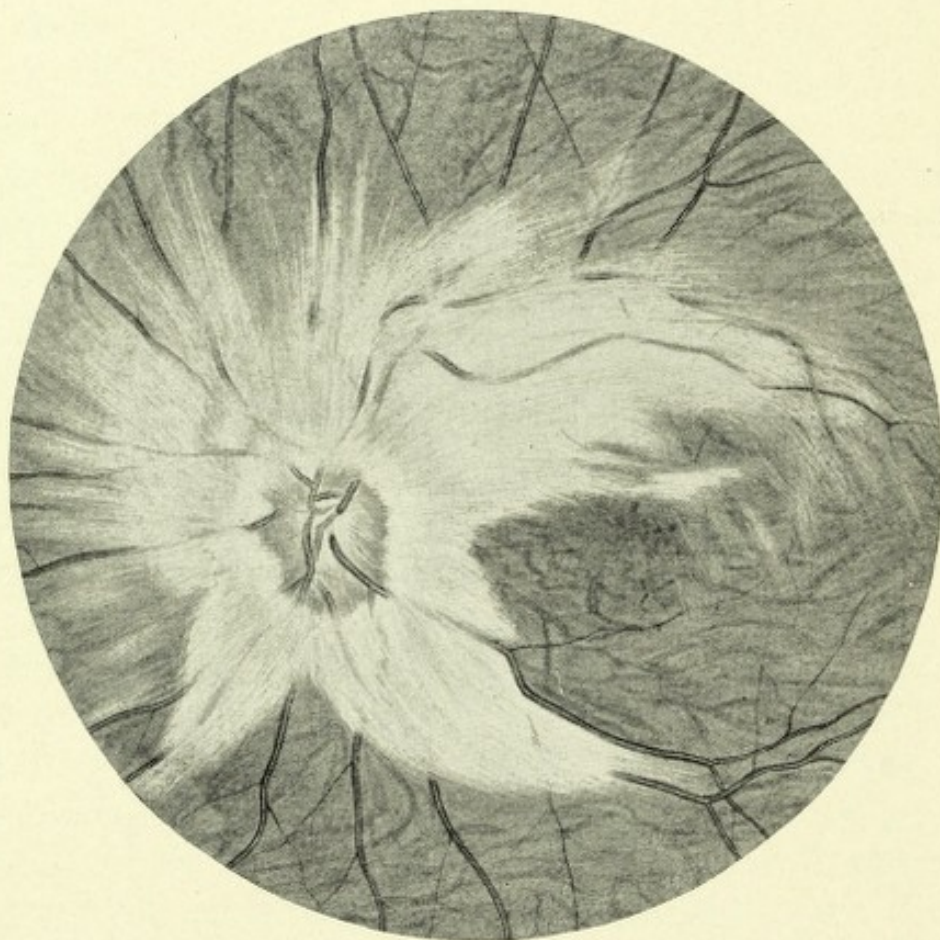


FIG. 24.—Opaque nerve fibres.

suggestive of a fairly thick layer of combed cotton, the fibres of which follow the direction of the retinal vessels. The white, however, is more brilliant than that of cotton—if one could liken it to "spun-snow," the simile would be more appropriate. The form of the plaque is like that of a flame of fire blown by a blast that comes from the centre of the papilla, and it has a flare like a burning gas-jet. The



larger projections of opaque nerve-fibres follow the four large pairs of vessels, sometimes far into the fundus, even into the macular area, and are so wide as to cover the entire peripapillary region. Where thickest, they entirely hide the vessels. The disc also is, at times, partially or completely concealed. When partially so, the visible portions look grayish red, partly through contrast and partly, perhaps, from crowding of the fibres and condensation of their color. Everywhere the fine striation indicates the course of the nerve-fibres, but it is at the outer extremities of the snowy patches that the combed effect is most pronounced. Fig. 24 shows an unusually extensive display of this phenomenon.

Myelin fibres of the retina are differentiated positively in the manner first described; or negatively, that is by the absence of all signs of inflammatory or degenerative processes. Blood-vessels and other structures in the midst of the phenomena are unchanged. The papilla is flat, and the choroidal (or retinal) pigment epithelium, right up to the edges of the white areas, is undisturbed. Such signs as pigment splotches, hemorrhages, new vessels, haze of media, etc., are not present. The blind spot is enlarged in the extreme cases, on account of the thick covering around the papilla; otherwise the vision is unaffected. Atrophy of the optic nerve and retina is said to cause the disappearance of opaque nerve-fibres. They are less often associated with other anomalies of the fundus than are most of these natal irregularities, though they have been observed in conjunction with remains of the hyaloid artery.

**Glaucomatous Halo at the Papilla.**—In well-established glaucoma, chronic or irritative, there is usually to be seen partially or wholly surrounding the papilla, a pale, yellowish-white band of choroidal atrophy. Its internal border coincides with the scleral ring, while its external border, sinuous and irregular, contrasts rather sharply with the color of the surrounding fundus. It varies in width in different parts of its circuit, being usually wider at the temporal than at



the nasal side. In some instances it may be mistaken for an extra broad scleral ring. The confusion is heightened if, as not infrequently happens, the inner edge of the frayed choroid simulates the normal pigment ring. Sometimes it is incomplete, the interruption occurring most often inward or upward. When this happens, the segment of the band, in one or the other of these situations, gradually tapers to points. It is seldom that the tint of the band is anything but a faint yellow, though this deepens slightly near the outer limits, but exceptionally a few traces of choroidal detail are found in it. The halo, or **glaucomatous girdle**, is rarely lacking in chronic idiopathic glaucoma. It differs very materially from the zone of lighter tinted, or faded, choroid, that surrounds the papilla in optic atrophy. The latter is not a clearly defined ring, but is a sort of indeterminate areola about the papilla, through which the choroid has a frayed, washed-out look, and which loses itself by insensible gradations in its surroundings. The glaucomatous girdle generally develops along with the excavation, beginning at the temporal side and extending both ways around. It has, however, been known to precede or to follow the formation of the cup (Fig. 25).

**Excavation or Cupping of the Papilla.**—Depressions in the papilla are of two kinds, **physiologic** and **symptomatic**, and the symptomatic excavations are divided into two classes, the **glaucomatous** and the **atrophic**. Upon one's ability to recognize the distinguishing features of these varieties often hinge questions of great moment. The glaucomatous excavation characterizes all forms of well-established glaucoma, that is, those in which the intra-ocular lesion has long been excessive, for it is of purely **mechanical** origin, while the physiologic excavation is due to **structural peculiarities**, and the atrophic to **metamorphosis**, in the tissues of the papilla.

**The Glaucomatous Excavation.**—In proportion as the intra-ocular tension is augmented, changes occur in the



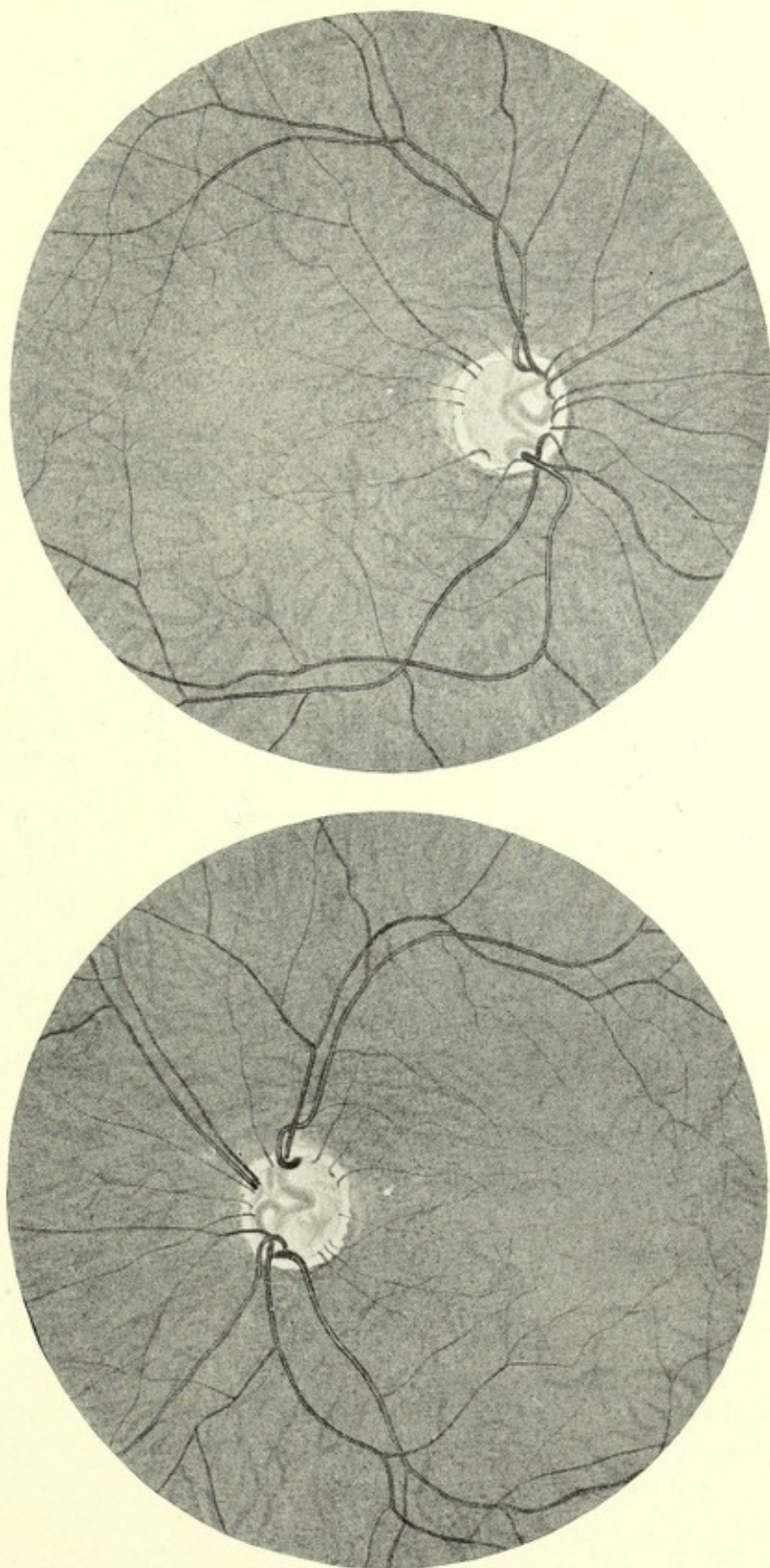


FIG. 25.—Bilateral glaucomatous excavation with surrounding halo.



level of the optic disc, which is the most yielding spot in the walls of the globe. First it is the softer portion, just external to the emergence of the retinal vessels, that gives way. At this time the ophthalmoscopic image of the excavation is not different from that of a physiologic cup. The differentiation must be made by other symptoms. Later, the outer segment of pink nerve-fibres is pushed out of view and the small vessels at that side disappear **immediately** over the

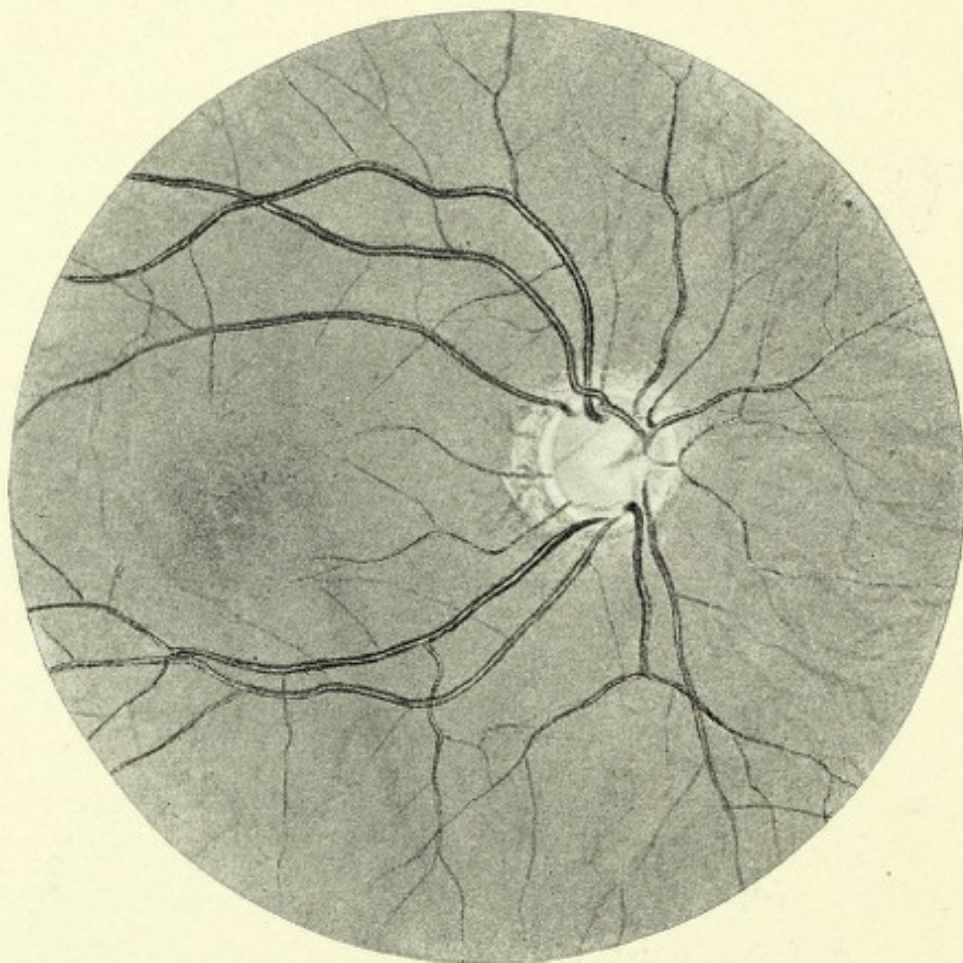


FIG. 26.—Glaucomatous excavation of moderate depth. Conus outward.

concave edge of the scleral ring (Fig. 26). It is at this time that the true nature of the excavation begins to show. The pressure is exerted by an ever-increasing button of the vitreous that is forced into the scleral aperture, driving the normal tissues before it and usurping their place. Next, the pink zone disappears in its upper and lower segments and, last to succumb is the inner portion, in which the nerve-



fibres are more abundant (the nerve-tissue is heavier) and which is further fortified by the great trunks of the vessels. Once past the constricting scleral ring, the button of vitreous expands in the ampulla of the optic nerve, assuming precisely the form of an old-fashioned iron pot or kettle (Fig. 27), and giving, of course, the same form to the excavation. The optic nerve-fibres are crowded outward in all directions against the walls or sheaths of the nerve, the lamina cribrosa is pushed backward and made concave, and the central vessels are all shoved to the nasal side. The picture is now about complete (Fig. 28). The explanation of this invariable disposition of the vessels is as follows: In the absence of a physiologic excavation the first sprouting, so to speak, of the pression button of vitreous is in the **porous opticus**, or little depression at the exact centre of the papilla. Now the retinal vessels emerge from the papilla somewhat to the nasal side of the centre, hence the entering wedge of vitreous forces them always to that side. Obviously, if a physiologic excavation already exists, the formation of a glaucomatous one is much facilitated. It is said that it is possible sometimes to see the remains of a former physiologic excavation appearing as a second cup at the bottom of the symptomatic kind.

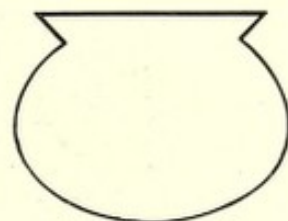


FIG. 27.—Pot, or kettle form of glaucomatous excavation of the papilla.

**Ophthalmoscopic Features.**—The glaucomatous excavation is best studied by the direct method and light of moderate intensity. The first thing that strikes one is the appearance of the branches of the central vessels as they emerge from the disc. While passing over the whitish band encircling the disc they seem to be slightly dilated, become darker in color, and lose their cylindric reflex as they near the inner edge of the band, over which they abruptly disappear. Those that approach the disc in a meridional line and disappear at a right angle, seem to terminate *straight*, not *flush* with the scleral ring, but just a trifle *full*; those that do not



follow meridians, or that veer to one side in disappearing look like red hooks, caught snugly on the inner edge of the scleral ring (see Figs. 25 and 28). The edge of the ring is marked all the way round by a fine line of dark Prussian blue. Within this line it is very evident there is a concavity, being evinced by the shading and by the lively parallax between objects on the border of the disc and those within it. The continuation of the blood-vessels is seen at the

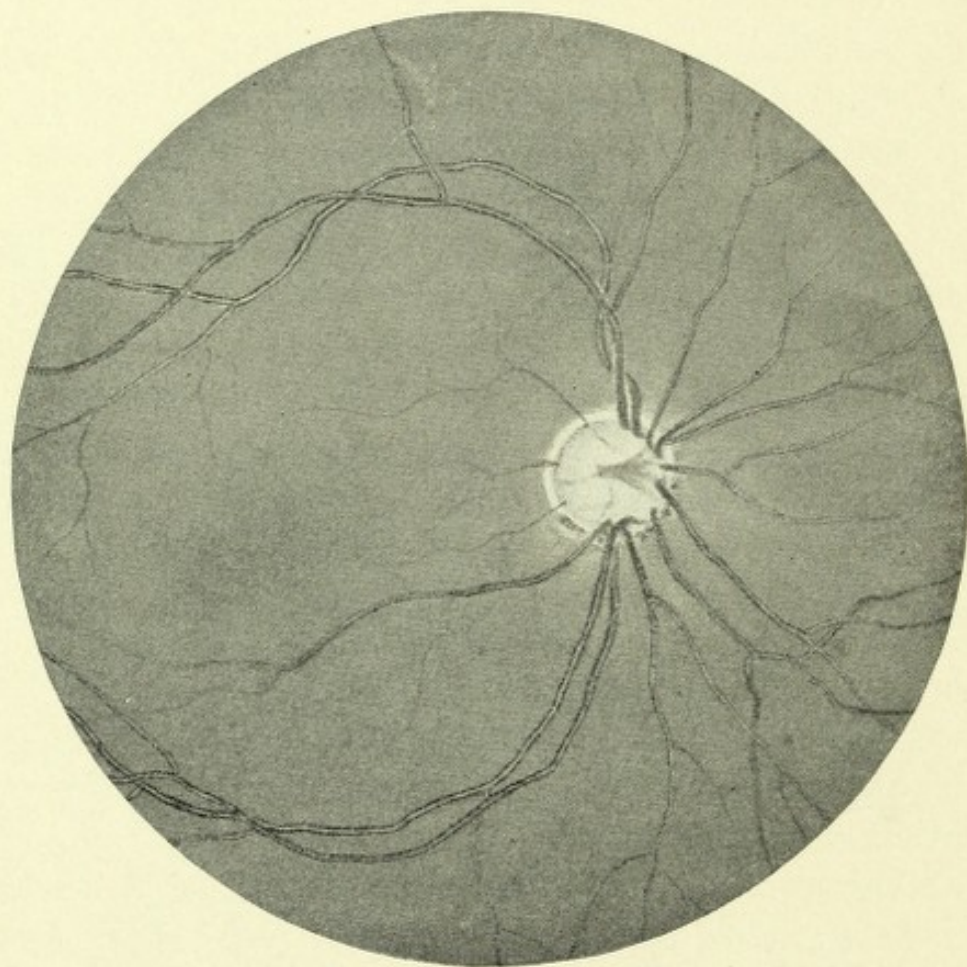


FIG. 28.—Glaucomatous excavation.

bottom of the concavity, but they are faint and fuzzy from being out of focus. They are seen to have their beginning in the inner portion of the concavity, to run a short distance toward the nose and pass from view. No continuity is discernible between those at the bottom of the cup or pot and those on the brink or rim. They have gone beneath the overhang in order to climb up the sides (arteries)—or *vice*



*versa*, they have gone under the overhang to climb down the sides (veins). The parallax and the shading are accentuated in proportion as the depth of the cup is great. The color of the interior, aside from that imparted by the vessels, is greenish-blue or blue-gray in the shadows, fading to white at the centre. Over the bottom, or floor, nearer the temporal side, are dark gray, irregular dots. These are openings in the outer wall of the globe for the entrance of the nerve-

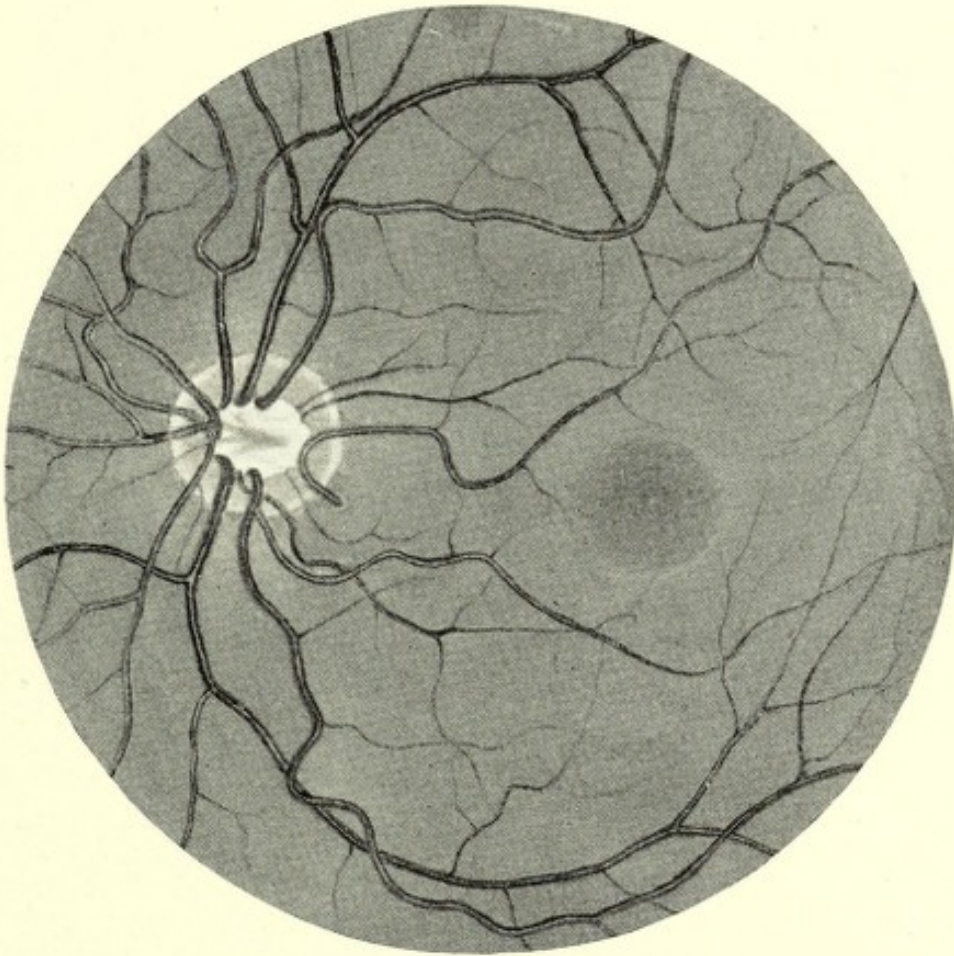


FIG. 29.—Physiologic excavation. Large cilioretinal artery.

fibres. According to von Arlt, we see deeply into these openings because the fibres are already transparent—because divested of their myelin sheaths. The color in the excavation does not depend so much upon the depth of the cupping as upon the stage or rapidity of the glaucomatous process. In the earlier period, green predominates, later blue, then gray and white; green and blue so long as there



is blood in the capillaries of the nerve-tissue of the papilla; gray and white in the stage of atrophy of this tissue. In this stage there is also diminution in the calibre of all the retinal vessels, and often tortuosity besides. The last quality is more characteristic of glaucoma secondary to active inflammatory disturbances.

**Points Distinguishing the Physiologic and the Atrophic Excavation from the Glaucomatous.**—In the physiologic form there is a complete ring of the rose-pink nerve-tissue surrounding the excavation (Fig. 29). It may be exceedingly narrow at the temporal side, but it is not broken. The only exception to this is seen in that anomalous condition described under the heading "temporal cleft of the nerve-head." During the first period in the development of the excavation of glaucoma more or less of the pink zone may be left standing, but in a comparatively short time it all disappears—*the whole disc becomes excavated* out to the scleral ring. It goes without saying that when a physiologic cup is associated with complete atrophy of the optic nerve there is no pink zone, and this point of distinction is not available. There is here a moderate "under-cut" of the nerve-fibre zone at the nasal side, often also above and below, so that the blood-vessels in these localities present the same features as in the glaucomatous excavation. The temporal segment of the pink zone is rarely overhanging, the sides of the excavation here incline outward—though it may be but slightly, and vessels passing over show no breaks in continuity. There is a healthy glow to the interior of the cavity—it is not "sicklied o'er with the pale cast of" green like that of active glaucoma, nor does it present the lifeless gray and white as do late stages of this disease. Of course, there must come a time, in the early period of the glaucomatous cup, when it so nearly resembles the physiologic as to make it necessary to rely entirely upon other symptoms in making the diagnosis. The physiologic cup never exceeds in depth the normal level of the lamina cribrosa, whereas in glaucoma the structures here are often pushed back so far as to make



the cavity of extraordinary depth. The physiologic cup hardly ever exceeds three dioptries, or 1 mm., while that of glaucoma may measure five or six dioptries—1 1/2 to 2 mm.

**The excavation of optic atrophy**, except when there had previously existed a physiologic cup, is a regular concavity—"saucer-shaped," as it is called, and never measures more than one and one-half to two dioptries at its deepest part—the centre. There is a slight shading or grayish-blue which, in the form of a crescent, keeps to the side opposite to that whence comes the light. As the atrophy is far advanced when the excavation is developed there is seldom any true blue, and never any green in the color of the disc. The significance of the excavation is wanting in atrophy *with* physiologic cupping. The many other signs of atrophy of the optic nerve and of glaucoma, both objective and subjective, ought positively to prevent any confusion, unless, as often happens, the case be one of atrophy and glaucoma combined. Physiologic cupping is, as a rule, bilateral, while the symptomatic is often unilateral.



## CHAPTER XIII.

### MODIFICATIONS AND ALTERATIONS REFERABLE TO THE BLOOD-VESSELS OF THE PAPILLA AND RETINA.

**Normal Appearances and Arrangement.**—The ophthalmoscopic view of the fundus is peculiar in the highest sense of the word, in that it constitutes a picture unlike anything else in the world. But there is one feature of the scene that is unique, not only because of singularity of aspect, but because of the exceptional opportunity it affords for study and observation. This is particularly true as regards the ramifications of the retinal vascular system. Here one looks virtually at the naked blood-vessels (see frontispiece).

**The retinal circulation** is derived from the central artery and vein of the retina, which are continuous with the ophthalmic artery and vein, a branch from each of which enters the optic nerve about 20 mm. or less behind the globe, and run forward close together near its centre, to emerge, ophthalmoscopically speaking, a little to the nasal side of the centre of the papilla. Somewhere in their passage through the lamina cribrosa these central vessels divide into two branches—more often than not this branching takes place before the vessels are visible in the papilla. One branch turns upward and the other downward, constituting the **superior and inferior retinal artery and vein**. What we first see of them on the disc is not the main trunk of each central artery or vein, but the first two branches of each, one pertaining to the upper half of the retina, the other to the lower. The arterial trunks, as a rule, keep to the inner or nasal side of those of the veins, and lie deeper, *i.e.*, nearer the centre of the globe. Hence, in crossing each other the artery is



most often on the side of the observer; though veins often cross arteries. Arteries, however, do not cross arteries nor do veins cross veins. The two kinds of vessels are easily distinguished by their appearances.

**The blood-column** of the arteries appears as three stripes, two of deep carmine with one of bright orange between them. That of the veins also appears as three stripes, two of crimson with a fine line of light salmon-pink between them. The three stripes composing the artery are truer, straighter and more regular than those of the vein. The carmine stripes are somewhat broader than the orange. The crimson stripes of the veins are slightly wider than are the carmine of the arteries, decidedly wider than the central or salmon one, and all three are less uniform than those of the artery. The central stripe in each instance is the cylindric reflex of the vessel, and that of the arteries is broader, more even, and characterized by more color than that of the veins because the blood-column outward bound is brighter and more transparent than in returning. That the veins are also translucent is shown when, in crossing, the vein lies on top of the artery. The orange reflex of the artery can then be plainly seen through the vein. When the artery is on top, its reflex is almost obliterated at that point by the dark body of the vein beneath.

The reflex follows the vessels into the extremely small branches, yet cannot be traced in those that are barely visible. Neither are these last distinguishable by difference in color, but they can be readily identified by tracing them back a short distance. The venous reflex is not only very narrow and irregular, but is broken, or missing, at many points. Over it, or along with it, especially in young subjects, there is the flicker and the glint of a still brighter, or pure white reflex, as if it came from silk or spun-glass, and is probably due to the presence of fine, knotted strands of connective tissue. A similar effect is apparent at intervals along the sides of all the larger arteries and veins, and is especially marked about their main stems in and near the



papilla. In this region the white knots or tufts are so large and dense as to conceal parts of the vessels. It is real tissue, and not a mere reflection, like the effect that is called "shot silk" and "watered silk." The arteries are smaller than the corresponding veins, and run a straighter course. In the papilla their red or crimson is gradually merged into the white at the centre unless they come out perpendicularly, or straight at the observer, as then their stems of origin are hidden. The arrangement and distribution of the retinal vessels follow no invariable rule.

The second division, that is, the branching of the superior and inferior retinal trunks, usually takes place on the disc, sometimes just beyond its borders, and from their location and direction the subdivisions are called **supero- and infero-temporal artery and vein** and **supero- and infero-nasal artery and vein**. The temporal divisions being destined for larger areas are of larger size than the nasal. Aside from this arrangement, which is far from constant, there is but little system or order in the manner of division. Lesions and other things are often located by such terms as "at, or near the first large branch of the supero-temporal artery or vein," "well out along the course of the lower nasal," etc. The four large temporal vessels, the largest of the retinal system, while running toward the **ora serrata**, two above and two below, make a wide detour around the posterior pole. The result is that not only the macula, but a wide area around it, is devoid of large vessels; at the same time it is highly vascular by reason of the smaller vessels which converge toward the macula from all directions. Moreover, the larger vessels, except in the vicinity of the papilla, instead of running close together in pairs, as is usual in membranes in other parts of the body, are seen to intercolate. A vein is placed in the middle of the space bounded by two diverging arteries. In consequence of these methods of distribution the most sensitive and important region of the retina is least deranged, and the interference of the shadows and reflexes of the trunks is reduced to the minimum.



The arteries of the retina are further individualized by being **terminal** or **end-vessels**, which means that, like certain of the cerebral arteries, they do not anastomose with each other except through the medium of their capillaries, and are incapable in an emergency, of a saving collateral circulation. All the arteries and veins lie just beneath the internal limiting membrane of the retina, appearing in relief upon its inner surface, *i.e.*, superficially in the nerve-fibre layer. Another peculiarity of the retinal circulation consists in the fact that the capillary network reaches only to the external plexiform layer. This implies that the neuro-epithelial layer is without blood-vessels, and that there is a "hole," so to speak, in the capillary system at the centre of the macula, where the retina is thinned down to the several outer layers. Tiny branches can sometimes be traced, with the ophthalmoscope, almost to the rim of the foveola, and by entopic images one can easily demonstrate how small is the non-vascular space around this depression.

In the region of the optic-nerve entrance, besides branches of the central retinal artery, there are to be found those of two other systems. The arterial network from the pial sheath, whose office is the nourishment of the optic nerve, comes forward and anastomoses with a circumpapillary network in the sclera, which is derived from the short posterior ciliary arteries, and is called the **arterial circle of Zinn**. Branches of three systems thus unite in the lamina cribrosa, viz., **pial**, **choroidal** and **central**. The anastomosing vessels here are too small to be of great physiologic importance. In point of size it is well to remember that by the direct method we see retinal vessels magnified twelve to fifteen diameters; this would give to the largest stems an actual diameter of some tenths of a millimeter, or but slightly more than 1/100 of an inch.

**Anomalous Appearances.**—The disposal of the vessels in the papilla and in the retina is so varied within normal bounds that only those irregularities that are very striking can be called anomalous. Many freaks of growth are to be



observed if one will habituate himself to observe closely, just as a study of trees in a forest will disclose curious deviations from the natural in the development of the trunks and branches of the trees. There are few that are of any semio-logic value. To the unimportant types belong the queer twisted loops that project from the papilla, the twining of a vein around an artery like a vine around a twig, the bifurca-

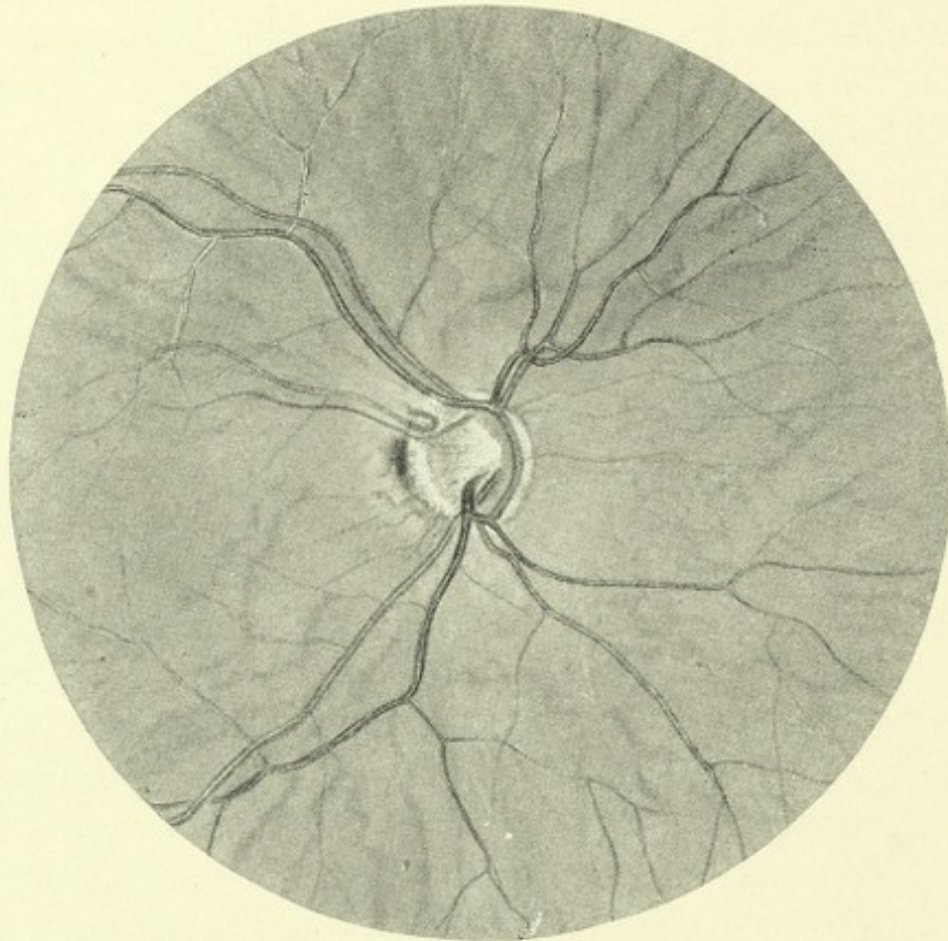


FIG. 30.—Anomalous central retinal vein. Cleft of nerve-head. Cilio-retinal artery.

tion of a vein just before entering the disc, the short sections that cause communication between two veins, the single enormous vein that emerges from the lamina cribrosa (Fig. 30), turned either upward or downward, then makes a half-circle on the border of the disc, at the nasal side, and gives off branches for the whole retinal venous system—and many more.

**Cilio-retinal Vessels.**—This name has been given to those



vessels of small calibre situated on the temporal side of the disc, which, in lieu of springing from or rejoining the central vessels, disappear, or appear, as the case may be, with an abrupt bend either on the border of the papilla or just outside of it. Such, at least, *seems* to be the case. We can have no assurance, in a given instance, that the vessel does not regain the central system by a route that renders it invisible. That there are occasionally arteries derived from

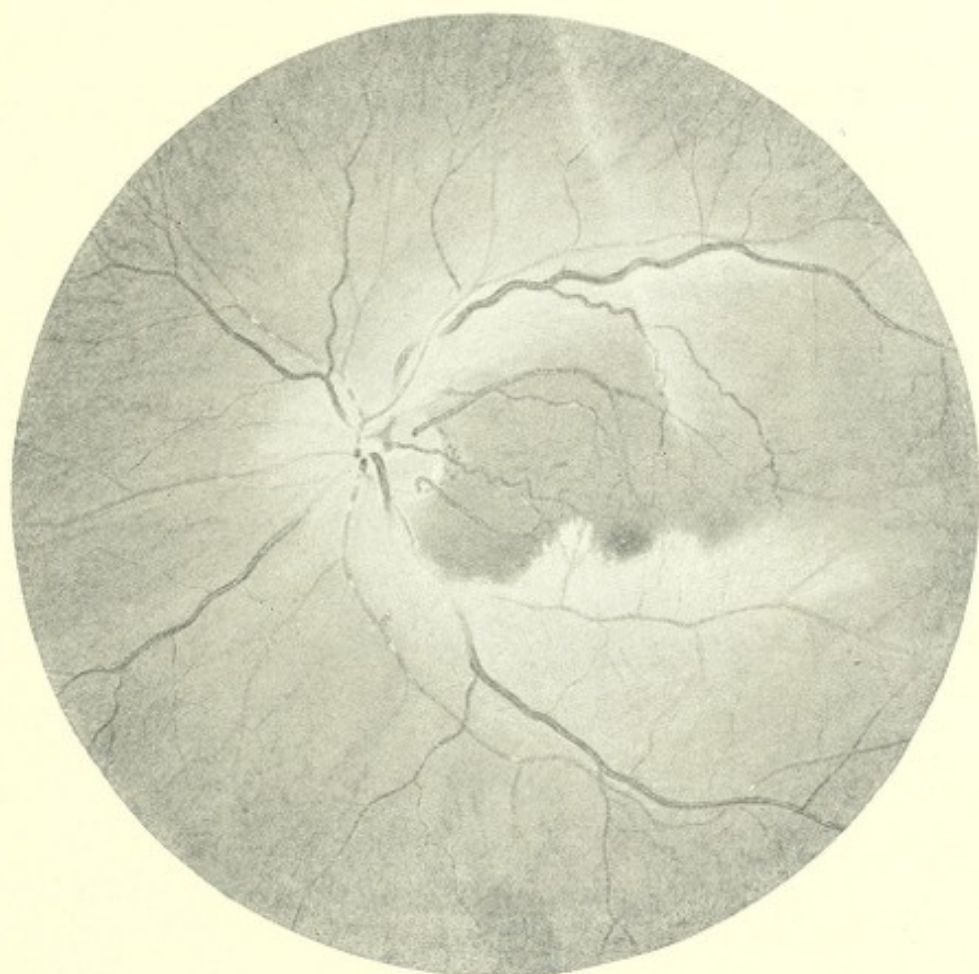


FIG. 31.

the choroidal system that preside over the nutrition of a limited district of the retina, extending even as far as the macula, can no longer be denied. They exist in man only as an anomaly, but in certain of the lower animals, as the cat, for example, they are a part of the normal circulatory apparatus. Czermak proved that there are two kinds of cilio-retinal vessels, those that come from the arterial circle



of Zinn, and those that come directly from the short ciliary arteries in the choroid. Of eleven cases examined anatomically by Elsching, the vessel in each was derived from the circle of Zinn. Cilio-retinal arteries are supposed to occur in more than one eye out of ten. Jackson, of Denver, found them in 20 per cent. of a thousand eyes he examined. Cilio-retinal veins are extremely rare. By the ophthalmoscope it is not always possible to identify positively cilio-retinal vessels. It is not rare to see an artery of considerable size appear like a hook stuck into the border of the papilla and course outward across the greater width of the fundus. Fig. 29 shows such a one. Many instances are on record of the sudden stoppage of the central artery, and in which a portion of the retina at the posterior pole was enabled to maintain its integrity by means of blood furnished by an artery of this description; but how many of these were cilio-retinal and how many proceeded from the central artery back of the obstruction it is impossible to state. Fig. 31 is from a case of decomposing thrombus of the central artery of the retina in which central vision was preserved by an artery of the kind in question.

**Inordinate Physiologic or Congenital Tortuosity.**—Hyperopia is frequently accompanied by retinal vessels that are sinuous beyond the ordinary. Schön thought this tortuosity was the result of excessive effort in accommodation. Others, that the vessels, being destined for a globe of normal dimensions, were crimped and bent in adapting themselves to the narrow confines of the highly hyperopic eye. These explanations fail when the anomaly is unilateral in eyes of the same dimensions—as it is in *rare* instances. Levin attributed it to an insufficiency of elastic vascular fibres—especially of the longitudinal. It is sometimes observed in connection with other anomalies of the fundus, such as extensive physiologic excavation. The fact that the vessels do not disappear by segments, or loops, in any part of the background, that the arteries participate as freely as the veins in the anomaly, together with the absence of



all signs of inflammation, or changes in their calibre, are sufficient proofs of their congenital character. If the tortuosity is congenital the bending of the vessels is not so much in the antero-posterior plane as in the acquired form, but is chiefly in the lateral plane—that is, the loops are flatter. With extreme tortuosity is sometimes found that condition of the papilla known as **pseudo choked-disc**, which has already been described.

Schirmer and Pantaenius have observed the curious association of tortuosity of the retinal vessels with **telangiectasia** or **nevus** of the lids and face.

**Acquired or Pathologic Tortuosity of the Arteries.**—This form of tortuosity is produced by both **mechanical** and **degenerative** influences. Mechanically, for instance, in engorgement of the vessels which lengthens them—gives them too much “fulness,” as is said of cloth in a garment that is corrugated when it should be smooth. Degenerative, as when the result of effacement or relaxation of the longitudinal elastic fibres in the different varieties of vasculitis of the retina. Hyperemia of the retina, or congestion of the veins, either passive or prolonged, causes them to be more tortuous than in the normal state. Hyperemia is of two kinds, (1) **active**, depending upon increased afflux of blood to the retina, and (2) **passive**, consisting in decreased **efflux** from it. The first concerns the arteries and is characterized by expansion of the visible blood-column, which may approach the width normal in the veins. The second by dilation of the veins, the enlarged vessels sometimes becoming two or three times their normal size. The inevitable effect of hyperemia—or **congestion**, if one chooses so to term it—is to produce **lengthening** of the vessels. This is evinced by sinuosities both of surface and of depth, which are proportionate to the degree of congestion. Superficial sinuosity is made evident by the kinks that show in *profile*. In that of depth, which is best studied by the direct method, one notes that the cylindrical reflex and the coloration of the blood-column are not uniform along the course of the vessel.



The high convexities of the loops have a wide, brilliant reflex, and their contents are bright red, while the color of the loops that are situated in the substance of the retina is much more mat or dull, and, in places, is quite indistinct. The intermediate portions of the vessel trunks, having a direction more or less perpendicular, or rather, *oblique*, with respect to the plane of the retina, are foreshortened, and the blood-column in them is seen "end on," and appears much darker—almost black as regards the veins. It is the alternation of sections of trunks that are bright with sections that are dull—of luminous reflexes with absence of reflexes—that enhance the impression one obtains of the third dimension in the sinuosities.

In simple hyperemic tortuosity it is nearly always the veins alone that are concerned to any degree, the arteries retaining practically their normal appearance, while the veins are distorted into various forms. The tortuosity is ordinarily most pronounced, or, at least, most readily occurs, in the smaller vessels, which take the forms of cork-screws, and of the tendrils of vines. The larger vessels, in extreme cases, form a complicated system of undulations, loops and twists, in which the observer finds it difficult or impossible to follow the order of succession. Sometimes there are, here and there, marking the courses of the vessels, groupings or rows of what look like miliary tumors which, from their color, might be taken for small hemorrhages, or for varicosities, but which attentive study will demonstrate to be due to series of sharp kinks in the veins, like the yarn raveling of a stocking, and appear thus because of being viewed on edge. It has been ascertained by anatomic investigation that the tops of these loops may project decidedly above the level of the retina, thus raising minute hummocks upon the inner surface of the internal limiting membrane, and adding to the deception. We know of instances in which simple tortuosities occurring in the manner thus described, have been taken for varicosities of the retinal veins, or for the strings of miliary aneurysms or varicosities that are not



infrequently seen in the retinal vessels of luetico-albuminuric subjects. When one reflects that these three species of vascular alteration are common to a certain diatheses of elderly individuals, the confusion is not surprising. This sort of tortuosity is occasionally seen in the young with thrombosis of the central retinal veins, but the prevailing type there presented is a tortuosity like a mass of tangled string (Fig. 32). The vessels here are softer and lack that

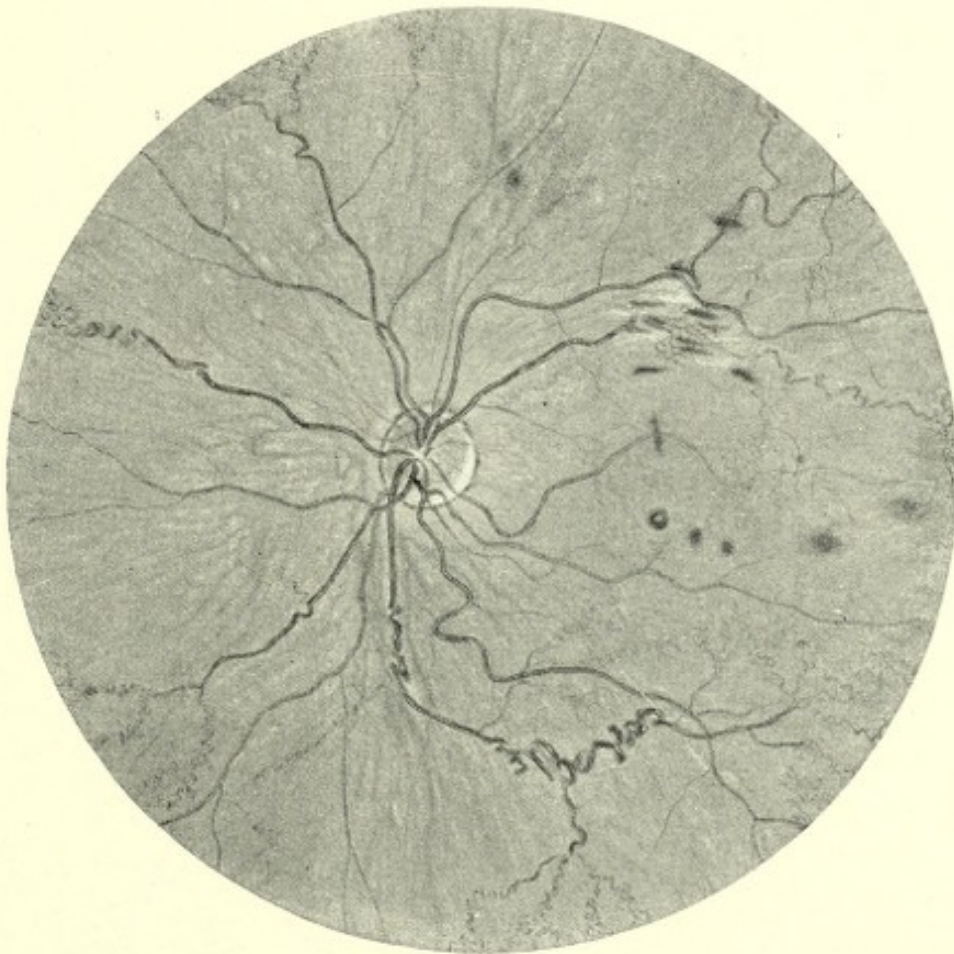


FIG. 32.—Excessive venous tortuosity in retina of young subject.

wiry resilience of the hardened ones of age. The loops and coils are more regular in subjects past middle age.

**In Venous Thrombosis.**—The degree of tortuosity and the extent of the modifications in the appearances of the retinal vessels in general depends upon whether the obstruction is in the central vein itself or in one of its branches, and also upon the extent of the obstruction—whether **partial** or



DESCRIPTION OF FIG. 33.

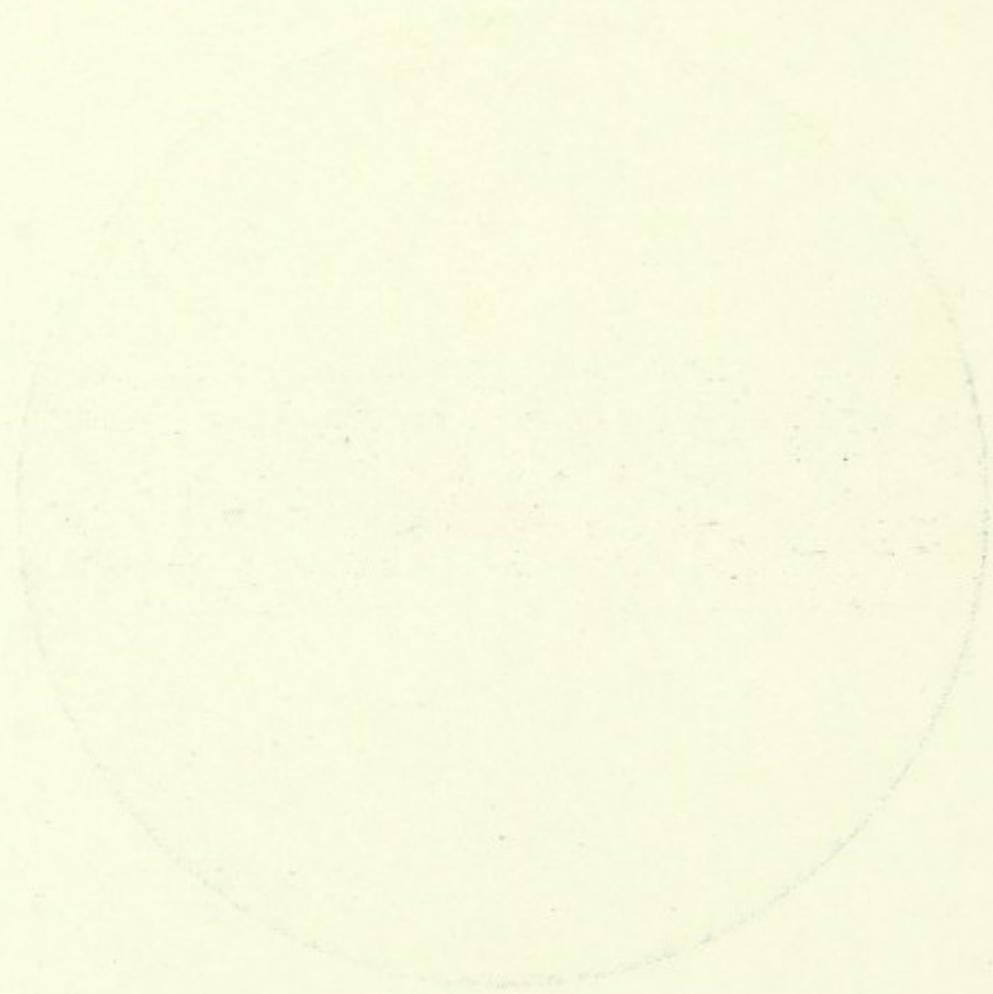
Thrombosis of central vein of retina in an elderly subject.





FIG. 33.







**complete.** In thrombosis of a branch, only the affected vein exhibits tortuosity and the other features, such as dilatation, hemorrhages, white effusions, etc. If the central vein is the seat of the obstruction, and the degree of closure is marked, the entire fundus is profoundly involved, giving a typical and impressive picture (Fig. 33). The veins, as a rule, are inordinately tortuous, and dilated to twice or three times their natural size. But their calibre varies in different localities, being in some places partially or totally effaced. This is shown chiefly by the lack of uniformity in the blood-column. Occasionally the column is broken into a series of disconnected cylinders, or joggy, a sign of the extreme sluggishness of the retinal circulation. Red splotches of various sizes, and in form linear, fusiform, and

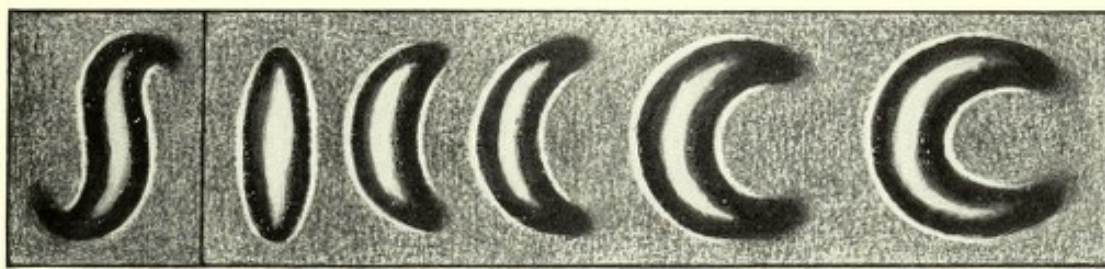


FIG. 34.—Forms of loops in tortuous retinal vessels. *a*, The S-shaped loop, indicative of spirality.

flame-shaped, radiate in every direction from the papilla, their deep red contrasting with the dusky crimson of the swollen and sinuous veins. The arteries are twisted threads of pale orange, often hard to follow in the scene of disorder. The background is of light tint, like that of the visible portions of the papilla.

**Tortuosity in Leukemia.**—When there is dense **leukocytic infiltration**, **exudations** and **hemorrhages** of the retina, or, in other words, when the retina is abnormally opaque, as in **albuminuric retinitis** and in **thrombosis** of the **central retinal vein**, the loops of tortuosity in the larger vessels are characterized by apparent forms suggestive of crescents of different arcs, and of horse-shoes. Owing to the opacity of the retina,



only the upper part of the loop is visible—one sees the convex but not the concave loops, the last being hidden in the tissues. If the loop is exactly perpendicular all one sees of it is a small ellipse (*b*, Fig. 34). In proportion as it inclines from the perpendicular the appearance it presents is that of a crescent of increasing arc (*c*, *d*, *e*, *f*, Fig. 34). The broadest part of the crescent is clearly defined and bears a big, bright reflex, also of crescent shape. The red line on either side of the reflex is distinctly *red*. At the points, which disappear in the swollen retina, the color is much darker and, of course, devoid of any reflex. While being examined by the direct method, vertically inclined loops will seem to disappear in perfectly transparent tissue, but this is because the further part of the bent vessel is out of focus, the blurred portion being made distinct by turning on the proper lens. In this position it has not the sharp crescentic outline that it assumes when in the opaque retina.

The appearance of the retinal vessels in acute leukemic retinitis or pernicious anemia is most distinctive. Both arteries and veins are enormously enlarged, and the ground of the fundus is a pale orange. The arteries are salmon and the veins are light rose. Viewed by yellow artificial light, there is but little difference between the tint of the arteries and that of the veins. The tortuosity in this affection is highly distinctive. It is immoderate, and extends to both arteries and veins, the veins sometimes attaining a diameter of five times and the arteries twice, the normal. In consequence of the relative thinness of the retina—to these huge vessels—and to its slight opacity, the loops in the trunks of the inordinately tortuous vessels do not disappear at any point, but present the appearance depicted in Fig. 35. If the expression “like links of sausage” as sometimes used to describe sinuosity in a vessel is ever appropriate it is in this connection. One gets the impression that the larger of the veins have a strong tendency to get their bends or undulations perpendicular to the plane of the retina, but that there



is not room enough, and they must perforce stand at an angle; moreover, the cylindric reflex on the more prominent loops is also peculiar in that it is too broad to be that of a true cylinder of the diameter represented by the vessel to which it belongs. This indicates that the vessel is flattened at this point by pressure against the vitreous body—as also, it is presumed, is the case with the inverted loop against the parts external to it if they are not too yielding. Framing the most prominent of the big loops are delicate grayish-white lines denoting the walls of the vessel and representing their thickness.

**Retinal Cyanosis.**—Under this name Liebreich described the aspect of the fundus that accompanies general cyanosis from defects of the central circulatory apparatus (*Morbus Ceruleus*). General cyanosis is of two kinds, **congenital** and **acquired**, and its presence indicates serious interference with the circulation, or, at least, insufficient oxygenation of the blood. Congenital cyanosis is, most often, the result of a developmental defect of the heart, or of the pulmonary artery or of both together.

The chief defects leading to general cyanosis are:

1. Considerable absence of the interauricular septum.
2. Defects of the base, leaving interventricular openings.
3. Transposition of the two great vessel trunks, with reversal of the ventricles, or, as the Germans call it, *Recht-slage*.
4. Pulmonary stenosis and atresia.

These, singly or in combination with other connate circulatory defects, constitute the most frequent cardiac anomalies.

1. It has been popularly supposed that the most common cause of congenital cyanosis is a widely **patent foramen ovale**. This orifice persists, to a greater or lesser degree, in about 15 per cent. of adult individuals; hence, to be termed anomalous it must be *widely* patent, say, capable of admitting the first phalanx of the index finger, or larger. Yet it has been ascertained that practically the entire septum may



DESCRIPTION OF FIG. 35.

Leukemic retinitis.



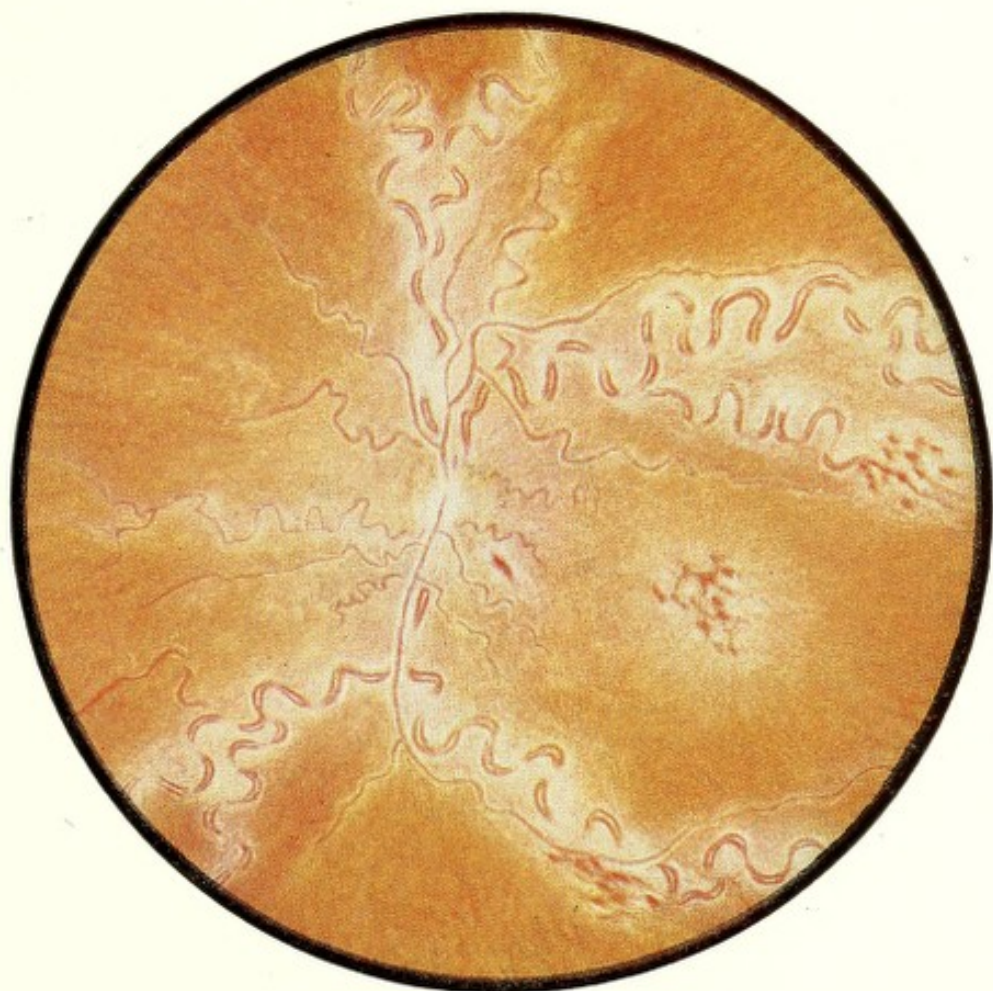


FIG. 35.







be lacking without giving rise to cyanosis and its attendant symptoms.

2. Considerable absence of the interventricular septum, with presence of the auricular septum results in a three-chambered heart, called **cor biatriatum triloculare**. The symptoms are similar to those from absence of the auricular septum, and in both conditions cyanosis may be absent or long delayed, another proof that this condition is not inevitably consequent upon mingling of the two blood-currents. Clubbing of the extremities and dyspnea are not always in evidence with the cyanosis.

3. **Rechtslage**.—If the transposition is complete the duration of life is very short, that is, from a few days to a few years. In measure as the reversal is incomplete the life of the subject is apt to be prolonged.

4. **Pulmonary stenosis and atresia** are the commonest of all the cardiac anomalies, according to Maude E. Abbott, and they present the classic picture of congenital cyanosis in all its details. So frequent is the association between the two conditions that morbus ceruleus and pulmonary stenosis have come to be considered as almost synonymous terms." The symptoms vary in accordance with the character and extent of any coexisting cardiac defects. The difference between stenosis and atresia, as the words are here employed, refers mainly to the degree of the cyanosis, it being extreme in atresia. This form of general cyanosis constitutes true morbus ceruleus in that the blue or purple tint of the body is *constant*. It may increase to blue-black upon exertion. In pulmonary stenosis, with closed interventricular septum, the duration of life is relatively greater. It has been known to be as great as forty-five years. In stenosis with considerable septal defect death occurs much earlier, the subject seldom if ever reaching adult life. The cyanosis may appear only in after years. In pulmonary atresia life is less prolonged. Children with this condition, and closed intraventricular septum, die within the first few months. If, on the con-



trary, the septum is largely perforate the individual may live a number of years. The highest recorded age was thirteen.

Congenital cyanosis affects all the tissues of the organism. The conjunctival vessels are enlarged, so that this membrane partakes of the same magenta tint as that of the optic nerve and retina. The color of the lips, the tongue and the buccal lining is deep purple, as if from eating mulberries. The superficial cutaneous vessels are dilated, so that the face and extremities are what Parker, of Detroit, has described as "a dusky, slaty purple;" while over the rest of the integument the tinge is usually less pronounced, being a sort of opaque lavender, or a milky ultra-marine. The tongue, in advanced stages, assumes that patchy appearance called "geographic." The tips of the fingers, toes and nose become flattened and bulbous—"clubbed." The respirations are heightened, and dyspnea, if not constant, is brought on by slight exertion. Those afflicted with congenital cyanosis are stunted physically, and sometimes mentally, or they are effeminate in body and mind. Polycythemia is now recognized to be a common feature of the disorder, though not a constant one, excepting in the severe forms and last stages, when the red blood-cells may number as high as six to eight, and in extremely rare cases and in the late periods, as much as nine millions per cubic millimeter. Marked cyanosis may exist for many years without other signs of venous stasis than clubbing of the extremities.

**Ophthalmoscopic Appearances.**—In certain respects the appearance of the retinal vessels in this condition constitutes the antithesis of those of leukemic retinitis. They were first fully described by Knapp in 1861. The entire fundus—every detail of it—looks as if stained with magenta—or the peculiar purplish-blue of the analine pencil. The retinal vessels are all greatly augmented in calibre, and all are more or less tortuous. They were all very tortuous in the case from which the drawing was made that furnished



Fig. 36. Of the twenty-odd published cases of the affection the veins only were referred to as dilated in some. The tortuosity is of the lateral kind—that is, the loops do not dip and rise but lie flat. The color of the arteries is of the same crimson as that normal for the veins, while the latter are of a proportionately deeper purple. The finest vessels are injected, those around the macula barely visible under ordinary conditions, forming a conspicuous crown in that region. The purple tint in the colored zone of the papilla occurs in patches. The details of the macula are accentuated, a peculiarity before noted by Babinski.

The conditions just described must not be confounded with those that characterize an acquired affection of after life, which has, within the past twenty years, been referred to by the rather trite expression “a distinct clinical entity,” and has been called “Vasquez’s Disease,” in honor of him who first mentioned it, or termed the **“Osler-Vasquez Disease,”** because the distinguished Oxford professor was the first fully to describe the symptoms. It is, strictly speaking, rather a *quidditive* entity than a *positive* one. Of the comparatively few published cases of this curious affection the subjects were verging upon or near middle life. The three most important symptoms, or those that constitute the syndrome of the disorder, are general cyanosis, polycythemia and enlargement of the spleen. Other more or less constant symptoms, all of which are in great measure paroxysmal, are asthma or dyspnea, headache, nausea, vertigo, palpitations, tinnitus aurium, disturbances of vision, albuminuria and high arterial tension. The general cyanosis is not essentially different from that of the congenital variety, save in that it is characterized as being more florid, especially as regards the face and hands. It is less constantly present, and its intensity is more variable. The appearances in the fundus oculi, however, aside from the cyanosis, present certain distinctive features. The retinal arteries are less tortuous in this form, and they are characterized by signs of degeneration of their walls. The



DESCRIPTION OF FIG 36.

Retinal cyanosis.



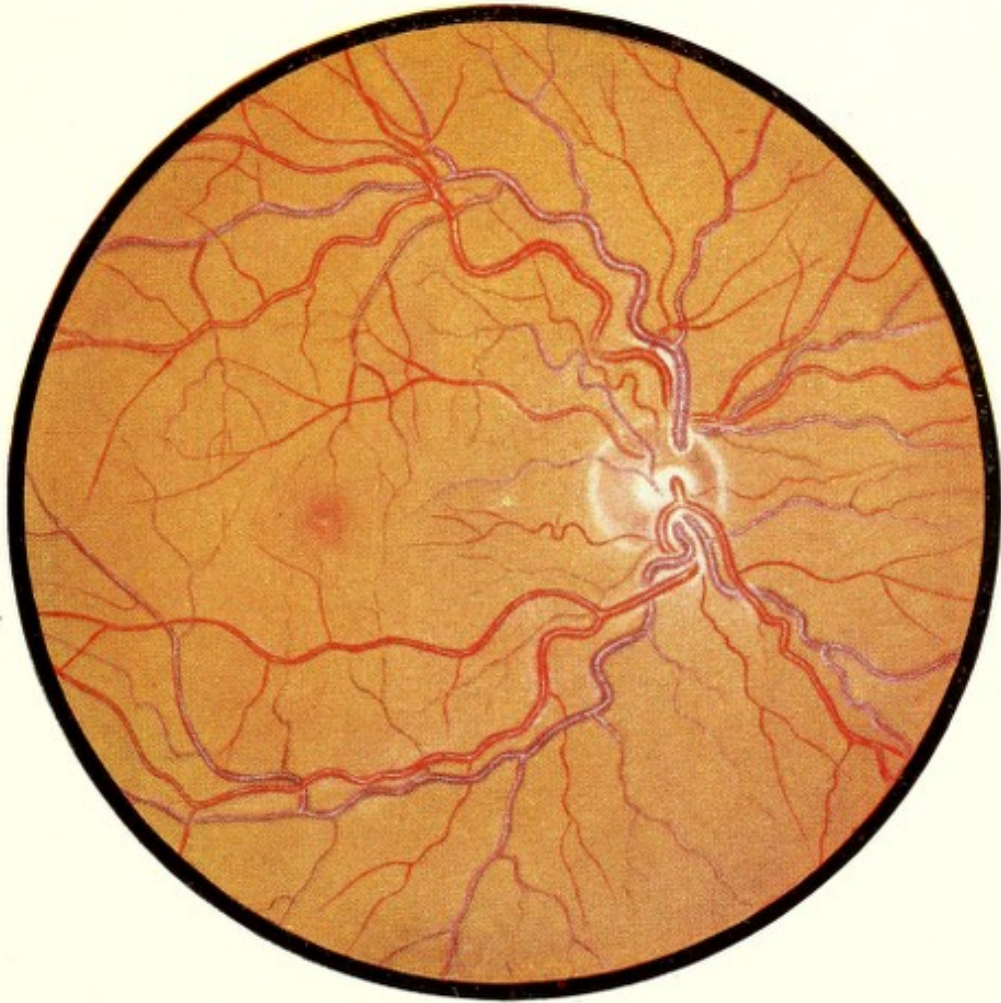


FIG. 36.







loops of the sinuous vessels are more vertical, often dipping out of sight in the swollen retina. Hemorrhages of the retina are not infrequent; on the contrary, they occur in most all the cases. The optic papilla and the retina are likely to be edematous, and the outlines of the disc to be blurred. The details of the macular region are lacking. The arteries are inclined to pulsate, while the veins do not.

The numbers of the red blood-cells are *always* increased, on an average reaching a count that is much higher than in the congenital form. As many as eight or nine—even as high as twelve—millions per cubic millimeter, have been recorded, the other cells remaining numerically about normal.

There is a third variety of general cyanosis that is sufficiently distinctive to merit mention. It is called **methemoglobinemia**. It, too, is acquired, and is produced by certain toxic agents, such as hydrogen sulphid, and some of the analin products. The coloration of the integument is more gray than purplish-blue, the hue of the mucosæ is more blue than red, and that of the optic nerve and retina more purple than bright magenta. Heart signs, polycythemia, splenomegaly, and the paroxysmal character of the symptoms do not exist. The history will often disclose the toxic factor.

**Persistence or Remains of the Hyaloid Artery and Canal of Cloquet.**—The fetal hyaloid artery, which is a part of the central system of the retina, and whose principal branch traverses the canal of Cloquet and ramifies to form the posterior vascular sac of the crystalline lens, normally disappears just before the completion of the term of gestation. Not infrequently, however, the artery or relics of it, or some part of its envelope—canal of Cloquet—is found to have persisted. In some instances the remains seem to be those of the artery, in others only those of the canal, and to which to refer them is often largely a matter of assumption or conjecture. Those that can be positively identified as referring to the artery are seen to be connected with the



central retinal artery or one of its principal branches toward the temporal side—the point of origin being most often within the boundary of the papilla, though occasionally external to it. It turns forward in the vitreous and can sometimes be traced as far as the posterior surface of the lens. In rare instances it carries blood, when it is said to be *permeable*. It then has a reddish or crimson color, and digital pressure upon the globe will cause it to become empty.

Remains of the hyaloid canal are always attached to the papilla. They rarely reach the lens, but project varying distances into the vitreous, the proximal end being free. The attachment at the disc may be anything from a slight shred to a gauzy funnel-shaped or nondescript affair that partially or completely conceals the papilla. More often the attached portion appears small, and the projecting portion gradually enlarges toward the observer, like a cornucopia. This is partly real and partly the effect of refraction. When the remains contain no blood, they appear dark and shadowy by reflection, and by incidental light, white or grayish-white, sometimes with slight admixtures of blue and green. They are rarely fixed, but undulate and wave freely with the movements of the globe. A few are drawn to one side and fixed, and others dangle from the papilla. Their free extremities exhibit a variety of forms, such as conical tubes, single or multiple points, plain or fimbriated bulbs, or they terminate in a formless shred. It is always well to examine the posterior capsule of the lens for evidences of attachment there, either present or past. These show by transmitted light as black dots, and by reflected light as grayish-white ones. They are often of characteristic outline—*i.e.*, discoid, oval, triangular—or what has been likened to the shape of a leech-bite. They are near the centre of the pupil and single or multiple.

The possessor of such an anomaly is most often unaware of anything abnormal in the eye. If the amblyopia is pronounced, it may have attracted attention—as also might the effect upon the field of vision caused by the moving



shadow of a waving remnant. The sight is either not affected, or there is partial or total abolishment of it, in accordance with the extent of the remains themselves or of that of the **concomitant anomalies** that so frequently exist. These last are rather distinctive as to their appearances. On the papilla, or standing out boldly from it, are glistening white formations, with suggestions of iridescent coloring,

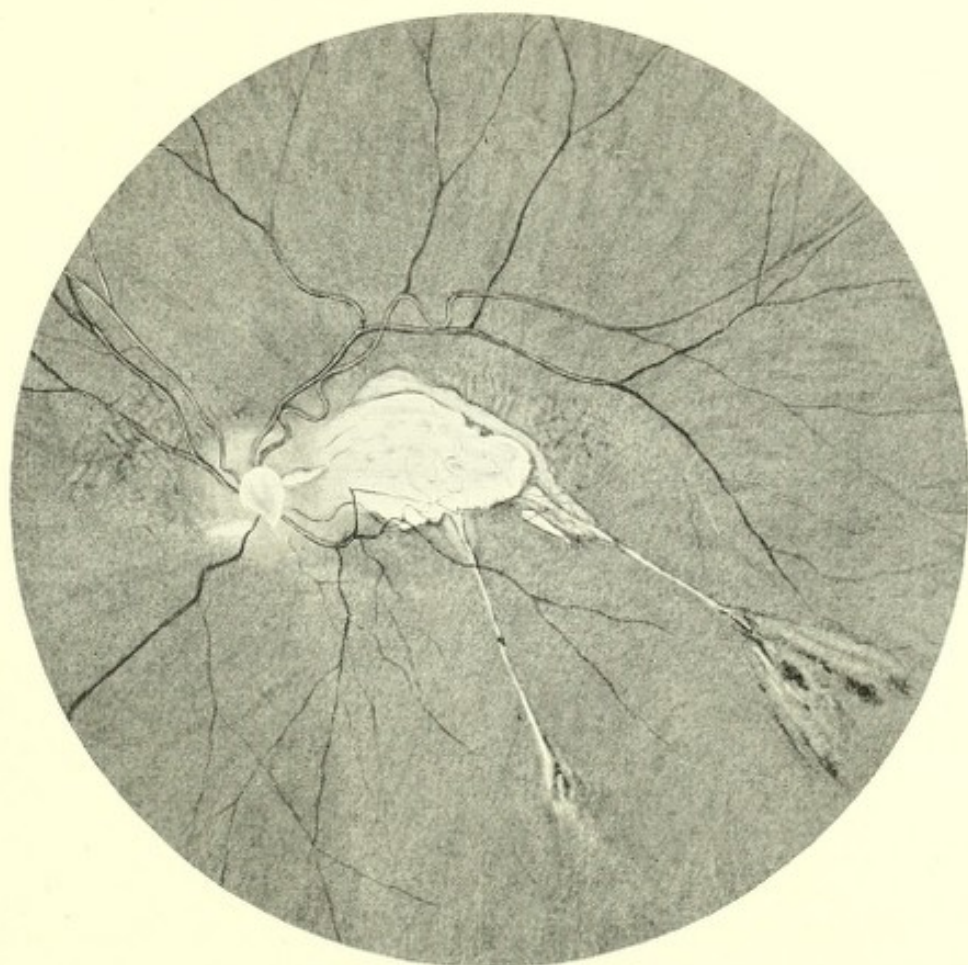


FIG. 37.—Anomalies associated with remains of hyaloid artery.

and often of fanciful or grotesque forms. There are often associated with these certain irregular streaks of pigment and connective tissue, that go out from the papilla as if following the course of an extinct retinal blood-vessel, but which are really in the choroid (see Fig. 37). Pigmented coloboma of the choroid of varying extent, and connected with the papilla or isolated from it, as well as coloboma of other parts of the uveal tract, are very frequently present.



The papilla is apt to be deformed, and the aspect and distribution of the retinal vessels is wholly anomalous.

Persistent hyaloid remains are rarely, if ever, present in both eyes of the same subject. Several observers have noted their association with congenital pigment degeneration of the retina. The writer has made a drawing of one such case (see Fig. 38).

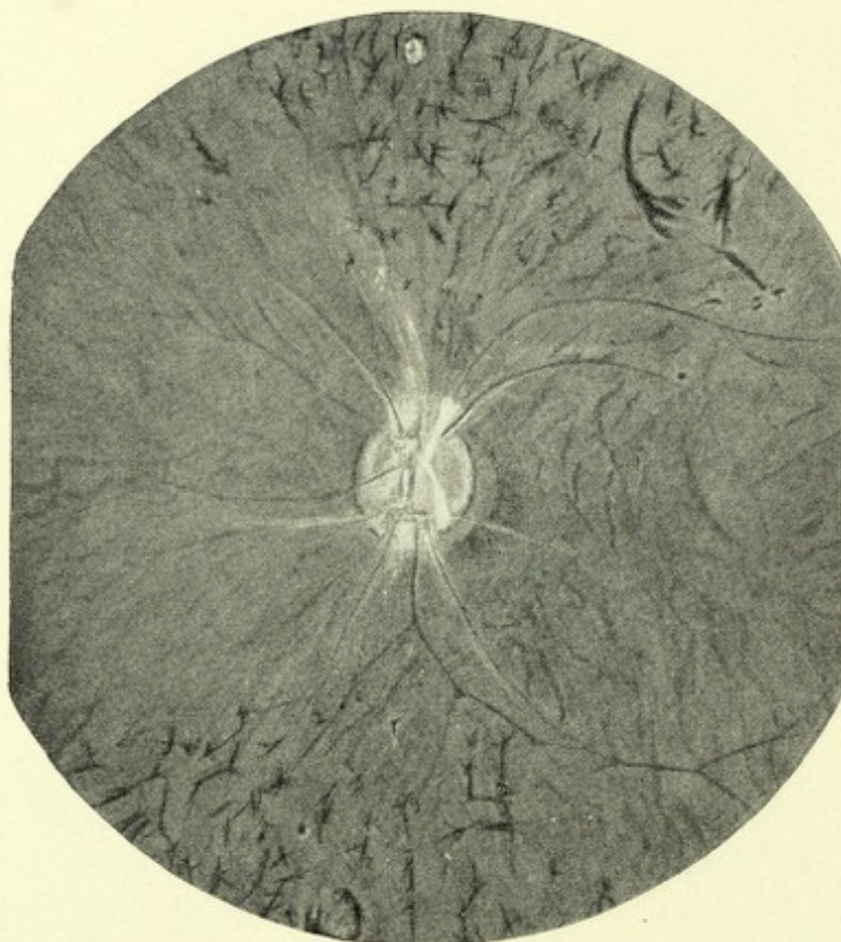


FIG. 38.—Remains of hyaloid artery, and pigment degeneration of retina.

**Differentiation.**—The points just given should ordinarily enable one to recognize hyaloid remains and its associate defects. They are, perhaps, more liable to confusion with **retinitis proliferans** than with anything else. This disease is of two kinds, **primary** or **idiopathic** and **secondary**. The primary usually owes its origin to an intervaginal effusion, mostly hemorrhagic, about the nerve-head. The **secondary** form is consequent upon traumatic hemorrhage of the



retina, or upon the kind of hemorrhage which is such a frequent accident in degenerating and obliterating diseases of the retinal vessels (Fig. 30). In any event it may be said to have its inception in a hemorrhage, and it is essentially a proliferation of the connective tissue of the nerve-fibre portion of the papilla (or retina) and that of the outer coats of the retinal vessels. It is not a disease either of the



FIG. 39.—Retinitis proliferans in arterio-sclerosis.

vitreous or of the neural tissue of the retina. It is characterized by the formation of tendinous white membranous expansions over the surface of the retina, from which often tent-like projections and tufts of new blood-vessels reach out different distances, crowding and disorganizing the vitreous. Neoform vessels are distinguished from the normal retinal veins and arteries in that they grow in bunches, wherein they are disposed in a parallel manner and,



that, instead of branching dichotomously they anastomose with each other. These newly born vessels are prone to hemorrhages, which serves greatly to complicate matters. The proliferation or hyperplasia sometimes begins in several places at the same time, or, at least, is seen in several parts of the fundus, each site having its features of connective tissue and new vessels. Another peculiarity of the connective-tissue formations is their tendency to curve into hooks, scrolls and cylinders.

We have seen that in rare instances a hemorrhage may occur from a permeable persistent hyaloid artery, in which case one might be able to see waving before the disc, in the perfectly healthy vitreous, the vascular appendix. In retinitis proliferans the vessels are multiple, and always either entangled in a mass of cellular tissue or they are connected with the membranous expansions of connective-tissue proliferation. It is well then to remember when one finds abnormal blood-vessels in the depths of the eye, especially when they give rise to hemorrhages, that those vessels can only be of congenital or of inflammatory origin. In the latter case they are always preceded by other phenomena of the fundus, more or less violent, and accompanied by other lesions that point almost certainly to their inflammatory nature. Retinitis proliferans always has its origin in a hemorrhage, whereas, if there is any hemorrhage in the vicinity of a persistent hyaloid artery it has, of course, appeared subsequent to the vessel—the vessel has caused it, not it the vessel. Primary proliferating retinitis is, as a rule, bilateral, while the secondary is most often unilateral. Both are characterized by disorganization of the vitreous. The primary variety is more common in the younger individuals and the secondary, particularly that following hemorrhages from disease of the retinal vessels, is more frequent in elderly subjects. The primary seems to have a preference for adolescence, and for males rather than females—a class of subjects in whom spontaneous hemorrhage other than that of the retina, such, for example, as epistaxis, is a special



attribute. These cases are most likely to come under observation when the connective-tissue proliferation is the conspicuous feature of the ophthalmoscopic picture, while with the atheromatous subjects it is often the neovascular epoch, as shown in Fig. 40. In this picture we observe how the blood trickles or filters down through the vitreous. The

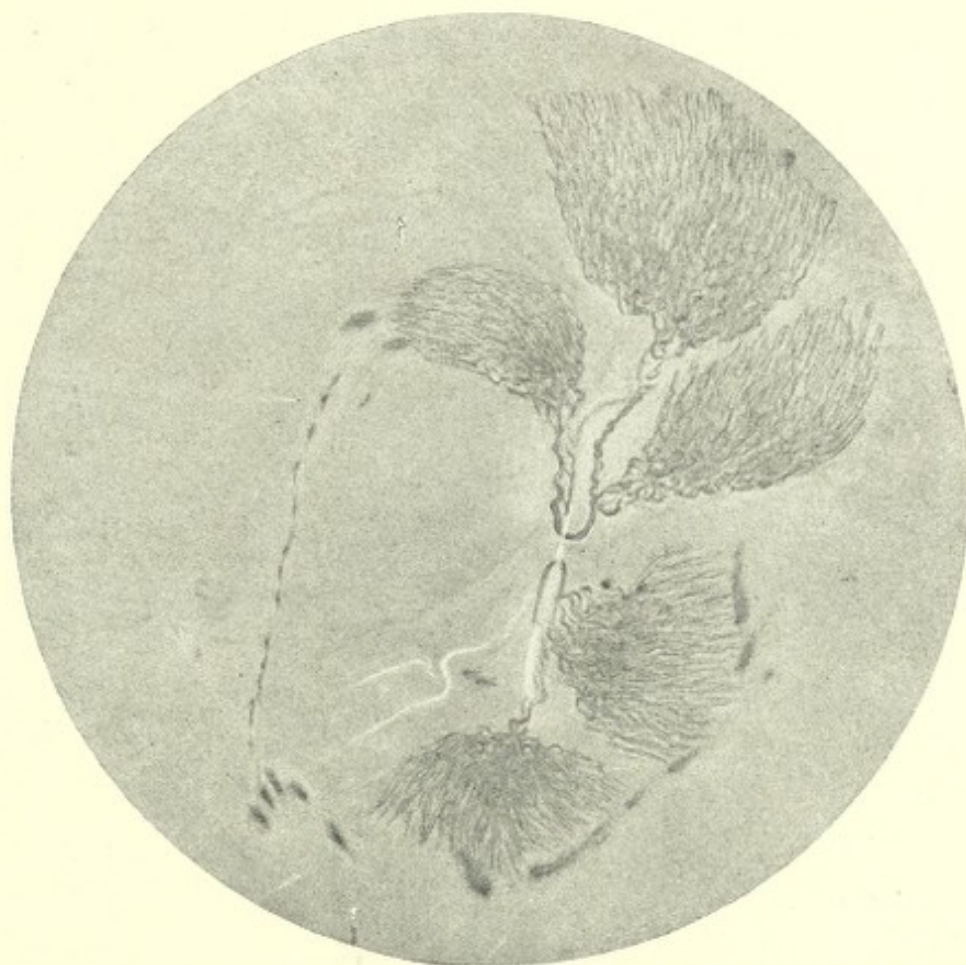


FIG. 40.—Extensive new blood-vessel formation in the fundus.

prognosis is grave as regards both kinds, as to detachment of the retina in the one, and total obscuration of the vitreous in the other, with resultant ultimate blindness. This affection is not to be confounded with that known as **retinitis striata**, described elsewhere.



## CHAPTER XIV.

### TOPIC MODIFICATIONS OF CALIBRE.

**Aneurysms.**—The ophthalmoscopic study of the vascular changes in the retina is now regarded as one of the most important departments of ophthalmic semiology. The signs and warnings given in the fundus concern the integrity not alone of the eye but of the whole organism. By heeding them, and acting in strict accordance with the information they give, the gravest of accidents local and remote may often be averted. Not the least of the local accidents are retinal hemorrhages. **Miliary aneurysms** are fruitful sources of retinal hemorrhage. Before ophthalmologists began so diligently to search for alterations in the walls of the retinal vessels, the tiny aneurysms were considered rare. Now they are frequently found in atheromatous subjects. They appear as fusiform or spheroid dilations, and are chiefly noticeable in contrast with the endarteritic contractions that are seen in other places. A number have been observed on the papilla, where, on account of their proximity to the central retinal artery, they can be seen to pulsate synchronously with the radial pulse. They occur in any part of the fundus up to the equatorial region, or they are visible in many parts of the fundus at the same time. Again, a number of them occur in succession on a single artery. A favored site is a little back of a bifurcation. This is explained as due to the mechanical action of the blood-column which is here subjected to sudden checking and change of direction.

**Varicosities.**—We understand by venous varicosities those circumscribed anatomic alterations, having for effect the local relaxation of the vessel wall, and consequently the production of irregularities more or less pronounced in the



calibre. These varicose dilatations in the retinal veins are analogous to the aneurysms just mentioned, as concerns their etiology and the rôle they play in the production of retinal hemorrhages. Like the aneurysms, they may be situated on the papilla or at some distance from it. The trunk is sometimes dilated throughout a fairly long section—again the enlargement presents a more or less circumscribed sacculation; or a fusiform dilatation shows additional ring-like ectasias; or a single branch may be made up entirely of a series of almost uniform short sacculations that give to the vessel precisely the appearance of a string of beads—**moniliform** (Fig. 46). Other similes are, to say, like a **rosary** or like a **necklace**. Both aneurysms and varicosities will be again referred to as **dilatations**, under “Signs of Arteriosclerosis of the Retina.”

**Modifications of Aspect and Calibre.**—As before stated, in the normal state vessels of the retina are invisible. It is true that in the papillary region of young individuals, one can discern parts of the walls. They appear as beautiful, soft, pearly outlines to the red stripes of blood, like the waxy bloom that covers the grape when viewed in certain lights. By very close scrutiny a double outline can be distinguished about the largest trunks on the disc—one for the wall and another for the sheath, the so-called perivascular lymph space being between. Aside from these vague glimpses what we see is not the vessel but its contents. The vessel is said to be wide or narrow, large or small, dilated or diminished, engorged or empty, according to the appearance of the blood-stream within it. Visible modifications of calibre are, therefore, not necessarily criterion of the actual dimensions. The alteration is *real* or *apparent* in proportion as it *is* or is *not* partly concealed by abnormal opacity in the vessels. Both real and apparent reductions in calibre are more or less characterized by the appearance of a grayish or white outline on each side of the blood-column. In conditions bordering on ischemia of the retinal vessels, or even in sudden complete emptying of them, these lines



appear in faint degree. This can be verified with the ophthalmoscope by steady, rather forceful digital compression upon the relatively soft globe of a youthful person. The vessels are thus deprived of their blood and their walls rendered partly visible. The effect is somewhat more marked in the arteries because of the presence of the muscular fibres which make them thicker and more contractile than the veins.

By way of illustrating why the vessel will not exhibit the white lines so long as it has its proper volume of blood, imagine a small thin tube of white or transparent India rubber, filled tightly, so as to put it on the stretch, with a red liquid, and laid on a background of lighter red. Save for the cylindric reflex, it appears like a red stripe. No part of the tube is in evidence. Now partially empty the tube, again lay it down, and the red stripe has borders of white. In distention the walls of the tube are greatly thinned, and in contraction they are proportionately thickened. At either side the rays of light have to pierce much greater thickness by reason of the foreshortening of the walls, hence the lines. So with the blood-vessel, while expanded it does not show, but when depleted it shrinks from the resilience of the circular, elastic fibres, to which, in the arteries, are added the muscular fibres, which not only follow the diminishing column of blood, but actually tend to force it out. The white lines thus formed, if measured from outside to outside, would give the external diameter of the vessel, but their distance apart would not represent the diameter of the lumen—in other words, the *real* width of the blood-stream—because the latter is somewhat reduced on each side by the overhang of the opacity. Ophthalmoscopic estimates, then, have not so much reference to the size of the lumen as to the seeming width of the contained blood-column. The more nearly normal the structure of the vessel walls, the more nearly coincide the real and the apparent widths of this column.

**Reduction in Calibre.**—The principal causes of real reduced



calibre of the retinal vessels are lack of volume or force in the blood-stream, spasm of the muscle tunic (of arteries), thickening of the intima, and atrophy of all the tunics. In ordinary simple and secondary atrophy of the optic nerve and retina it is manifestly real, for even the outside measurements of the accompanying white streaks are less than the corresponding normal ones. The white lines in atrophy are relatively wider, however, than in simple contraction of a vessel, because of the connective-tissue degeneration of the walls. This is particularly true of those of secondary atrophy. Consequently more of the blood stripe is concealed. The latter also appears of lighter color, partly because of the thinning and partly because seen through and *against* a white film.

**Anemia.**—Attenuation and pallor of the retinal vessels are not constant symptoms of general anemia. One no longer pretends to ascertain the degree of anemia by ophthalmoscopic examination. It is none the less certain that in more than half the cases of pronounced benign anemia and chlorosis there is diminution of both arteries and veins with lack of color in their blood. The relative difference in size between arteries and veins is not maintained but is accentuated, the latter being but slightly reduced, and sometimes even enlarged.

**Ischemia.**—This refers to local and sudden depletion of the retinal vessels. It may or may not be accompanied by general symptoms. Its chief causes are collapse, less cardiac energy, occlusion of the retinal blood-vessels and vasomotor reflex influences; the last inducing spasm of the retinal arteries. Von Graefe and deWecker examined victims of cholera in the stage of asphyxia and noted attenuation of the retinal arteries so extreme that they could be traced but a short distance from the papilla. The veins, too, were greatly narrowed. Certain cases of bilateral ischemia sufficient to cause complete blindness have been attributed to an enfeeblement of the heart's action to such a degree that the force of the blood-current in the retinal



arteries was no longer able to overcome the normal intra-ocular tension. It would seem, and it is doubtless true, that given diminished cardiac impulses associated with hypertension of the globe, or with obturation of the retinal circulation, that blindness from ischemia would be imminent.

**Vessel-cramp.**—Certain local vasomotor phenomena seem to be responsible for many instances of ischemia of the retinal vessels; some being due to physiologic and others to pathologic reflexes. A number of cases of sudden complete transient blindness have been observed in perfectly healthy normal individuals, mostly young, in some of which the ophthalmoscope has demonstrated not only the ischemic condition of the vessels, but has revealed the spasmodic nature of it. Most of the cases of blindness accredited to vessel-cramp have been in connection with affections that could act as exciting causes. Among these are Jacksonian epilepsy, (deWecker) intestinal parasites, Raynaud's "local asphyxia of the extremities," intermittent fever (Schnabel), whooping cough (Knapp), and migraine (Galezowski and Berger). Wagenmann and others have reported transient recurring blindness in arterio-sclerotic subjects. It is considered by some authorities as one of the prodromic symptoms of endarteritis. One of the more recent of these reports is that of Zentmayer, reported to the American Medical Association, at Boston in 1906. Zentmayer had the exceptional opportunity of making repeated examinations of the fundus phenomena in his case. He says concerning his findings: "Ophthalmoscopic examination showed a moderate contraction and distinct flattening of the arteries and veins at the onset of an attack of blindness. When the blindness is complete, the arteries become ribbon-like and lose their reflex, and the veins, especially the superior branch, become very narrow. After a brief period there is a gradual refilling of the arteries and veins, the latter becoming greatly distended, particularly the inferior branch. During the latest attack that was observed, the last of a long series occurring at half-hour intervals, there



appeared to be some retinal haze about the disc. There were never any macular changes. The pupil dilated synchronously with the loss of vision, and for a while after the attack exhibited hippus."

Wagenmann's observations, as quoted by Zentmayer are: "A man, aged sixty-nine years, had for two months noticed frequent, and of late almost daily, attacks of blindness in the right eye, lasting from a few minutes to a few hours. He had had epileptiform attacks in childhood, but was now healthy except for arterio-sclerosis. The changes observed were, in a short time perception of light was gone, and with it direct and consensual pupil reaction. Externally no change. Ophthalmoscopically, disc pale, arteries appeared as shiny streaks in which no blood-columns could be seen, even with the direct method. Veins thread-like. No pulsation on pressure. Soon the retina became cloudy and the fovea (foveola) stood out as a round red spot. About ten minutes after the beginning of the attack a fine, red line was seen by the indirect image to appear in the arteries, and immediately afterward the veins became large. Thereupon the patient had perception of light, and the pupil reacted a little. In a few minutes the circulation, and with it the vision, was completely restored."

In two cases, one by Benson the other by Sachs, also quoted by Zentmayer, the vessel-cramp was actually visible with the ophthalmoscope. For more than four years a man, aged thirty-two, in good health, had had numerous attacks of transient blindness, sometimes complete, at other times involving only a section of the field. During an attack of complete blindness the ophthalmoscopic examination revealed the inferior temporary artery entirely bloodless for a distance of four disc's diameter from its point of emergence. The bloodless section was seen to shift toward the periphery until it reached the next large bifurcation, when it suddenly disappeared, leaving the blood-vessels normal. The same phenomena were noticed during attacks of partial blindness.

Sachs' case resembled that of Benson: In the affected eye



a circular constriction would start from the papillary end of the inferior artery and travel to the point of first bifurcation, taking from three to five minutes to make the journey, a distance of one-half the disc's diameter. The fundus typified that of embolism. When again seen, three days later, only the picture of embolism was present. The patient had arterio-sclerosis.

Spastic ischemia is, as a rule, monolateral. For this reason one might be led to suspect embolism or thrombosis. The ophthalmoscope should clear the diagnosis.

It is remarkable how long a time the eye may remain blind in these recurrent paroxysms, yet completely regain its integrity. The period has been reported as lasting from a few minutes to a few hours—in one case sixteen hours—without ultimate impairment. In bilateral ischemia of a girl five and one-half years old, in which blindness had lasted a number of days, Graefe restored the sight by iridectomies, and Rothmund was enabled to perform a similar fortunate office for two girls, one thirteen, the other eighteen, who had been totally blind in both eyes for several days. The reason why the retina suffers so little permanent injury in these cases is that during the spasm of the vessel the arrest of the circulation is not complete, but that there remains a tiny stream of blood, too feeble to admit of visual function, yet sufficient to safeguard the anatomic integrity of the retina. This explanation does not satisfy those who have noted that very pronounced ischemia of the retina was not incompatible with good vision, and who believe that to cause complete blindness the interruption must also be complete. Ischemia, purely spasmodic, is generally of shorter duration than that whose determining cause is obstruction of the lumen. Wagenmann aptly states that in thickening of the intima, which is common in certain individuals past the age of forty, spasmodic contraction of the retinal arteries that would otherwise pass unnoticed would produce ischemia. Hence the graver prognosis of the affection is precisely as regards this class of subjects. For them the vision



once lost from sudden transient spasm would be less likely to be restored with the passing of the spasm. Leber is one of those who believes that vessel-cramp may be sufficiently prolonged to produce permanent blindness. The prognosis is, nevertheless, favorable, for most of the cases are, from the start, transitory and many of the others are amenable to surgical treatment. Knapp and Berger cured their patients by paracentesis, and Wagenmann relieved his of intermittent blindness by iridectomy.

**Segmentation of the Blood-stream.**—This curious phenomenon occurs in the same classes of individuals and under the same conditions as ischemia. It is indeed the result of sudden stoppage or retardation of the retinal circulation. The agglutination of the red or of the white blood-cells gives rise to the formation of small granular corpuscles carried by the plasma, and whose volumes augment in such measure that the current is retarded. The process results in the appearance of grains so small that each might seemingly be taken for a single blood-corpuscle—for a red cell, magnified fifteen to sixteen times by the ophthalmoscope would acquire an apparent diameter of about  $1/10$  mm. These corpuscular bodies may be seen gliding slowly along the interior of the vessels, and their aspect was compared by Jaeger to grains of sand passing through a glass tube. If the interruption of the circulation is prolonged the entire blood-column becomes a succession of red cylinders separated by yellowish semitransparent spaces (Fig. 41). These last are not, as was formerly supposed, mere empty spaces, but are alternating cylinders of uncolored blood-plasma, while the red cylinders are agglutinated red cells. The segments vary in length, and if two come in contact they fuse into a single segment. They change their dimensions as they progress—those of the arteries becoming thinner and longer to accommodate themselves to the diminishing calibre; the inverse of those in the veins, which grow thicker and shorter till they disappear in the lamina cribrosa.

The behavior of the segments is sometimes paradoxical.



DESCRIPTION OF FIG. 41.

Segmentation of blood column in embolism of central retinal artery.



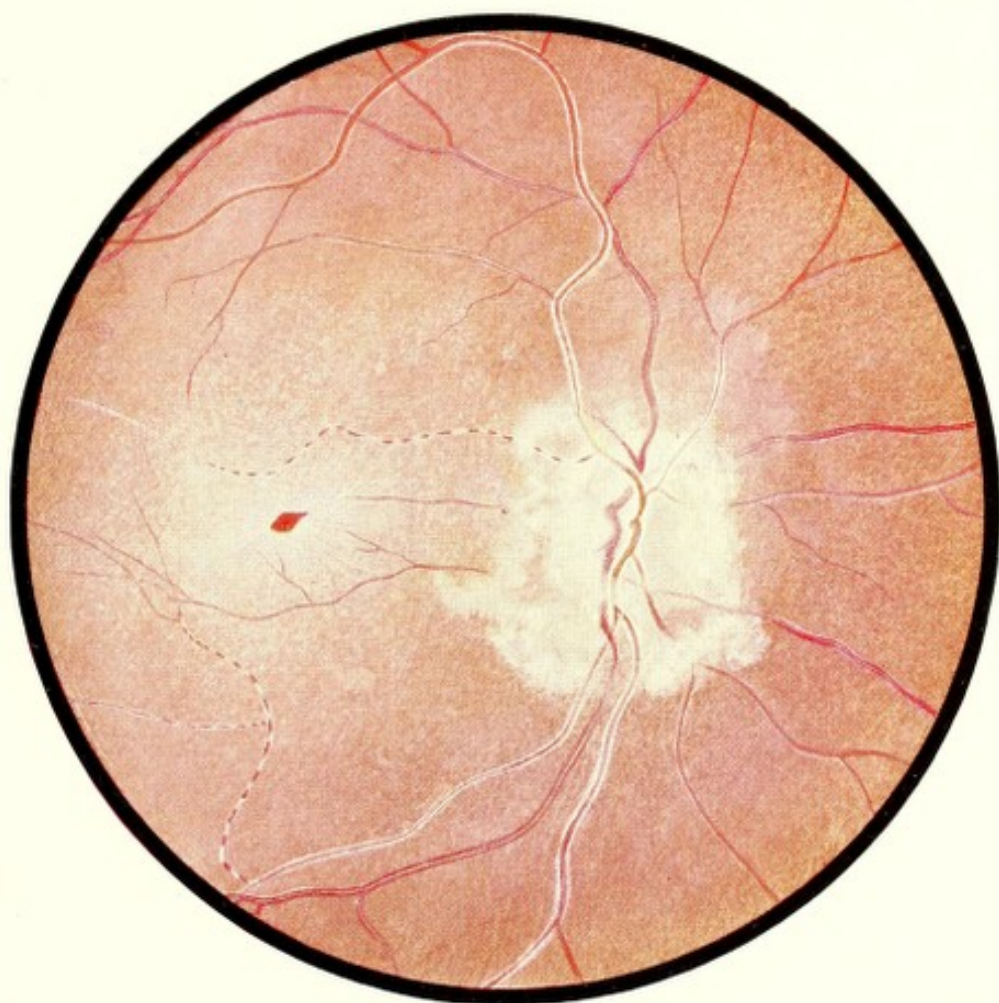


FIG. 41.







That is, instead of moving steadily onward, they become stationary or move backward, or they advance and retreat alternately, in the same vessel. The vessel meanwhile presents a semblance to an old-fashioned necklace of red and yellow cylinders. Segmentation of the blood-column constitutes one of the signs of death. It occurs after traumatic section of the central vessels behind the globe or from pressure or strangulation severe enough to arrest the circulation. It has most often been observed and described in connection with obstruction of the **central artery — embolism and thrombosis**. It affects the veins more often than it does the arteries, and may occur simultaneously in both vascular systems. If the red and yellow cylinders rest immobile, or limit themselves to slight to-and-fro movements without definite progress, it signifies that the interruption is one of *afflux* and concerns an artery, and, according to Reimar, may be taken as proof that the stoppage of the circulation is complete. It does not follow from this, however, that complete arrest of the retinal circulation always results in segmentation. The blood-columns in the retinal vessels of enucleated eyes, for instance, are often found intact, although the eye had previously been made blind by a central thrombosis. In ischemia from vessel-cramp, segmentation is sometimes observed.

**Phases of Real and Apparent Modifications of Calibre.**—Real reduction in the calibre of the retinal arteries is common to the several allied diseases that are characterized by thickening of the intima, viz., **syphilitic vasculitis, albuminuric retinitis** and **arterio-sclerosis**. These affections merge one into another with never any sharp lines of distinction, yet each, at times, presents individual ophthalmoscopic signs that are unmistakable in their significance. The alterations in the retinal vessels, as we see them when isolated, or not accompanied by such distinctive pictures as those of acute diffuse chorio-retinitis (that of chronic interstitial nephritis) are attributable, according to Michel, to phases of primary arterio-capillo-phlebo-sclerosis, and are



usually diagnosed as **endovasculitis obliterans**, **endarteritis deformans**, or simply as **arterio-sclerosis of the retina**, the veins being included by inference. The earlier stages of the arteritis, in most instances, is a thickening of the intima and consequent narrowing of the lumen. This is brought about by cellular infiltration followed by hyalin and fatty degeneration. There is not necessarily proliferation of the endothelial cells, though they are, through their exfoliation and accumulation in the narrower places, instrumental in producing some of the ophthalmoscopic phenomena. For example, through the loss of epithelium the part becomes eroded and favors the lodgment there of cellular detritus, and in this way thrombi are formed.

The disease has a preference for the vessels of small and medium calibre, and it is in them that one must search diligently for evidences of beginning trouble, and it is also in them that are most frequently seen the miliary aneurysms and varicosities of the later periods. Slight departures from the normal are made more manifest by comparing the corresponding vessels of the other eye, or those of another individual of known health. The arteries suffer more than the veins. The process extends to the muscle tunic, where the infiltration and hyalin degeneration are repeated. The walls, deprived of the support of the circular elastic and muscle fibres, bulge outward into sacculations of various forms and dimensions. Eventually the adventitia becomes the seat of hyalin deposits more or less pronounced, and all around the vessels, tending to fill the perivascular space, are accumulations of lymph cells and proliferation of the connective tissue, **perivasculitis**. Veins and capillaries participate though to a lesser extent. The vessels become tortuous, chiefly from elongation due to swelling of the intima, which acts like engorgement, and to relaxation and degeneration of the longitudinal elastic fibres. The final stages are marked by foci of cholesterin and calcareous degeneration, while everywhere are the streaks and plaques of connective tissue. The ophthalmoscopic study of these vas-



cular changes with the view to their recognition and significance, is an interesting and important branch of ocular semiology. We endeavor to know the stage of advancement by the identity of the lesion, and to arrive at some notion as to the probable or possible consequences.

**Ophthalmoscopic Signs of Arterio-sclerosis to be Found in the Retinal Vessels.**—In order the better to perceive and appreciate these signs, some knowledge of the pathologic anatomy of the disease is necessary; hence, the brief résumé contained in the preceding section. So long as the vascular disease is limited to infiltration and transparent hyalin degeneration of the intima, the only visible evidences of alteration in the vessel will be a **narrowing of the blood-stream** by the encroachment of the swollen lining, and, as before stated, to mechanical elongation of the vessel from the swelling itself, which results in increased **sinuosity**. The last is similar to the tortuosity caused by vascular engorgement. The smaller vessels are obviously more susceptible of tortuosity from this cause than the larger ones; hence, an early sign of arterio-sclerosis is the "cork-screw" or the "yarn raveling" distortion of tiny arterial or venous twigs. This symptom is significant, of course, in proportion that the larger vessels are not abnormally sinuous, or that it is accompanied by other corroborative signs. Later the kinking of the larger vessels is accomplished or intensified by the giving way of the longitudinal elastic fibres.

Since the disease is rarely diffuse, the vascular alterations are not uniform over the entire fundus, but, in the beginning, at least, are confined to certain branches, or even to only a portion of a given branch. Incipient endovasculitis, therefore, is often detected by the constriction it causes in a short section of a vessel, and shown by the perceptible narrowing of the blood-stripe at that point. At this time there may be no distentions of the walls; the calibre is either narrowed or it is normal. If the constricted segment chances to cross another vessel so as to be on the side of the observer, the underlying vessel is seen to be obscured for a



distance corresponding to a width somewhat greater than the normal portions of the blood-column in the affected vessel. To illustrate, if one will note closely in observing the retinal vessels of the normal fundus he will see that, whenever an artery, for example, crosses a vein and the two are of fair size, there is a blank or whitish space on either side of its blood-column where the vein is hidden by the walls of the artery. The width of these spaces corresponds, approximately, to the thickness of said walls. It is well to familiarize oneself with their proportions as viewed in health. Should the lumen of the artery be encroached upon by thickening around it, in just that measure would the width of the spaces in question be increased *in excess of the normal*. Were the entire artery constricted, as in vessel-cramp, while the spaces *would still appear*, they would represent only the usual thickness of the vessel's walls. It is an error to assume that only diseased branches show the two blank spaces in crossing. To use a simile that is by no means original with the writer, the diseased artery seems to pass through a notch in the vein that was cut for a much larger vessel. This sort of constriction in the calibre of a vessel must not be confounded with that *apparent* constriction corresponding to the width of "accompanying white lines," to be referred to later.

**Signs Indicating Opacification of the Vessels. Modifications in the Color of the Artery and that of its Central Light Reflex.**—The alterations treated of in the preceding paragraph do not of necessity interfere materially with the transparency of the vessel. If, instead of the transparent hyalin substance, there is an admixture of fatty material, the wall of the vessel becomes translucent, and later, when the period of sclerosis has arrived, the blood-column is still further obscured by a gradually increasing opacity of connective-tissue proliferation. Among the first signs of this opacification is a slight waning in the normal golden-pink glow of the central light-streak of the arteries. It loses warmth of color, but gains in brilliancy in proportion as the



vessel whitens. The tint of the blood-stripe also fades, though hardly to such a degree. The effect of the pallor, together with that of the changed light-reflex, is one of coldness, the two looking first like a "copper wire" and ultimately, when the opacity is complete, like a "silver wire." The only color in the opaque white cylinder, or in its reflex, is such as may be imparted by that of the light that is employed for the ophthalmoscopic examination. As regards the fading of the blood-stripe and the central reflex, the appearances are the same whether the opacification takes place deep in the vessel—*i.e.*, is of the median and

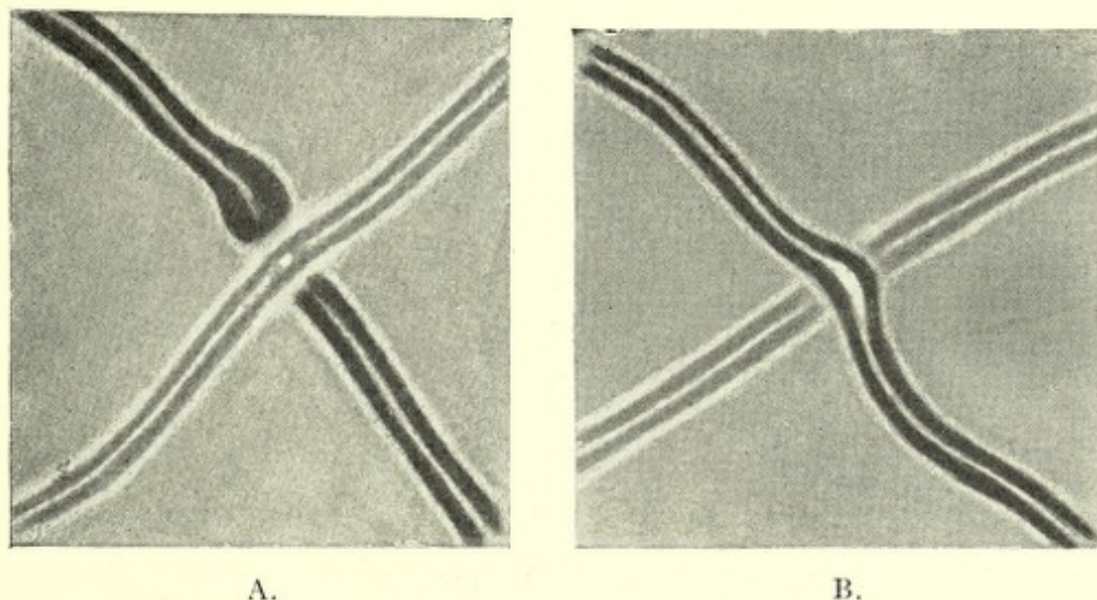


FIG. 42.

internal tunics—or is superficial—*i.e.*, is of the external tunic, or the sheath. While these changes are under way the lessened diaphaniety of the artery may be verified by seeking out a point where it is crossed by a vein. In place of its warm orange, the dot in the middle of the dark red square, or check, has been touched with white. It is the difference that exists between the glowing coal and the burning flame. The lustre of the dot is heightened as the artery grows harder, for the reason that the more the overlying vein is pressed upon, the more it yields—is transversely grooved by and spread out upon the artery—thus



thinning and rendering more translucent and redder its own blood-stream (Fig. 42, A). It is possible, too, that the vein may be seen to rise, or climb, over the artery, and its broadened light-reflex emphasizes the impression of the flattening it undergoes in doing so. At this period, other phenomena may be noticed where the sclerosed artery lies upon a vein. The pressure of the stouter vessel with the aid, perhaps, of the impingement of high arterial tension, so narrows the lumen of the vein as to retard its blood-current; the consequence is that where the vein meets the artery on the peripheral side, or side of afflux, it becomes perceptibly clubbed, or bulbous (see Fig. 42, B). On the side of efflux the vein may be even slightly attenuated. Moreover, it is also evident that the artery has driven the vein deeper into the substance of the retina, where the crossing occurs. This is evinced by the **dip** of the vein on either side of the artery, and the darker color of the contained blood at those points, produced by viewing it endwise. In the event of an endophlebitis at this place the scene is all set, as it were, for the production of a thrombus. As a matter of fact, a favorite site for thrombi, as well as for hemorrhages, is at just such crossings.

**White Outlines.**—A characteristic sign of all varieties of contraction of the lumen, and of inflammatory and degenerative changes in the vessel walls, is the appearance of white lines that border the edges of the blood-stripe. They are common to edema of the retina and to all acute forms of neuro-retinitis, in consequence of serous and cellular infiltration of the perivascular spaces. They are then fine, and of uniform breadth and **grayish** or pearly whiteness. They are constant at certain periods of arterio-sclerosis; their advent being more prompt as regards the arteries, and their development always relatively less as regards the veins. They are peculiarly broad and white and of less uniform width in the retinal vascular sclerosis of old syphilitic cases and that of elderly subjects—**senile arterio-sclerosis**. In these cases there is occasionally seen an exaggerated develop-



ment wherein the trunk of every vessel in the papillary region is attended by strips of white so broad that in converging toward the lamina cribrosa they almost hide the disc (see Fig. 43). They appear to grow in width from the outside toward the mid-line of the vessel, and the width of the blood-column seems to correspond to the distance between the lines. This is not, of necessity, true—in fact

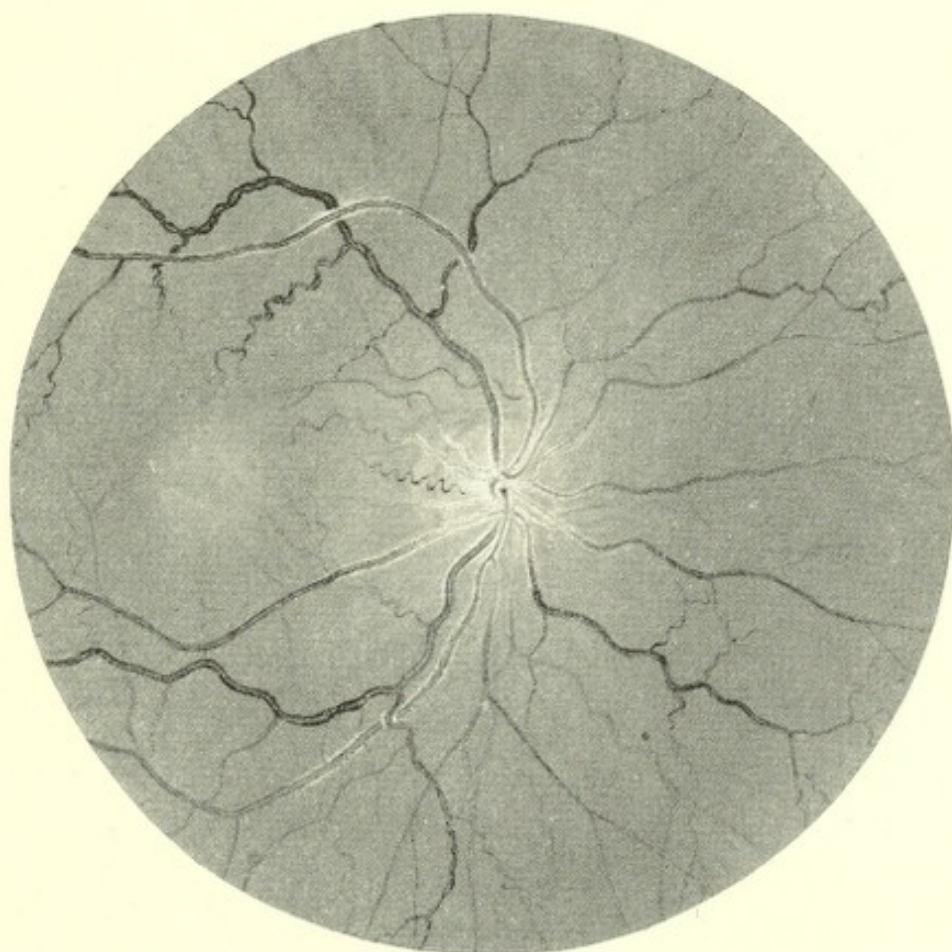


FIG. 43.

it seldom is, for the outer limits of the column are usually concealed by the inner limits of the white stripes. In other words, the reduction in the calibre of the vessel is only **apparent**, or specious. The progressive growth from without inward is also but seeming, as it is but another effect resulting from the cylindric form of the vessel. The whitening is, in reality, uniform over the entire vessel, but is intensified at either side where the walls are presented perpen-



dicularly to the eye of the observer; and the blood-column is encroached upon in accordance with the *density*—not the *width*—of the intervening white substance. The obscuration of the blood-stripe is *partial* or *complete* owing to the permeability or impermeability of its covering to rays of light. The degree of permeability of its **lumen** depends upon the seat of the material that imparts the opacity. If it is something which transplants the internal and middle tunics there will be great narrowing of the lumen, often even to obliteration. If it is a proliferation of connective tissue which concerns only the external tunic and the space immediately surrounding it, while it may be more or less contracted, the calibre is usually patent, and likely to remain so.

Unfortunately, one cannot with the ophthalmoscope always make fine distinctions in the phases under discussion. The primary seat of the disease is commonly the intima, and its progress is from within outward, so that one has relatively few opportunities of observing vessels in which the thickening or opacity is superficial and unaccompanied by any deeper affection. The blood-column is most often visible in some part of the vessel, or in one or more of its branches, though not infrequently stem and ramifications are mere white streaks. If the trunks on and near the disc are all whitened, as is often the case in senile arterio-sclerosis, and the medium and smaller branches are free from the streaks, or the latter, if present there, are barely perceptible, and their blood-columns are about normal for the age of the subject, we may conclude that the degeneration of the middle and inner tunics, if present, is not grave. It is supposed that this heavy coating of protective tissue may be provided to re-enforce the walls of the vessel and fortify it against the enormous blood-pressure which often exists, particularly when there is hypertrophy of the heart. It must not be forgotten that, owing to the extreme variability of the lesions in these circulatory diseases, what is true of one vessel of the fundus can by no means be ascribed to the



others. Some escape entirely, while others are profoundly involved (Figs. 44 and 45). Some exhibit one phase of the disorder, and some another; and all periods and all degrees may be represented in the same fundus (Figs. 46 and 78)—it is hardly going too far to say in the same vessel. Not infre-

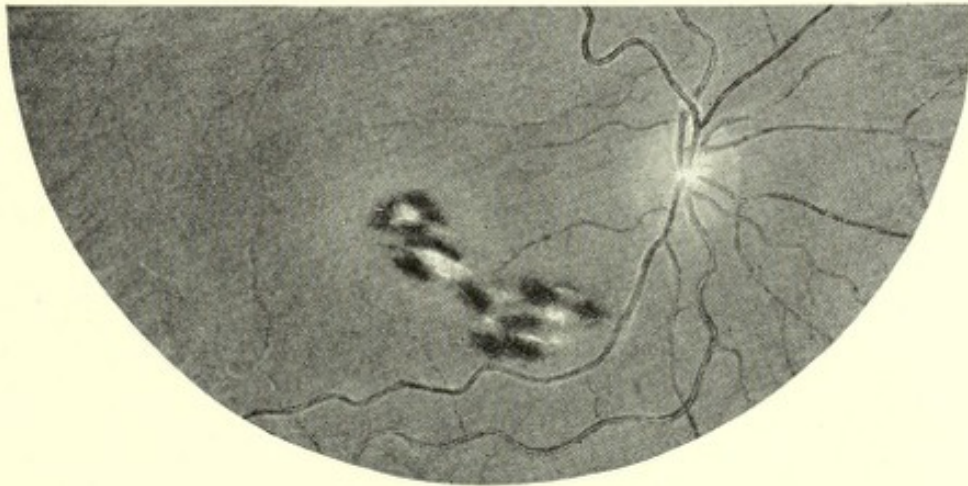


FIG. 44.—Shows recent endarteritis obliterans of the infero-temporal artery at its second large bifurcation.

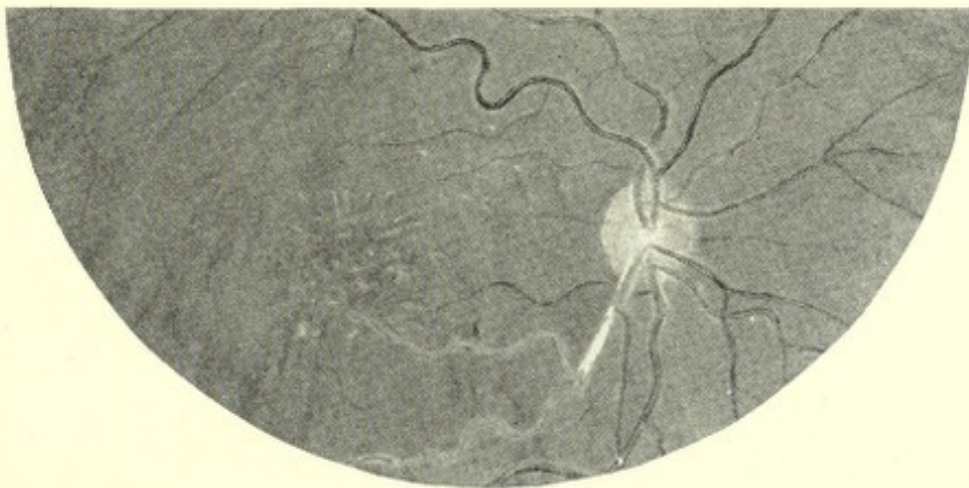


FIG. 45.—The same case nine months later.

quently the retinal vessels of one eye are in an advanced state of inflammation and deterioration and those of its fellow are essentially sound.

**Haab's Scales.**—Professor Haab attaches considerable importance to this sign, which he believes to be pathognomonic of syphilitic degeneration of the arteries. The



lesions consist of tiny white patches, suggestive of scales, scattered here and there over the walls of the retinal arteries. They may be so small that only close scrutiny, by the direct method, will disclose them. Haab considers them to be the beginning of opacification of the vessel.

**Dilatations.**—When any part of the lumen of an artery is nearing obliteration as a result of endarteritis, the blood-

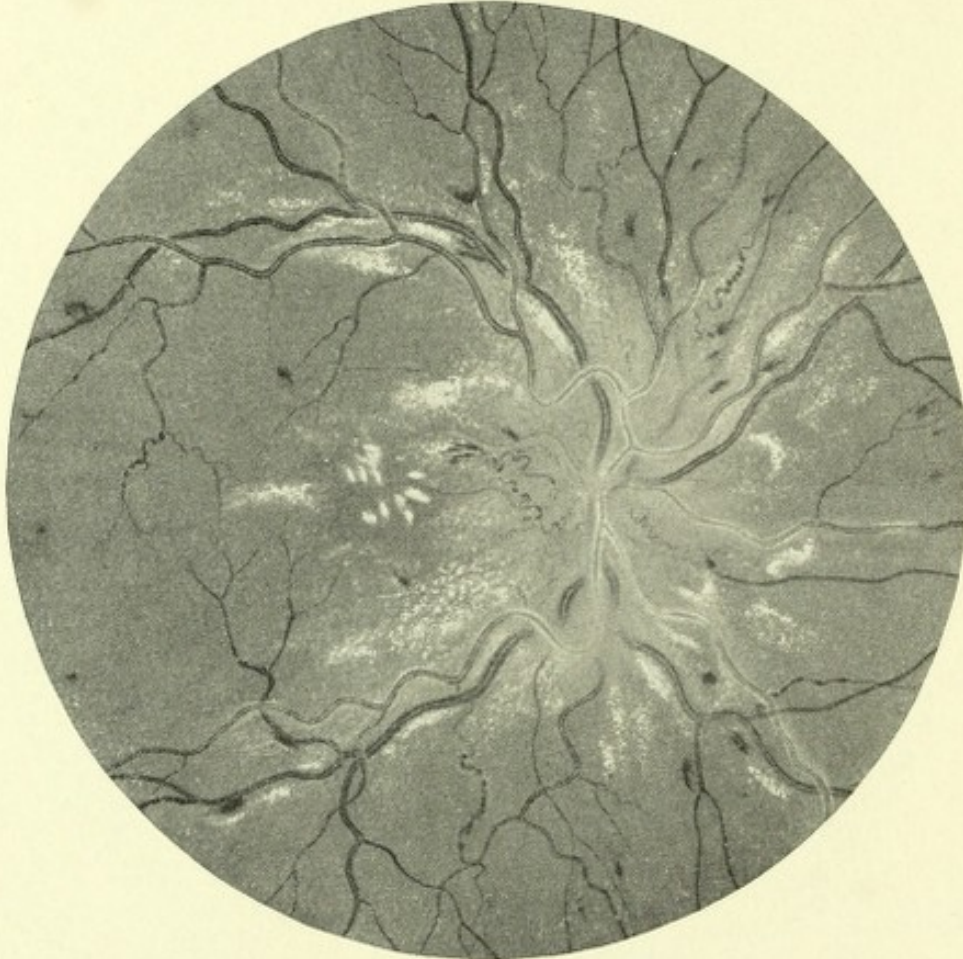


FIG. 46.—Many phases of vascular degeneration in the same retina.

pressure is not transmitted through the capillaries and there is stasis of the corresponding vein, shown by engorgement with increased sinuosity and darker color. This is stasis from lack or cessation of *vis à tergo*. When any part of the lumen of a vein is nearing obliteration, all the branches on the peripheral side of the obstruction become congested, etc., from excess of *vis à tergo*. This is a most critical period. In addition to the retinal lesions



there is probably hypertrophy of the left verticle. In consequence of the stasis, the high blood-pressure and lax and softened tissues composing the walls of the vessels, the veins become the seat of ectasias of varying forms and dimensions—**varicosities**—either sacciform or more or less elongated, and the weakened inner and middle tunics of the arteries give way and their thin external tunics bulge into spheroid cysts or **aneurysms**. Serous and cellular infiltration of the papilla and retina ensue, a few neoform twigs may appear in the vicinity of the disc, and the smaller vessels and capillaries lose their tonus. The time is ripe for thrombi to form in the narrow places within the vessels, and for hemorrhages to occur along the courses of the tiny porous twigs and capillaries, and from the sacculations, both venous and arterial. It is not to be presumed that this is the regular order, or disorder, that is to be definitely looked for or expected, as the picture is something of a composite one; yet practically all these features obtain at some period in all well-marked cases of retinal arterio-sclerosis, if not simultaneously, at any rate serially.

**The Retinal Vessels in Retinitis Pigmentosa.**—This would seem a fitting place to refer to the peculiar nature of the vascular changes that characterize this curious disease—called also **congenital pigment degeneration of the retina**. The vascular alterations here are in marked contrast to those just described. Their primary seat is in the median tunic, the intima remaining but little altered and while the vessels grow smaller their calibre is but slightly reduced, relatively speaking, for a very long time. Hyalin degeneration ensues and the entire vessel finally becomes a homogeneous, feebly translucent, waxy cord, without cells or elastic fibres. In cross-section the lumen of the arteries remains wide open. The peculiar ophthalmoscopic feature of the vessels is the unique manner in which they seem to dwindle away after leaving the papilla. They are not followed by white lines, and their gelatinous walls are just impervious enough to mask the blood-stream laterally, and



of just the taint to mingle best with the surroundings. By carefully seeking along the trajects of the vessels one may find vague, shadowy outlines, paralleling the blood-columns. These outlines occasionally show faint tints of blue or yellow, or, rather green. They represent the partly transparent, thickened vessel walls. The blood-columns, in the advanced stages, appear as thin, pale streaks of carmine, without the central reflex—the veins and arteries differing but slightly in depth of tint. In advanced cases the arteries lose themselves a short distance from the papilla, but by searching in the direction indicated by the vanishing trunk one can often succeed in picking it up again and tracing its thread-like stripe of red for some distance, even into the ramifications of retinal pigment (Fig 11, page 209). At the same time microscopic examination of the vessel may show that its external dimensions remain unaltered. Allusion has already been made to certain distinctive characters of the papilla in this disease.

**Arterial Pulse.**—Ordinarily, pulsation of the retinal arteries is either invisible or so slight as to require the closest attention to make it perceptible. Haab says that if one of the retinal arteries at the disc is sharply curved, particularly if it is bent in the form of an S, one can always detect pulsation in it at the loop. This is a pulsation of *locomotion*, and is characterized by a rhythmic dilation, like the *bounding* of the radial artery—or an actual *displacement* of the loop in question, with augmentation of its curve. An exaggeration of this physiologic pulse occurs in aortic insufficiency, as first noted by Quincke, in 1868, and often verified since. Added to the valvular defect of the aorta, however, must be cardiac hypertrophy, so that there is a very rapid rise of arterial tension followed immediately by as rapid a depression. Under such conditions, and in an eye of normal tension, the motion may be propagated even to the peripheral arteries of the retina. The physiologic venous pulse is also exaggerated and greatly extended over the fundus, and there is sometimes a corresponding capil-



lary pulse, manifested by a flushing of the papilla in systole and a blanching of it in diastole. The changes in calibre of the arteries are most noticeable just where the trunk is about to bifurcate. They cease on light compression of the carotid, and are either augmented or not affected by pressure upon the jugular.

The more violent the heart's action, the more intense the pulsation, and any tendency to aortic stenosis will counteract it. It is indicative of pronounced differences or alternations in the arterial tension. Becker frequently observed this pulse in Basedow's disease, and Raehlmann noted it in two-thirds of the cases of the retinal hyperemia of chlorotic patients, and Schmall saw it in twenty out of fifty-five cases of chlorosis. It was slight, however, and save in grave cases, did not extend beyond the papilla. In the lightest cases the only visible evidence of a pulsation was a simple alternate broadening and narrowing of the central light-streak in the bends of the artery on the disc. It is frequently observed in pernicious anemia, being usually accompanied by dilated veins and retinal hemorrhages. A number of explanations have been offered to account for the presence of this pulse in chlorosis. As, for instance, diminution of the specific gravity of the erythrocytes—they being smaller and less numerous, permitting the heart to push the blood-columns further at a single beat (Raehlmann); and lessened weight, whether of the red cells or of serum (Zumpf). More plausible seems to be that of Thoma, who attributes it to an atony of the arteries due to the alteration in the composition of the blood. Pulsation is said to be of greater relative frequency in cilio-retinal arteries than in those of the central retinal system.

Another retinal arterial pulse, and one of greater semio-logic value, is that first observed and described by von Graefe in connection with glaucoma, of which it is frequently a prodromic sign. It is a spontaneous rhythmic phenomenon of the retinal arteries at the papilla, which is the result of a disproportion between the blood-pressure and the intra-ocu-



lar tension, with preponderance of the latter. In the normal eye, these two pressures, being in equilibrium, the arterial current is kept virtually constant; but as soon as the intra-ocular tension increases (as in glaucoma) or the blood-pressure diminishes (as in syncope) the central artery is compressed, the blood can only enter it by spurts, and this form of pulsation is established. It consists in a regular intermittence of the afflux of blood in the retinal arteries whereby their papillary extremities are seen to be alternately filled and emptied in time with the heart beats. The filling corresponds to the cardiac systole, and starts at the centre of the disc and travels toward the border, where it usually ceases. Then follows the emptying, which is synchronous with the diastole of the heart, and passes from the border of the papilla to the point of emergence of the vessels. It may be limited to one or two branches, or it may extend to all the large stems in the papilla. As before stated, it expresses a want of equilibrium between the arterial and the intra-ocular tensions.

When the pressure of the vitreous body is in excess of that of the blood, the arteries are depleted in the manner just described, and it is only by another cardiac impulse, or systole, that the blood can again penetrate the eye at the lamina cribrosa. So it is in glaucoma in which to an elevated vitreous tension is joined a normal blood-pressure. The same imbalance would obtain in reduced arterial tension, that of the vitreous or the intra-ocular, remaining normal. It is to the latter condition that is due the retinal pulsation in syncope—from feeble heart-beat—as also the arterial retinal pulse sometimes seen in connection with retro-bulbar tumors and neuro-retinitis, where the tension of the central artery is so diminished by the strangulation that it is overcome by the physiologic tension of the globe.

It follows from this that a spontaneous pulsation of a retinal artery is not an exclusive sign of glaucoma. This pulse can be readily produced experimentally by making progressive compression with the finger upon the globe of



the eye under examination. This is true particularly as regards the young and the middle-aged. In elderly subjects, especially those characterized by arterio-sclerosis or high arterial tension, it is sometimes difficult to produce the pulse even by considerable pressure. Jackson, of Denver, states that under these conditions it is sometimes even impossible to elicit the pulse by pression. Hence, absence of the arterial-retinal pulse upon induced hypertension becomes a sign of high blood-pressure. If there is the slightest hypertension of the eye, the lightest pressure, sometimes merely a touch, will suffice to cause a perceptible pulsation, whereas, if the tension is physiologic, a much stronger pressure is required to produce it. It is not then a phenomenon peculiar to only *immoderate* hypertension. Therefore, its value as a prodromic sign of glaucoma, as *it may be the only sign present*. Of course, the tonometer might indicate the plus tension, when palpation would not. It is spontaneous and fairly constant in acute glaucoma, but seldom exists in the chronic forms, even when the tension is inordinate.

In chronic glaucoma not accompanied by excessive arterial tension, or by arterio-sclerosis, there would seem to be some compensatory changes in the retinal vessels whereby their patency is maintained. These facts led Schnabel to suspect the concurrence of other factors in the apparition of this pulse, chief among which his investigations induced him to regard hyalin degeneration of the retinal arteries and spasmodic contraction of their muscular tunics. The arguments upon which he bases his opinion are as follows:

1. When the tension of the globe is raised by compression with the finger, for example, all the vessels at the papilla can be observed to flatten, and the disc to pale, while in glaucoma, the arteries are attenuated, the veins dilated, and the disc reddened, showing that there must exist in the glaucomatous eye some circulatory disturbance independent of mere vitreous pressure.

2. As an expression of that disturbance one may some-



times perceive a dilatation of the fine papillary vessels even when the optic nerve is not yet excavated.

3. If the pulsation were due solely to the hypertension it should characterize all the retinal trunks and all the glaucomatous eyes, which is not the case.

4. Sometimes the pulsation diminishes or again disappears after the instillation of a solution of eserine, the tension meanwhile remaining unaltered. This proves (to Schnabel) that, in the retina as in the iris, the drug acts directly upon the spasmodic condition of the vessels.

DaGama Pinto says, "The clinical observations upon which Schnabel bases his arguments are, for the most part, contestable, nevertheless, we believe that the dynamic alterations in the vascular walls constitute an important factor in the production of the arterial pulse of glaucoma."

**The venous pulse** is the true correlative of the arterial pulse last described. It consists in a regular intermittence in the *efflux* of the blood in the retinal veins, whereby their papillary extremities are seen to be alternately enlarged and diminished in time with the heart-beats. Like the arterial pulse the enlargement, or *filling*, corresponds to the cardiac systole, and the diminution, or emptying, to the diastole. In one sense only is there any reciprocity of the venous stream with that of the arteries, namely, while the artery is filling the current of the vein is shut off. The arteries and veins empty synchronously and in the same direction. Usually the venous pulse is limited to one or two of the large stems and is visible only within the papilla. The veins are much more easily influenced by trifling imbalance in pressure within and without, and visible pulsation in them is too common to be of special significance. It is well named "**physiologic venous end pulse.**"

In this connection, however, attention is called to two other phenomena relative to the veins at the disc, and to excess of intra-ocular tension: the flattening of the veins and pallor of the nerve-head, the last evidently an expression of capillary ischemia in this location. These modifications are



barely appreciable and possess no real diagnostic value except in that period of glaucoma which precedes any signs of pathologic cupping. During the access of hypertension the venous trunks, compressed at their exit, are seen to dilate a little outside the centre of the disc, and to be actuated by lively pulsation which affects a greater number of vessels than is usual. At the same time the papilla loses some of its rosy tint and becomes paler. To better appreciate these appearances, the degree of pigmentation of the eye-ground should be taken into consideration, as the contrast between disc and surroundings is proportionate to the amount of pigment. Moreover, it is always best in trying to arrive at conclusions relative to what one sees or does not see with the ophthalmoscope, to compare the corresponding parts of the other eye, if it be normal. In instructions given by Haab for seeking retinal pulse phenomena, which apply equally well here, he says, "To study them carefully it is well to examine the patient while seated in a chair instead of in bed. The surgeon should, if possible, also be seated, and support the arm that holds the mirror on a table. If these precautions are neglected, the pulsating movements of the surgeon's arm and trunk will deceive him when he looks into the eye and make him think that the vessel he is examining is pulsating. The same error may be produced by pulsating movements on the part of the patient. Auto-suggestion plays an important part. If the observer is very anxious to see a pulsating vessel he will seem to see things pulsate which in reality do not show a trace of pulsation."



## CHAPTER XV.

### MACULAR AND POSTERIOR POLAR REGIONS.

The **macula** or **fovea**, together with its **foveola**, constitute the optic or geometric centre of the fundus, while the papilla is the anatomic centre. The **posterior polar region**, roughly considered, is the area bounded outwardly by the fovea and inwardly by the papillary region, and on all other sides by the larger branches of the upper and lower temporal arteries and veins. Ordinarily, it is a zone free from the larger vessels, but owing to the extreme variability in the distribution of the retinal vessels, one of considerable size is often seen to traverse the entire area, all but touching the margin of the macula. It is most often an artery. Just as in other regions where the retina, save for the small arteries and veins whose thin red lines converge from all directions toward the macula, the retina here is invisible, these vessels and certain reflections from its surface, constituting the only landmarks. For the rest, any physiologic markings occurring in the regions in question, are those of the corresponding portions of the choroid.

The following description of the **normal appearances** has reference to the retina of a child or an adolescent, as that of an older individual would be considered less representative. In accordance with the latest and more consistent nomenclature the word **fovea** will be used as synonymous with **macula**, and **foveola** as meaning the tiny depression with its central reflex which marks not only the exact centre of the macula but also that of the fundus (see frontispiece). The foveola is situated slightly outward and downward from the center of the papilla. The papilla—as it is magnified by the direct method with the ophthalmoscope fourteen to fifteen diameters—should have an apparent diameter of  $7/8$



inch. Imagine a horizontal line drawn  $1/8$  inch below the inferior margin of the disc, follow it outward—toward the temple,  $2\ 1/4$  inches from the central line of the disc, and there is the centre of the foveola. In reality it is about one millimeter downward and four outward from the centre of the disc. All that one sees of it is the light-reflex that indicates its bottom, just as a similar reflex will appear in the bottom of a china cup. Its reflex, then, is of microscopic dimensions, in reality about  $1/250$  inch in diameter, or less. This is crescentic, or circular, owing to the incidence of the light, that is, it is circular only when the light is reflected exactly in line with the optic axis. As the rays of the mirror approach the perpendicular, they are occasionally shot out from the foveola like a tiny fan or the tail of a comet, an effect called by Gunn "**policeman's lantern reflex.**" The circle in our magnified image has an apparent diameter not exceeding one millimeter. When the foveola is exceptionally broad and shallow, as, for example, in certain instances of axial myopia, its fundus-reflex sometimes becomes peculiarly spectral, and may be made to shift its position **bodily** with the movements of the observer's head.

The brink of the foveola is slightly elevated and marks the point where the layer of ganglion cells suddenly drops from a thickness of eight or nine cells to that of a single cell. The rim is invisible with the ophthalmoscope except under a few pathologic conditions, such as in that strange malady known as **amaurotic family idiocy**, when it is clearly defined, and its apparent diameter is seen to be about 4 mm., or  $5/32$  inch (see Fig. 52). The enclosed shallow bowl almost makes a hole through the retina, for the thickness at its fundus is merely that of the layer of cones, the outer nuclear layer, the fibres of Henle and the scant layer of single multipolar cells. The foveola is devoid of vascular circulation. The choroid appears of darker color seen through this thin spot. In a light colored fundus one can get a pretty fair idea of the apparent dimensions of the foveola by its bright red color as contrasted with the slightly



more veiled tint of the surrounding macula. A very faint reflex has occasionally been seen playing about the outer limits of the depression.

Very common errors are those of calling the foveola the **fovea centralis**, or simply **fovea**, and taking the tiny crescentic reflex, which *indicates the deepest part of the depression*, or **central fundus-reflex of the foveola** for that of the **rim of the depression**. The macula or fovea is a shallower, and much larger depression, of ellipsoid shape—long axis horizontal—of an apparent diameter about equal that of the

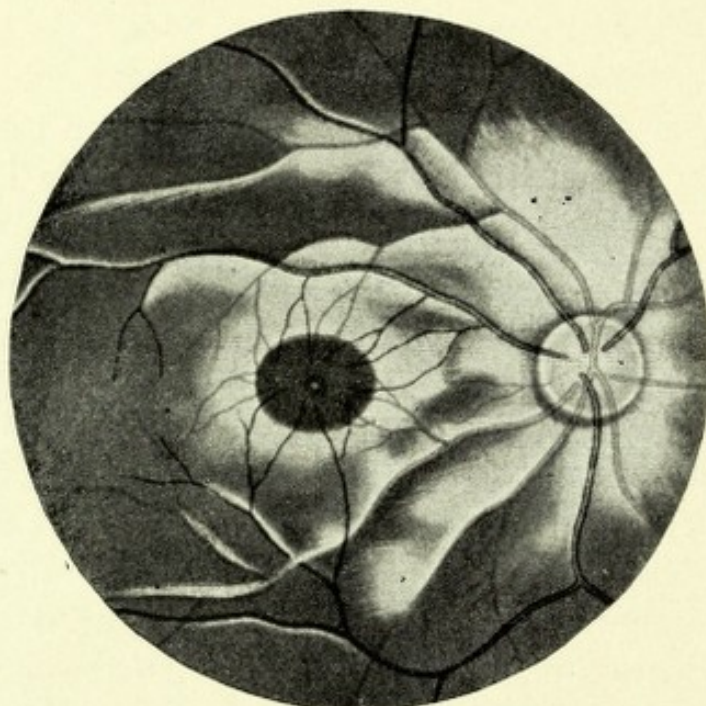


FIG. 47.—Retinal reflexes. (After Haab.)

disc—say  $7/8$  inch vertically, and 1 inch horizontally. Its outer limits are made manifest in the young subject by a barely perceptible spectral reflex, in the form of a thin halo. By the direct method, a concave mirror, good light and with the pupil moderately dilated, this play of light about the rim of the fovea may sometimes be very strikingly brought out. The sheen darts and flickers, now broadening and extending far into the surrounding polar zone in a manner suggestive of the auroraborealis, again narrowing down to a thin ring, but never extending into the area of the fovea



itself (see Fig. 47, taken from Haab's Atlas). This phenomenon is identical with the brilliant shifting reflexes that also follow the courses of the retinal vessels in these young subjects, commonly described as "shot silk," "watered silk," and "moire" effects. They are caused by light reflected from the surface of the *membrana limitans interna*, where it is slightly lifted or pushed inward by the blood-vessels. The halo about the macula is seldom visible in adult life. The central fundus reflex of the foveola, however, is often visible, by the direct method, till middle age.

The hexagonal epithelial cells of the macula are richer in pigment than elsewhere in the retina, which in many instances gives to this area a dusky hue. It is the variation in the distribution of the pigment granules in these cells that gives the retina, as a whole, its granular, or *granitoid* appearance. In most eyes, certain cells or groups of cells in the fovea are transparent, or free from pigment, permitting the red-orange tint of the chorio-capillaris to show as tiny dots. These have been named **Gunn's dots**, after the late Marcus Gunn, of London, who first described them. They are located chiefly about the brink of the foveola, and have no pathologic significance. They are to be differentiated from **drusen** of the lamina vitrea, from **Tay's chorio-oiditis**, and from forms of macular **chorio-retinitis**, such as the **subacute syphilitic** and the **senile**, or **sclerotic**, discussed later.

The original name, **macula lutea**, as given to the fovea, has been responsible for much confusion, for so long as the retina is in its normal state it is perfectly transparent, remaining so for a short time even after death or enucleation. There is no color in the normal retina at the fovea. The name was bestowed because of the yellow tint that appears there in the dead and opacified retina, and which shows only when the membrane is stripped from the globe and viewed by transmitted light. At the same time the foveola, being so thin, looks like a pin-hole—colorless and transparent. Yet one often reads that the macula can be distinguished,



in the ophthalmoscopic examination, by its yellow hue. Moreover, the outline of the yellow portion does not necessarily coincide with that of the brink of the fovea, but may extend beyond it. Another fallacy, frequently reiterated, is that the macula (or fovea), is deprived of blood-vessels. In truth it is very vascular—the vessels being of just the sort that count. Small twigs of arteries and veins may be traced with the ophthalmoscope, sometimes as far as the outer limits of the foveola, while entopic images and injected anatomic specimens attest, by the radiating and anastomosing loops of capillaries they show, to the abundance of the blood-supply of the fovea.

The capillary loops that constitute the inner limits of the system are situated in the rim of the foveola. It is possible, at times, to perceive an extremely delicate striation radiating from the foveola, either confined to the area of the macula or extending somewhat beyond it. In the normal eye this is not usually a reflex, but is in the substance of the retina, and is due to the obliquity here assumed by the fibres of Müller, which in other places are perpendicular to the plane of the retina. In edema of the macula, the striation becomes more distinct, takes more the nature of a reflex, and may extend in an inward direction as far as the papilla. The lines indicate the course of the optic nerve-fibres, by which they are formed. These anatomic features, that is, the radiation of macular blood-vessels and of neural fibres is doubtless, in great measure, accountable for the spoke-like disposition often assumed by inflammatory and degenerative products in this locality, as, for example, in albuminuric retinitis.

**Modifications in the Aspect of the Macular and Posterior Polar Regions. Coloboma.**—A congenital defect in this locality is not a true coloboma in that its origin can have had nothing in common either with the normal fetal cleft nor with the formation of the fovea and foveola. Usage, however, sanctions the terms **coloboma of the macular region** and—simpler and more appropriate—**central coloboma**. It is,



then, an atypic coloboma. It consists in congenital absence of the choroid at the centre of the fundus. It is either ellipsoid, irregularly oval with long axis horizontally, or of bizarre outline, and its size is from one to ten disc diameters. It is usually framed more or less completely with pigment. The entire area of the defect presents naked sclera, or it is mottled throughout with pigment (Fig. 15). Again, it is bluish or grayish in places, indicating ectasias of the sclera at those points. A few choroidal vessels are usually present, either meandering across the spot, or one or more of the short posterior ciliary arteries may be observed perforating the sclera within its borders. It is often crossed by the larger retinal vessels, which, by their *dip* and their refraction, often show that the coloboma is considerably excavated or otherwise indicate its topography. It is more often monolateral, and affects the left eye by preference.

It is likely that some of the densely pigmented of these defects, and those of very irregular outline, are of inflammatory origin—from prenatal specific choroiditis, for example; and it is more than probable that a number of them are the result of those extensive hemorrhages that are so often to be observed in the funduses of new-born infants (see chapter on "Hemorrhages"). The more symmetric among them, however, especially when they contain permeable choroidal vessels, and when the retinal vessels that traverse their surfaces are of normal appearance, plainly bespeak errors of development. The papilla, as a rule, is separated from the defect in the choroid by a bridge of normal tissue. The exceptions are likely to be of the irregular and pigmented kind. The papilla is often anomalous—that is, it shows conus outward, or temporal cleft of the nerve-fibres—and the central vessels deviate from the normal in number and distribution. It goes without saying that the retina over the defect is devoid of function.

**Alterations caused by Age.**—As has already been stated, the halo around the macula disappears with adult life. Later the fundus-reflex of the foveola also goes. The pigmentation



of the hexagonal epithelium appears less and less uniform as the years advance, resulting in accentuation of the physiologic granitoid stippling, and the color of the underlying choroid looks faded. So far the changes are only such as may be naturally expected in the macula of an aged subject, and are so constant as to be considered physiologic; yet it is only a step to a more serious condition, that is, to a

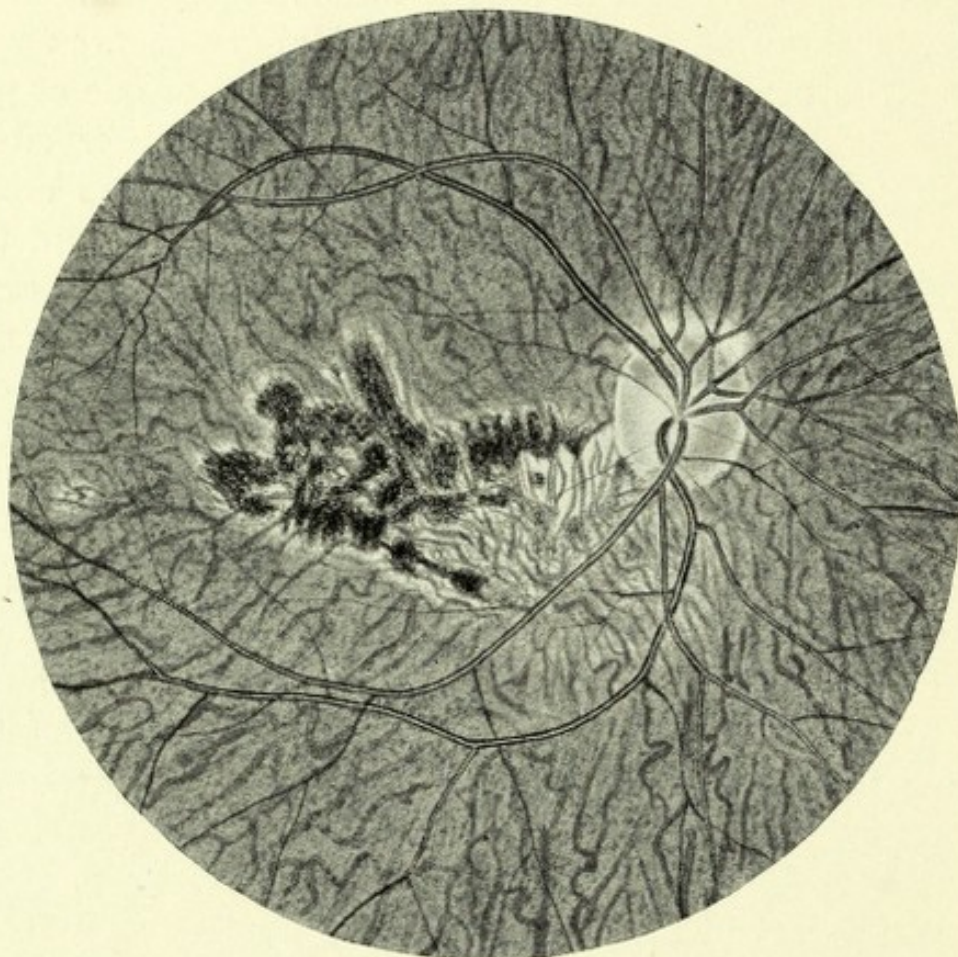


FIG. 48.—Central coloboma.

more pronounced mottling—a coarser and darker stipple of pigment, interspersed with small pinkish spaces. The patient complains of reduced visual acuity, inability to read, etc. There is a decided central scotoma. The stage has passed that of mere depigmentation. It has become a senile atrophy of the deeper portions of the choroid, and as it progresses the sclera often participates in the ophthalmoscopic picture, as shown by a gradual fading of all color at



the centre till only yellowish or grayish-white, with a sprinkling of pigment, remains. The latter usually takes its place about the periphery, leaving the clearer space at the centre. The alterations are slow to occur, save in exceptional cases, when central vision may be lost in a few months. It is usually bilateral and self-limiting—not extending beyond the fovea. The sight is rarely affected before the age of sixty. Its frequency after that period is in direct ratio to the age of the subjects. In connection with beginning cataract it would be grounds for a guarded, or an unfavorable prognosis, or for actual non-interference, owing to its extent and to the general circumstances of the case. Certainly a fair degree of *peripheral* vision is better than the blindness of mature cataract. We are indebted to Haab, of Zurich, for most of the observations relative to this **senile macular atrophy**.

**Modifications due to Injury. Contusion of the Macula. Commotio.**—According to an older nomenclature this is concussion of the retina. Although the macular appearances in this affection are, in most instances, but a small part of the disturbance to be seen in the fundus, they are sufficiently peculiar to deserve description in this place. The appearances in question are most often seen in conjunction with a much larger area of **commotio retinæ** in another part of the fundus. As first described by Berlin, in 1813 (**Berlin's Opacity**) the larger opacity corresponds to the area of *direct contusion*, the smaller, or macular, to that form *contre-coup*. Either may be present singly. The lighter blows are not often productive of the *contre-coup*. The latter is opposite the point of attack, hence to be at the macula, the cornea, or some other portion of the anterior half of the globe must receive the blow. The two are not of necessity diametrically opposite, for it is the *direction* of the blow that partly determines the seat of the secondary injury. It is probable, too, that the macula suffers because of its greater susceptibility to this sort of traumatism. Often the effects of both *coup* and *contre-coup* upon the retina are visible with the oph-



thalmoscope. The opacity resulting from the coup is the first to be seen and last to disappear. It will be treated of differentially later. The opacity of *contre-coup* is often in the macular region. It takes the form of a crescent whose curve is about concentric with the border of the fovea. The arc of the opacity may extend so as to subtend nearly the entire circumference of the fovea. It is milk-white in the

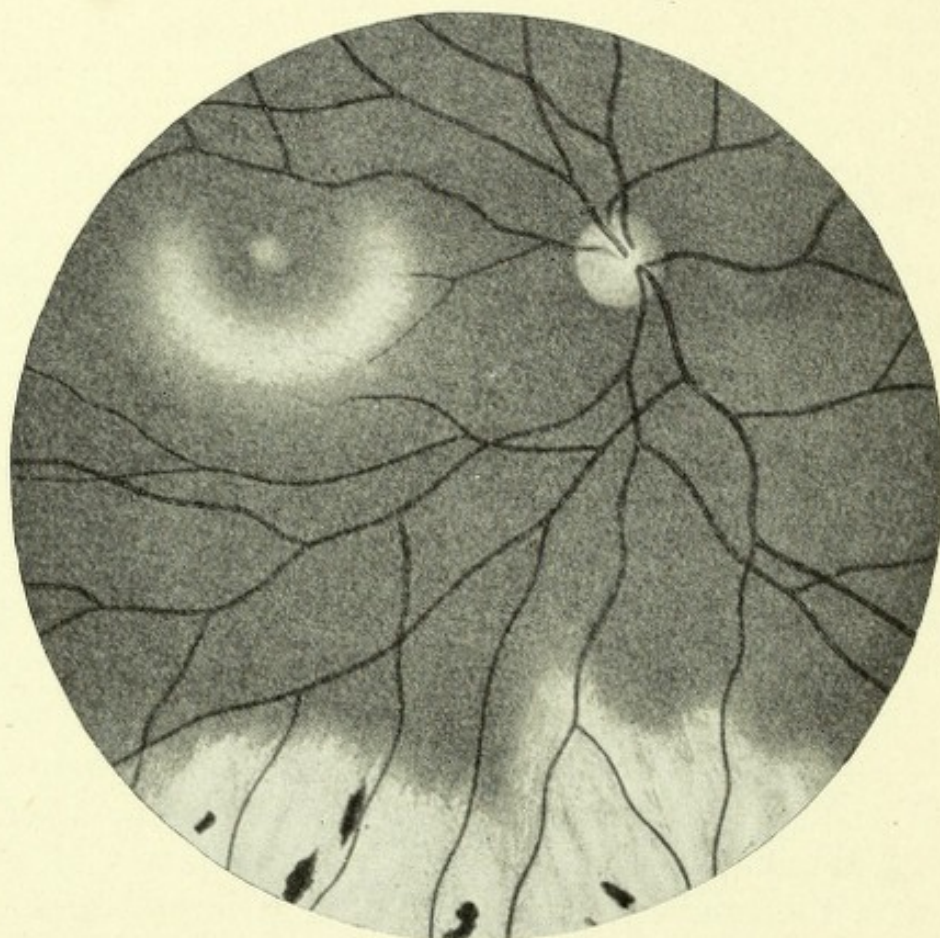


FIG. 49.—Commotio retinae.

denser portions, and its limits gradually merge into the color of the fundus.

Haab has noted an interesting peculiarity of the foveola that is sometimes observed in this connection. It appears as an isolated, more or less distinct, circular gray opacity. It is probably situated deeper than the crescentic opacity. It also is transitory and its size about equals that of the foveola itself. Haab's drawings of the macular region in



these cases suggest a picture of the Turkish emblem of **star and crescent**, that is out of focus (Fig. 49). Among the commoner bruising agents that produce **traumatic opacity of the macula** are billets of wood that fly in chopping, pieces of metal, balls, fists, and corks. The opacity becomes almost immediately manifest, and, barring complications, such as extensive retinal hemorrhage, rupture and detachment, it clears up in a few days without leaving a trace. Favorable appearances, however, do not warrant an unreserved prognosis of the same nature, for according to Haab, who is an authority in this matter, traumatisms that in other parts of the retina would pass and leave no visible sign, when located in the macula are sometimes followed by permanent alterations. These are most often revealed by an abnormal stippling, either black or yellowish, that develops very slowly, terminating after several months or even years, in circumscribed atrophy and central scotoma. The outcome is specially unpromising, therefore, if there are any early evidences of pigmentary irregularities, say within the first week after the injury. These appearances are to be differentiated from the senile changes just alluded to, when seen in aged subjects, by being monocular and by the anamnesis.

**Perforation of the Macula.**—Kuhnt and Haab have independently observed another variety of macular alteration that most often occurs in consequence of a contusion. It is a curious lesion, and as yet little understood, and appears as a perforation, real or apparent, of the foveola, involving its entire area, or extending beyond, and characterized by the presence of a red spot, regularly rounded or oval, at the centre of the fovea. The borders are clean cut, without tags and indentations, as if made by a punch. Its diameter may be one-half or two-thirds that of the disc. Within it the red tint is found, on close inspection, to show a faint yellowish mottling. It is surrounded by a zone of grayish retinal opacity made up of points and striæ. The fundamental tint of the red spot varies with the individual. Sometimes its uniformity is interrupted by a sort of brownish



granulation, giving one the impression as of looking directly at the surface of the choroid with its capillaries and epithelial pigment-cells. The centre of the spot is depressed, as shown by parallax displacement, and verified by dioptric measurement. Haab considers the phenomena as expressive of a veritable "**hole**" in the centre of the macula, and named it **traumatic perforation of the macula**. Haab has seen a similar punched-out appearance in the macula associated with senile arterio-sclerosis, and de Schweinitz has reported them as having been seen with non-traumatic iridocyclitis.

**Myopic Macular Chorio-retinitis.**—In the higher degrees of myopia it is of the utmost importance to study the appearances at the macula, with the view to forestalling central blindness by early recognition of the changes at the posterior pole—or central retinitis and chorio-retinitis. Their frequency is in direct ratio to the degree of myopia. Following the classification of Sulzer, there are four grades of the disease, each characterized by certain ophthalmoscopic peculiarities.

**The first grade**, or that in which the changes are least pronounced, is distinguished by a mass of brown or slaty pigment in the macular region, its shape conforming to that of the fovea, darker at the centre and merging insensibly into the tint of the eye-ground. It may extend even beyond the fovea. It causes more or less amblyopia, but the latter is not of necessity permanent. If restoration does not take place the next stage appears.

**The second grade** is recognized by a fine orange stipple, that becomes apparent in the brown of the macula, but which is composed of dots so exceedingly fine as to require the most minute ophthalmoscopic research to discover them. If the patient is quick to observe, he will probably be aware of a relative central scotoma, and of disquieting phosphenes, with the vision reduced. The changes bespeak a film of exudation which threatens to disorganize the pigment epithelium. This, too, may all clear up, dispelling both objec-



tive and subjective symptoms. If, on the contrary, exacerbations occur, the orange stipple is likely to give place to the third stage.

In the **third stage** of the myopic macular affection, the part presents a reticulation of yellow streaks, which traverse the brown or slaty ground, suggestive of Sienna marble or of the *craquel* of crazed pottery. Sometimes the yellow fissures exhibit an arrangement that recalls the radiate figure of albuminuric retinitis. In proportion as the yellow fissures augment in width and number, the affected area appears depigmented. A complete cure is no longer possible, and progression is the rule.

**The Fourth Stage, or Stage of the Heavy Exudation of Chorio-retinitis.**—Through a single exacerbation, or a long series of slighter ones, the focus of exudation may completely overwhelm the pigment elements of the choroid and the retina. It either displaces or masks them to the extent of transforming the macula into a spot of clear yellow, contrasting strongly with the darker ground of the fundus. In one instance it is single, in another multiple. It is often roundish. Again its contour indicates that it has been formed by the confluence of several original foci, or it is of irregular form, with deeply indented outline. The exudate terminates in resorption. It is then obvious that there has been complete disappearance of the pigment elements, for through the transparent retina may be seen the site of former activity outlined in black, and crossed by the large vessels belonging to the outer layer of the choroid, with interspaces of bare sclera. Whatever the period of evolution of this circumscribed chorio-retinitis, it may be complicated by retinal hemorrhages occurring in the diseased locality.

**Macular Apoplexy.**—The hemorrhage may be small or extensive. If small, it is usually intra-retinal, if more profuse, it may be pre-retinal or subhyaloid (see Fig. 50), or it may break through and invade the vitreous. Occasionally there is rarefaction of pigment extending beyond the site of more pronounced atrophy, or an extensive area



of depigmentation at the posterior pole outside of the circumscribed central changes.

**Macular Chorio-retinitis. Posterior Choroiditis. Diffuse, Syphilitic Macular Chorio-retinitis.**—By these terms are indicated virtually a circumscribed form of that disease which, when generalized, is known as **diffuse syphilitic chorio-retinitis**. It is a tardy complication of syphilis,

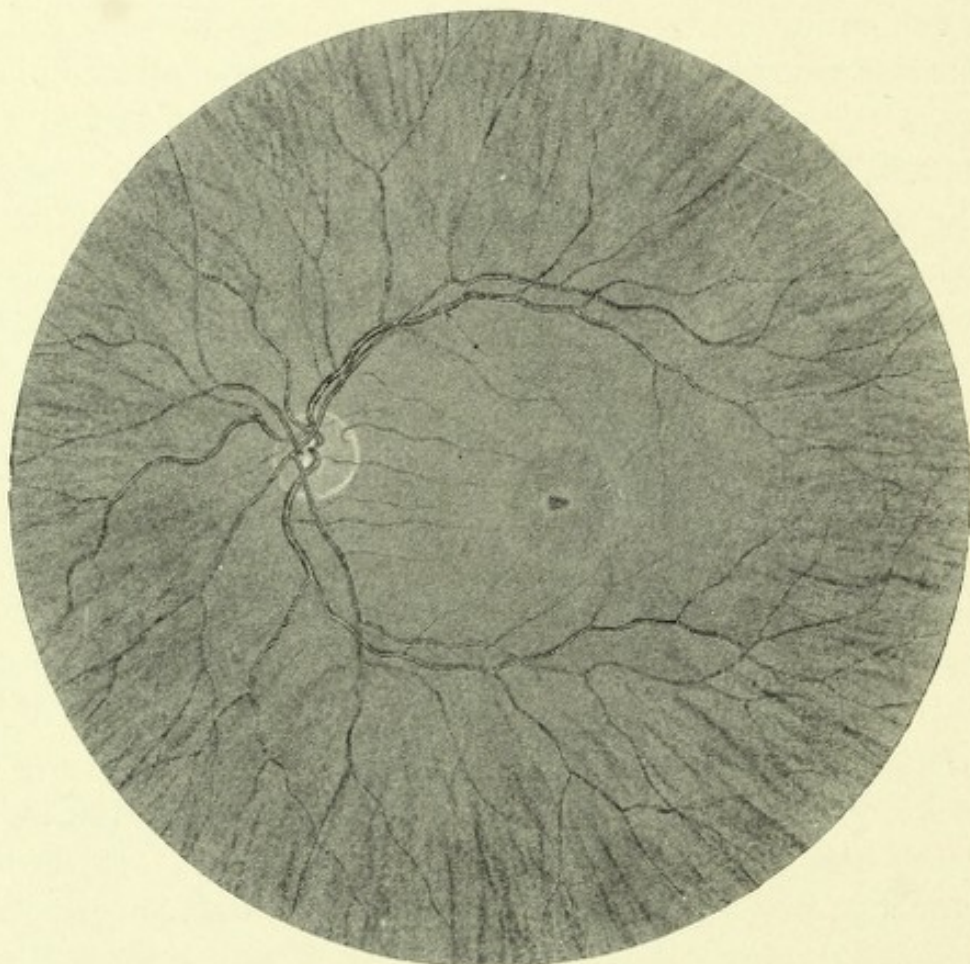


FIG. 50.—Hemorrhage in foveola.

appearing at from eighteen months to five years after the initial lesion. In many instances the disease in question has been preceded by acute diffuse chorio-retinitis, the latter having supervened several months or several years previously, just as in case of disseminated choroiditis of syphilitic origin. Less often it occurs in an eye that had before been sound.

**The ophthalmoscopic signs** are in many respects identical



with those of the myopic form just discussed. They are usually pronounced in the same measure as are the subjective symptoms. So long as these consist of merely a vague uneasiness concerning the vision there is little that is abnormal to be found in the fundus. The macula may seem a little too highly pigmented, or the foveola a trifle too red, yet, on reflecting how greatly normal eyes vary as to these points, one can only give wholesome advice and await developments—the result of the examination is negative. It frequently happens that a later examination yields revelations much more definite. It becomes quite evident that the tint of the macula is not normal. Moreover, there are often present certain markings that are absolutely characteristic of the disease. In the very acute form, however, the first evidence is a uniform discoloration of the entire macula, manifested by a big homogeneous spot. At first it is of a chamois tint, but later, from distention of the retinal and choroidal capillaries, it becomes deep red. The first tint is more conspicuous in light colored eye-grounds—the second, in dark ones. If the yellow discoloration takes place rapidly, it means successive blanching of the hexagonal epithelial cells and stellate connective-tissue cells, and a dense round-cell infiltration of the choroidal stroma. This is a grave condition; just the kind which ultimately results in a plaque of choroidal atrophy that resembles central coloboma of the choroid. The retina may have suffered but little, and its vessels are sometimes seen to pass unaltered over some part of the plaque.

In the **subacute** form the choroidal pigment has an irregular, "washed-out" look, and the yellowish tint is less uniform, indicating that the infiltration is concentrated in nodules. These changes may be distinctly defined as spots of deeper yellow. This is a much more frequent form of the affection. This pigmentary and yellow mottling sometimes extends to the surrounding, or perimacular, region, involving an area two or three times that of the papilla, measuring somewhat more horizontally than vertically. Less often



it is round, and still less often of trefoil, quarterfoil, or bizarre shape. These forms, unusual here, are more common in the senile and traumatic varieties of central choroiditis. Like that of myopic chorio-retinitis, the aspect of the affected region is frequently suggestive of Sienna marble, or the fine broken zigzag yellow lines recall the surface of crazed pottery. Later, upon the pale ground of the region appears pigment, in coarse powder, in rounded dots, in delicate figures, in angles or stars, or in formless blots. Again the pigment may be disposed in fine arcs, framing more or less completely the light spots that are destined in the future to become white spots of atrophy, as in disseminated choroiditis. Small hemorrhages in the fovea are by no means an unaccustomed feature of the picture.

**The permanent or residual ophthalmoscopic image** left by diffuse macular chorio-retinitis varies in accordance with the phases of rapidity and severity that mark the progress of the original affection, both formative and regressive. When the absorption of the intra-choroidal transudates is complete, the macula exhibits no sign of former trouble. Nevertheless the quantity of pigment—epithelial and stromal—may be reduced. Again, all that remains in token of the dread disease is an obvious uniform depigmentation, gradually shading into the unchanged eye-ground; or it may be that what would otherwise be an even area of pale red, or chamois, is sprinkled over with coarse grains of dark gray or black powder (**"powder grain"** effect). In this event the vision may have suffered little or not at all. In another case, in which the resolution has been less complete as regards both retina and choroid, the erstwhile light area is spotted, and seamed with cicatrices which are gray or white, owing to the degree of atrophy. In addition, the borders of the affected region are then sometimes indented or fringed. The image is further modified by the black outline that nearly always surrounds the white spots of atrophy. Again, as before intimated, the process is so overwhelming as to involve the entire thickness of the choroid in a mass of



cicatricial degeneration, leaving but a mournful white panel bordered in black.

**Complications.**—The most frequent of these is retinal hemorrhage or macular apoplexy. Owing to the thinness of the central portion of the fovea the blood readily finds its way into the space between retina and vitreous, and if the flow is free and rapid the vitreous itself is invaded. In view of the fact that these hemorrhages nearly always entail the permanent destruction of some portion of the retinal neuro-epithelium, their advent is serious. Those occurring in the nerve-fibre layer, if not too copious or too long present are often absorbed, leaving the retina unharmed. Those of deeper origin, and those that are abundant, are most to be dreaded (see chapter on "Hemorrhages"). Other serious and not infrequent complications of all forms of central chorio-retinitis are **posterior polar cataract** and **detachment of the retina**. A less serious complication, and the most frequent of all, is constituted by **opacities** or floating bodies **in the vitreous**. They are usually present to some extent throughout the progress of the affection, being often the prodromal symptom, and even lingering after it has run its course. Their volume may be so slight as to be visible only to the patient, or so great as to form extensive membranous shreds that obscure the view of both observer and observed.

**The differentiation between myopic and syphilitic macular choroiditis** is often difficult, and not always possible; indeed, the two are often identical. Rarely, also, there is the traumatic form to be identified, wherein the anamnesis is the principal key. Monolateral myopia is frequent in the traumatic form. The syphilitic type is prevalent in both sexes, and in all ages up to middle life. The purely myopic is rather peculiar to adult females. The syphilitic is, *as a rule*, *unilateral*, while the myopic, in more than half the cases, is *bilateral*. The syphilitic when acute is always accompanied by papillitis, and sometimes also when subacute. The myopic is usually characterized by hyperemia of the papilla,



but never by papillitis. In the absence of retinitis and opacities of the vitreous, with central choroiditis, and with sudden and great increase in the myopia, say of several dioptries in a few days, or a few weeks, the indications are of a posterior **sclero-choroiditis** or **supra-choroiditis**. A peculiarity common to ordinary myopic and traumatic macular choroiditis is that there is likely to be a high degree of amblyopia when the ophthalmoscopic evidences of the disease are comparatively slight. Whereas, in the luetic form one is often astonished to find what extensive destruction of the macular choroid can be consistent with fairly good vision. The subjective symptoms are much the same in all three forms. After all, it must be admitted that progressive myopia, malignant myopia, macular chorio-retinitis, central choroiditis, posterior sclero-choroiditis, and diffuse circumscribed macular chorio-retinitis, are often one and inseparable.

**Alterations due to Intense Glare.**—That brilliant light concentrated upon the retina may cause transient obscuration of vision has been known for ages, but Saint Yves, in 1722, was the first to call attention to the fact that the ill effects may be lasting. He observed cases of permanent central blindness from the glare of molten metal, from the lightning, and from looking at the sun during an eclipse. The first recorded description of the ophthalmoscopic manifestations from the cause last named, emanated from the Ophthalmic Hospital of Lausanne, in 1879. There is never a solar eclipse that is not followed by a great number of central scotomata, relative and positive, many of them permanent, all through neglect of the precaution to safeguard the retina by the use of a smoked glass or other protective medium. The scotoma is bilateral or unilateral according to whether one or both eyes are directed to the sun. Owing to the conditions under which the injury is sustained it necessarily follows that the part of the retina upon which the rays are concentrated, and which suffers in consequence, is the fovea. The ophthalmoscopic findings, as reported from Lausanne,



relative to a particular case, are considered typical, and are here quoted from Sulzer (*Enc. franc. d' Opht. t. VI, p. 962*): "Diffuse, very light opacity of the retina around the optic disc, like a thin vapor over the vessels; hardly noticeable except at the side toward the macula. The macula is dark red, and, by the indirect method, seems to be the seat of an effusion of blood about the size of the papilla (*Fig. 51*). Its centre is occupied by a white spot, contrasting strongly

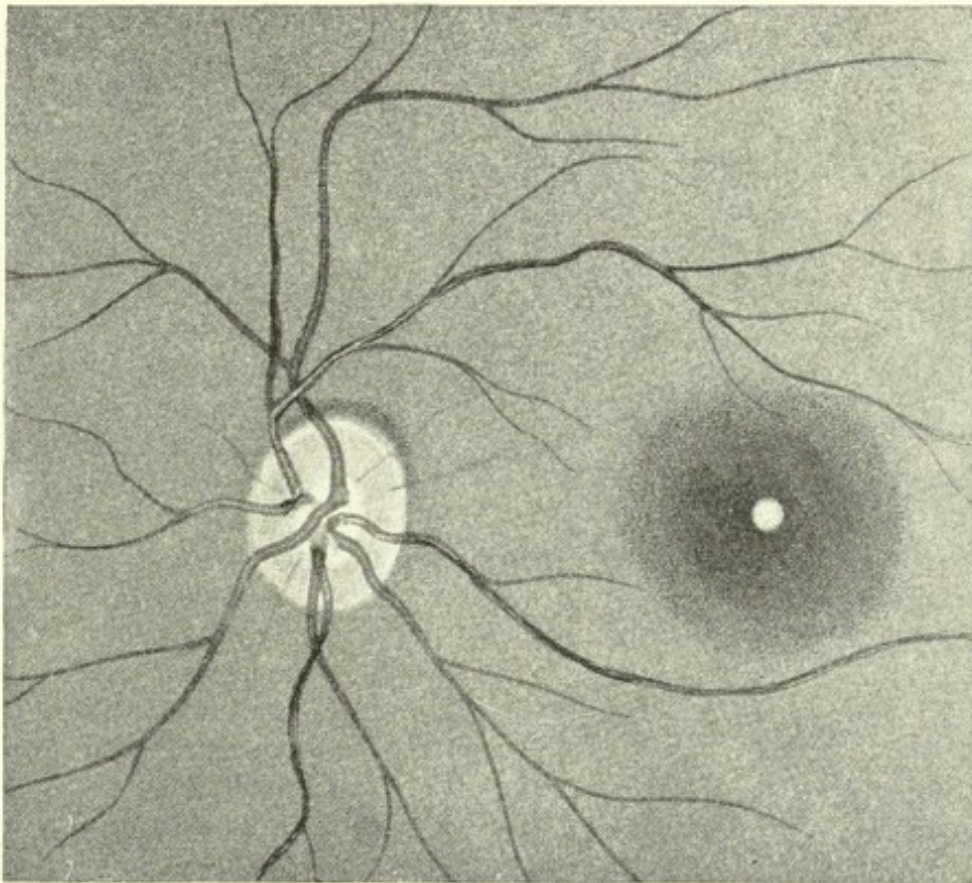


FIG. 51.—Effects of intense glare upon the macula. (*After Dufour.*)

with the dark red around it, and, by the indirect method, seeming to stand out in relief. Observation by the direct method corrects these two impressions. It shows that what seemed to be an effusion of blood is something different, for at no place is there any definite limit to the taint; its color, dark brown near the centre, thinning more and more toward the periphery till lost in that of the normal retina. Nevertheless the dark brown is, at least, a violent capillary con-



gestion, for its aspect is manifestly different from that of the brown pigment so frequent at the fovea. It is also of much deeper color than the corresponding spot in the other eye, which, in this patient, has a high degree of convergent squint, and could not be directed toward the sun. The central white spot, by the direct method, appears as a perfect disc, yellowish-white, and with an outline as clear cut as if made with a punch."

The foregoing observations were made three days after the eye was injured. In a few days the central spot became effaced, first turning flesh color. The red-brown areola disappeared in a week, and the fundus was considered normal in eleven or twelve days. The visual acuity was still reduced one-half after the lapse of several months. Sulzer further states that three of Haab's cases, reported by Siegfried, closely resemble the one just cited. In these also there was the light spot at the centre of the fovea, which was likened to an optogram of the sun; and, strange to relate, this spot had the form of a half-moon or crescent, identical with that of the sun at the moment when the eyes were directed toward it. Siegfried reports further that the central spot after a few days turned reddish-brown, and its areola became grayish. The Doctors Dufour (of Lausanne) examined a victim of bilateral sunblindness in 1905, and confirmed the typic nature of the first findings, but the white of the foveola and the red of the fovea were of lesser intensity.

Alterations in the fundus attributable to the effects of **blinding flashes** from accidental **electric arcs**, or short circuits, are, in most instances, visible with the ophthalmoscope, but they are less strictly localized in the macula than those from the sun. This is easily explained by the fact that a workingman, for example, who is surprised by the sudden glare, strives his utmost to avoid looking straight at it, and thus escapes the production of its optogram on the foveola. The result is a diffuse opacity of the papillo-macular region.

**Appearances in Amaurotic Family Idiocy.**—The macular



changes seen in connection with this disease constitute an almost exact reversal of those just described as consequent upon looking directly at the sun (Fig. 52). In the first the foveola appears brownish-red or, as has been described by the writer, **liver-color**, and the macular region around it white, shading gradually into the red of the fundus. The two pictures are strikingly suggestive of the differences in the same design on the two sides of certain fabrics woven in two colors, or of those between a photographic negative and its transparent positive.

The following is a description of the eye-grounds in amaurotic family idiocy. Surrounding the foveola and extending considerably beyond the ordinary limits of the macula, is a zone of grayish-white, which gradually fades away till it is lost in the normal color of the eye-ground in the posterior polar region. This whitish zone is highly characteristic. Seeing that it is the result of a degeneration of the ganglion-cells of the retina, the white is densest, or almost pure, around the rim of the foveola; for here the ganglion-cells are most numerous. Thence outwardly the density decreases, as does the number of ganglion-cells, by insensible degrees, and in a perfectly regular manner, becoming nebulous a little way outside the macular region, till, still well within the confines of the posterior polar region, it is thinned to nothing. The whitened area is everywhere characterized by some color, or a certain translucence—opalescent, as it were. The foveola itself appears in its true dimensions, and as a liver-colored disc, as round and clean-cut as a coin: not irregular of outline as when it is the centre of infiltration—nor is it cherry red nor carmine, but is distinctly brownish. The whitish zone does not obscure the diminutive retinal vessels which traverse it, but, on the contrary, only serves to render them unusually distinct, by contrast. In consequence they can be clearly traced almost to the brink of the livid disc of the foveola.

The data were obtained in making colored drawings in



DESCRIPTION OF FIG. 52.

Fundus in amaurotic family idiocy.



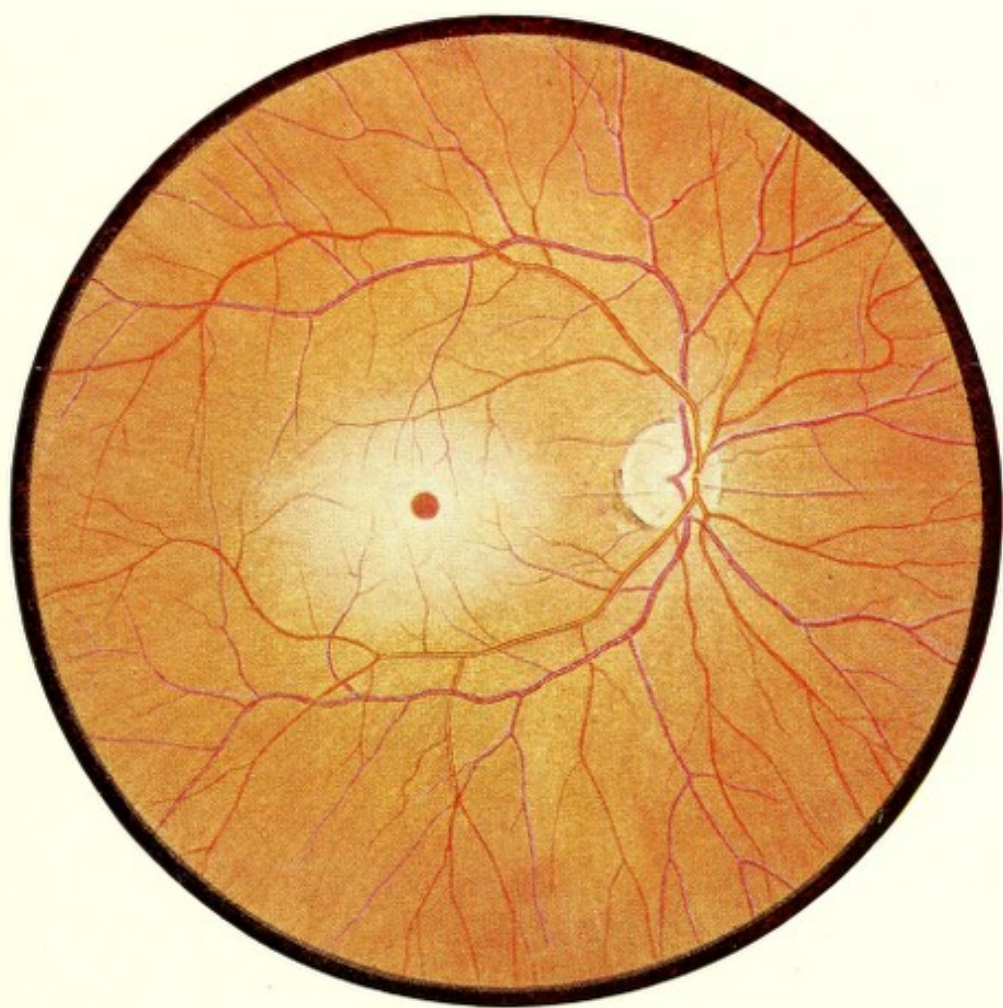


FIG. 52.







two cases of the disease, from the clienteles of Doctors Patrick and Kuh, of Chicago.

The ophthalmoscopic appearances were first described by Tay in 1881. They had been observed by several others before the disease was named by Sachs, of New York, in 1887. This neurologist noted the principal clinical features, lack of mental development, progressive general muscular weakness, defective vision—terminating in complete blindness; also that it was a disease of childhood, that it was familial, affecting more than one child of the same parents, and that it was generally fatal—the child dying in a condition of complete marasmus before the end of the second year of life. Later the fact was established that the disease is one peculiar to the Jewish race. It was not till 1895 that Kingdon discovered that Tay's fundus findings belonged to the affection thus named by Sachs. In 1898, Holden gave a correct explanation of the changes that furnish the distinctive ophthalmoscopic picture. The writer was the first to make an accurate colored drawing of the fundus. That the disease is not very rare is proved by some seventy-five cases that are already to be found in neuro-ophthalmic literature.

**Cherry Spot, or Red Spot at the Centre of the Macula.**—Many fallacies have been uttered and printed relative to this appearance at the posterior pole. It was formerly thought to be an exclusive sign of embolism of the central artery of the retina. Later it was found associated with choked disc, central detachment of the retina, and, in modified form, in connection with traumatism of the retina (Commotio). We now know that it is made possible purely by the anatomic arrangement of the retinal constituents in the macular region. These have already been described on page 300. Anything that causes a **white** opacity of the fovea will cause the foveola to appear redder by contrast. The cause of the opacity may be from serous infiltration of the entire retina with swelling, as in choked disc, it may be from beginning necrosis of the optic nerve-fibres, as in



arterial thrombosis and embolism, or it may be from degeneration of the ganglion-cells as in amaurotic family idiocy. In these several instances the color of the choroid is obscured but slightly or not at all by the thin foveola, but is much more, or completely concealed by the thicker or opacified retina of the surrounding region. In edema the foveola itself is sufficiently affected by the serous infiltration to cause

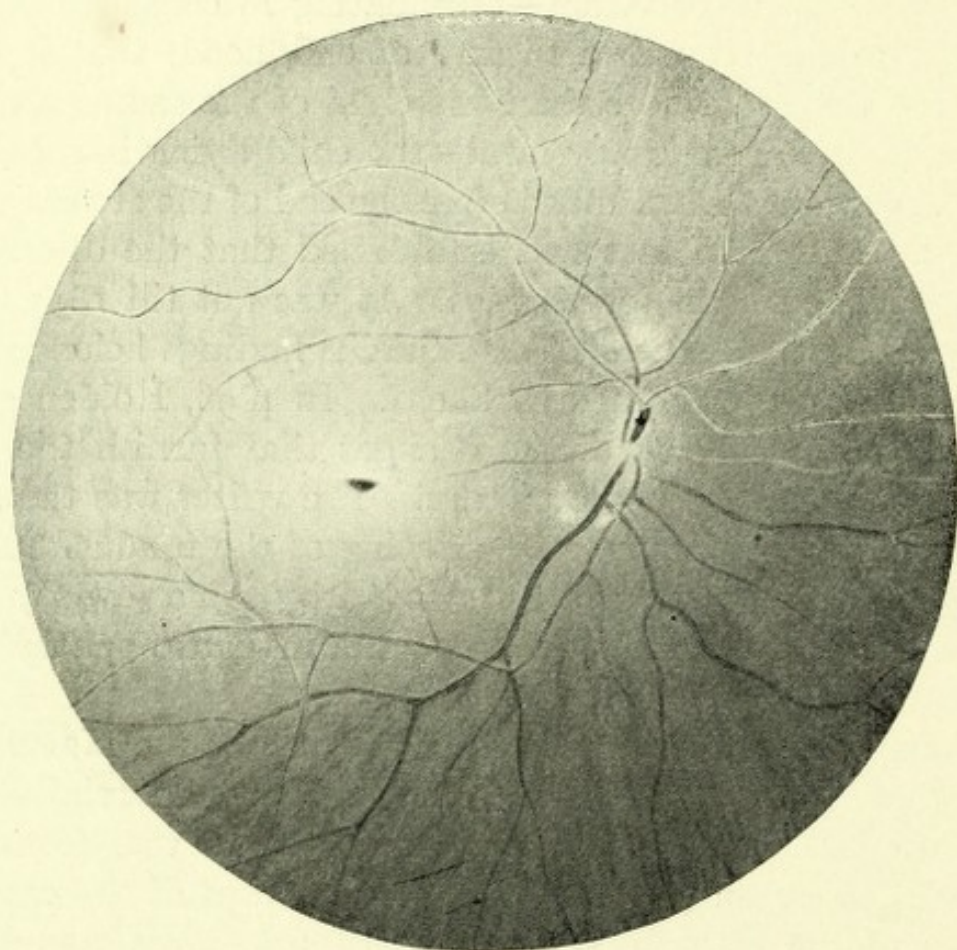


FIG. 53.—Embolism of upper branch of central artery and the cherry spot at the foveola.

a veiling of the color of the underlying choroid, and the spot is red, cherry or pink, in proportion to the degree of veiling. Moreover, the form of the spot varies also with the extent of the infiltration. If the latter does not involve the entire rim of the foveola, but merely encroaches upon its upper and lower borders, the red spot will be elongated horizontally, its long axis being sometimes much greater than the corresponding diameter of the foveola (Fig. 53). If, on



the contrary, the swelling encroaches on all sides of the foveola, the spot will be of smaller diameter than that of the normal central pit. It may then be no longer circular, but polygonal (Fig. 54).

When there is no veiling of the choroid, the depth of color depends not only upon the degree of contrast between fovea and foveola, but also upon the normal tint of the choroid

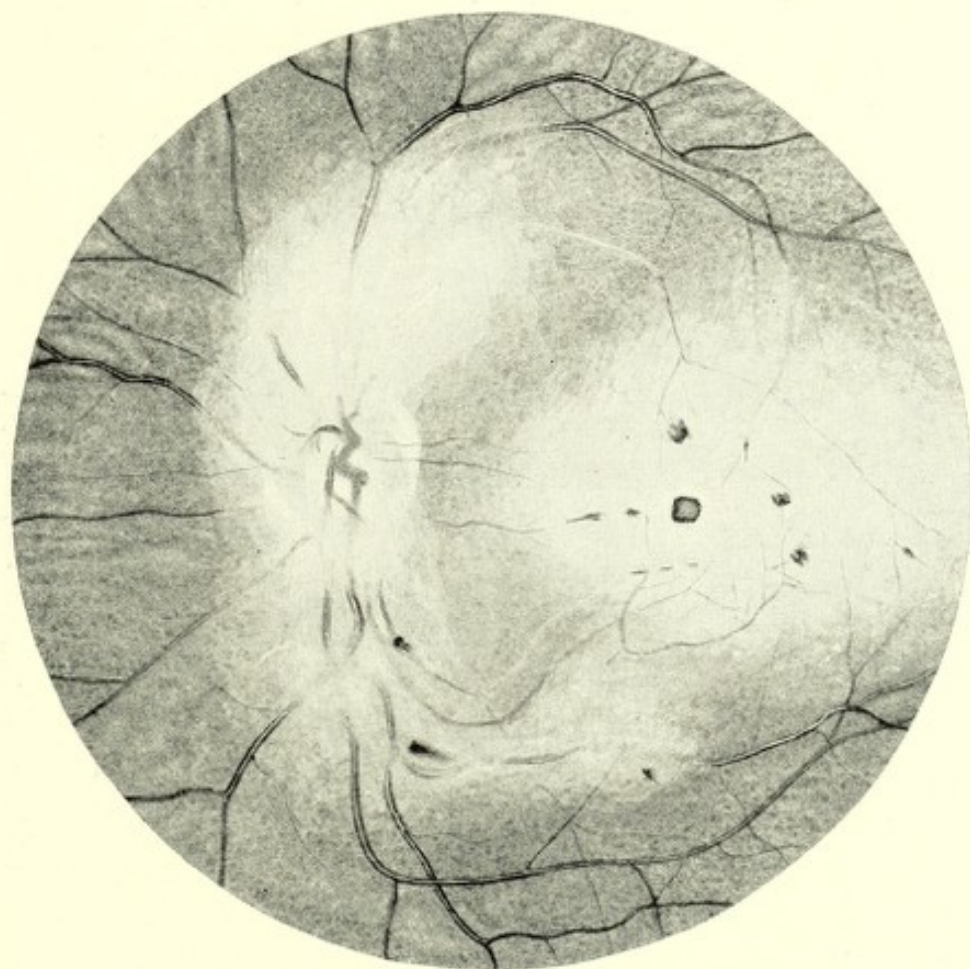


FIG. 54.—Embolism and foveola.

of the particular individual. The more velvety the choriocapillaris, and the more abundant the pigment in the hexagonal cells, the darker will be the tint. Hence, it occasionally assumes shades of brown—almost of sepia. When there is no swelling and invasion at the borders, the spot appears as a perfect disc, and its diameter exactly coincides with that of the foveola itself. When there is no veiling of the choroid beneath the foveola the red spot cannot be due



DESCRIPTION OF FIG. 55.

*Retinitis circinata.*



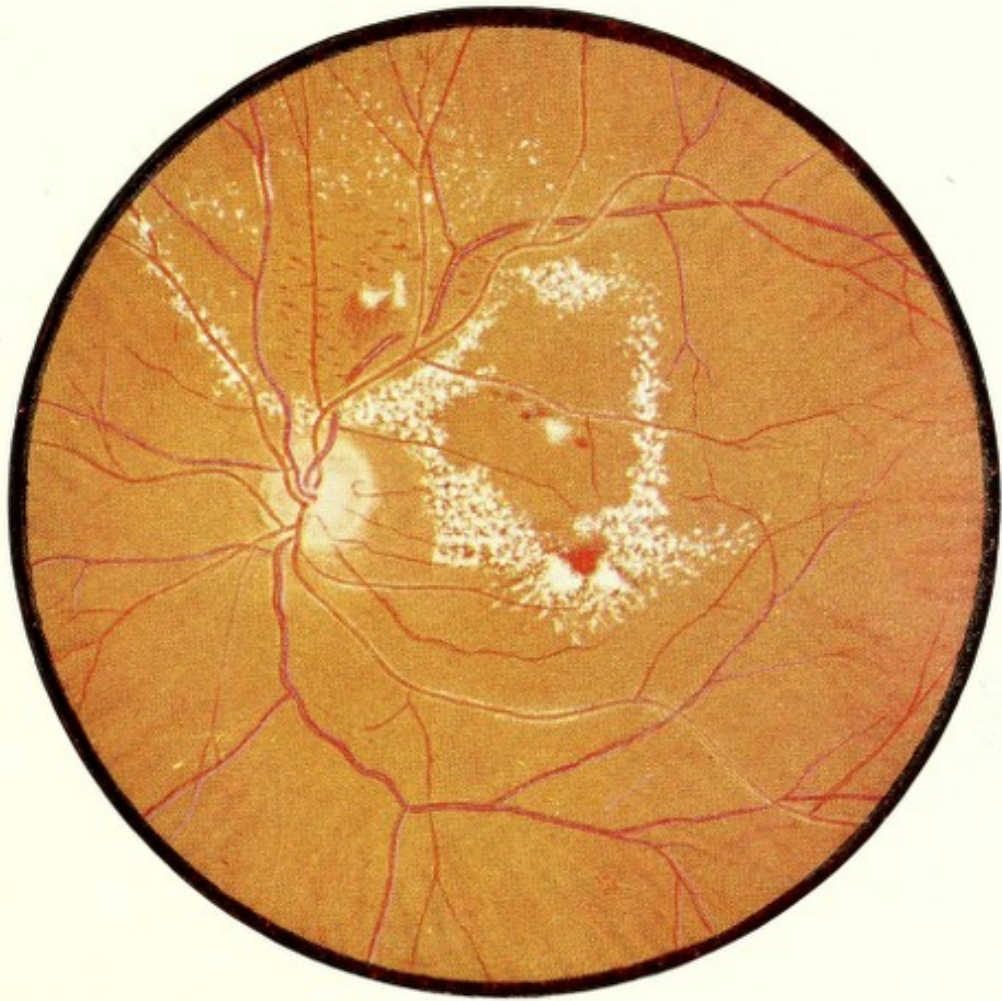


FIG. 55.







to the presence of either cellular or serous infiltration. The most striking contrast of all is that afforded by the changes in amaurotic family idiocy just described, for here the white around the foveola being the result of degeneration of the ganglion-cells, is most intense at the rim of the foveola, where these elements are piled high, thence gradually becoming less so as the distance from the centre increases. In the absence of any swelling the foveola appears in its true (magnified) dimensions, and as clean cut, and as true a circle, to use a hackneyed phrase, as if made with a punch (see Fig. 52).

**Retinitis Circinata. Circinate Degeneration of the Retina.**

—The first name was bestowed by Fuchs in 1893 upon this rare form of disease, originally recognized as a clinical entity by Hutchinson in 1876. A year later deWecker substituted "degeneration" for "retinitis." The affection is mentioned here because of the highly characteristic appearances in the posterior polar region; the alterations being confined exclusively to this locality. The typic white spots are not seen in the fovea, though it sometimes contains small hemorrhages and, in very old cases, minute pigment patches or crystals of cholesterin. The unique distinctive feature is a girdle of white spots, of irregular elliptic form, larger diameter horizontal, that encircles the fovea (Fig. 55). The girdle is rarely complete, being either composed of a superior and an inferior arc, or disposed in the shape of a horseshoe, opening temporally. It seldom extends beyond the temporal trunks of the retinal vessels, when these are normally distributed. At times the spots seem to group themselves along the secondary branches of the vessels.

The perimacular changes under discussion are characterized as much by their detail as by their peculiar situation and general arrangement. The white spots that compose the girdle are of rounded outlines, and milk-white, like those of recent albuminuric and diabetic retinitis. In coalescing they form fantastic lobulated designs, which were likened by Fuchs to that of the *arbor vitæ cerebelli*. They are not



bordered by pigment, but by reason of their pure whiteness they contrast so strongly with the eye-ground that by comparison the latter looks many shades darker than normal. Some of the spots may seem in relief, or cameo, but that they are always situated beyond the blood-vessels is shown by the fact that these, even to the tiniest twigs, pass uninterruptedly over the white. The rest of the fundus exhibits nothing that is not normally consistent with the age of the subject, except that signs of arterio-sclerosis are usually pronounced. The girdle enlarges or grows from within outward by the development of additional spots of white. That there is a constant tendency to progression as regards the white foci of degeneration is shown not only by their augmentation in number but also in that of size, with mutability of shape; their growth being by additions of infinitesimal snowy globules. Indeed, they are identical in this respect with the macular white spots of albuminuric retinitis. Hemorrhages, few in the beginning, later become a constant feature of the ophthalmoscopic picture. When their seat is in the midst of the zone of degeneration they are sharply circumscribed, exceedingly small, and of ovular or fusiform shape. When situated anywhere without the limits of this zone they are in no way different from ordinary extravasations.

The disease is essentially chronic, and has been known to last for from ten to twenty years. According to some authors it is incapable of amelioration; others have noted, exceptionally, instances of improvement—even to disappearance of the salient ophthalmoscopic signs. Weeks saw them disappear in one year, and de Schweinitz in a little more than a year. The rarity of the disease is attested by the fact that only about one case is seen in some 10,000 examinations of the eye-grounds. It is rarely seen before the age of fifty, and affects women much more often than men. It is bilateral in only about one-half the cases.

Pathologically considered, the white spots consist of fibrinous or hyalin masses, or of aggregations of the granulo-



fatty globules that displace them, situated in the lacunæ between the external and internal nuclear layers. By the majority of investigators they are believed to be the ultimate products of former hemorrhagic foci. The disease is to be differentiated from albuminuric and diabetic retinitis and from **punctata albescens**, also from certain other punctate appearances in the fundus, such as Tay's spots, or Tay's

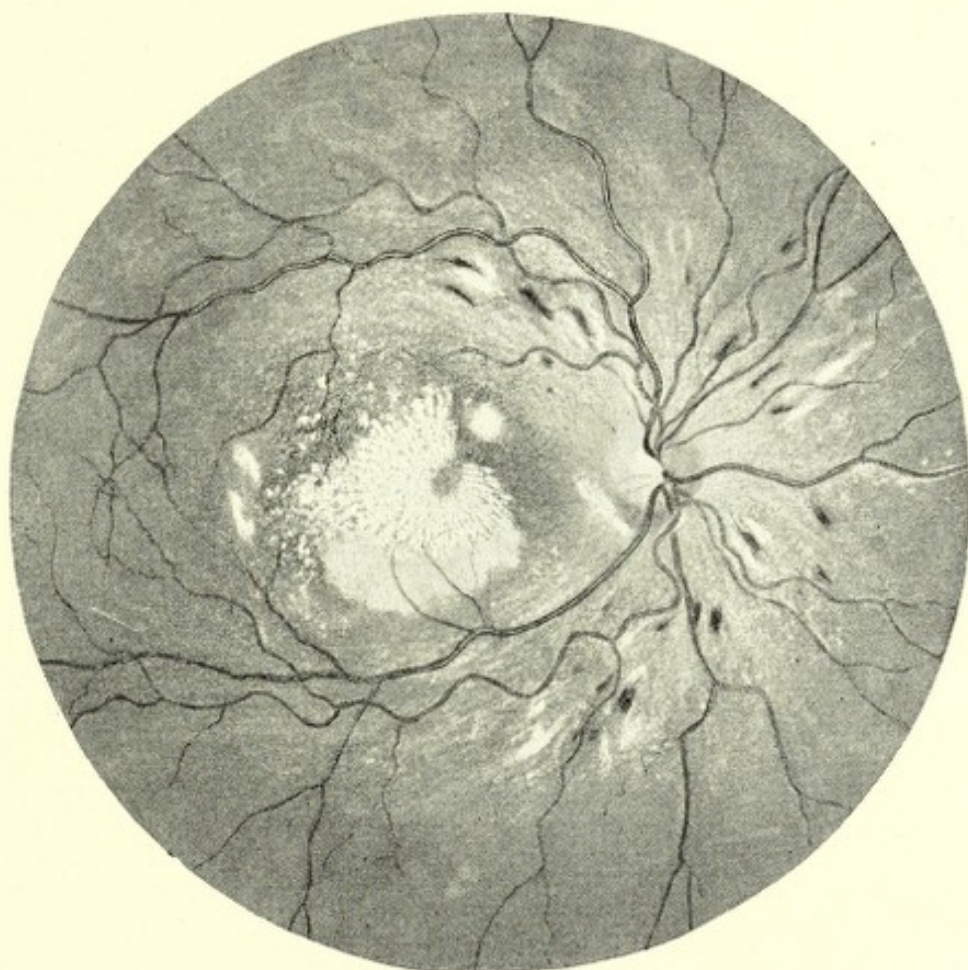


FIG. 56.—Closely packed, radating white spots in albuminuric retinitis.

choroiditis, and the more generally distributed dotting associated with verrucosities of the lamina vitrea of the choroid, or **drusen**. The last are more regularly round and discrete, and have no tendency to coalesce. They do not affect the vision centrally or peripherally, while retinitis circinata always causes a corresponding scotoma, relative or absolute. (See "Retinitis punctata albescens" for further differentiation.)



**Macular Spots of Albuminuric Retinitis.**—There are two varieties of white spots characteristic of albuminuric retinitis—the peripapillary and the macular. The first are larger and more superficial. They are considered on page 341. The smaller and deeper seated macular spots are very variable as to size and shape. They begin as tiny fusiform dots that radiate from the foveola. There may be only a

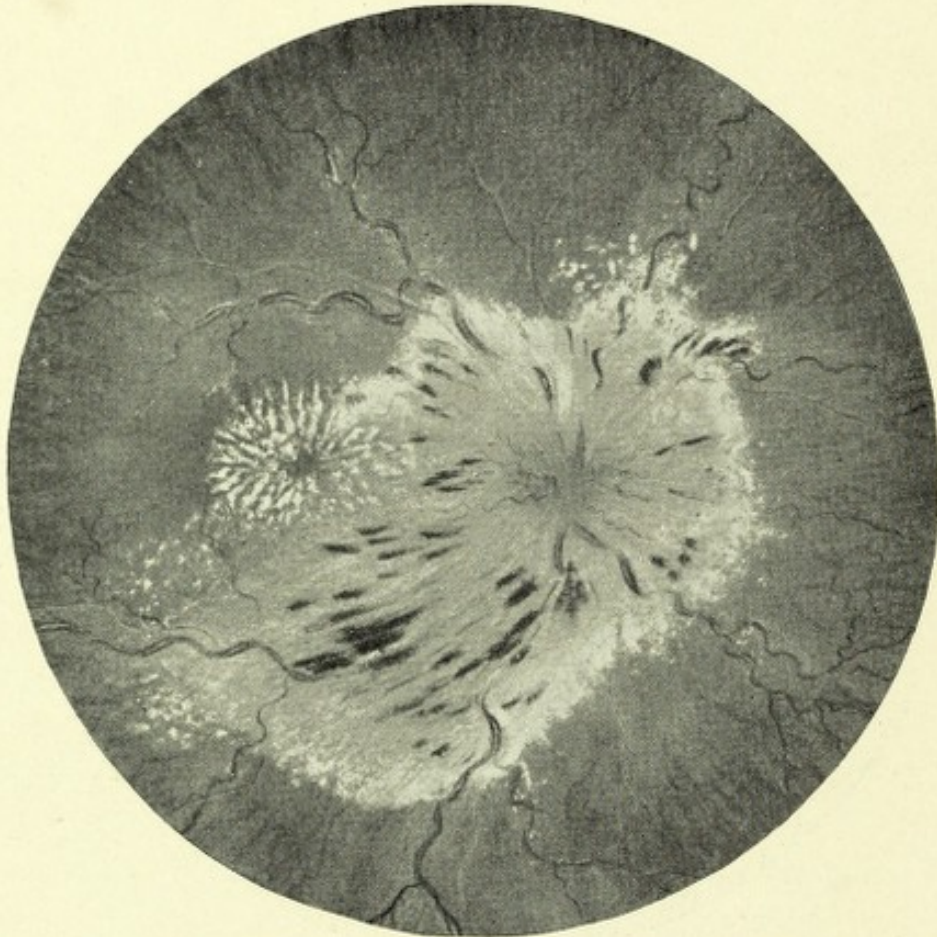


FIG. 57.—The macular white spots in albuminuric retinitis.

single dot, but its long axis will point toward the centre. From this beginning other dots may appear, in the same radius, or in other radii; they become confluent in places, forming broken lines, or entirely confluent, forming solid lines, like the spokes of a wheel, but of irregular width, owing to the fact that the original focuses are of different ages and in different stages of development. The spokes may represent the segment of a circle or the entire wheel



(Fig. 56). They are of a silvery white, which is enhanced by the dark background of the non-edematous macula; now appearing exceptionally dark through contrast. After having attained their numerical limit, the spokes often continue to increase in length and breadth. In length they sometimes transcend the limits of the fovea, see Fig. 57. Projections from the wider ones often reach across and unite with those of neighboring spokes, resulting in the formation of fantastic figures. In rare instances the crosswise coalescence goes on

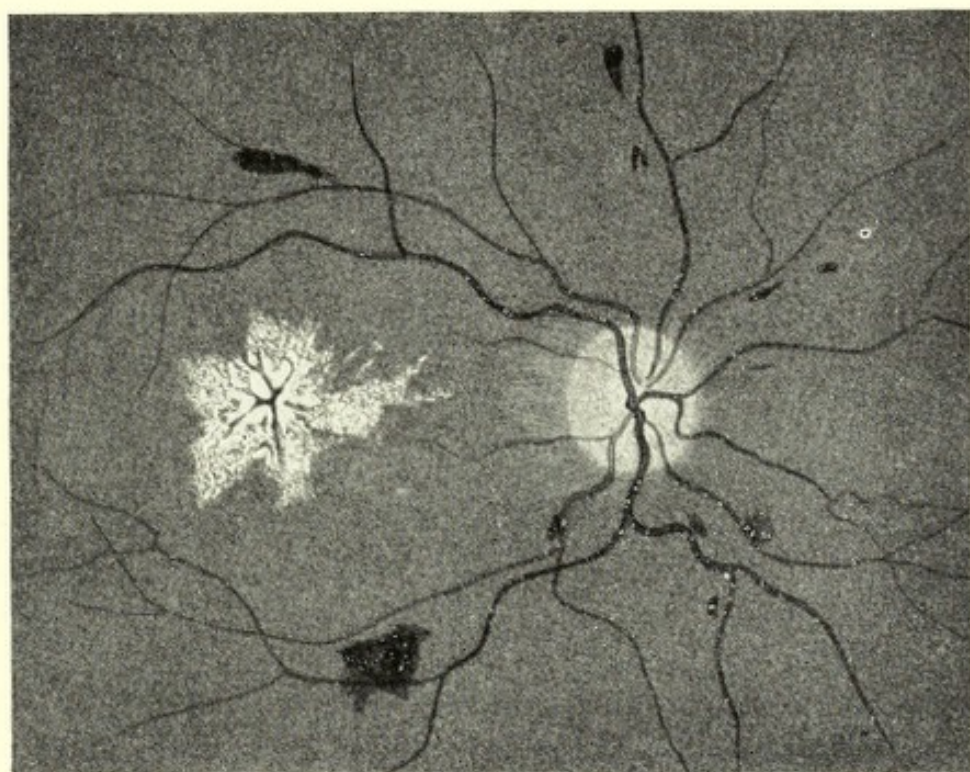


FIG. 58.—Stellate figure in albuminuric retinitis. (After Dufour.)

till the wheel becomes more or less solid, the only radiation that is visible now being that of the narrow interspaces of dark ground tint, and in the star-like processes in the white about the periphery of the macular region. This constitutes the extremely rare true **stellate** figure of the macula (see Fig. 58), as distinguished from the common **radiate** figure. Their growth is much slower than that of the large cloudy spots around the papilla. If one will take the time to make a painstaking drawing of the exact outlines of the spots



constituting the whole or only part of the macular design, he can convince himself not only as to the fact that they do increase in size, but can, in a measure, observe their manner of growth. By means of the direct method, an extra bright white light and the sharp focusing of the spots, it is evident, particularly around their edges, that the spots are made up of agglomerations of extremely minute white round bodies that might be likened to a miniature reproduction of that form of snow which falls in the shape of tiny opaque spheres. These spots then are not, like certain other white fundus spots, consecutive to corresponding hemorrhages.

If life is sufficiently prolonged the spots will usually disappear; though the process of removal is slower than that of their formation—requiring months to complete. Permanent traces of the larger and more lingering spots are occasionally left in the form of radiating streaks of connective tissue or of sparkling aggregations of cholesterin crystals.

Notwithstanding the fact that radiating spots are found in the macula associated with retinal affections other than that of albuminuric retinitis, this sign is hardly less characteristic of Bright's disease than it was when Liebreich first described and pictured it. A few thin spokes, or a full complement of them, are seen from time to time, in conjunction with certain forms of acute syphilitic and of leukemic retinitis; also in that consecutive to some of the acute infectious diseases, like typhoid, especially that of young subjects. Such macular designs, however, never attain the proportions, and the fullness of detail, that is so peculiar and so constant a feature of those in albuminuric retinitis. None of these figures are to be confounded with the radiations often present in edema of the macula, which, as will be presently shown, are of another nature.

Several **explanations** have been offered for the **radiate arrangement** assumed by infiltrations and degenerations of the retinal tissues at the fovea; they all, however, rest upon an anatomic basis. In recalling the structures within the



limits of the macula it will be remembered that all those of any relative length are disposed in a radiate manner. The entopic figure made by the shadows of the tiny blood-vessels of the macula upon the rods and cones, in the experiment of Purkinje, is an exact silhouette of the arrangement of these vessels. The cone-fibres of Henle, that form the intermediary between the percipient and the conductive elements of the retina, are specially well developed in the macular region, where, instead of standing perpendicularly, as they do elsewhere in the retina, including the foveola, they become more and more oblique, from the centre outward, till, at the periphery of the macula, or a little beyond it, they are almost horizontal. Corresponding to these, as to development and arrangement, are the sustentacular fibres of Müller. Both sets are largest and most numerous at the nasal side.

As a result of his anatomico-pathologic researches, Dimmer concluded that the radiate figure at the macula in albuminuric retinitis is caused by an accumulation of fat-granules between the fibres of Henle. Nuël later, and after similar investigations, announced his conviction that the cause was the transudation of round white blood-cells from the macular vessels. These force apart the fibres of Müller with the fibres of Henle, and ensconce themselves in the spaces so created. The ophthalmoscopic findings would seem to verify this view. Koppen found homogeneous masses in the lacunæ of the layer of Henle, and big granular cells that were probably phagocytes. The homogeneous masses he thought were derived from old hemorrhages, and, according to his view, the radiate figure had reference more to the arrangement of the small macular vessels than to that of Henle's fibres. It is possible that, in many instances, extravasations, with consecutive fibrous deposits and hyalin and fatty degeneration, account for the ray spots in the macula unassociated with albuminuric retinitis.

As regards the radiating striæ seen at the nasal side of the macula in choked disc and allied conditions, they are



the trajects of the macular optic nerve-fibres rendered visible by the edema. These are the only nerve-fibres that really radiate from the foveola. These striæ are more of a reflex than an actual opacity. The serous infiltration also causes fusiform dilatations, or lacunæ, between these fibres. An identical radiation surrounds the edematous nerve-head in these cases.



## CHAPTER XVI.

### MEDIAN AND PERIPHERAL REGIONS OF THE FUNDUS.

Under physiologic conditions, whatever of detail exists in the ophthalmoscopic tableau of these parts of the fundus, aside from those of the retinal vessels, belongs to the choroid. The same is true, to a very great extent, of visible pathologic processes. Before proceeding to a discussion of the semiology of these regions it may be expedient to recall something of the **anatomy of the choroid**. First, as to the blood-vessels, of which it is mainly composed. The ophthalmic artery, a short distance behind the globe, gives off three or four branches, which constitute practically the entire blood-supply of the choroid. Almost immediately, these several arteries subdivide into some fifteen to twenty branches that run forward, surrounding the optic nerve, and pierce the sclera close to the borders of the papilla. These are the **short posterior ciliary arteries**. Immediately upon entering the globe they begin to give off numerous branches that break up into arterioles, and these again into capillaries, all anastomosing freely with each other, and a few of those nearest the papilla anastomosing also with small twigs of the central artery of the retina (see under "Retinal Vessels"). In fundi sufficiently free from pigment, the entering trunks of the short posterior ciliary arteries and their larger ramifications may sometimes be seen with the ophthalmoscope. They are usually full of queer crooks, and vary considerably in size. Perhaps, were the conditions less anomalous, their sizes would be more nearly uniform (see Fig. 59).

The blood returned from the tissues by the capillaries is delivered to a multiplicity of venules that correspond to the arterioles, and by these to larger and larger veins till the



venous system of the choroid is reduced, at its exit through the equatorial portion of the sclera, to four large trunks—the **vorticosae veins**. Often in converging to form these four trunks, the veins of the vortices all fail to meet within the sclera, in which event the number of perforations by which they get clear of the globe is increased. There may be as many as two for each of

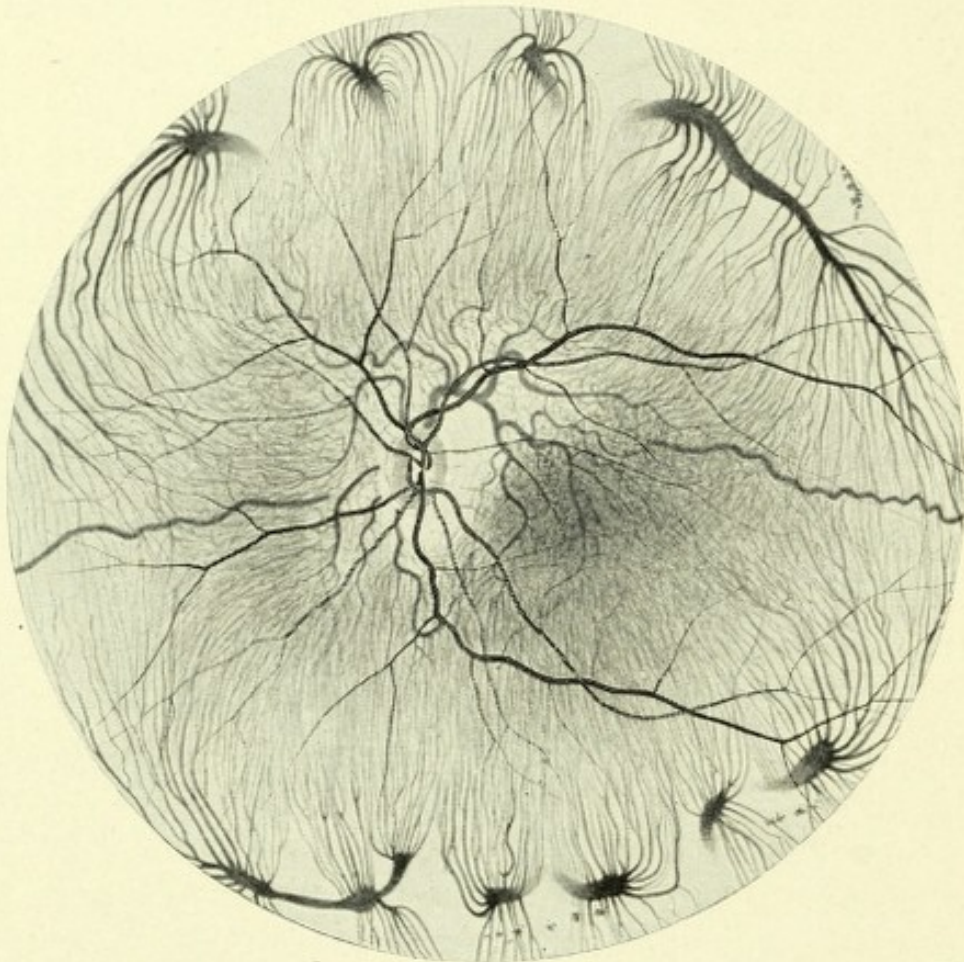


FIG. 59.—Unusual showing of circulatory system in the fundus.

the four ultimate divisions. Such was the case in the fundus from which was made the drawing here reproduced (Fig. 59). It was that of an Italian boy, with dark hair and eyes, but whose eye-grounds were devoid of pigment. In addition there was a myopia of 14 dioptries, and this, together with maximum dilatation of the pupil, was highly instrumental in bringing to view the points where the veins



effect their egress. The view thus afforded was certainly a unique one in the experience of the writer.

Both the venous and arterial systems of the choroid anastomose anteriorly with the anterior ciliary systems. The two long posterior ciliary arteries, that penetrate the globe about five millimetres from the papilla, one on either side, and run forward just within the sclera, and external to the choroid, do not concern us materially, as they give off no branches till they reach the ciliary body. They, too, are visible in this fundus, though dimly so. They pierce the sclera very obliquely, which causes them to enter the supra-choroidal space well forward of the entrances of the short arteries.

**Structure.**—If we begin with the layer of hexagonal pigment cells, which pertains to the choroid inasmuch as it is sustained by it, the choroid is composed of no less than **six layers**. From within outward they are:

1. Layer of pigment epithelium, made up chiefly of hexagonal cells in a single course, joined together by a transparent cement, like a mosaic. At the centre of each cell is its nucleus, around this a circular clear spot, while in the rest of the cell is packed, more or less densely, dark brown pigment granules.

2. **Lamina Vitrea.**—This is a very thin, featureless membrane that serves as a foundation for the pigment cells, and to which they are cemented by the same transparent material that unites their edges.

3. **Chorio-capillaris.**—This layer consists almost wholly of capillaries.

4. **The Intervascular layer, or elastic lamina, of Sattler,** contains elastic and connective-tissue fibres but no pigment.

5. **Layer of Larger Blood-vessels.**—This tunic consists of two vascular networks, one of arteries and one of veins, the interspaces being normally filled with pigment cells, and the whole supported by a stroma of connective tissue. The larger vessels are disposed rather externally in the layer.

6. **Lamina-fusca.**—This is a pigmented layer of spongy



connective tissue, traversed by trabeculae that unite the sclera with the vascular layer. By reason of its spongy construction it constitutes the **supra-choroidal space**. In the immediate vicinity of the papilla this space is about obliterated by the passage of the posterior ciliary arteries and nerves.

The several vascular networks in the choroid, capillaries, arteries and veins, are densest in the posterior polar and papillary regions of the fundus. In and around the macula the capillaries form a solid red lamina, their diameters being greater than the width of the spaces that separate them. They are narrower and longer at the posterior pole—becoming thicker and shorter, as well as less numerous, toward the ora serrata. The anastomosing branches of the arteries and veins are shorter and more numerous at the centre, and longer, less numerous and more independent of any inosculations in a peripheral direction.

**Physiologic Modifications.**—When the choroid is fully developed, and the eye has undergone no senile changes, the sclera is entirely hidden from view, and the eye-ground presents a uniform tint. If pigment is abundant in the layer of hexagonal cells this tint is an even reddish-brown. If the layer of hexagonal cells is fairly transparent, from sparseness of pigment, the predominant tint is that of the chorio-capillaris, or an even scarlet. In neither case, however, does the uniform tint extend to the peripheral zone of the fundus, for, as first stated, here the vascular networks are normally less dense, so that the longer *reaches* of the larger vessels, with their intervening pigment spaces, are only partly or not at all concealed by pigment epithelium and capillaries. Hence, the radiating streaks of red, alternating with wider ones of brown, that are to be seen even in the deepest colored grounds in this location.

In proportion as the growth of capillaries is less luxuriant the choroidal vessels toward the centre of the fundus become visible. This most frequently occurs in the nasal half and about the papilla. Then the interspaces are dark or light



in accordance with the amount of pigment in the choroidal stroma. Rich pigmentation of the stroma, in contrast with the luminous red of the vessels, imparts a most striking tassellated effect, that has often been mistaken by the tyro for some serious pathologic condition, like retinitis pigmentosa, or disseminated choroiditis (see Fig. 60). The tassellated fundus is characteristic of Mongolians and white

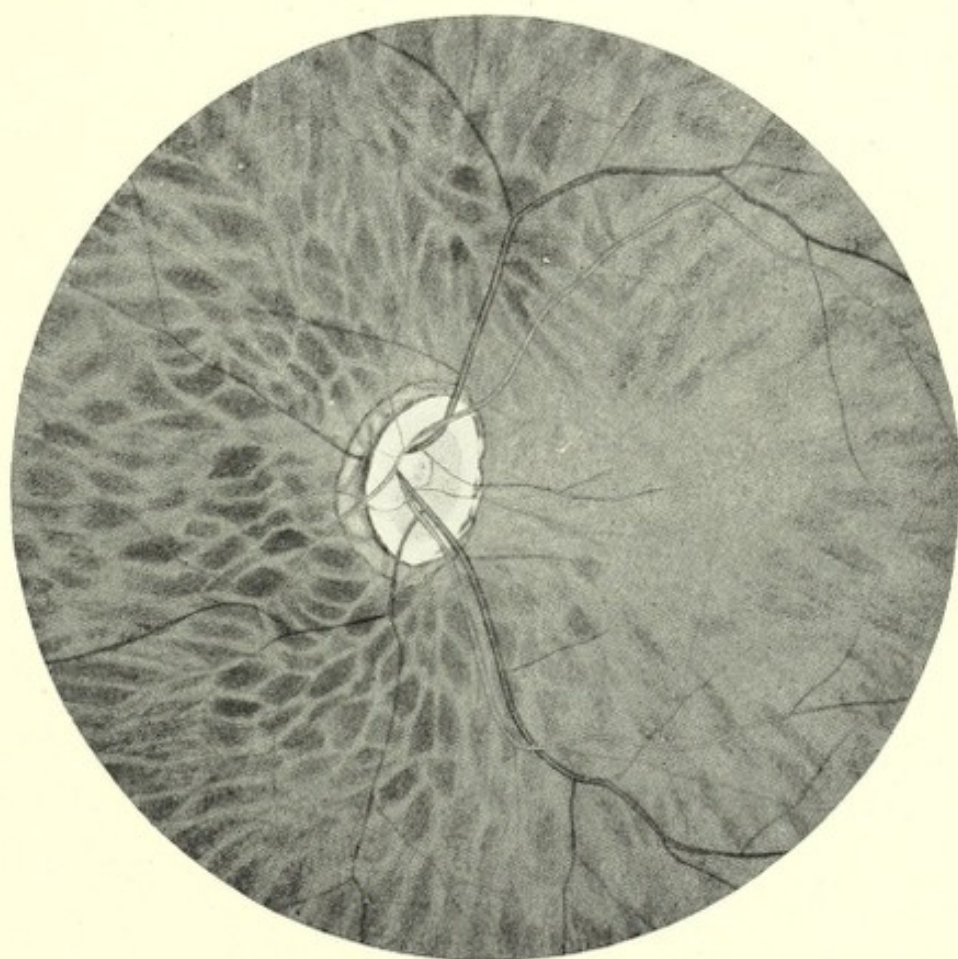


FIG. 60.—Tessellated fundus.

Orientals. Owing to the more numerous and shorter anastomoses of the vessels as the centre is approached, the checkering here is on a smaller scale than that further out. The different grades bear a close resemblance to the mottling over the different portions of the body as seen in the giraffe and the jaguar. It is in senile sclerosis of the choroid, in which the islands of pigment are thin, and veiled with connective tissue, and the vessels are pale yellow and white,



and the whole fundus has a tawny look, that we get the "**fundus tigrée**," which is well named, for the picture here seen is suggestive of a shabby old tiger-skin rug.

In the measure that the fundus is devoid of pigment of any description, epithelial or stromal, the sclera becomes visible in the spaces between the vessels. In the total absence of pigment of the albino, the interstices have the faintest

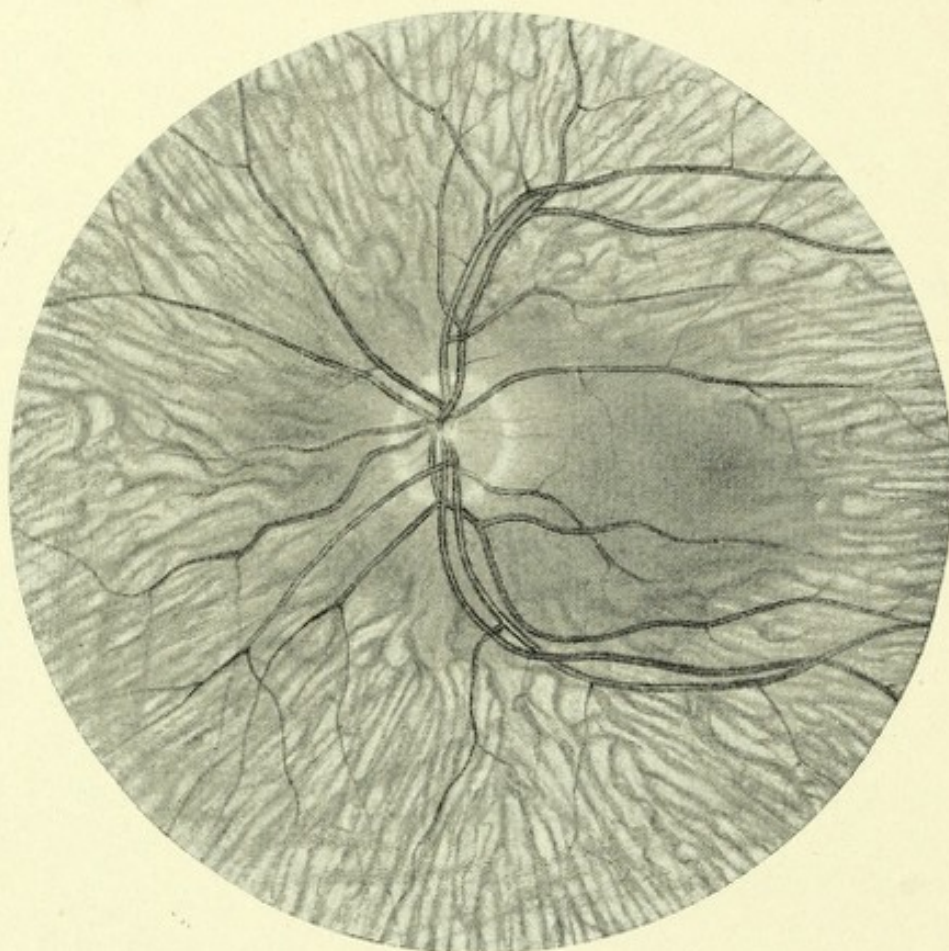


FIG. 61.—The fundus of an albino.

possible tinge of pink, owing to the capillaries of retina and choroid, whereas in pigment deficiency from atrophy of the choroid the sclera in these spaces is glaring white. Figure 61 is the fundus of an albino. It gives a fair idea of the appearance of the choroidal vessels, particularly as to the manner in which they inosculate. The designs they form are precisely like those made by the gravitation of the granular film of liquid that remains on the sides of a tumbler that has



just been emptied of butter-milk. Usually there is no way of distinguishing the arteries of the choroid from the veins. In the fundus which is represented by Fig. 59, the vortex trunks were of a decided crimson tint, but over the rest of the fundus the vessels had one color in common. Both this and the fundus of the albino (Fig. 61) show very small central areas of chorio-capillaris, and the fields are cor-

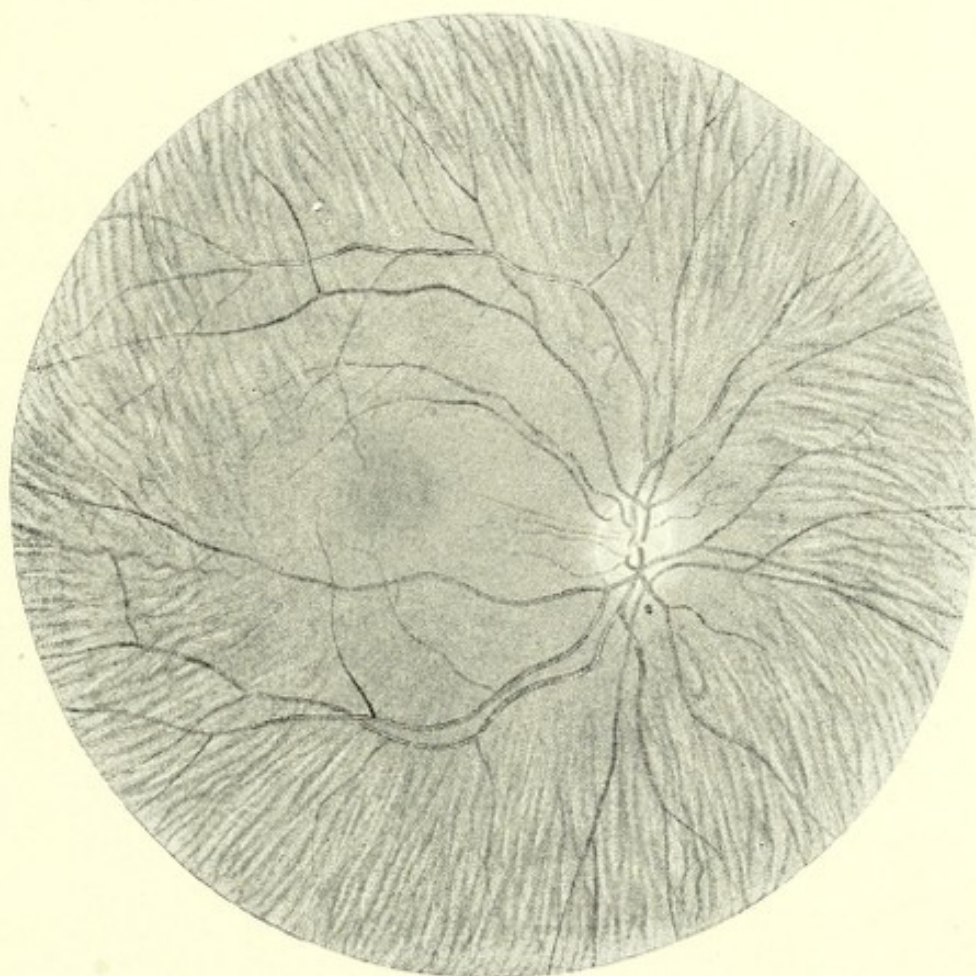


FIG. 62.—“Poor” choroid.

respondingly contracted. The choroids are little more than rudimentary.

**Absence of Choroid.**—In this connection we are reminded that there have been published a few reports of rudimentary choroid or, as they have been most often termed, **absence of the choroid**. Such a case was published by Cowgill in Knapp's Archives, Vol. XXI, p. 105, 1892.

**Poor Choroid.**—This is a term used by the writer in his



private case records, to designate a certain type of fundus. The retina and papilla are normal save perhaps that the latter shows a slight lack of color. The choroid appears, on the whole, thin and meagre. It is devoid of stromal pigment, the vessels are narrower and more sparse than normal, a conus is frequently present; the chorio-capillaris is developed sufficiently to mask the vessels only in the macular and perimacular regions, and even here the choroidal vessels

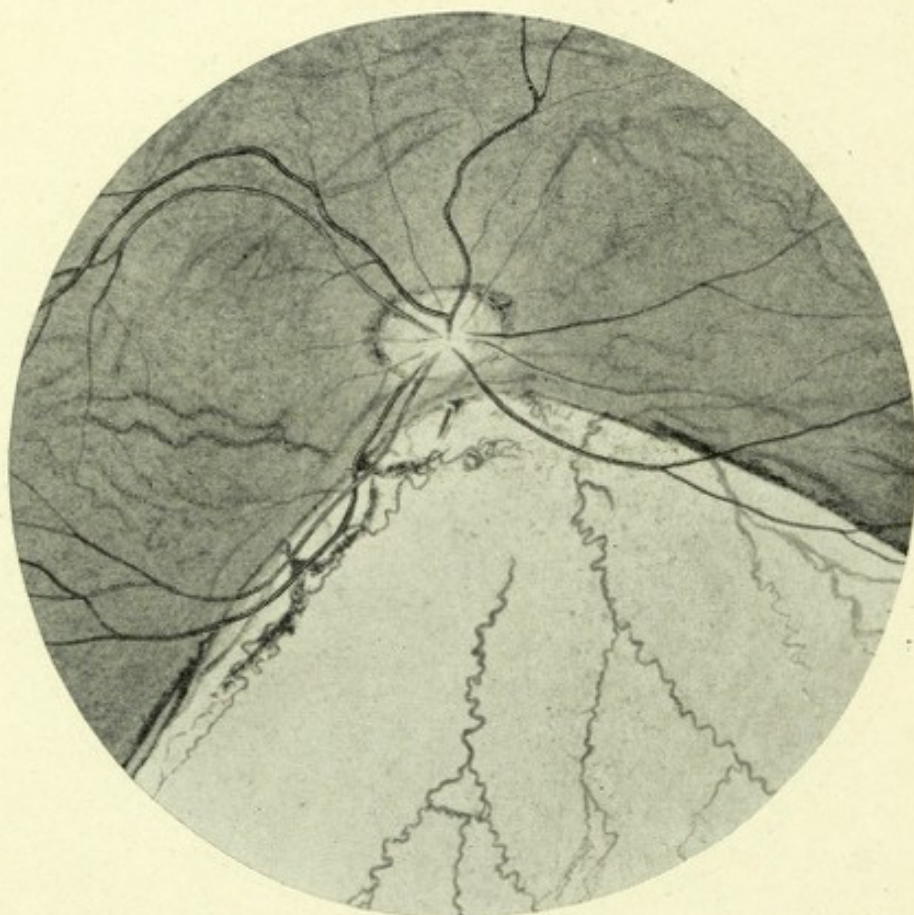


FIG. 63.—Right eye. Papilla not included in coloboma.

are not wholly concealed. Over the entire area characterized by evidences of any considerable capillary development there is that granitoid appearance, due to irregular distribution of the pigment of the hexagonal cells, known as "powder grain," or "peppered" eye-ground (Fig. 62). There is likely to be pronounced astigmatism, but the vision is subnormal in any case. There is a lack of robustness in the whole organism, but no lack of mentality; indeed, if



young, the subject is probably intellectually precocious. In a small child, there is always the prospect of an improvement of the conditions, particularly if wholesome precautionary measures are adopted in the rearing.

**Coloboma of the Choroid.**—Typical coloboma of “the floor of the eye,” as the French say, is most often bilateral, and may exist independently of any anomaly of the iris,

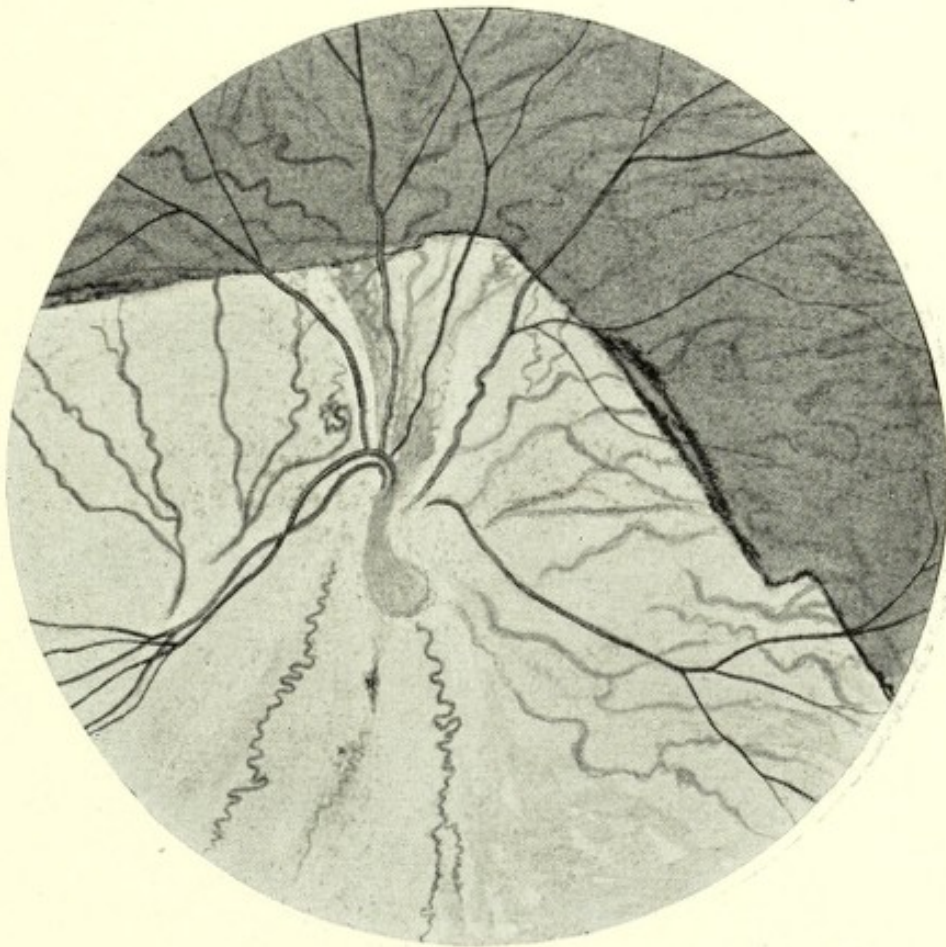


FIG. 64.—Left eye. Same case as preceding. Papilla included in coloboma.

though the greater number of these cases are accompanied by cleft of both iris and ciliary body. In the ophthalmoscopic examination one is perhaps first struck by the bluish-white of the pupillary reflex, especially in looking a little downward. On moving the head from side to side the white alternates with the normal red of the fundus. The intensity of the pearly reflection depends upon the amount of tissue that covers the sclera, and upon the topography of the



defect. The defect varies in extent from a circumscribed oval, no larger than the papilla, to one that involves the lower third of the fundus. There is, in most instances, a strip of normal appearing choroid between the summit of the coloboma and the papilla, but frequently the nerve-head is included. Each of these conditions may be represented in the eyes of the same individual, as shown in Figs.

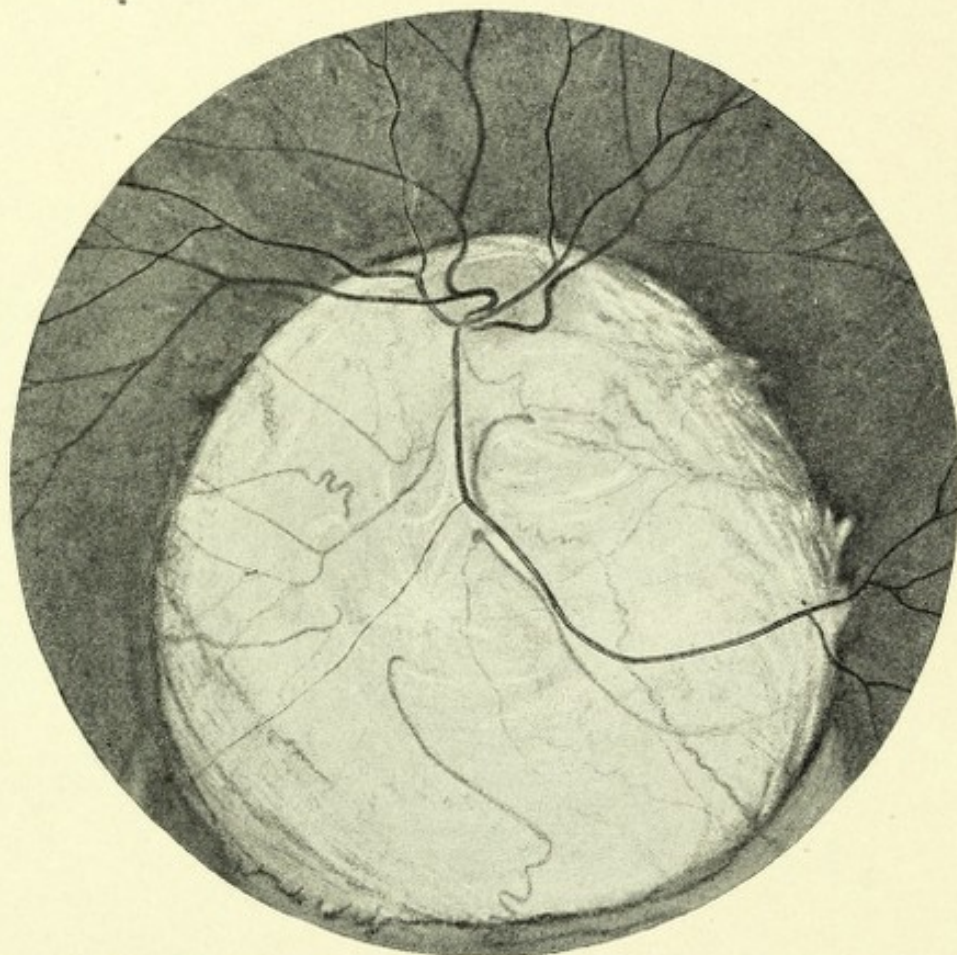


FIG. 65.—Coloboma of the choroid showing irregular topography of the "floor."

63 and 64. The floor of the coloboma is usually ectasic, either as a whole, or irregularly. The papilla often contains an anomalous physiologic excavation. In crossing the defect the retinal vessels are seen to bend to adapt themselves to the uneven character of the ground. Straggling and sinuous choroidal vessels are seen here and there, sometimes being distorted, and dwarfed posterior ciliary arteries, that perforate the sclera in the midst of the defect. An irregular



border, of densely massed pigment, surrounds the coloboma, and varying patches of the same material, without definite contour or arrangement, are frequently scattered over the white of the sclera (Fig. 65).

Jaeger's rendering of coloboma of the choroid is classic. All who are familiar with his Atlas can get a tolerable idea of the excellence of the master's work, but only those who have seen the original drawings can fully testify to their true beauty and their faithfulness to nature. The few who have had the fortune to examine ophthalmoscopically funduses that he had already depicted can, in addition, appreciate his conscientious attention to minute detail. The present writer is one of those so favored. In the matter of this coloboma, for instance, he made comparison, detail by detail, between drawing and living fundus, and, so far as concerned the likeness, it was as if the fundus had been photographed in colors. At this time, which was fourteen years after the Atlas was published, only the fundus of the right eye was visible, the left having been obscured by a cataract.



## CHAPTER XVII.

### LIGHT AND DARK SPOTS.

Pathologic alterations manifest themselves in the fundus by patches of white, or light red, and black, or some intermediate shade. White and yellowish spots refer to exudative, destructive or cicatricial lesions, red to hemorrhagic, and black to pigmentary lesions. The three varieties appear singly or combined—most often combined. By the use of the ophthalmoscope one endeavors to ascertain their physical properties by translating their visible signs. Not that it is always possible to make a diagnosis by the ophthalmoscope alone, for in some instances a definite conclusion cannot be arrived at without the aid of the interrogatory and that of the functional examination. It may be that the diagnosis hinges upon the information to be gained from repeated ophthalmoscopic examinations extending over considerable time, by which progress or absence of progress is noted. Leaving the red for the present we will consider certain affections in which light or dark spots, or the two in combination, are conspicuous features. The area of the "spot," as the word is here employed, includes that of all dimensions between the smallest visible and that which is equal in area to the optic disc. The larger ones are considered elsewhere.

First a few words as to the **general characteristics of light spots**. These, either white or slightly tinted, appearing in the fundus, singly or multiplied, in groups or disseminated over considerable territory, discrete or confluent, are the expression of a retinal, a choroidal, or a chorio-retinal lesion, and indicate cellular infiltrations, non-cellular exudations, or cicatrices. The number and dimensions of the spots are of less diagnostic value than are their situation and arrangement. White spots, whether occurring in retinal or cho-



roidal affections, are usually located behind the retinal vessels; that is, the vessels are seen to pass clearly over them. If they are more superficial than these vessels they almost surely belong to the retina, and are due to infiltrations and exudations of the nerve-fibre layer, or they correspond to verrucous hypertrophy of the fibres themselves, or they are due to serous effusions that escape from the retina into the subhyaloid space—**pre-retinal**. Among the white spots that cover the retinal vessels the most distinctive are those that accompany the neuro-retinitis of acute parenchymatous nephritis, and they are most frequently observed in young or relatively young subjects—a form of **albuminuric retinitis**. They are situated in the peripapillary zone of the fundus about the limits of the papillary edema which is always present. By their growth and coalescence they form an irregular girdle of fleecy white masses, varying in size from one-fourth to one disc area, that by their round outline bear a close resemblance to the form of clouds known as **cirro-cumulous**; hence the ophthalmoscopic tableau is called “mackerel-sky effect” (see Fig. 66). Similar white cloud exudations are sometimes seen in acute syphilitic retinitis, but they are more isolated—often single, and their seat is usually the macular or posterior polar zone. A characteristic of the cotton-like and cloud-like superficial white spots of the retina is a sort of phantasmal iridescence that plays over them. The deep retinal opacities are smaller, and of a silvery whiteness. About the superficial spots there is a suggestion of translucence, but the deep ones are as opaque as metal. The largest vessels pass **through** or **beneath** the spots of the nerve-fibre layer; the deepest (smallest) retinal vessels pass cleanly over the deeper spots. These last are between the granular layers, as in albuminuric and diabetic retinitis, or they are in the pigment epithelium (see “Macular Spots of Bright’s Retinitis”).

Occasionally, in angio-sclerotic subjects, one sees exceedingly small, perfectly round, shining, white spots scattered over the fundus and that lie immediately *upon* the vessels.



DESCRIPTION OF FIG. 66.

“Mackerel sky effect” in albuminuric retinitis.





FIG. 66.







They are most numerous in the peripapillary region, where they are sometimes as thickly studded as stars in the firmament. They were formerly supposed to pertain to the "hyaloid membrane" or capsule of the vitreous, but as it is now believed that there is no such membrane, they are relegated to the membrana limitans interna, and are probably centres of hyalin degeneration around the bases or feet of Müller's fibres. The white spots of the choroid, as a rule, denote cicatrices, and are brilliant and solid, like mother-of-pearl. If the spot represents a lesion that is **active**, it is more apt to have a yellow, or buff, tinge. When the retina over the spot becomes edematous, as often happens, that membrane is rendered opaque there and hides the spot. Whether white or yellowish, *if the outline of the choroidal spot is indefinite, the lesion is in an early period of evolution.*

A decided tint, blue or green in a retinal or choroidal spot is indicative of **elevation** or **tumefaction** as well as of activity. It might be a button of **disseminated choroiditis**, a **conglomerate tubercle** or a **tumor** (Figs. 67 and 75). Its appearance would then be as if composed of soft, tinted pastel, or chalk. A circumscribed **ectasia** of the choroid will also have a tint—but one of gray, not blue nor green. Besides, it looks hard and pearly, and often has a border of pigment. That which, above all, serves to distinguish a choroidal spot from one of the retina is the **presence of pigment**. A **white and black** spot is necessarily of the choroid, or of the retina and choroid. The only retinal spot that can simulate it is one that is in the last stages of evolution from a hemorrhage to a white spot, when the old, changed and darkened blood-remains might appear black in contrast with the new white. Such a transformation of a retinal hemorrhage that has escaped outward, or in contact with the pigment epithelium, could have an admixture of real pigment. Black spots without white may occur in the retina, but they are of distinctive, moss-like forms, as in retinitis pigmentosa. Choroidal spots, whether white or black, or black and white, are



DESCRIPTION OF FIG. 67.

Active button of disseminated choroiditis.



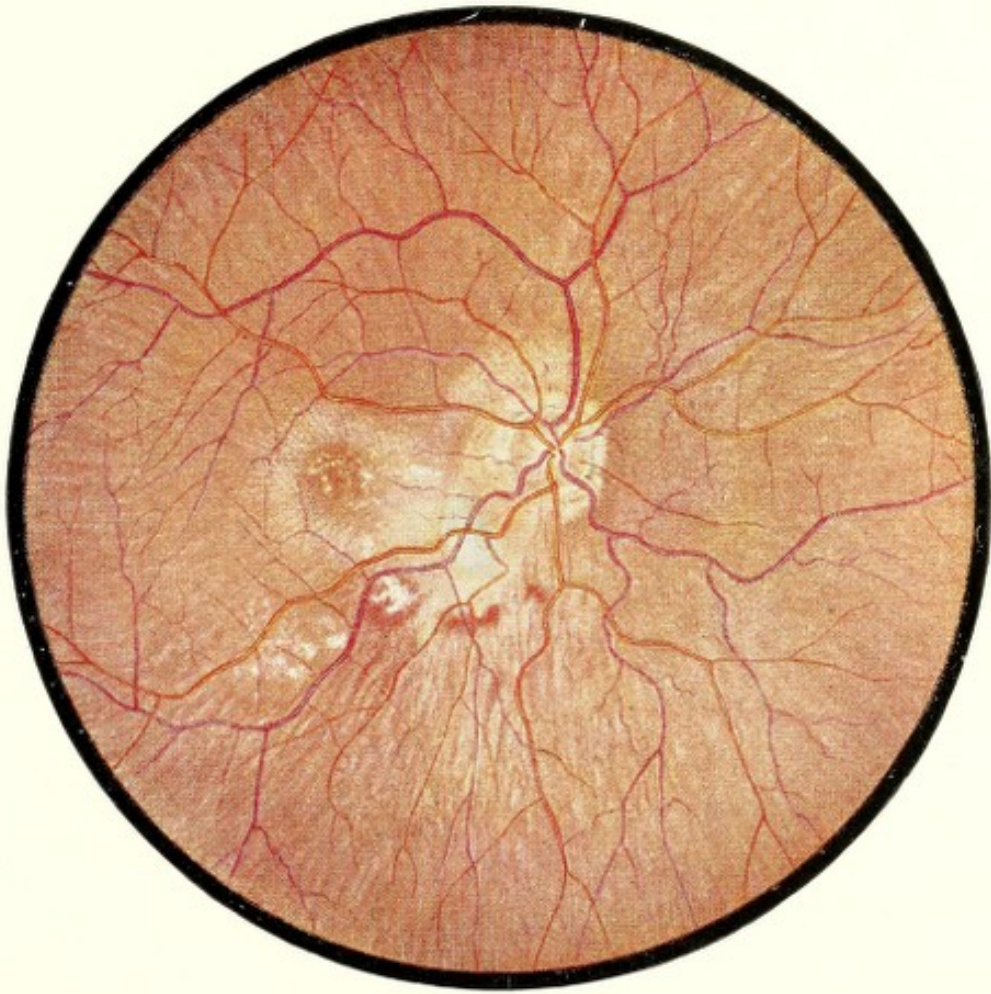


FIG. 67.







strongly inclined to circular, or rounded forms. The result is that the larger spots, and even the great areas of choroidal degeneration, exhibit polycycle effects, showing that they have been made up of the coalescence of several, or many, circular plaques.

**Retinitis Punctata Albescens.**—This is a term applied by Mooren, in 1882, to an affection of the retina wherein the fundus was dotted with small white spots by the hundreds, and wherein, after an interval of several years, there was no appreciable change. The disease is rare, but of distinctive

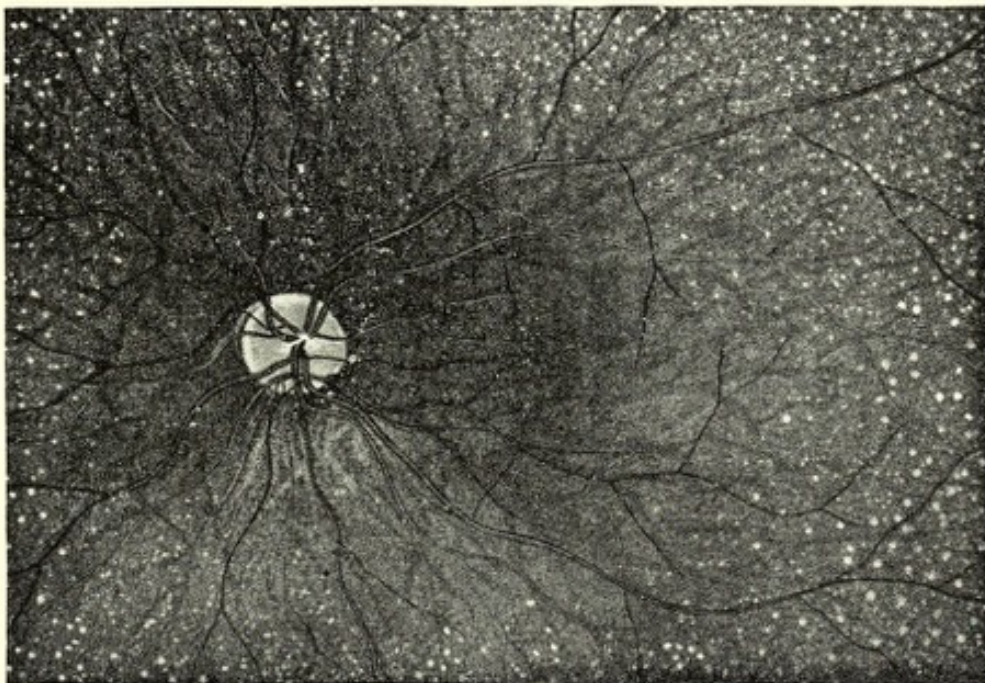


FIG. 68.—Retinitis punctata albescens. (After Dufour.)

type ophthalmoscopically (Fig. 68). The number of spots is extremely great; they are strewn over the fundus from the papilla to the extreme periphery, but *none is found in the macula*. Around the posterior pole they are smaller than toward the ora serrata. They are pure white or very slightly grayish. Their form is rounded, and their outline sharply defined, without any bordering of pigment. The diameter of the largest does not exceed that of a retinal vessel of the first magnitude; from that down they appear of all sizes till no longer visible. They are peculiar in being



so netly discrete; their freedom from any tendency to coalesce having caused Gayet to liken them to the white spots on the plumage of the guinea-fowl. They do not make apparent any parallax displacement, and they are, without exception, located in a plane deeper than that of the retinal vessels, but anterior to that of those of the choroid. The general tint of the eye-ground in retinitis-punctata albescens is usually quite dark, even when the subject is of blond complexion.

The disease is bilateral, congenital and familial. It has been observed in several children of the same parents. It has for subjective symptoms hemeralopia, concentric diminution of the visual field, and, in the ulterior stages, more or less pronounced reduction in the acuity of vision. These symptoms, together with the fact that, in case of a brother or a sister of the patient, there have been found in the retina a few of the characteristic moss-agate pigment figures, have led to the belief that there exists a very close relation between the affection in question and retinitis pigmentosa. This has been made all the more probable since a number of observers, among them Fuchs, Gayet, Spengler, Querin and Galezowski, have noted consanguinity of the parents. At yet there has been no anatomo-pathologic examination of an eye with the disease. All that can be said relative to its precise seat is that the white spots seem to be in the deepest layers of the retina—perhaps in the pigment epithelium. It is possible that, also like retinitis pigmentosa, the retinal changes may be secondary to an affection whose point of departure is in the choroid. There is little if any tendency to progression.

**White Spots and Hemorrhages in Diabetic Retinitis.**—The earliest reports of retinitis in conjunction with diabetes, as, for example, those of Jaeger and Desmarres, were to the effect that the ocular disease was a coincidence, and attributable to renal complications. Noyes, of New York, in 1869, was the first to show that retinitis characterized by white spots of degeneration, could develop in the course of



diabetes independent of nephritis. Notwithstanding the somewhat extensive chemical researches of Galezowski, Lagrange and Hirschberg, some ophthalmologists still seem undecided as to the existence of an ophthalmoscopic picture typical of diabetes. After having collected and analyzed nineteen cases, Leber concluded that the various retinal alterations, taken as a whole, presented nothing characteristic of diabetes. Nevertheless, all are agreed that certain fundus appearances have come to be regarded as pathognomonic, and demand an immediate search for glycosuria.

According to Hirschberg there are three principal types of visible diabetic retinal changes.

1. A central punctate retinitis. Retinitis punctata diabetica.

2. Retinal hemorrhages with focuses of degeneration.

3. Rarer forms with inflammatory or degenerative changes whose connection with diabetes is not clearly established.

The first form, Hirschberg considers as absolutely typical, and the lesions quite distinct from those of albuminuria (Fig. 69). They consist of **numerous small white spots** situated in the retinal tissue, mostly in the posterior polar region. By their brilliant whiteness, irregular form and varying sizes the writer has always associated their appearance with that of the snow that falls in flakes. They group themselves in a desultory way near the large temporal vessels of the retina, and about the macular region; (*but never show an arrangement in the last-mentioned locality approximating the radiate figure of albuminuric retinitis*). A few of the dots are scattered in the peripapillary region, and still fewer occasionally in remoter parts of the fundus. The largest of them seldom covers an area equal to one-fourth that of the disc, and they are never pigmented. Interspersed with the spots, and dotting the more peripheral parts of the eye-ground, are a number of minute hemorrhages. These are less numerous than the white spots, and they are continually coming and going. The focuses of degeneration are more fixed, and their changes consist



DESCRIPTION OF FIG. 69.

Diabetic retinitis.



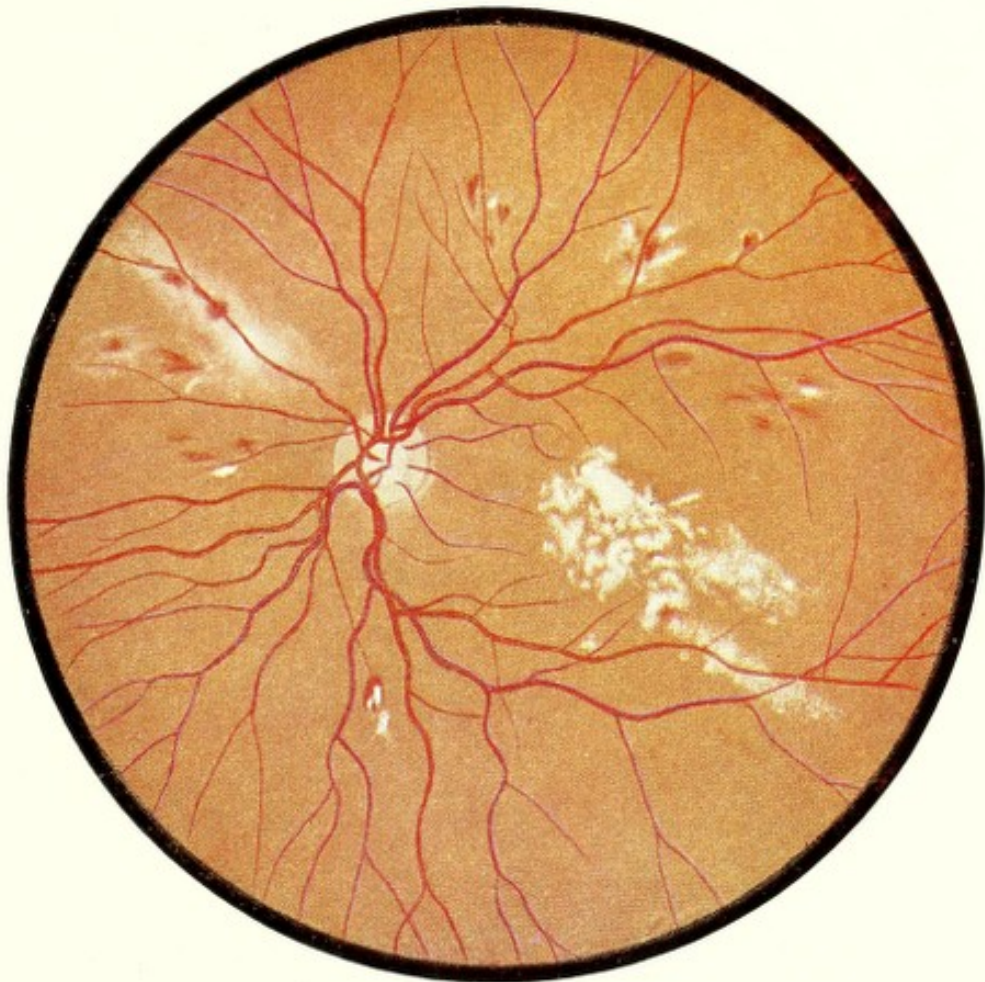


FIG. 69.







chiefly of augmentation in size and number. They are not confluent as in nephritic retinitis.

**The hemorrhagic forms** of the affection may be combined with the punctate, or they may precede the latter. They are less characteristic than the first type, and subject to greater variations. Hirschberg divides them into four categories, according to the degree of intensity. *a.* Punctiform hemorrhages; *b.* more extensive hemorrhages, with or without opacities of the vitreous; *c.* hemorrhagic infarcts of the retina; *d.* hemorrhagic glaucoma.

The white spots, when present in these forms, are larger and fewer. The intra-retinal hemorrhages are in the nerve-fibre layer, and it is most likely their invasion of the vitreous that gives rise to the opacities often found there. The papilla is usually free from edema, as is also the retina in its vicinity. Mild neuro-retinitis has been occasionally observed with the hemorrhagic forms. Rarely are there any visible alterations in the retinal vessels, and when present these may be the expression of coincident renal disease, or of arteriosclerosis—two very common complications. Among the characteristic associated affections of the eye are **cataract** and **hemorrhagic glaucoma**.

Retinitis is a tardy symptom of diabetes, in most instances the general disorder has existed for a long time before the advent of the fundus signs. The expectancy of life is much greater, especially when the patient's manner of living is well ordered, than is the case with albuminuric retinitis, and in subjects of similar age. The author has known a number of instances wherein the diabetic retinitis has existed for from twelve to twenty years. In some the retinal disease has remained apparently stationary for long periods of time, notwithstanding the fact that large percentages of sugar were constantly present in the urine. The degenerative spots, as they become older, lose their whiteness, and appear yellow—later taking on a soiled yellow tint. Hence, if there are no fresh white spots—if they are *all* dull or “dirty”-looking—especially if there are no recent hemor-



rhages, one may conclude that the progress of the retinitis has been arrested—temporarily at least. This retinitis is a relatively rare complication affecting only about 4 per cent. of all diabetic subjects. It is bilateral, sooner or later, in the majority of cases, though it may be confined to one eye for a considerable period.

The ophthalmoscopic signs have sometimes been confused with those of albuminuric retinitis, and their atypic forms are occasionally impossible of differentiation, requiring a urinalysis to clear the doubt. Besides, there is always the possibility of a combination of the two.

The white spots of diabetes are always of sharp outline—never diffuse like the large ones of Bright's retinitis, and they never cover the retinal vessels. They do not coalesce. They are of more irregular form than the smaller ones of the albuminuric variety. The hemorrhages in the more typical cases of diabetic retinitis are rounder, not flame-shaped, and do not radiate from the papilla, but are more disseminated than in Bright's retinitis (see chapter on "Hemorrhages in the Fundus").

**Verrucosities of the Lamina Vitrea. "Drusen."**—This affection may be confounded with retinitis punctata albescent. The anatomic feature consists in more or less numerous minute nipple-like prominences which spring from the inner surface of the lamina vitrea, elevating and causing pressure atrophy of the pigment epithelium. They manifest themselves as *light* spots or *white* spots in accordance with the density of the epithelial pigment, and the altitude of the excrescences. In the darker eye-grounds, with only moderate elevation of the growths, the dots are smaller and of orange tint. With lighter eye-grounds or excessive development of the "**drusen**" the spots are larger, rounder, and brilliant yellow or white. Their diameter, however, is never greater than 1 mm. ophthalmoscopic magnification. They are not outlined with pigment, and they are most frequently bilateral, though not symmetrically so. Their favorite localities are the peripapillary and perimacular



regions. They appear in clusters or constellations, and this constitutes one of their most distinctive traits. Individually their form is not so round as that of the white spot of white punctate retinitis.

At times these growths reach such proportions as to disturb the function of the rods and cones upon which they impinge, but ordinarily they do not interfere with vision.

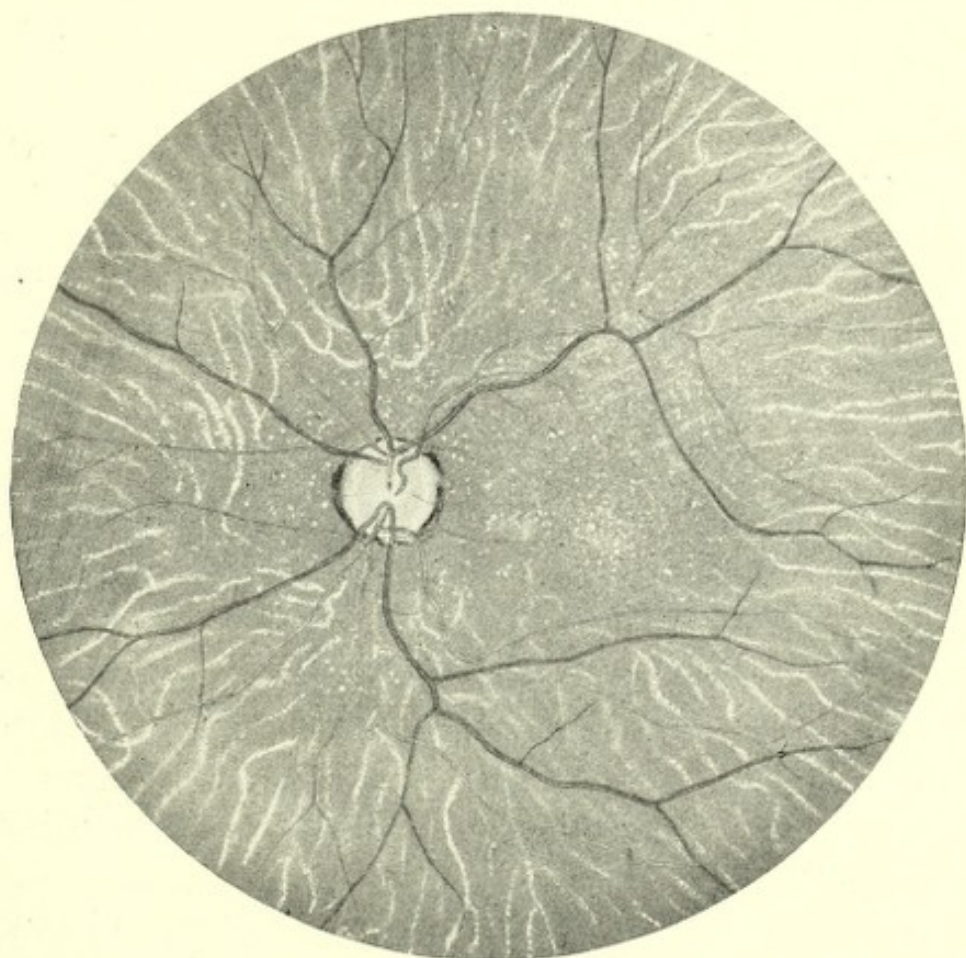


FIG. 70.—Tay's choroiditis.

They are essentially an expression of senility. This is the affection that was described in 1881 by Mr. Warren Tay, then assistant to Hutchinson, and that has, in consequence, been often referred to as **Tay's choroiditis**; and probably the same that, when situated at the posterior pole, was by Nettleship called **central senile guttate choroiditis**. Fig. 70 is from a drawing of the fundus of one of the cases included in Tay's report, the woman having afterward migrated to



Chicago. These spots are not to be confused with **Gunn's dots**, which appear only in the macula and in young subjects; nor with the irregular light or white patches, alternating with pigment, in central senile atrophy. The disease is slowly progressive.

**"Oil Spray" Effect.**—There is yet another modification of the spotted fundus, that, while it may not be classed as a separate affection, has clinical features that are sufficiently distinctive, and presents an ophthalmoscopic picture so different from either of the two last-described diseases as to warrant special mention. For want of a better name the writer has designated it by the caption that he gives this paragraph. That it is not the result of senility is proven by the fact that it is found as often in the young as in those of advanced years. Neither is it secondary to any pronounced pathologic processes in the blood-vessels of the choroid, as there is never a symptom, objective or subjective, other than that which suggested the name, although the case may be for years under observation.

Fancy the magnified ophthalmoscopic picture as created in the following manner: First, as the foundation, or basis, the entire fundus is painted with a coat of soft buff. Over this when dry is spread an exceedingly thin but perfectly uniform layer of fine dust or powder of a color somewhat redder—say brownish-pink. Then fancy this dust sprinkled with a fine spray of transparent oil, but in such a way that no two of the tiny globules are superposed, or confluent. Lastly, imagine a setting, or inlay representing optic disc and retinal vessels normal in every particular. The whole background will be thickly studded with minute, barely perceptible circles of dark brownish-pink, each enclosing its tiny disc of soft buff, the intermediate spaces being tinted an even brownish-pink intermediate between the soft buff of the discs and the other brownish-pink of the circles. Each globule of oil as it fell on the dust washed the dust to its periphery forming a ring and leaving exposed a disc of the priming coat of buff. Such is the finished picture (see



Fig. 71). Nowhere are the spots obtrusive—nowhere in decided contrast with the background. The spots invade every region of the fundus—even the fovea not being exempt, for here are situated the largest. There is no tendency to grouping. The spots in the macula—the largest—are about one-fifth the disc diameter. From that they grade down to infinity. The smallest visible retinal vessel passes straight

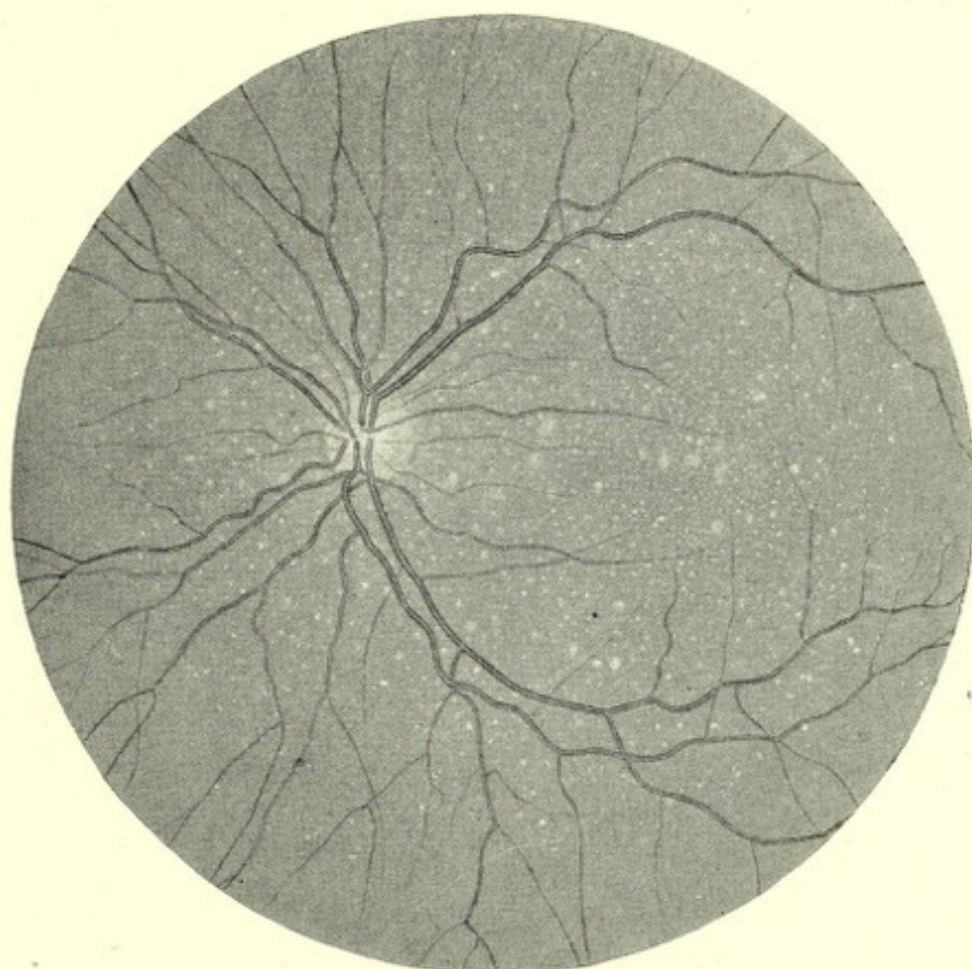


FIG. 71.—"Oil spray" effect.

over them—they exhibit no paralaxes. As in other speckled fundi the spots here seem to refer to the hexagonal epithelium, and answer fairly well to what Treacher Collins, in 1888, termed **colloid degeneration of the pigment epithelium**. The phenomenon is bilateral, but not hereditary nor familial, although it may at times be congenital.

**Choroiditis Disseminata.**—This is by far the most frequent cause of spots distributed over the fundus. The name is



derived from the fact that the disease is characterized by discrete focuses of inflammation disseminated over different regions of the fundus or over its entire extent. Sometimes the focuses are limited to the posterior polar and equatorial zones, but avoiding the macula (**areolar choroiditis of Foerster**). Sometimes the equatorial zone alone is the seat (simple **disseminated choroiditis**) and again it is the retro-ciliary zone (**anterior choroiditis of Fuchs**). This division of this form of choroiditis by regions rests upon an important anatomic principle—the delimitation of certain circulatory zones in the choroid. The focuses are situated in accordance with the classification of the terminal branches of the short posterior ciliary arteries. Thus we distinguish focuses corresponding to the macular branches (**central disseminated choroiditis**) those corresponding to the perimacular and peripapillary branches, those to the equatorial, and those corresponding to the domain of confluence between the longest of the short posterior ciliary and the recurrent vessels of the arterial circle of the iris. Under ordinary conditions the equatorial zone is most often the seat of the choroidal eruption. In the consecutive eruptions the buttons of inflammation invade the retro-ciliary zone in front or the perimacular zone behind. The macular and the peripapillary are the regions least often affected.

While the hyperemia is only in a measure confined to certain relatively limited territories, the infiltration, serous or cellular, is perfectly circumscribed, or focalized, in small, isolated points. It is at these points exclusively, where the choroidal stroma is edematous, and there that the young, round cells accumulate in nodules of inflammation. Their location in the choroid is in the intra-vascular or dermic tunic, among the terminal arterioles and initial venules. They are never found in great abundance either in the chorio-capillaris inward or in the stroma of the tunic of larger vessels outward. The primary nodule is microscopic, and invisible with the ophthalmoscope. It often undergoes resorption and disappears without leaving a trace. From



being a **centre of accumulation** of white globules in and around the vessels it passes to become a **centre of proliferation**. It then constitutes the infectious tubercle, or focus of disseminated choroiditis, and first manifests itself ophthalmoscopically by the alterations it produces in the overlying pigment epithelium. Like the nodule, the tubercle, and the spot it causes, may disappear and the fundus at the point resume its normal tint. More frequently there ensues necrosis and caseous degeneration at its centre, with connective-tissue formation at the periphery or throughout—*i.e.*, **sclerosis**. In consequence there is complete effacement of the choroid at this point, and with it a corresponding destruction of the retina; so that instead of the *elevation* of the first stages of the process there now exists a *depression* from the atrophic thinning of the two membranes. In the period of proliferation and swelling the focus is a **button**, in that of destruction it is an **excavation**, but it is always a **spot**.

In progress of development both the stromal and the epithelial cells are deprived of their pigment. It is this discoloration, or local fading, of the eye-ground, and not, as formerly supposed, the presence of exudation, that first makes visible the focus. A spot of paler red or chamois-yellow appears. In proportion as the pigment-cells and the original connective tissue are preserved, the spot assumes a grayish, or soiled-chamois appearance. Near the end of the time of resolution the pigment-cells immediately surrounding the focus begin proliferation. The young cells, black like their parents, pile one upon another, forming around the blanching focus a more or less complete—more or less regular—framing of pigment. Sometimes the young ambient cells move toward the centre of the spot and there depose themselves in irregular masses. The appearance of the pigment around the spot marks the final stage in the evolution of the choroiditic focus. From this moment there is no further change excepting that which is wrought by the advent of new buttons; an occurrence that is very frequent,



tor disseminated choroiditis is a disease essentially prone to recidivations.

Sometimes, instead of an initial depigmentation of the focus and the formation of a **yellow button**, there is a superpigmentation, resulting in a **black button**. The last presupposes a less active local inflammation than that of the yellow button. Occasionally the black button on the first appearance is angular or elongated, instead of round, and very small, but it enlarges rapidly, becoming meanwhile of rounded contour. Like the yellow, the black buttons are discrete, and it is only by successive eruptions that polycyclic plaques are formed. Black and yellow buttons often intermingle. Around each black button is a zone of discoloration—**zone of attack**. The apparition of this light border coincides with the cellular disintegration that follows the inflammatory hypernutrition. When the disintegration does not occur the button remains without its border. At a given stage the centre of the black button begins to clarify, changing to gray, yellow, or white. In case of a small button it seems to be illuminated at the centre. The clarification extends to the periphery—the entire spot becomes white, save for an irregular border of black. In short, the final stage is identical with that of the yellow button. The summits of the black button never reach the altitude attained by those of the yellow, so that the retinal vessels do not rise in an appreciable curve over them. As to the number of choroiditic focuses, it varies from one or two to dozens.

**Appearances in the Stage of Progression.**—At the beginning one finds in one or more parts of the fundus, spots of lighter tint than that of the unchanged choroid; their faded yellow outlines are not sharp but blend rapidly with the ground tint. No pigment is to be seen within nor encircling them. In those eyes in which the choroidal vessels are normally visible, their grillage is hidden where the spot intervenes; but the retinal vessels pass over unbroken. The dimensions of the spots vary greatly. The smallest, as seen magnified,



measure 1 or 2 mm. in diameter; the largest may reach a size equal to that of the optic disc—or even one greater, but these dimensions are rare, the usual diameter not exceeding one-fourth to one-third that of the disc. The small buttons are usually located more superficially in the choroid (*dermic*) and the largest deeper (*sub-dermic*).

Both small and large are elevated above the surrounding level, as evinced by the abrupt curve in the retinal vessels that chance to pass over them, by the parallax of the curve with the eye-ground, and by the refractive measurements (see Fig. 67). They form separate islands scattered promiscuously over the fundus, or they are disposed in archipelagoes. The last is truer of the very small spots.

**Retrogression.**—After a given season the spots brighten, gradually approaching a clearer, lighter yellow tint. Then, as regards the smallest focuses, they disappear, or (particularly the larger ones), become white. The white is that of the sclerotic showing through the atrophied choroid. This white is purer and more shell-like in the ratio that the degeneration of the choroid has been complete. In the event that any of the choroidal vessels there have escaped destruction they are seen as stripes of bright red on the white ground (Fig. 72). A tinge of gray or blue in the white denotes ectasia, which in turn bespeaks adhesion between the remnant of choroid and the sclera—an adhesion that probably includes also the thinned retina. During the transformation in the original color of the spot, its limits become defined by the appearance of a dusky outline indicative of pigment. As the white intensifies, so does the black. This black border is never above the level of the layer of pigment epithelium, and always permits the smallest retinal vessels to pass straight over it before they dip into the atrophic cup that replaces the focal button.

*There is never any pigmentation of the retina in simple disseminated choroiditis.* Out to the very edge of the spot, *i.e.*, throughout the area where the retina is soldered to the choroid, there is no pigment. It appears only around the



site of the adhesive inflammation. True, in the midst of the white spot of atrophy are often to be observed splotches of the pigment like that of the border, and it is really of the same origin. This supplementary pigmentation occurs when, in a large choroiditic button, the atrophic process has several distinct centres, thereby permitting the uveal pigment to persist in the intervals. The cicatricial spot does

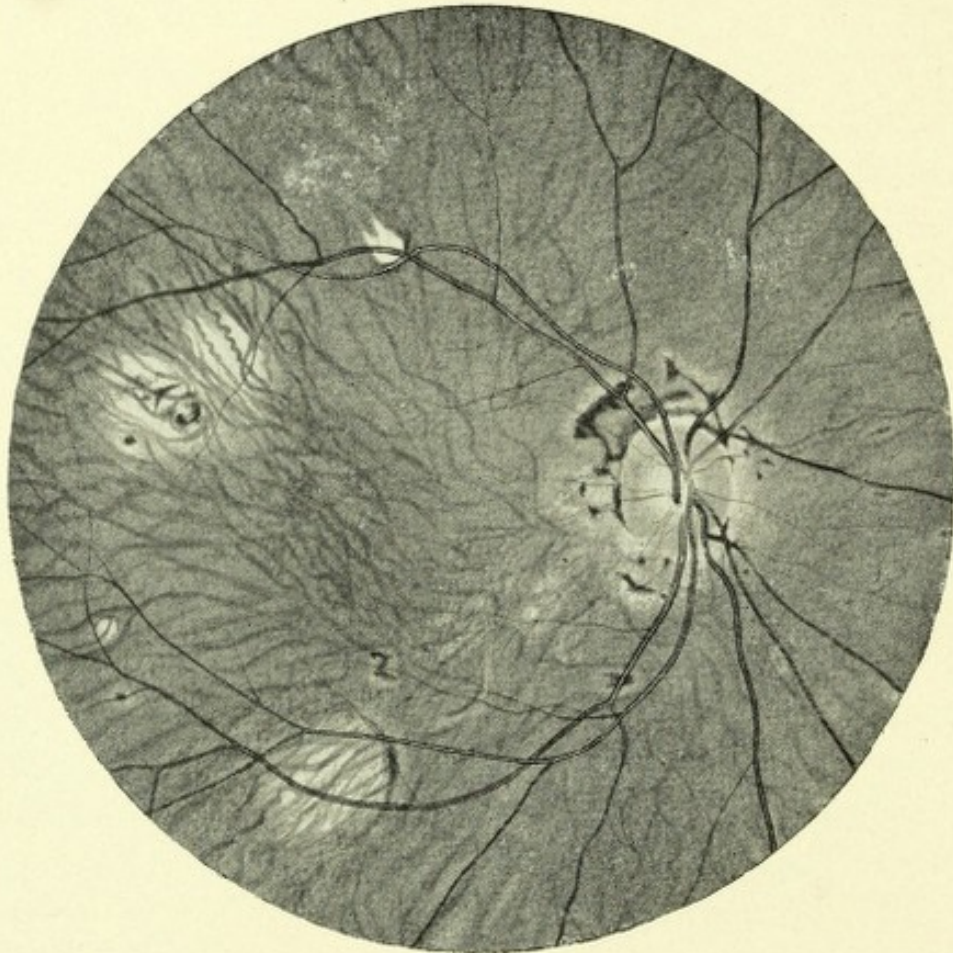


FIG. 72.—Disseminated choroiditis.

not necessarily correspond in size with that of the original button, but may be much smaller. Moreover, it may not be rounded like its predecessor, but occasionally becomes crescentic, triangular, or otherwise distorted, and having an outline that is clean, indented or fringed. When the spot is larger, the atrophy may be complete only in the primitive part of the focus. Here, solely, shows the brilliant white of the sclera. Elsewhere, there has been resorption of the in-



flammatory exudates, while the vessels and the elements of choroidal pigment that have been spared make a figure in red and black on a white ground. Between the spots and about the periphery of the fundus, appears a grillage of larger choroidal vessels, with either brown or white interspaces, owing to whether the stromal pigment is present or not. This would seem to imply that during the evolution of the disease in the focuses, the inflammation in a diffuse form had extended to the chorio-capillaris in these portions, with an intensity sufficient to cause discoloration of the epithelial pigment.

The foregoing descriptions refer to **equatorial** or **simple disseminated choroiditis**, the most frequent of all the varieties. It begins near the equator, and by successive crops of buttons, the disease works its way toward the posterior pole. Usually, however, the macula and peripapillary regions are immune. It is not peculiar to any period of life. It is more frequent among females than among males. Its progress is slow, and by its recurrences its course may be indefinitely prolonged. It has been called the manifestation of chronic choroidal syphilis, in contradistinction to diffuse chorio-retinitis of the acute form. Not that common disseminated choroiditis is always of syphilitic origin, as has been asserted.

**Posterior Polar Disseminated Choroiditis. Areolar of Foerster.**—The button of this form of choroiditis is constructed histologically, after the same type as that of the commoner form first considered, excepting as concerns the alterations of the pigment epithelium. As regards the fitness of the title, according to Venneman, it is only so because of the exceptional appearance in the atrophic plaque, which succeeds the button, of a network of hypertrophic neuroglia, representing the disorganized retina, and the prolongations sent by it into the cicatricial tissue of the choroid to which the retina has become adherent: a form of retinal sclerosis common to retinal atrophies. We have seen that when the localized, or focal, inflammation is violent, the swollen hex-



agonal cells become translucent from loss of pigment. On the contrary, if the inflammatory process is mild, these cells increase their quota of pigment, and the button instead of appearing lighter than the adjacent choroid is darker. This is precisely what distinguishes the choroiditis of Foerster, more than any other symptom, from the ordinary form, for here the button first shows as a coal-black spot. The initial location of the buttons and their march of invasion are also different in the two affections; for they are first seen in the posterior polar region, and the new buttons of successive attacks are situated further and further **outward**—toward the equator. The outlook is more favorable than in that of the equatorial form. The focuses of inflammation are much fewer, and its progress slower than the first form; but its duration is not shorter, nor its recurrences less frequent. While the termination is generally favorable, such is not always the case, for blindness from foveal and papillary complications is quite possible.

There is another peculiarity of the button of Foerster worth mentioning. While it clarifies from the centre toward the periphery, like that of the equatorial button, it is at the same time slowly enlarging by growth laterally, so that when the final stage is completed there is a broad border of black around the white, like that which characterizes the period of evolution of the common choroiditic spot. Moreover, the cicatrice is not cupped as in the other form. It is in the choroiditis of Foerster that are observed those great **perimacular, polycyclic, charcoal** plaques. The cause is most often an infarct of the chorio-capillaris, and is often seen in the young. In the beginning, the spot of Foerster is not so constantly rounded as the common one, being often irregular of contour; but it assumes both form and appearance practically the same as those of the equatorial variety. The choroid outside the spots, and at the periphery, is apparently not so subject to alterations in this form, hence there is not the grillage on the part of the larger choroidal vessels. The affection is doubtless of thrombotic



origin (see Fig. 73). The writer has likened the appearances of the cicatrix here remaining to that of a knot in a freshly planed pine board.

In **anterior disseminated choroiditis (Fuchs)** the focuses of inflammation occupy the retro-ciliary (or pre-equatorial) zone of the choroid. It is probable that, instead of being an essential variety of the disease, it is rather an extension

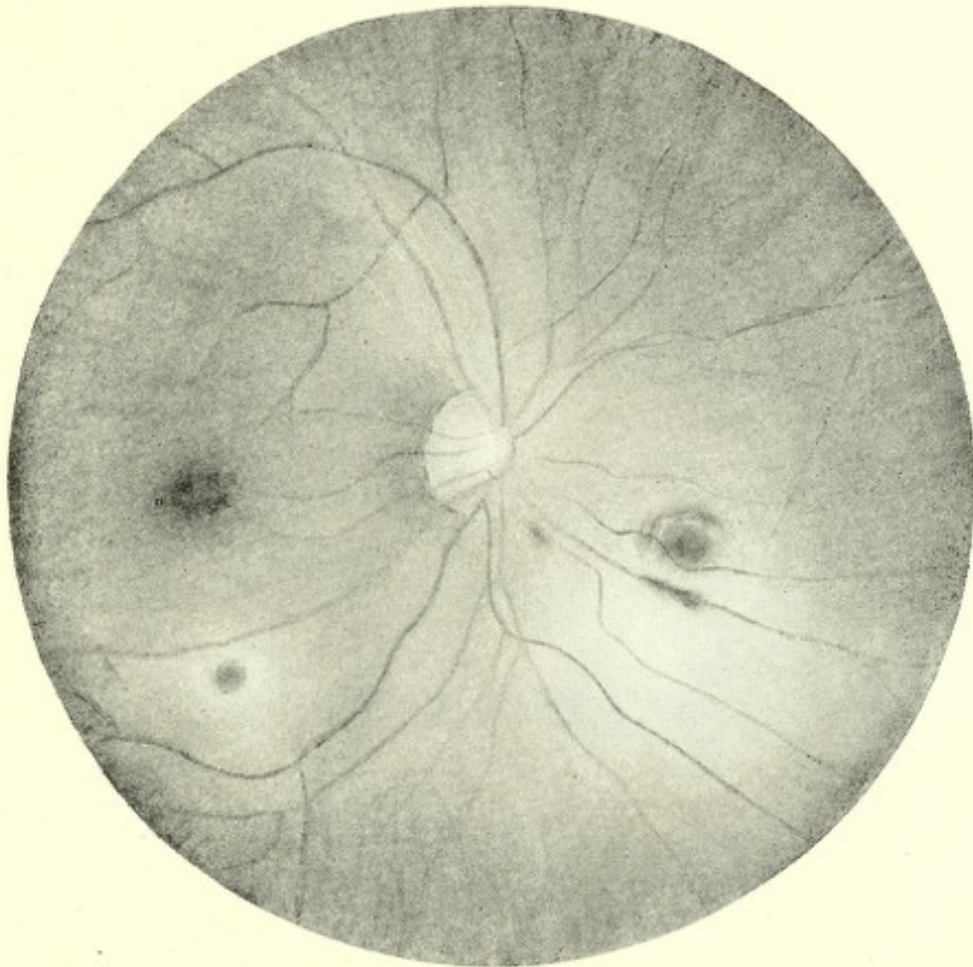


FIG. 73.—Foerster's areolar, disseminated choroiditis, with choroidal thrombus and pine-knot cicatrix, at the right, and a typical areola low down at the left.

of an inflammation of the ciliary zone, with which it is often associated. Besides iridocyclitis, anterior choroiditis is also observed in connection with parenchymatous keratitis and sclero-uveitis. These often hide by their own symptoms those of the accompanying anterior choroiditis; for this reason the ophthalmoscopic evidences of the last named are often discovered only after the concomitant inflammation



has long since passed away. For the same reason the disease has been termed rare—for it has been overlooked, which is not surprising. According to Fuchs, anterior choroiditis is sometimes combined with the peripapillary form.

The ophthalmoscopic lesions of anterior disseminated choroiditis are, in most instances, an indication of hereditary syphilis. They are occasionally visible in an eye of sound cornea, while that of its fellow is opaque from interstitial keratitis. The spot is very black, but smaller than that of the areolar form, and characteristic of the syphilitic choroiditic focus. They are isolated, or joined to others; arranged in line, or grouped *en bloc*, but it is always evident that the different dispositions are made up of the coalescence of a great number of small, distinct spots. The more abundant the pigment, the more certainly the luetic origin. In these syphilitic focuses, evolution seems as a rule to stop with proliferation of the uveal pigment, for the black spot does not change. Aside from this, the spot in question undergoes the changes normal to the focuses of disseminated choroiditis in general. The spots are disposed in a sort of irregular, radiate fashion, that is, in a desultory manner they follow the meridians of the zone they occupy. This is doubtless accounted for by the anatomy of the region—by the long vessel reaches and extensive interspaces, which also parallel the meridians.

**Central Disseminated Choroiditis.**—It is seldom that the typic focus of disseminated choroiditis is observed isolated in the macula. It is more often found in connection with Foerster's areolar form. The button does not differ from that of the equatorial kind. Fig. 74 is from a series of drawings made by the writer to illustrate the changes in the spots as observed from time to time. Here the advent of the spot was announced by a relatively large area of retinal edema over the site of the choroidal focus. By the time this had cleared enough to reveal the focus the latter was in the stage of pigment proliferation. The subject was a girl of 16 years, with normal refraction and otherwise sound and



vigorous save that the choroids were "poor," that is, lacking in pigment and capillary elements. The affection was unilateral. The rule in all varieties of disseminated choroiditis

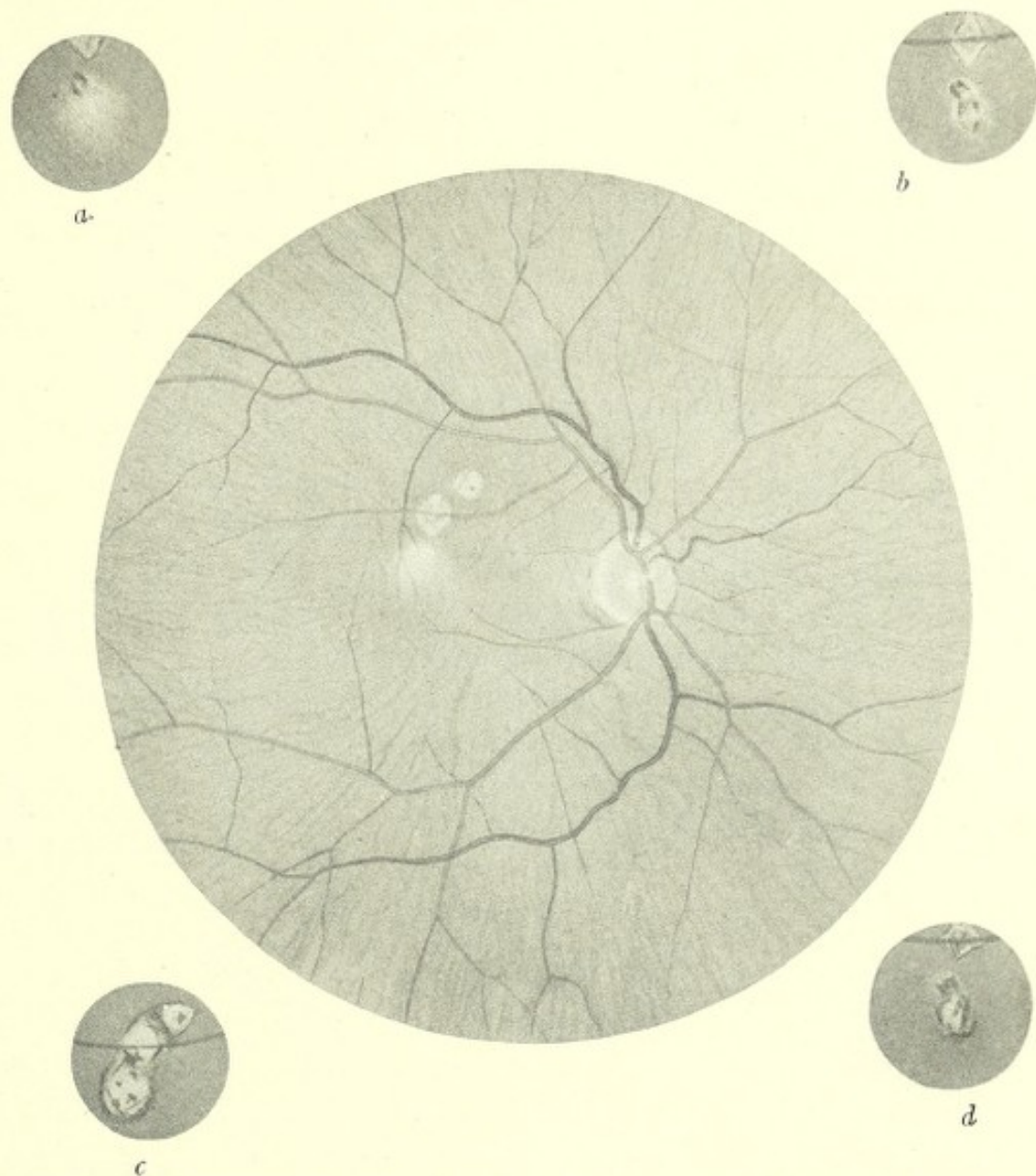


FIG. 74.—Disseminated choroiditis of posterior polar and macular regions. Confined to the right eye of a girl. The large drawing was made April 25 and 26, 1901. It shows a fresh button just above the fovea, and two cicatrices higher up. The new focus appears as a yellowish-white, rather glistening, spot without definite outline. It pushes up the retina, as indicated by the retinal vessels that run over it. *a*, Shows the same spot one week later; *b*, two weeks later; *d*, three months later, and *c*, the three spots on January 12, 1912, after a lapse of more than eleven years. No further trouble has occurred, and the vision of the right eye is 20/20.

is bilaterality. Both macular and peripapillary choroiditis, characterized by discrete focuses, are peculiar to myopia.



**Tuberculous Choroiditis.**—Distinction is made between **tubercular choroiditis** and **tuberculosis of the choroid**. The first is a diffuse inflammation of the choroid, the second more circumscribed. They bear much the same relation to each other that diffuse chorio-retinitis does to disseminated choroiditis. Ultimately, however, the chief objective symptom, that is, the appearance of **miliary** or **conglomerate** tubercles in the choroid, is the same. Tubercular choroiditis presupposes the existence of a tubercular focus somewhere outside of the eye, capable of contaminating the blood with an abundance of toxins or multitudes of bacilli. So long as it is a question of toxins only, the tubercular nodules present in the choroid with the diffuse inflammation, remain microscopic, *i.e.*, invisible with the ophthalmoscope. When the tubercle bacilli also enter the circulation, the nodules begin to develop, and, if death is not too imminent, become the ordinary miliary tubercles, visible with the ophthalmoscope as buttons of disseminated choroiditis. The affection is rare, but in **generalized miliary tuberculosis** it represents the miliary tuberculosis of the eye. It is seen in tubercular meningitis as neuro-retinitis, though here it is *rather toxic than metastatic*. It is never a complication of pulmonary or intestinal, nor any local form, until the tubercular process becomes generalized. Tubercular choroiditis is usually grave, and fatal to the eye. The neuro-retinitis of tubercular meningitis, if not complicated later by disseminated miliary choroiditis is sometimes curable.

**Tuberculosis of the choroid** is much more frequent than the preceding. It constitutes a true parasitic metastasis of an extra-ocular infection, manifested by one or several tubercular focuses, or miliary tubercles, in the choroid. In slow and lingering general tuberculosis these tubercles are nearly always visible before death. Tuberculosis of the choroid is relatively benign, though its gravity increases in accordance with the number and the location of the focuses. Then there is always the danger of a tubercular granuloma; for, on account of the presence of miliary tubercles, there may



develop, in the choroid, a great **conglomerate tubercle**, a veritable tumor—**tuberculoma**. This massive tubercle may extend outward through the sclerotic, or inward, detaching the retina, and partially filling the vitreous chamber, unless its development be stopped by the habitual forms of degeneration. Tubercular choroiditis occurs more often in conjunction with tubercular irido-cyclitis than it does separately; not so often from extension as from simultaneous invasion by the virulent or toxic elements of the blood. Simple isolated tubercular iritis is more frequent than that of the choroid.

Tuberculosis of the choroid is pre-eminently a disease of youth. Before the age of ten years any ocular traumatism, as a contusion, an external or a penetrating wound, or any spontaneous ocular malady, may prepare the way for an endogenous tuberculous infection of the choroid. Primitive tuberculosis of the choroid, not of traumatic origin, is practically and anatomically impossible. Tuberculosis of the choroid is rare after the twentieth year. When it occurs in elderly subjects it is usually in the form of granuloma.

**Ophthalmoscopic Symptoms.**—The miliary tubercle of the choroid first appears as a rounded spot of yellowish-white. At first very small, it extends rapidly, so that at the end of twenty-four to forty-eight hours it has been known to attain dimensions equal to those of the optic disc. This rapid augmentation in the apparent size serves absolutely to distinguish this form of choroidal focus from that of common disseminated choroiditis. The last, if it increases at all under one's observation, does so with extreme slowness; the lesions having attained at the start, so to speak, their definitive dimensions. That it is elevated is shown by the in-curve of the retinal vessels as they pass over it. The altitude is greater and more perceptible in the examination than is that of the ordinary focus, and is, usually, in ratio to the lateral measurements of the spot. When the elevation is marked, and the growth is rapid, the mass is characterized by decided coloring—tints of gray, blue and green. Fig.



#### DESCRIPTION OF FIG. 75.

Tubercle of the Choroid. It appears here as a tumor about the size of the disc, pushing up the retina between the papilla and the foveola. It is much lighter in tint than the eye-ground, its colors consisting of faint tones of blue, green and gray. Its height, measured by the ophthalmoscope, is about  $1\frac{1}{2}$  mm. By changing the direction of the light thrown by the mirror through the dilated pupil one could cause a shadow of the mass to fall in different directions. A retinal vessel is observed to run over the tumor. There is a zone of slight retinal opacity surrounding the tubercle, and the temporal border of the papilla is obliterated.



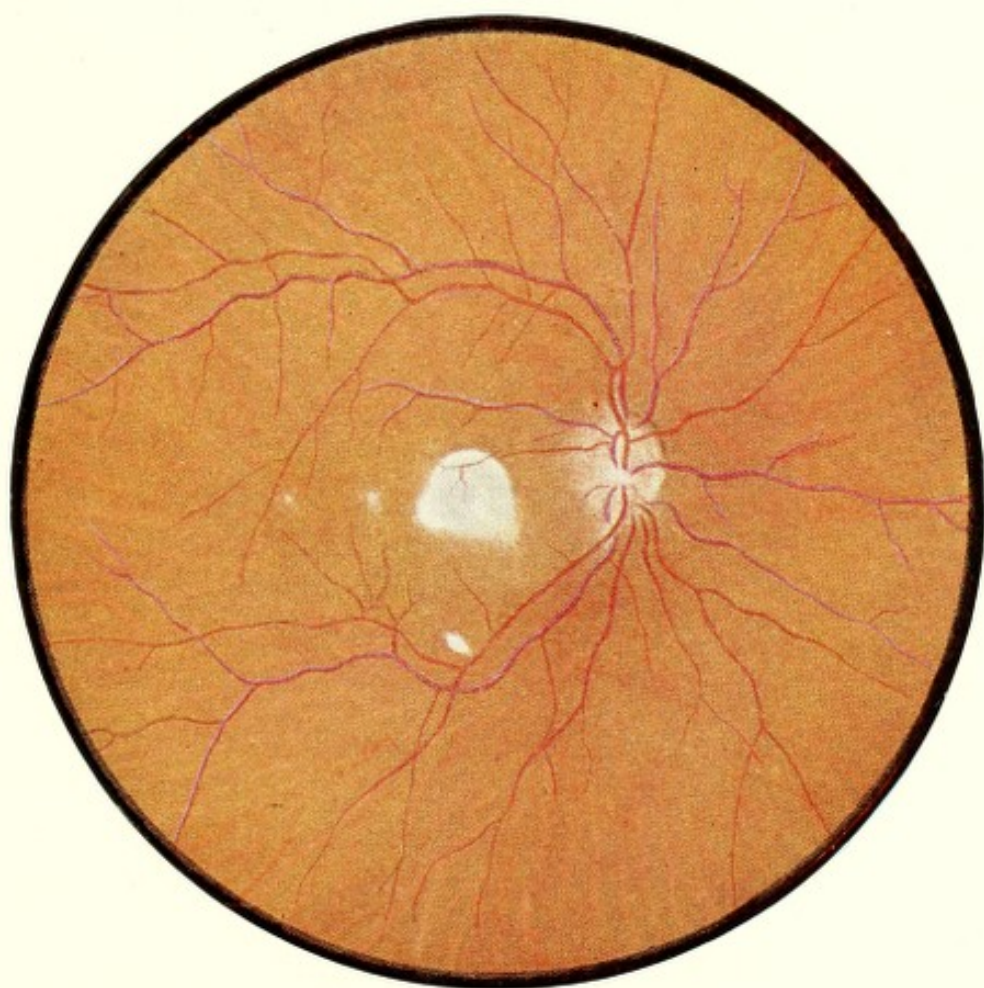


FIG. 75.







75 shows such a spot. Under treatment this tubercle disappeared absolutely.

Unlike that of ordinary disseminated choroiditis, the border of the spot is seldom pigmented—*another important distinction*. It may disappear without leaving a trace, but oftener it increases in size, is joined by neighboring tubercles, and rises higher and higher, yet not, of necessity, resulting in a tuberculoma. It then undergoes all the typic phases of degeneration, viz., necrosis, casefication, cicatricial retraction, and hyalin, calcareous, and even ossific degeneration. When very prominent in the vitreous chamber it may cause the pupil-reflex known as **amaurotic cat's eye**—or a form of **pseudo-glioma**. Opacities of the vitreous, detachment of the retina, and secondary glaucoma are frequent consequences. The hypertension is never of long duration, but is succeeded immediately by immoderate hypotension, denoting detachment or perforation of the sclera. Reduced tension from the start is not characteristic. The favorite situation of the miliary tubercle is the vicinity of the posterior pole. Their number is never as great as is sometimes that of the focuses in equatorial disseminated choroiditis. Usually it is limited to from one to half a dozen. Their average full size is somewhat less than that of the disc.



## CHAPTER XVIII.

### HEMORRHAGES IN THE FUNDUS.

In the classification of **spots** in the fundus we will have come now to the *red* ones. Owing to their semiologic and prognostic importance, these hemorrhages, of all the ophthalmoscopic lesions, have attracted the greatest attention. The only intra-ocular hemorrhages under discussion are those of the retina; choroidal hemorrhages, in so far as they concern the fundus picture, being too infrequent, and of too little significance to mention. A retinal hemorrhage, on the contrary, no matter how small, or otherwise how apparently normal the fundus in which it is found, should never be ignored; it may be the forerunner that announces an impending catastrophe as regards the eye or a more vital organ. It demands a searching investigation, optic and general. The two things to ascertain are, why it is there and what it portends.

The hemorrhages in question are classed as **intra-retinal** and **extra-retinal**. The first are **superficial** or **deep**. The extra-retinal are **pre-retinal** when between retina and vitreous, and **pre-choroidal**, or **retro-retinal** when between retina and choroid. The apparent form of the intra-retinal hemorrhage is determined principally by its seat in the retinal tissue, and its situation in the fundus. Those having their seat in the thicker portions of the nerve-fibre layer, *i.e.*, over or near the border of the papilla, take elongated forms, as **linear**, **spindle**, **lanceolate** and **elliptical**, long axes paralleling the direction of the axis cylinders, in whose interstices they find lodgment (Fig. 76). When in considerable number, as in certain thromboses, they make a girdle of radiation, with the porous opticus as the centre, and their dimensions are small. A linear hemorrhage may be so



drawn out, or two or more may be so joined, end-to-end, as to simulate a small vessel of irregular calibre. In the peripapillary zone, intra-retinal hemorrhages are often of much greater extent, say of an area one-fourth to one-half that of the disc, and their characteristic contour is what is popularly known as **flame-shaped**—or like an old-fashioned gas jet, and they are very frequent in venous thrombosis and albuminuric retinitis (Figs. 33 and 77).

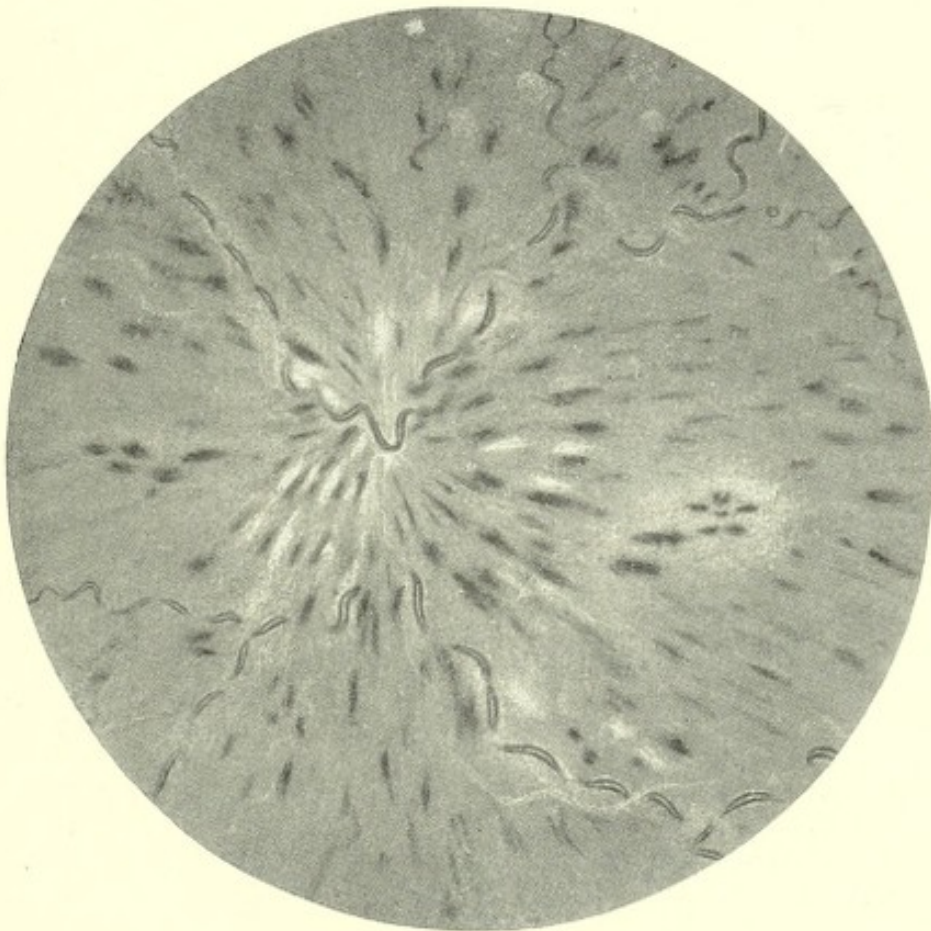


FIG. 76.—Intra-retinal hemorrhages.

The flame-shaped hemorrhages are also radiating, and often hide the trunks of the large vessels. The further from the disc, the less the radiation and *flare*, and the more rounded the hemorrhagic spot. Peculiarities of the **deeper retinal hemorrhages** are their small size—being often mere dots—their ovular form, and their tendency to appear in groups. Their tint is often less bright, due to the slight veiling from depth. They are frequently suggestive of the



petechia in the skin accompanying certain diseases. The greater the swelling of the retina, and the higher the intra ocular tension, the more attenuated, flaring and striated, the hemorrhages. In the more lax conditions the spot of blood is more rounded and thicker. It may then form in veritable pools in the tissues. These are isolated or confluent, and occasionally attain dimensions equal to, or greater than, those of the disc.

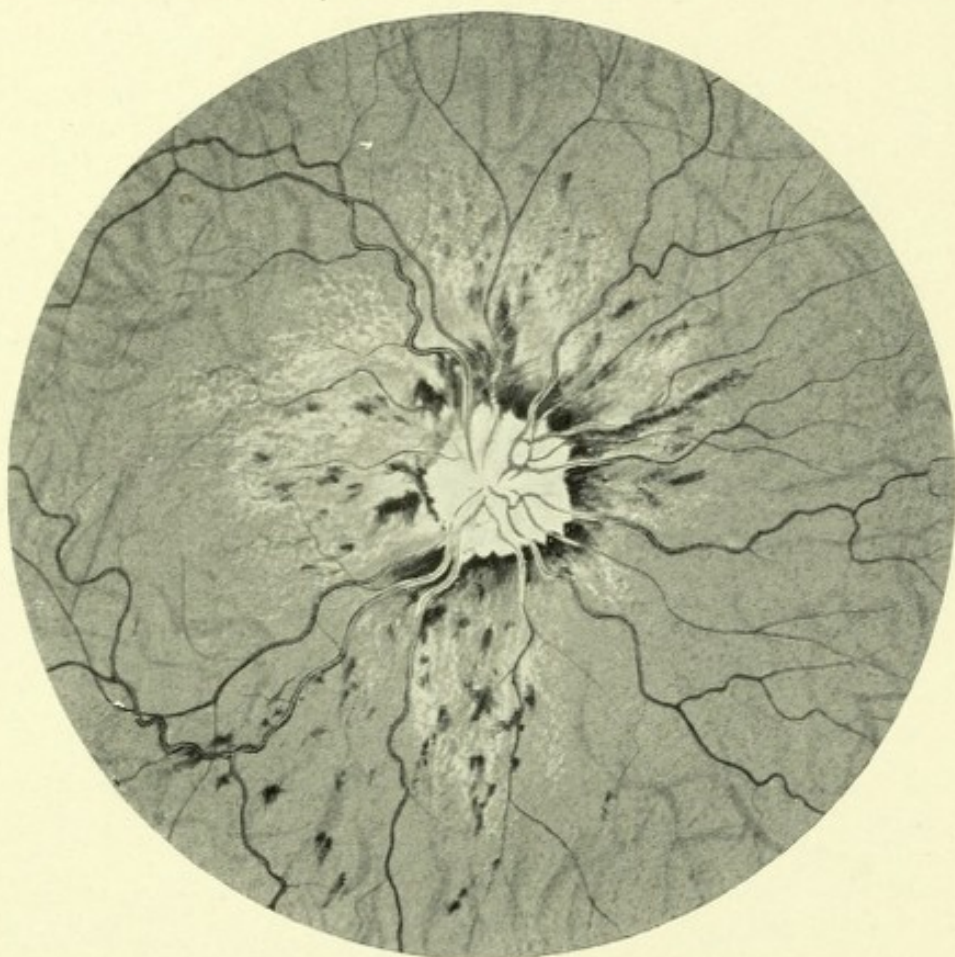


FIG. 77.—Flame-shaped hemorrhages in albuminuric retinitis.

**Pre-retinal Hemorrhages.**—These are extravasations that break through the membrana limitans interna and insert themselves between the retina and the vitreous. They are also called **subhyaloid** hemorrhages. They are easily recognized, and offer interesting peculiarities. They are formed in various regions of the fundus, but they show marked predilection for that of the posterior pole, or where



there is least adhesion between the retina and the vitreous, hence easily separated within certain limits. The hemorrhage, in conforming to these limits, takes a rounded outline, and, if sufficient to fill the space, becomes almost a circle or oval, long axis horizontal, and smaller end toward the papilla.

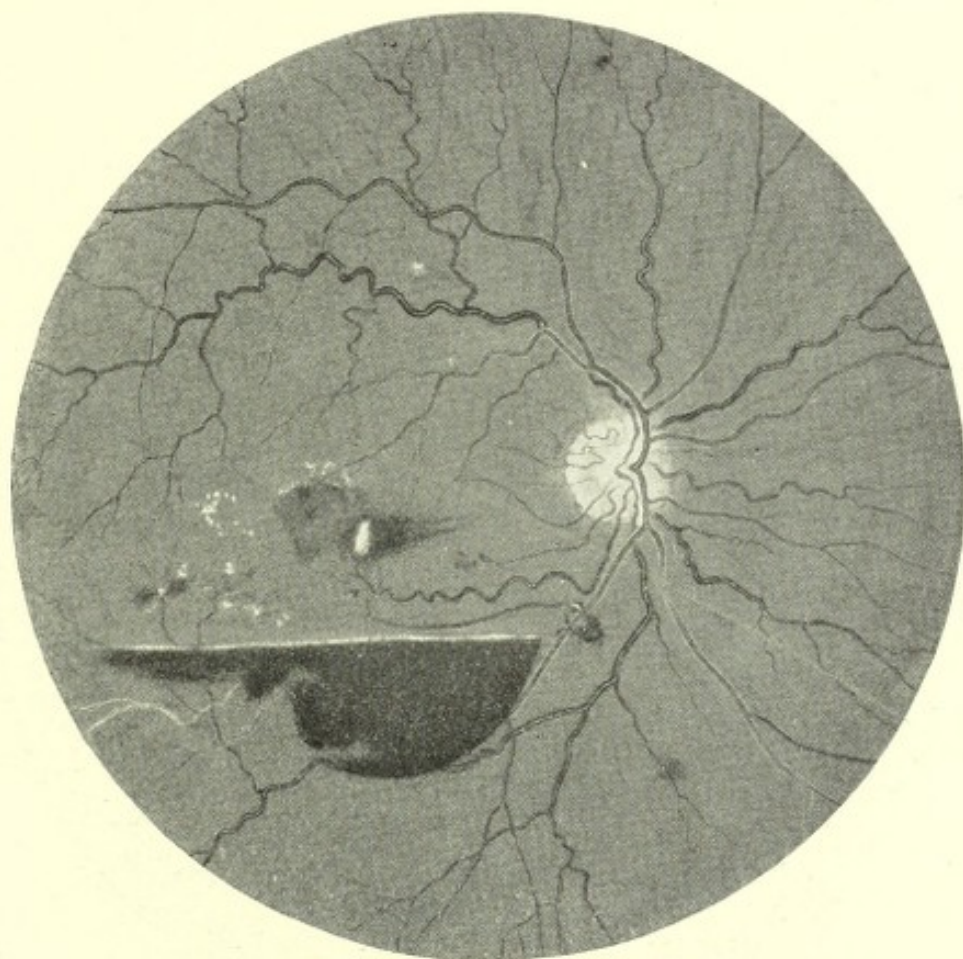


FIG. 78.—Pre-retinal hemorrhage, with arterio-sclerosis of retina. Note the tortuosity of the veins; how a large branch of the supero-temporal artery completely hides the vein, although the *apparent* caliber of the artery is much less than that of the vein. Note the sacculation of the vein just where it passes beneath the artery; the thrombus in the macular branch of the infero-nasal artery, the blood in this artery being of very dark tint; the apparent great reduction of the venous trunks overlying the optic disc; the pressing backward of the veins by the arteries above and below the disc; that the infero-temporal artery, where it emerges from behind the pre-retinal hemorrhage, is a mere white streak; lastly, note the white line at the top of the pre-retinal hemorrhage, and the dark one at the bottom.

If less copious, only partially filling it, the circle or oval is flattened above, or the form is that of a half-moon, convexity downward. The uppermost portion of the outline is a straight horizontal line, or **water-level**. From gravity and



sedimentation, after some days, the lowest portion appears thick and dark, thence the tint gradually lightens toward the top. The straight surface line is maintained with the inclinations of the head, in obedience to the laws of gravity (Figs. 78 and 79). Sometimes the superior edge of the spot is not a straight line, but curved, convexity *upward* when the space is *crowded*—downward when not. In the

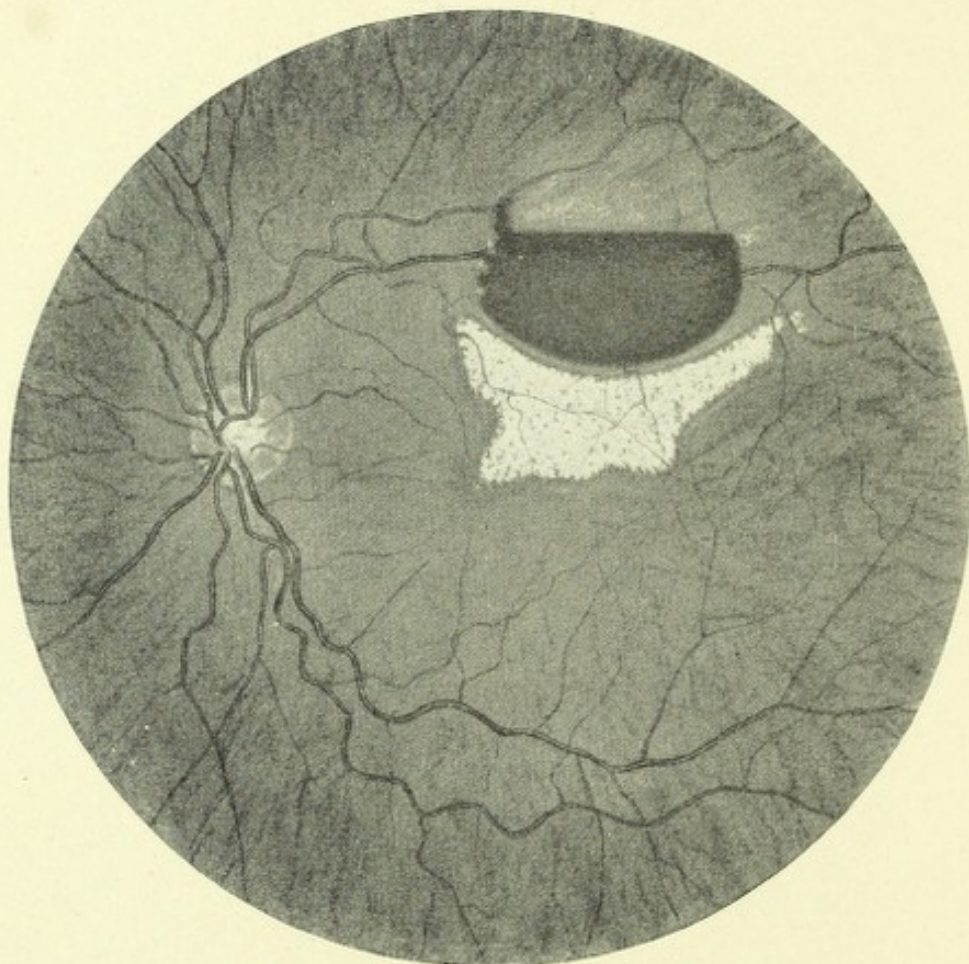


FIG. 79.—Recent pre-retinal hemorrhage, and, below it, an old transformed one.

last instance, capillary attraction draws it upward somewhat at either side, causing the upper outline to become concave. When coagulation takes place, of course, these rules are set at naught. The vessels abruptly disappear behind the spot.

It is not necessary that the breach in the vessel through which the extravasation poured should be in the region occupied by the hemorrhage. The source is often much



higher up, and the blood, trickling downward, finds the watch-crystal-like place in which to accumulate. Pre-retinal hemorrhages occur most frequently in adolescents, particularly in males of recurrent hemorrhagic tendencies, in elderly subjects with degenerative disease of the vessels, and in the menstrual disorders incident to both the puberal and the climacteric periods.

**Effects of Time.**—Recent hemorrhages are of clear outline and bright red or crimson color. As the extravasation is most often from a capillary, whose blood is as dark as that of the veins, it follows that the spot is much more often crimson than red. If small, or of only moderate extent, the hemorrhage disappears in a few days or weeks, leaving no sign. The heavier portions go last, so that, in process of absorption, these only may be visible, giving the appearance of a group of sanguinary splotches. Old hemorrhages turn darker, lose their translucent red or crimson, and look blackish, or liver-colored, or the thick places look black and the thin ones carmine. Their outlines become vague or very irregular, and, in places, taints of yellow and brown are observed. The clot is then slowly and entirely removed, or else it is transformed into a more or less white (yellowish or grayish) mass of fibrous material, or of granulo-fatty cells, with probably admixtures of very dark or black hematogenous pigment (Fig. 79). It is not always possible to distinguish these focuses of secondary degeneration in hemorrhagic spots from those that are of independent origin, as, for example, the white spots seen in the course of albuminuric or diabetic retinitis. Through degeneration of the clot a white spot may be substituted for the red in a very short time—as brief as one week; ordinarily, however, the process requires several months for its completion. In their turn, the resulting white (or light) spots may disappear in a few weeks or months or they may persist for an indefinite period.

When a hemorrhagic spot represents a rather thick extravasation lying so superficially in the retina as to arch the



internal limiting membrane, there appears at the point most elevated a bright reflex—the reflex of convexity. This, from its shape and whiteness, may easily be mistaken for a focus of beginning degeneration. A pre-retinal macular hemorrhage, when extensive, nearly always bears the reflex. If the blood-spot were symmetrically round, and centrally located, the reflex and blood might closely resemble the picture of the macula produced by looking at the sun (see page 313). If the surface of the hemorrhagic spot is uneven there will be a separate reflex for each of the high places. The shape of the reflex corresponds to that of the hemorrhage.

Large pre-macular hemorrhages, on account of their insulation from the retinal circulation by the limiting membrane, provoke little reaction in their vicinage, and are slow to show signs of alteration. Color and fluidity are preserved for an unusual time, and in being absorbed the hemorrhage may retain its straight upper line throughout, the blood disappearing just as if drained out from below. It rarely shrinks concentrically. Fig. 79, however, shows the old, white and mottled remains of a hemorrhage of this description. Pre-retinal hemorrhages that invade the vitreous occur most often in the peripheral zones of the fundus, or where the union between vitreous and retina is closest. Blood in the vitreous is chiefly an accompaniment of traumas or of recurrent hemorrhages, when the vitreous is disorganized. It is either absorbed or transformed into pseudo-membranes, or furnishes the soil for the growth of new vessels and connective-tissue formations, as in retinitis proliferans.

As before intimated, a bright red color in a fresh hemorrhage denotes that it was from an artery; a crimson color, that it was of venous or capillary origin. The particular vessel responsible for the extravasation is often indicated by the contiguity of the spot; but this indication is made much more pointed when a series of blood-spots follow the traject of a given vessel for a space. Alterations in the walls of



the vessels, on one hand, and in the constituent elements of the blood on the other, are, separately or jointly, accountable for the escape of the extravasation. Certain it is that the actual break is never visible save with the microscope. Many have thought that these hemorrhages occur principally by diapedesis, the star-shaped fissures in the walls of the capillaries being quite ample, when relaxed, or distended, to admit of the passage of the sanguinary corpuscles. Indeed, it is not necessary, in many instances, to pre-suppose changes either in the vessel walls or in the composition of the blood in seeking the cause of retinal hemorrhages. Extravasations, are doubtless possible from violent congestion of the retinal capillaries, especially if there is exaggerated cardiac action, though they are probably very rare in the presence of a sound retinal vascular system. A number of cases of spontaneous hemorrhage of the retina have been reported as happening in connection with suppression of the menses, especially when through the influence of cold or emotion. It is well known that the blood may be ejected from the normal retinal vessels by inordinate external pressure brought to bear either upon the entire circulatory apparatus, as in crushing injuries of the chest, or upon that of the head, as in strangulation from constriction of the neck, and in compressive traumatism of the head. Struggles for life, and extreme efforts, like those made in severe paroxysms of whooping-cough, and in straining at stool, have frequently produced such hemorrhages.

**Natal Hemorrhages.**—The fact has been established that a large percentage of the newly-born are subjects of retinal hemorrhages. They are due either to the enormous congestion sometimes occasioned by constriction of the neck, or to compression of the yielding skull. The percentage is obviously higher when the labor is prolonged, as when the straits are narrow and the head is large; and is raised still higher by forceps-deliveries. These natal hemorrhages are usually bilateral, and situated in the posterior polar regions. When they are small, and well within the substance of the



retina, they are soon absorbed without leaving a mark. When they occupy the fovea they may be the cause of grave retinal lesions. It is more than likely that the lack of vision in many of the cases diagnosed as "congenital amblyopia" and "**amblyopia ex anopsia**," especially when there is central scotoma, is the result of such hemorrhages. A macular hemorrhage could remain just long enough to permanently destroy the visual elements, then be absorbed and leave the region apparently normal. It has been demonstrated that the extravasation is sometimes retro-retinal, in which event nothing more likely than that detachment of the retina with lesion of the pigment-epithelium might ensue. Thus is afforded a possible explanation of the existence of many of those large pigmented plaques of central choroidal atrophy, known as "**pseudo-coloboma** of the macula" (see "Coloboma of the Macula.") The most damaging of the central hemorrhages seem to be the intra-retinal and the retro-retinal. The pre-retinal are relatively harmless. In his "Ophthalmoscopy," 1910, Haab shows a drawing of a fundus wherein a large pre-retinal, natal hemorrhage covered the macular region. Four and a half years after the drawing was made "the eye was examined, the eye-ground was found normal, and the vision was good."

**Retinal Hemorrhages of Adolescents.**—Under this head are ranged the great number of profuse, recurrent, intra-ocular hemorrhages, occurring in young subjects, mostly males, who for lack of any evidence to the contrary, are pronounced healthy. By the fundus appearances, many of the hemorrhages may be identified as of the retina; though it is probable that some of them have their source in that part of the uveal circulatory system corresponding to the *pars ciliaris retinae*. In fact, in the majority of cases, the vitreous is so filled with the blood, that it is impossible even to guess whence it came. Without any warning the vision rapidly declines, with, perhaps, photopsia, and a feeling as if the eye had suddenly grown very large. Judged by their ophthalmoscopic aspect, these hemorrhages present nothing



specially characteristic, though they have certain features in common. For example, they are nearly always venous; they are copious; and they are frequently of the pre-retinal type. After a few weeks, or months, the blood may disappear, but recidivations are the rule. These are sometimes numerous and at short intervals, as many as 6 having been observed in one year; again, *vice versa*. Generally, after two or three years, the recurrences cease. Both eyes are occasionally involved.

These hemorrhages constitute the beginning of most of the cases of retinitis proliferans. Detachment of the retina is possible at an early period, and is almost certain with proliferating retinitis. According to Abadie, when the vitreous is all obscured by the blood there is no detachment so long as there is a diffuse dull red reflex *from every part of the fundus*, "like the glow of a distant conflagration on a dark night." This is verified if pupillary reactions and light-projection are preserved, whereas fixed pupil and faulty projection would favor detachment. Various causes have been assigned, but many have agreed upon hypertrophy of the heart and upon endophlebitis. The few cases seen by the writer wherein ophthalmoscopic examination was possible, gave indications of venous thrombosis. As to frequency, statistics seem to indicate that one case is seen in about three or four thousand consultants.



## CHAPTER XIX.

### DETACHMENT OF THE RETINA.

In treating semiologically of retinal detachment it is specially incumbent that we should regard it in its true light—that of *an anatomic incident*; or, keep constantly in mind that “there is no one affection bearing the name of detachment of the retina, but that there are a great number of very diverse pathologic conditions having nothing in common among them but retinal detachment” (Dufour). In any case it is but the separation of the retina from the choroid by a purely mechanic force that either pushes or pulls, the intervening space being filled at the expense of the contents of the original vitreous chamber. Varieties of detachment are classified according to their primary causes, and the particular mechanical force that accomplishes the separation.

Of all the forms of detachment, the most important, from both the clinical and the pathognostic view-points, is that which is generally named **spontaneous** or **idiopathic**. It is also the one that is understood in a generic sense by the term “detachment of the retina.” The specific conditions under which it is produced, its initial symptoms, its ophthalmoscopic aspect, its accustomed tendency to progress, its most frequent complications, even the manner in which, exceptionally, it eventuates in recovery—all conspire to make of this form a characteristic affection. “They lend it the qualities of a morbid entity—qualities it does not possess in the anatomic meaning of the term” (Dufour).

Following the classification of this author, the two great divisions of detachment are into **traumatic** and **non-traumatic**.

Among the commoner traumatisms are:



*First*, concussions and contusions of the globe, with or without rupture of the sclera.

*Second*, perforating and incised wounds with or without consecutive infection; especially when attended by loss of vitreous.

*Third*, the presence of a foreign body in the eye.

Among the principal **non-traumatic causes** are :

- a. Focuses of disseminated chorio-retinitis in the anterior regions of the fundus, with abnormal adhesions between vitreous and retina, and disturbed nutrition of the vitreous. According to recent researches these are the conditions usually responsible for **spontaneous** detachment.
- b. Hemorrhages into the vitreous, or over the surface of the retina, leading to the formation of connective-tissue bands between retina and vitreous, and shrinkage of the vitreous, as in the recurrent hemorrhage of adolescents, with its frequent sequel, retinitis proliferans.
- c. The development of an intra-ocular tumor, such as glioma, sarcoma, or of a cysticercus (the last being extremely rare in this country).
- d. Circulatory diseases—albuminuric retinitis, choked disc, tumors of the orbit, etc.
- e. Consecutive inflammatory processes of the ocular tunics, or adjacent tissues, like metastatic choroiditis and retinitis, strias and purulent choroiditis, scleritis, retro-bulbar abscess and sinusitis.

Contrary to a widespread opinion, myopia cannot be considered as a veritable cause of detachment, but only as a condition that predisposes to it. There is no special form of detachment peculiar to myopia. Most high myopes escape detachment, while many with low degrees, or none at all, do not. Myopia encourages traumatic detachment as much as it does idiopathic. Shocks, contusions, and operative interventions that would leave a normal eye unharmed, may result in detachment when in relation to a



myopic one. Nevertheless, the fact remains that considerably more than half the spontaneous detachments are in myopic eyes; 11 per cent. of these are bilateral. It is more frequent in males than in females; occurs most often between the ages of twenty and thirty, and next often between those of fifty and sixty.

Concerning the **anatomic causes**, there have been many theories, and, while at present the opinions of ophthalmologists are more nearly unanimous than heretofore, the questions are not definitely settled. The leading theories will be mentioned in the order of their conception or promulgation. About the close of the first half of the nineteenth century, pathologists considered detachment simply as an effusion beneath the retina. In 1853, in trying to account for sudden extensive detachment, without corresponding hypertension of the globe, von Arlt was the first to conceive the rôle that might be played in detachment by a rent in the retina produced in the course of a chorio-retinitis. Von Graefe, in 1854, believed hemorrhages and serous effusions outside the retina to be the most probable cause. To explain the detachment so frequently associated with progressive myopia, in 1857 he proposed his **distention theory**. This was to the effect that the retina lacked the extensibility of the outer tunics, and was prone to tears or ruptures in the stretching backward of the globe.

In 1858, H. Müller and von Graefe, having observed in a number of enucleated eyes, with detachment, how the vitreous was fibrinous, cicatricially shrunken, and strongly adherent to the detached retina, concluded that this state of the vitreous was an important *factor*, and not a *result* of the separation, particularly after traumatisms and operations. In 1869, Iwanof announced his conviction that the primary cause of most all forms of detachment, including those associated with myopia, wounds and tumors, was **detachment of the vitreous**, and an accumulation of fluid between this body and the retina. In 1875, Raehlmann



submitted his **diffusion theory**, which assumed a primordial alteration in the constitution of the vitreous, whereby, through the influence of osmosis, its watery constituents passed through the retina, seeking to equalize with the juices of the choroid, rich in albumin. In 1889, Leber advanced a theory embodying the fibrinous degeneration of the vitreous of Müller, the detachment of the vitreous and shrinking, with accumulation of pre-retinal fluid of Iwanof, and the rupture, or tear, of von Arlt. He argued, given the shrunken fibrillary vitreous attached more extensively and firmly than normal to the retina, and with its pool of fluid representing the amount of shrinking, and the train is laid for an acute "spontaneous" detachment, precisely the kind to which Leber applied his theory. A sudden rent furnished the match; through this the pre-retinal fluid rapidly becomes retro-retinal, and the detachment is accomplished. In 1887, Nordenson elaborated this theory and extended its application to include detachments from albuminuric retinitis and choroidal tumors. Leber's theory affords an explanation of the sudden extended separation without a rise in the intra-ocular tension, but it excludes the possibility of a spontaneous cure, an event that has frequently happened. Those who do not accede to this theory have their choice of the two others—Raehlmann's diffusion theory, and the primary retro-retinal exudation with compensatory diminution of volume in the vitreous, coming down from Arlt and Graefe.

The foregoing was deemed contributory to a better grasp of what is to follow.

Detachment of the retina may be partial or total, meaning that it is still applied to the layer of hexagonal cells throughout a certain area, or certain areas, or hangs free in the vitreous chamber save for its cord-like union with the optic nerve and its circular line of attachment to the ora serrata. This last condition is commonly referred to as "**funnel-form detachment.**" It is also, and more appropriately, likened to the wilted corolla of a convolvulus—the



French say, "corolle de liseron"; we would say "withered morning glory." The space within the flower is almost *nil* in comparison with that around it, hence, in total detachment the conditions are not favorable for ophthalmoscopic examination, though the more anterior folds of the loosened retina may be seen by focal illumination.

**Ophthalmoscopic Appearances.**—The detachment in question is more or less incomplete. In most cases the aspect of the pupil-reflex is sufficient to reveal the presence of a detachment. The normal scarlet glow of the pupillary area is shared or supplanted by a very light grayish, or greenish-gray reflex. If the reflex is normal with the eye looking ahead, and the grayish reflex appears only when the eye is turned from the direct line of regard, the approximate extent of the detachment can be estimated by noting how great a deviation of the globe is required to cause the gray to enter the pupil, and how great the excursions throughout which it remains visible; and its site in the fundus corresponds to the direction in which the globe must turn to bring the gray into view. In the early days of the ophthalmoscope it so happened that in most all the cases of spontaneous detachment that were reported the seat of the separation was in the lower part of the vitreous chamber. Hence, this came to be regarded as the chosen region. Graefe upset this belief by declaring that the seat was primarily upward, and that, unless the detachment became total, the upward location was exchanged for the downward; reapplication taking place above, *i.e.*, that displacement from the vault of the fundus to the floor is the rule, a fact that has been abundantly verified. In the interval of making the change the macula is usually somewhat involved and the vision affected. This is a critical time, for the separation then threatens to become total. Having effected the removal, and become settled in its new location, there is a much better chance of the detachment remaining stationary.

The grayish mass has a movement of its own in addition to that imparted by the excursions of the globe. At the



beginning of the ocular movement in a given direction that of the mass lags; at its completion, the mass oscillates for an instant in that direction. It is usually evident that the gray is not a uniform surface, but that it is creased, or corrugated, and broken by irregular crimson lines—the folds and blood-vessels of the detachment. Having obtained these general impressions, the biconvex lens is interposed, and the ophthalmoscopic lens set for a clearer and more comprehensive inspection—one that is freer from the disturbing results of violent perspective (Fig. 80). The details are now sharply defined; if the detachment is fairly recent the white and the gray of the mound of retina is interspersed with glowing orange-scarlet patches, the reflex of the choroid beneath. This is more or less modified by the tint of the retro-retinal fluid, and as the detachment ages, most of its delicate coloring is lost from thickening and opacity.

Over the whole undulating mass there is a play of iridescence. This, too, may be affected by the tint of the afore-said fluid, just as it is affected in the opal by the body-tint of the stone; *i.e.*, it inclines to green, or red, or yellow, as the case may be. For it must be remembered that although the two fluids—the one within, the other without—are of a common density, they are rarely of the same color. The contour of the detachment stands boldly out from the red of the fundus. The usual form of the detachment, as it rises from the periphery, is that of a rounded mountain, or cumulous cloud, that is partly hidden below the horizon—single or multiple. Curving over the bulky sides, or over the summit, are the darkened vessels of the separated retina, sending their terminal branches toward the centre, or toward the base, of the mountain. The aspect of the vessel stems and branches bears the same relations to normal vessels that fresh, crisp succulent sea-weed does to that which has lain in the sun for a day. The branchings are no longer the angles of health, they are but limp curves. Their translucent color has turned dark and lifeless.







*Mr W. W. Brenchley.*

*Crofton,  
Kingsmead Avenue,  
Worcester Park.*



DESCRIPTION OF FIG. 80.

Detachment of the retina.





FIG. 80.







This wilted sea-weed disposition of the branches has often been referred to as *tortuosity*, a term that is somewhat misleading in this connection. True, the vessels are more sinuous in the detached portion than in that which is still smoothly applied to the choroid, and this is one of the great diagnostic signs in a low detachment; but it is a peculiar sinuosity; it is not crisp like that seen in retinitis, but lax,



FIG. 81.—Low detachment showing vessels curving in unison.

as if the vessel had been overstretched and then allowed to recoil. The vessels perhaps look smaller and less plump than normal. A most distinct sign of detachment, as shown by the included vessels, is **curving in unison**. Two or more vessels running parallel, or in the same general direction, exhibit a coincidence in their curves that could not be attributed to mere chance (Fig. 81). Arteries and veins having traversed the adherent retina, straight and true,



upon reaching a certain line are all observed thereafter to be sinuous. A little careful study will often make it plain that the convexities of the curves of the several vessels correspond, as do also their concavities—they *rise* and *dip* together. By noting this feature the limits of a very *low* detachment, and the number and dimensions of its folds, can be roughly deciphered. The soft retina is lifted or pulled upward by the harsher vessels into tiny sharp ridges whose sides cause a play of white reflexes along their courses.

If the brink of the detachment overhangs the fundus, the vessels disappear in rounding it. When the mountain is high their continuations cannot be traced in the adjacent fundus. If low or of moderate height, they can sometimes be traced, but there is a decided break in their continuity. If there is no overhang their lines are foreshortened, but not broken. The same phenomena are true of vessels passing from one fold to another, as of those passing from fundus to detachment. The more opaque the affected retina, the darker the vessels, and the more devoid they are of any signs of color in their light-streak, and the less pronounced the difference in the shades of veins and arteries. The degree of relief of the folds, as regards the fundus, or as regards each other, is indicated by the rapidity of the parallax displacement caused by slight movements of the observer's head; the speed being in direct ratio to the height of the fold, or at least, to its proximity to the eye of the observer.

Another important indication of a low-lying detachment is **effacement of choroidal detail**. No matter how transparent the retina, nor how thin the layer of fluid that separates it from the choroid, the choroidal vessels throughout the area are lost to view. The *color* of the choroid may be, and often is, as vivid beneath the detached portion as elsewhere, but its *design* is lacking. The retinal vessels, besides curving in unison, are darker than normal, even in the lowest and most diaphenous detachments.

**Rents and Perforations.**—The presence of small openings



in the detached retina has frequently been noted, ever since the invention of the ophthalmoscope, but until a comparatively recent period they were not associated with the causation. The tearing of the membrane referred to as figuring in the theory of von Arlt was of a more extensive kind. It is now pretty generally conceded that a small rent is the determining cause of so-called spontaneous de-

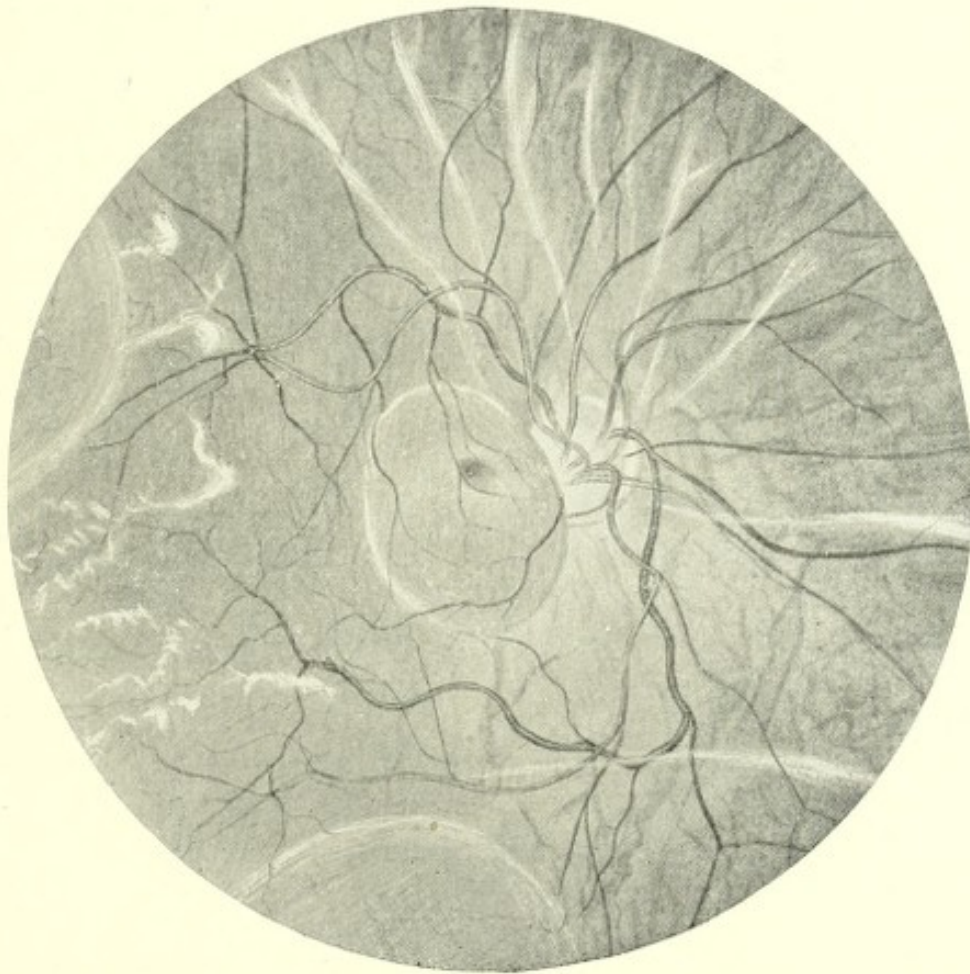


FIG. 82.—Oval detachment, overlapping optic disc, with clean-cut perforation.

tachment. Actuated by the sound logic of Leber's theory, Dufour made zealous anatomic investigation as to their rôle in this matter. He verified all that Leber had declared concerning them.

There are two classes of rents, viz.: the valve-like and the clean-cut perforation (**Perforation à l'emporte pièce**) as if made with a punch (Fig. 82). By diligent search they are to be found ophthalmoscopically in most cases of de-



tachment, recent or old. The round or ellipsoid openings are most often situated in the anterior portions of the retina, just where most detachments begin. Their location corresponds to the spots or focuses of anterior disseminated choroiditis. They are supposed to be the breaking away of the retina from its adhesion to one of these spots, from shrinking of the vitreous; thus the liquidized portion

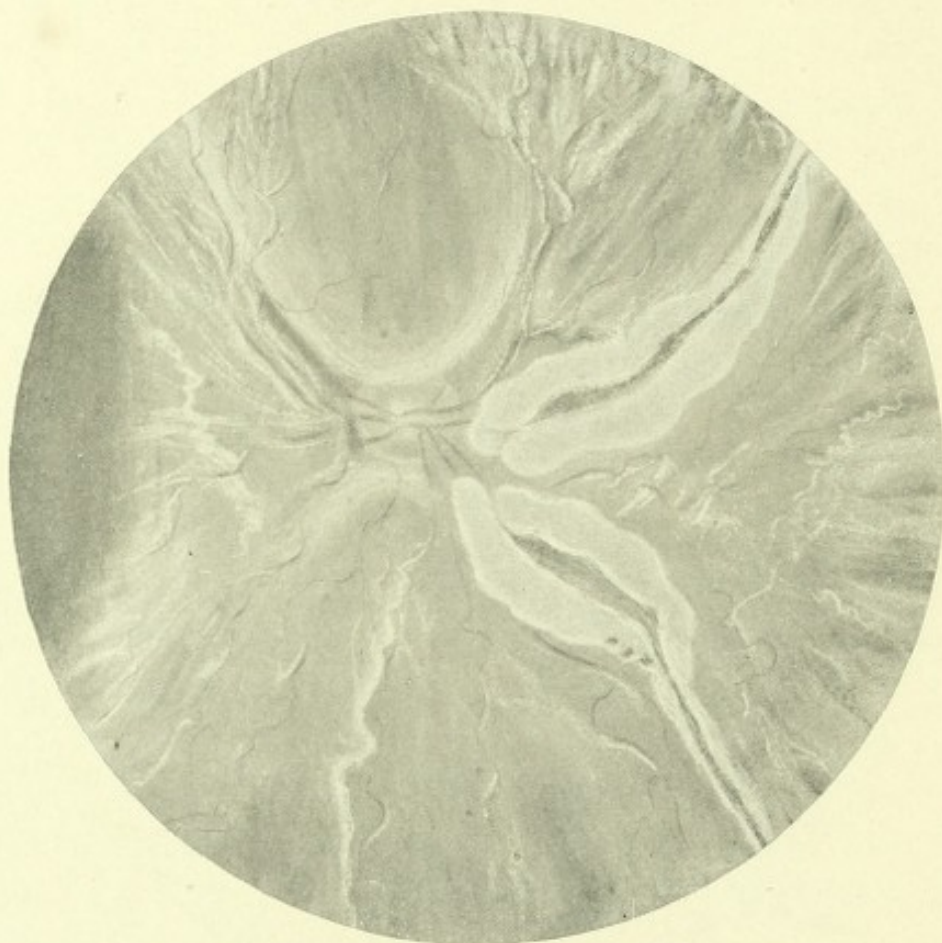


FIG. 83.—Traumatic detachment with large ruptures.

of the vitreous is permitted to flow beneath the simply *applied* retina. The valve-like rents are usually situated further back. They are, for the most part, shaped like the letter U or the letter V, with the free end, or apex of the flap, directed toward the optic-nerve entrance. This last is due to the fact that the strain brought to bear by the contracting vitreous upon the tiny point of abnormal adhesion between it and the retina is toward the ora serrata. These



openings are easily recognized by their fiery red glow as reflected from the exposed choroid. Sometimes they are so extensive that the vessels of the tunic are visible through them. Fig. 83 shows large ruptures in a recent traumatic detachment.

**Deposits of pigment** are often to be seen in the detached retina, either on the back or on the front, or both. Those



FIG. 84.—Pigment spots over area of former detachment.

on the front are less frequent, and usually bespeak the nearness of a perforation, through which they have found their way. They are all inclined to circularity of outline; those in front being intensely black, those behind less so. The retro-retinal spots evidently were integral parts of the focuses of chorio-retinitis before the detachment occurred.

The detachment is frequently traversed in places by white stripes, or striæ. They bear a sort of parallelism to



the advancing borders of the detachment, and are supposed to be derived from linear accumulations of fibrin beneath the retina that marked former limits of the separation (Fig. 81). Similar lines in the applied retina are of the same origin, but the retina in which they are situated has become reappplied. The striæ are always behind the retinal vessels, and are sometimes pigmented (see "Striated Retinitis").

In addition to the striæ there are other tokens that mark the areas of former detachments, or where the retina has managed to stick itself to the choroid again. Traces are left behind, even when the separation was of very short duration. The part that was involved is distinguished from the normally applied retina by the paler tint, especially as it approaches the line of demarcation. Instead of the white lines there are sometimes series of pigment spots (Fig. 84). In most instances there are numerous pigment mottlings over the entire territory of the old separation, imparting a marbleized appearance. The grillage of the choroidal vessels is conspicuous in places. The aspect of the "*restuck*" retina may resemble so closely the final stages of an old chorio-retinitis that it is only its strict limitation to a certain sector of the fundus, at least, in the absence of an appropriate history, that assures the differentiation.

**Spontaneous Detachment and that Secondary to a Tumor.**—Examinations of enucleated eyes have often demonstrated that total detachment has been produced by a very small tumor; and, on the other hand, limited or partial detachments have been found associated with choroidal tumors of considerable volume. In most cases the separation is due less to the neoplasm than to an exudate, probably choroidal. The maximum of massing of the exudate is not necessarily at the site of the tumor, probably because the retina is often adherent to the growth. It is not infrequently diametrically opposite to the latter. Nor has it any special tendency to accumulate at the lowest part of the fundus, even when



its density is greater than that of the vitreous. In total detachment, due to a tumor, one is occasionally afforded an opportunity of observing a characteristic phenomenon. By focal illumination, or the use of the ophthalmoscopic mirror, and of a widely dilated pupil, the retina is seen applied closely to the ciliary processes and to the posterior surface of the crystalline. This is quite different from looking into the cone of waving folds peculiar to total spontaneous detachment—it is the difference between detachment by *vis à tergo* and that by *attraction*.

It is often very difficult, if not impossible, to decide whether the upheaval of the retina is from the pressure of a tumor or that of an exudate; yet opportunities are not wanting of making the distinction and of watching the corresponding development of tumor and detachment. These are most likely to relate to tumors about the posterior pole, for the early disturbance of vision they incite leads to promptness in consulting an oculist. Indeed, the very location of a detachment in this vicinity is suggestive of a tumor. The same is true of a circumscribed detachment at the vault of the vitreous chamber. Both the limits of the detachment, and the scotoma it gives rise to, are more sharply defined in the detachment of a neoplasm. The secondary detachment is smoother, or, if thrown into folds, these are smaller and less numerous. It is more rigid—there is not the characteristic undulation. It is more opaque, and more devoid of color. There is not that “undulating, iridescent veil,” as has been said of the idiopathic form; the aspect is more that of a fixed dome. The retinal vessels running over the detachment do not branch so like wilted sea-weed nor curve so in unison, and they are less sinuous.

The surface of the detachment must be studied minutely by both the direct and the indirect methods. With the latter, and proper illumination, and with the aid of a large pupil, and the turning on of varying convex lenses, one may succeed in piercing with the eye a certain depth into the mass. In this manner there may be brought to view



the presence of a second set of vessels—*i.e.*, a set beneath those of the retina—pathognomonic of a neoplasm; or one may detect the dark brownish glint of a melanotic sarcoma. Even should this brown, or reddish-brown, be produced by blood, it is no less significant of a tumor; so also is a bloody infiltration of the detached retina. The “pushed up” retina is devoid of the rents and perforations, the pigment and the white stripes, so often found in the idiopathic form. In spontaneous detachment hypotension is the rule; from the first, if not normal, the tension is reduced. Hypertension, then, be it ever so slight, associated with detachment, is indicative of a tumor or a retro-retinal effusion. Unless the detachment is situated too far back, transillumination of the globe may be of great service in making the differentiation.

**Detachment of the Choroid.**—In 1868, Knapp described this affection as an accident consecutive to extraction. From that time until 1900, only ten cases were reported. Then Fuchs observed six cases in six months, all following extraction with iridectomy. At the same time Fuchs reported three other cases following simple iridectomy. In 1902, the same surgeon reported 37 new cases—all following extractions or iridectomies; the ratio of detachments to the whole number of cases examined being about 5 per cent. It appeared in from a few days to a few weeks after the operation. In the great majority of cases the separation of the choroid from the sclera is the result of a rent in the insertion of the ciliary muscle, allowing the aqueous to pervade the suprachoroidal space. In a few, it is due to slight hemorrhage into this space. Excessive hemorrhage in this locality usually means expulsion of the contents of the globe and loss of the eye. Exceptionally, the detached is the work of an inflammatory exudate. In the first two forms, spontaneous recovery is the rule in from one to eight weeks.

**Objective Signs.**—Rounded shadows are seen to encroach upon the pupil-reflex, springing usually from the nasal side;



rarely from the temporal side, and never from below. In height they may reach almost to the median line, or even beyond it. The swellings that give rise to the shadows are sometimes separated by deep furrows—at others the swelling is single. They are situated well forward, near the ciliary zone, so that they are visible by lateral illuminations. Their color is brownish- or yellowish-red. A few retinal vessels may be seen stretched horizontally over them, while beneath these are the choroidal vessels, ramifying over the mass. There is no movement in this detachment as in that of the retina. It is never white, gray or blue—always of a dark or warm tint.

**Retinitis Striata. Striæ of the Retina.**—In discussing detachment of the retina we have seen how certain white streaks are formed in the retina, supposedly by the transformation into connective tissue of the deposits of fibrin that accumulate where retina and choroid meet along the limits of the detachment. The stripes are long and slender, of tolerably regular width, disposed in gentle curves, and often confluent. Their direction is peculiar in that it is roughly *concentric with the papilla, or transversal to the courses of the larger retinal vessels*. They are in bright contrast to the eye-ground, though they are most always pure white—rarely showing a slight grayish or yellow tinge. We have also seen that pigment is often present in the striated territory of the fundus. This is the common type of the affection.

Another type of striated retina has been described. Here the stripes themselves are often pigmented; they are brownish, yellowish or grayish, of more irregular breadth, are straighter, and their course is generally radiate as regards the papilla, or *with* that of the larger retinal vessels; though they have nothing in common with these vessels. In several instances hemorrhages have been seen in the immediate vicinity of the stripes. For this, and other reasons, their origin is accredited, by the majority of the few observers who have investigated the matter, to retinal hemorrhages, particularly to the retro-retinal variety. As they seem to



pertain to a period of life less advanced than that wherein detachment is most frequent, it is not improbable that they are related to natal hemorrhages, or to those of adolescents; in the latter connection being an **external form of retinitis proliferans**. Their principal diagnostic feature lies in the fact that *they are always situated behind the retinal vessels*. In proliferating retinitis the white bands are internal to the retina, and cover the vessels (see "Retinitis Proliferans").

Both the affections just described are sometimes simulated by the cicatrices of the fundus that are consequent upon ruptures of the choroid and retina. Here, however, there is most always the history of an injury. Besides, these scars are nearly always in the posterior polar and papillary regions—the parts that bear the brunt of *contre-coup*—and the lesions are coarser and less regular.



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