

University of Nebraska Medical Center DigitalCommons@UNMC

MD Theses

Special Collections

1933

Ophthalmoscopic examination

S. P. Benbrook University of Nebraska Medical Center

This manuscript is historical in nature and may not reflect current medical research and practice. Search PubMed for current research.

Follow this and additional works at: https://digitalcommons.unmc.edu/mdtheses

Recommended Citation

Benbrook, S. P., "Ophthalmoscopic examination" (1933). *MD Theses*. 246. https://digitalcommons.unmc.edu/mdtheses/246

This Thesis is brought to you for free and open access by the Special Collections at DigitalCommons@UNMC. It has been accepted for inclusion in MD Theses by an authorized administrator of DigitalCommons@UNMC. For more information, please contact digitalcommons@unmc.edu.

OPHTHALMOSCOPIC EXAMINATION

Senior Thesis University of Nebraska College of Medicine S. P. Benbrook Omaha, Nebraska

TABLE OF CONTENTS

		Pages		
I	Importance of Ophthalmoscopic Examination			
II	History of Development of the Ophthalmoscope	2 - 3		
III	Technique of Using the Ophthalmoscope	4 - 6		
IV	Anatomy			
	A Papilla	7 - 8		
	B Retina	9 - 11		
	C Chorioid	11 - 12		
ν	Appearance of the Normal Papilla and Fundus	13 - 19		
VI	Pathology of the Papilla			
	A Atrophy	20 - 25		
	B Neuritis	26 - 29		
	C Papilledema	29 - 33		
VII	Pathology of the Retina and Chorioid			
	A General Pathology and Diagnosis			
	l Hemorrhages	34 - 35		
	2 White Spots	35 - 36		
	3 Opacities	36		
	4 Pigmentation and Depigmentation	36 - 37		
	B Important Diseases Pathology and			
	Diagnosis			
	l Syphilitic Retinitis	37 - 3 8		
	2 Albuminuric Neuro-Retinitis	38 - 40		
	3 Diabetic Retinitis	41		
	4 Leukemic Retinitis	41 - 42		
	4804	77		

\$			Pages
	5	Arteriosclerosis	42 - 43
	6	Tubercle of the Chorioid	44
VIII	Conclusions		45

IMPORTANCE OF OPHTHALMOSCOPIC EXAMINATION

In writing this paper, it will be our purpose to discuss the use of the ophthalmoscope as an aid to the general practitioner in making diagnosis. Our discussion will be limited to the more important diseases in which an examination of the eye grounds is of diagnostic value.

It is becoming more and more realized that the practitioner of medicine should familiarize himself with the use of the ophthalmoscope. Medical schools are falling in line and doing their part by offering courses in ophthalmoscopic examination to the students and helping the interns to become acquainted with the value of the instrument.

The importance of studying the eye grounds in our cases is impressed upon us when we realize that often a diagnosis can be made, before there are any general symptoms, by an ophthalmoscopic examination. Such an advantage should be of extreme importance in this day of preventive medicine.

The importance of viewing the eye grounds should further be impressed upon us when we are told that the eye is the only place in the human body where the circulation may be studied by a direct view, and this is only possible by the use of the ophthalmoscope.

HISTORY OF THE DEVELOPMENT OF THE OPHTHALMOSCOPE

The ophthalmoscope was invented by Babbage in 1848; however, its importance was not recognized, and it was rediscovered by von Helmholtz in 1851. The first ophthalmoscope made by von Helmholtz was merely a plane plate of glass as shown in Figure 1. A source of light was placed beside the observed eye, and holding a glass plate obliquely in front of it, so a portion of the light was reflected from the surface of the plate into the eye of the patient. Upon looking through the transparent plate, an observer could now receive some of the rays from the fundus into his own eye, and thus obtain an image of the illuminated fundus. It was found that the amount of illumination was very feeble by the use of this method, so von Helmholtz set out to improve upon this type of opthalmoscope.

The next type of ophthalmoscope was designed in an effort to increase the amount of light reflected from the plate glass. This was done by von Helmholtz, when he superimposed three plane plates of glass, silvering the back of the third mirror, thus converting it into a more powerful mirror. In the center a small round circular hole was left unsilvered, serving as an opening through which the observer might look. The illumination was still feeble, as the rays reflected by the plane mirror were divergent. The question of divergent rays was met and solved by Ruete in 1852. He introduced a



Diagram of von Helmholtz' ophthalmoscope. O_i , observed eye; O_2 , observer's eye; source of light; Limage of L formed by the plane mirror – immediate source of light; MEH, relative positions of retina in myopia, emmetropia, and hypermetropia respectively, showing the relative sizes of the areas of retina illuminated in each case. perforated mirror, which was concave, and thus held the rays down to a small field. A later modification was the addition of a battery of small lenses of various strengths, which might be brought into position behind the aperture.

With the advent of the flashlight came another step forward in the development of the ophthalmoscope. The instrument we now have is a combination of the reflecting mirror, lenses, and source of light, all in one compact unit.

There are several good makes of ophthalmoscope on the market today, and the physician and student should obtain the type which he finds best suited to his methods.

TECHNIQUE OF USING THE OPHTHALMOSCOPE

There are two chief methods of ophthalmoscopic examination, the indirect method, and the direct method. The indirect method is used to determine whether the eye is emmetropic or ametropic, by observing the effect of shifting the lens on the size of the image of the fundus. Such information is necessary in the refraction of eyes.

We shall be interested in the second, or direct, method, only, in our discussion. By using the direct method, the parts of the fundus are seen in their true position, giving an image magnified some 16 to 18 diameters, though a much smaller part of the fundus is seen than is in the indirect method. The amount of fundus that will be visible depends chiefly upon the size of the pupil, and also upon the amount of light used. Although only a small part of the fundus can be seen at once, yet by varying the position of the head and ophthalmoscope one is able to look over a considerable part of the posterior hemisphere of the eye. By this method minute changes can be detected, and one is given accurate information of any lesion as regards its level, being an upright image with everything being seen in its proper position. The refraction may be observed and corrected also.

The student should commence by taking a patient whose pupils have been dilated with atropin. In this way a larger field is seen, and the patient's accommodation is paralysed.

It is best to examine the patient in a dark room; how-

ever, this is often impossible, especially in large wards. The observer sits facing the patient, with his eye on a level with that of the patient. The patient is told to look at some fixed point on the wall or ceiling, disregarding the observer and all other things about him during the examination. The light is turned on, and from about a distance of 18 inches, the rays are allowed to fall upon the pupil to be examined, meanwhile the observer looks through the sight-hole. When the light falls on the pupil, he notices a red reflex. When the light is well on the pupil and the observer can see the red reflex, he approaches slowly nearer and nearer, watching that the light does not leave the pupil, until his brow is almost or just touching the patient's brow. When looking at the right eye of the patient, the observer holds the ophtnalmoscope in his right hand and uses his right eye for the inspection.

If he has lost the light, he draws back from the patient and repeats the same maneuver. If the image is not quite distinct, the lenses in the ophthalmoscope are slowly changed for stronger or weaker ones, while the observer watches until a sharply defined image of the papilla is seen. The best way to do this is to look constantly at a certain blood vessel, and to determine the correction. If the physician wishes to look at other parts of the fundus than that directly before him, he moves his head in the opposite direction to that in which the desired part lies. The patient should continue throughout to look quietly in the same direction.

By changing the lenses in the ophthalmoscope the different parts of the eye may be examined, starting with the cornea and observing it through a 4 20 D lens, and going right back thru, to the fundus, which is best seen at 0 in the emmetropic eye. A hypermetropic eye is too short, in comparison with the emmetropic, i. e., its retina is nearer to the eye of the observer than that of the emmetropic eye, all other conditions being the same. Hence, when in an otherwise emmetropic eye a certain part, for example, the papilla, is greatly elevated, as in a papilledema, so as to lie closer to the eye of the observer, such a part will be hypermetropic and form a contrast with its emmetropic surroundings. A difference in level can be calculated in millimeters from the difference in refraction, for a difference in refraction of 3 D corresponds to an elevation or depression of 1 millimeter.

The place and size of a lesion in the fundus may be indicated by reference to the papilla and its diameter, which is 1.5 mm. For example, it may be said, that a lesion lies 2 papillary diameters from the temporal margin of the disc of the optic nerve, i. e., 3 mm. distant.

ANATOMY

Before proceeding further it will be well to consider the anatomy of the normal papilla, retina and chorioid, in order that we may more readily recognize and understand pathological changes met in the various types of disease.

The optic nerve enters the orbit thru the optic foramen, in company with the ophthalmic artery, and passes into the eye ball about 1/10 of an inch internal to the posterior pole. The nerve is to be considered as a portion of the brain that has been projected forward, and like the latter, it is enveloped in three sheaths, the dural, arachnoidal, and pial membranes, the interspaces of which correspond to those of the brain and is furthermore connected directly with the lateral ventricles. This anatomical structure explains how any increase in intra-cranial pressure would be transmitted into the optic nerve and cause a papilledema.

When these sheaths reach the eye ball proper the two outer members pass over into the sclera, while the innermost joins with the chorioid and aids in making up the lamina cribrosa, thru which the optic nerve enters the eye ball. In the lower surface of the nerve, about 15-20 mm. behind, the globe, the central artery enters. This artery divides on or slightly below the disc into the main retinal trunks. In the orbital portion the nerve is round and about 4 mm. thick. It is made up of nerve fivers and connective tissue. The nerve fibers form bundles which run parallel to one

another and are interlaced together by an interchange of fibers, the number of which has been estimated at half a million. The nerve fibers have a medullary sheath and a supporting substance called neuroglia tissue lies between them. The pial sheath, which lies right next to the nerve, sends in numerous trabeculae and septa within which are found the lymphatic and blood vessels.

The average size of the individual fibers is 2 microns. The smallest fibers are those of the maculopapillar bundle, which supplies the macula. The bundle lies wholly in the temporal margin and occupies the lower, outer sector of the disc at the level at which the central vessels divide. The fact will be of significance later, when we consider the temporal pallor of multiple sclerosis.

At the level of the inner surface of the sclera there are numerous fibers of connective tissue cutting transversely thru the nerve, which together with fibers from the sclera and choroid form the lamina cribrosa, thru the meshes of which passes the optic nerve, which at this point is 1.6 mm. thick. This diminution in diameter is due to the loss of the medullary sheaths, which go anteriorly only as far as the posterior third of the sclera. This loss causes a change in color, the meduallated fibers appearing white and the non-medullated gray.

The fibers of the optic nerve bend outward in the papilla and are distributed in the layer of nerve fibers of the retina.

The retina is about 1/3 mm. thick and corresponds in extent with the choroid which it lines. It is connected to the subjacent tissue at the entrance of the optic nerve and at the fovea centralis. When the retina is pulled away from the choroid the pigment layer remains in close connection with the choroid. Nevertheless the layer of pigment epithelium belongs to the retina. It has been shown by embryological research that the retina has continued forwards as a double layer of epithelium as far as the edge of the pupil. The center of the retina appears yellowish, the macula lutea, with a dark brown point, the fovea centralis.

In the living eye the retina is perfectly clear and transparent, its presence being perceived only by means of the vessels that course in it. The color of the fundus is therefore not influenced by that of the retina itself, but is due essentially to the greater or less abundance of the pigment contained in the pigment layer and in the choroid. The color of the choroidal vessels plays a subordinate part. The peculiar color of the macula is suppressed by that of the subjacent tissue; it appears only a little darker than its surroundings. Its center, the fovea centralis, is still darker, because the retina is very thin at that point and consequently the chor**p**idal vessels and pigment show through.

The layers of the retina are known as outer and inner, according to their position relative to the contents of the

eye. Those lying nearest the vitreous are called the inner layers, those more distant the outer. The outer layers, i. e., the layer of rods and cones, the outer granular layer, and the membrana limitans, which lies between them, form what is known as the layer of sensory epithelium, while the others which occupy the inner portion of the retina are grouped together as the cerebral layer.

The nerve fibers radiate from the papilla, with the exception of those coming from the temporal side, which circle in a great arch about the macula. The macula itself is supplied by particularly fine fibers which run directly to it from the temporal margin of the papilla, as the papillomacular bundle.

The macula is situated about 4 mm. outward and a little downward from the entrance of the optic nerve. Its margins are a little raised and its center is a depression, the fovea centralis. The number of cones increases toward the fovea at the expense of the rods.

While the number of cones increases as the macula is approached, it decreases toward the periphery, where the rods predominate.

The nutrition of the retina is received from two sources, the central artery and the vessels of the choroid. The former supplies the cerebral layer, the latter the layer of the pigment epithelium. The layer of nerve epithelium is entirely without blood vessles and receives its nourishment by diffusion from the capillary network of the choroid; the

fovea centralis is likewise nonvascular, the fine retinal vessels end in a circle of capillary loops on its margin.

In as much as the percipient organs of the retina lie in the outer layer, rays of light must first pass through the entire thickness of the retina in order to reach the organs of perception. This explains why the retina must be perfectly transparent, with the absence of vessels in the region of the macula.

The choroid is in such close relation with the retina that it is difficult to discuss them separately. The choroid consists essentially of vessels. It is in contact everywhere with the sclerotic, though not firmly adherent to it, so there is a potential space between the two structures which acts as a lymph space. On the inner side the choroid is covered by a thin elastic membrane, the membrane of Bruch. The blood vessels of the choroid increase in size from within outwards, so that immediately beneath the membrane of Bruch there is a capillary plexus, the choriocapillaris. Following upon this is the layer of medium-sized vessels, while most external are the large vessels.

The equatorial portions of the choroid have the poorest supply, consequently this portion is the first, or main one to be affected when degenerative processes take place in the eye.

The venous outflow of the choroid is quite different from its arterial supply, as its veins carry away not only

blood from the choroid itself, but also from the ciliary body and iris. Consequently, they are far more numerous than the arteries and have many more anastomoses. They commonly pass from the choroid into the sclera behind the equator of the eye in the form of from 4 to 6 large vessels.

. The choroid contains besides vessels, many collagenous fibrils and elastic fibers, as well as a great quantity of chromatophores laden with pigment, being found especially in the spaces between the vessels.

APPEARANCE OF THE NORMAL PAPILLA AND FUNDUS

When the fundus is viewed with the ophthalmoscope the first object to be sought is the optic disc or papilla. This is readily done by locating a vessel and following it centrally until the papilla comes into view.

The name papilla dates back to a time when it was thought to be an elevation at the entrance of the optic nerve. This is an erroneous anatomical idea, as it is the same height as the surrounding retina in the normal fundus.

The examination should be systematic and faithfully complete, certain points being noticed, one after the other, as follows:

- (1) Form and size.
- (2) Color.
- (3) Margins.
- (4) Level, excavation or protrusion.
- (5) Vessels.

It will be noticed in the normal papilla that the form is round or slightly oval vertically. Variations in size are only apparent as a rule -- in hypermetropea, the papilla seems to be smaller; in myopia, larger. True, differences in size are met with at times in hypermetropic eyes.

The color of the papilla is a delicate red, with the temporal side usually a little brighter than the nasal. In many cases a bright spot is to be seen in, or a little to one side of, the center, which corresponds to the excavation which will be described later. The color is also influenced by its environment. If this is very dark, as in brunettes, the optic nerve will seem to be particularly bright from contrast, and on the other hand it looks redder when the fundus is particularly pale.

The margins of the normal papilla are sharply defined. Surrounding the papilla two rings are frequently present, one white, the scleral ring, and one black, the pigment ring. These circles are complete only in exceptional cases; as a rule, only segments are visible, and these are usually on the temporal side. Both rings may be entirely absent.

In considering the excavation of the papilla, two types need to be differentiated, the flat papilla and the excavated. In the flat, the fibers of the optic nerve spread out on a level with the retina. The color of such a papilla would be uniformly red, with scarcely any difference in color between the temporal and nasal portions, and the white spot, which indicates the excavation in the other type, is nearly absent. The excavated type of papilla is due to the fact that the fibers of the optic nerve do not completely fill out the hole in the choroid, leaving a funnel-shaped or cupshaped cavity below the level of the surrounding tissue. The excavation may occupy only a small part of the papilla, or may be so large that only a small margin of color remains.

The transition from the tissue proper of the papilla to

the excavation may be gradual or abrupt, this being determined by the behavior of the vessels; in the former, they pass without visible bending into the white place, and in the other they bend like hooks.

The depth of the excavation varies and by use of the lenses of the ophthalmoscope, a fairly accurate estimation of the degree of excavation may be learned. Three diopters change between levels equals 1 mm. of depth. If the patient and observer are emmetropic and a -3 D lens must be interposed to enable the former to see the vessels at the bottom of the excavation clearly, this would then tell us that the excavation is 1 mm. deep.

The position of the cupping is usually central and often extends into the temporal portion. Every abrupt excavation that extends to the margin of the papilla should be considered pathological.

The central artery and vein passing out of the papilla divide, sending a branch upward and another downward, thus forming the superior and inferior artery and vein. The vessels divide and subdivide to form the schematic picture shown in Fig. 2 No anastomoses are present. Such regularity of subdivisions as pictured in the drawing is rarely seen. The vessels may divide within the optic nerve so that apparently three vessels emerge from the papilla. In the same manner we may see four vessels in cases where both of the main vessels divide within the optic nerve.

The vessels of the macula as a rule come from the super-



Schematic drawing of the Fundus, Upright Image.

- A- Superior Nasal Vein
 B- Inferior Nasal Vein
 C- Superior Temporal Vein
 D- Superior Papillary Vein
 E- Macular Vein
 F- Macular Artery
 G- Inferior Papillary Vein
 H- Inferior Temporal Vein

ior and inferior temporal arteries and veins.

The arteries and veins can be distinguished easily. It will be noticed that the arteries are more slender than the veins, being only about two-thirds the size of the veins. They are sharper in outline and follow a more direct course. They are bright red and have distinct reflexes, the breadth of which is about one-quarter the diameter of the vessel. The veins are a wine-red and have light reflexes, which are considerabley narrower, and are not so distinct as those on the arteries. The light reflexes from the vessels is thought to come from the surface of the blood column itself, and not the vessel wall, which is perfectly transparent under normal conditions.

The arteries and veins frequently cross. Branches of the same kind of vessels never cross, a fact that can be utilized to determine the nature of a vessel which cannot be seen distinctly.

In the principal venous trunks which lie near the papilla a pulsation is generally observed, although it is not very marked. The contraction and paleness of the vein begins just before the beat of the radial pulse, and extends from the center toward the periphery.

There are two types of vascular anomalies which are to be looked upon as normal. The first is the cilio-retinal vessels which emerge in the form of hooks of the periphery of the papilla, or in the region of the connective tissue ring, and pass into the retina. The second vascular anomaly is

the opticociliary vessels which pass into the vascular system of the choroid. They are rare normally, but more common, as newly formed vessels, in pathological conditions, such as glaucoma, papilledema, and wounds.

The color of the fundus is greatly influenced by the color and density of the pigment; the color of the vessels in the choroid is of less importance. The visual purple can in no way exert any influence, although its name might lead one to suppose it could. As we have mentioned previously, pigment can be found in the layer of pigment epithelium of the retina and in the intervascular spaces of the choroid. Three types of normal fundi are distinguished according to the quantity and distribution of the pigment in these two membranes.

I Uniform Stippled Fundus

A uniform appearance is seen in this type of fundus because the layer of pigment epithelium in the retina is dense, completely hiding the choroid underneath.

The tone of the color is red, brownish-red, or blackishbrown, according to the quantity of pigment.

II Tessellated Fundus

In this type of fundus the layer of pigment epithelium contains less coloring matter; consequently it is possible to see through the almost transparent retina and perceive the markings of the choroid quite well. The reddish choroidal vessels are seen to form numerous anastomoses, and the pigment of the choroid is massed into the intervascular spaces between them. The vessels appear as bright bands on the dark background.

III The Albinotic Fundus

In this type the layer of pigment epithelium contains little or no pigment, so that the markings of the choroid are again visible. In as much as the choroid has no pigment either, the sclera is seen to shine thru the retina and choroid, forming a yellowish-white background, upon which the choroidal vessels stand out as dark bands. They can be distinguished from the retinal vessels by the absence of light reflex, their abundant anastomoses, and their deeper position.

At times cases are met that do not belong exclusively to any one type of fundus. The layer of pigment epithelium may be dense only in places, allowing the choroid to snow thru in the less dense areas.

The pigmentation is usually thickest about the papilla and in the region of the macula. The amount of pigment in the fundus is usually in keeping with that in the hair and skin of individuals, so that we may hear the term, blonde or brunette fundus.

It is very striking to observe the fundus of a negro for the first time, having previously viewed the fundi of many white people.

Retinal vessels can be distinguished from choroidal

vessels by several characteristics. They appear to be round, have light streaks, have no anastomoses, and converge toward the papilla. The choroidal vessels appear to be flat, have no light streaks, form many anastomoses, and have no uniform direction.

The course of the retinal vessels varies according to the refraction of the eye. In myopia they are drawn out, while in hypermetropia a marked tortuosity, especially of the veins, can be seen. This tortuosity is due to the growth of the eyeball, being too little as compared with the design of the vessels.

The portion of the fundus called the macula has been termed such because of a yellow coloring matter which it contains. It is recognized by the behavior of blood vessels which surround and direct their points at it without reaching it, so the area has no blood vessels in it. The macula lies about $1\frac{1}{2}$ papillary diameters from and a little above the papilla. It has the form of an oval, 5 papillary diameters (P. D.) broad, and $2\frac{1}{2}$ P. D. high. The deeper red of the macula is due to the thinness of the retina, so that the specially vascular choriocapillaris of this region is seen more clearly. There is nearly always a foveal feflex, due to reflection of light from the walls of the foveal depression. This is most frequently seen as a silver ring, niding everything behind it.

PATHOLOGY OF THE PAPILLA

The only part of the optic nerve to be seen with the ophthalmoscope is the disc; changes taking place in the nerve may or may not cause alterations in the disc. Abnormal conditions in the disc may cause:

(1) Alterations in color.

(2) Alterations in surface level.

(3) Changes in the margins.

(4) Changes in the vessels.

Great variations are met with in color of the disc in different subjects, so that it is extremely difficult in many cases to decide when any increase in normal color has taken place. A comparison of the color of the discs on the two sides may be of great help. Hyperemia frequently exists, and will cause an increase in the normal pink color; this increase of color is due to fullness of the capillaries. This condition of hyperemia may remain for a long time and then gradually subside, or may pass on to inflammation, and will be referred to later under the head of papillitis; or it may pass on to the opposite condition, atrophy of the optic disc.

Anemia may cause the discs to look paler than usual; at the same time the retinal vessels will be badly filled. In permicious anemia the red of the chorioid may be diminished, while the retinal veins may be almost as light colored as the arteries. As age advances the disc becomes paler, so that a pale disc which when seen in an old person, may be normal, might in a young individual indicate a condition bordering on atrophy.

Atrophy of the optic nerve may be total or partial. We shall consider first the total atrophies.

In simple atrophy the only change seen in the disc is in the color which is a white to gray-white. The retinal vessels may become smaller in the later stages, so as to resemble nutritional atrophy, but this does not belong to the typical picture. The atrophy may be of a true neurogenous origin, caused by cerebral disease, when it is primary, or it may be due to injuries or compressions, when it is secondary. The same picture is seen in both cases, so the true diagnosis will depend on the findings in general and neurological examination.

Etiologically, tabes is the first disease to be thought of; then comes general paralysis and syphilis. Tabes passes everything else in importance. Tabetic atrophy begins, as a rule, very early in the disease and may be for years its only symptom. It rarely appears at the same time with the ataxia of the lower limbs. Tabetic atrophy almost always affects both eyes and leads to blindness, though with remissions.

Tabes frequently causes an ocular third; atrophy of the optic nerve, paresis of the ocular muscles, reflex immobility of the pupils.

None of the other causes are very frequent. Among them may be mentioned interruption of the conductivity of the optic nerve by direct or indirect injuries, as in fracture of the skull, compression in the optic canal, slowly growing tumors at the base of the skull, and pressure of arteriosclerotic carotid upon the intracranial portion of the nerve.

In simple atrophy of the optic nerve there is little proliferation of connective tissue; both the vessels and capillaries are preserved. Hence the white or gray discoloration cannot be caused in this way, but is probably due to reflection of light from the lamina cribrosa.

Nutritional atrophy may appear very much like simple atrophy, which has just been described. Only the absolutely negative evidence of neurological and internal examination, with the exception of arteriosclerosis that is often moderate, together with the condition of the retinal vessels, proves the diagnosis. This form of atrophy is to be met in old people. The visual disturbances are comparatively trivial in these cases.

In cases where there is an occlusion of an artery, the optic disc is white, with normal margins and level. The arteries are threadlike, no longer visible in some places. Patches of degeneration are often found in the macula. The vision is usually totally lost in these cases.

In glaucomatous atrophy the color is a gray white, as a rule; the margins often seem to be obscured by a surround-

ing ring, the so-called nalo. The veins are usually broad, sometimes varicose, while the arteries are engorged only at first, but later appear to be contracted. This form of atrophy is sharply differentiated from all others by the behavior of the vessels at the margin of the papilla, where they bend suddenly and seem to disappear in the cavity, and one must interpose concave lenses to see the floor of the excavation. From the difference in focus the depth of the excavation can be determined. The floor of the papilla usually lies 1 mm. behind the level of the retina. The fact that it is abrupt and extends to the margin distinguisnes the glaucomatous from the physiological excavation. Whenever a vessel is seen to make a sharp hook over the margin of the papilla the diagnosis of glaucoma is justified.

In old glaucomatous eyes we find obliterations of the vessels throughout the entire area of the retina, as well as new formation of large vessels at the margin of the papilla, or in the excavation.

Turning our attention to partial, or temporal atrophy of the optic nerve, we find the margins, level, and vessels are perfectly normal, the only variation from the normal to be seen is the paleness of the temporal side of the papilla. If the diagnosis of temporal pallor cannot be made from the ophthalmoscopic picture, the field of vision is to be investigated. If a central scotoma is found, with the outer portions of the field normal, a positive diagnosis of partial atrophy may be made.

Temporal paleness may be caused by several different diseases, hence the determination of the etiology is of great importance.

One of the most outstanding causes of pallor is multiple sclerosis. The visual disturbance caused by this condition is present, in about half the cases, and may precede by years all other signs. Nystagmus when the eyes are turned as far as possible to one side or the other is frequently present as an accompanying symptom. The discovery of temporal paleness often leads to the neurological examination that discloses the presence of this serious disease.

Chronic intoxications such as poisoning with methyl alcohol, lead, and arsenic, as well as auto-intoxication in diabetes, may cause the condition of temporal pallor.

The inflammatory diseases of the posterior ethmoidal cells and of the sphenoidal sinus may bring about a similar picture thru an extension of the inflammation to the optic

nerve, or through the influence of toxins some time after its subsidence.

Pathologically, this is considered as a secondary atrophy of the papillomacular bundle of the optic nerve fibers, which passes over the temporal margin of the papilla and supplies the macula; hence the central scotoma. Redness of the papilla without any other symptoms must be considered with great care, as it varies physiologically within rather wide limits. A papilla always appears redder in blonde than in a brunette fundus. Hyperemia may also be produced by prolonged examination with the ophthalmoscope.

Aside from the physiological causes, there is hyperemia which appears as an accompanying symptom of morbid processes, as in inflammations of the anterior and posterior segments of the eye, especially in iritis and iredocyclitis, in injuries of the eyeball, in empyemata of the accessory sinuses, and in such circulatory disturbances as are caused by heart disease. All the above possibilities must be kept in mind, recognized, and correctly estimated before a hyperemia can be called a forerunner to an optic neuritis or papilledema.

Optic neuritis should always be looked upon as a symptom of a serious disease, and it is the duty of the physician to ascertain the fundamental disease that has caused it.

In this condition we see redness and cloudiness of the papilla, with obstructions of its margins, peripapillary edema. There is little or no elevation of the papilla. The arteries are not changed, to speak of, but the veins are broadened and tortuous and are accompanied with streaks.

The most common cause of optic neuritis is syphilis, the next in frequency is albuminuria, all other causes being of secondary importance. They are tuberculosis, diabetes, basilar meningitis, typhoid fever, malaria, oneumonia, small

pox, diphtheria, scarlet fever, epidemic cerebrospinal meningitis, and myelitis.

Among the local conditions may be named suppurative inflammation in the orbit, in the ear, or in the accessory sinuses, and abscesses in the brain.

It should be noted that inflammation excited by general disease is commonly bilateral, while that due to local causes is apt to be confined to one side.

The onset may be acute and may, in that case, become fully developed in a few days, or it may take a chronic form, when the inflammatory lesion may affect only a portion of the papilla, as for example, its nasal half, and thence may spread to the whole.

When the inflammation disappears, the papilla finally snows a whitish atrophic discoloration which may be total or partial. As a rule the function returns to a greater or less degree.

After one has made a diagnosis of optic neuritis, what etiological conclusions can be drawn from the ophthalmoscopic picture?

It has already been mentioned that the forms of optic neuritis caused by general disease are for the most part bilateral, while those due to local lesions are unilateral. This is a guide to a certain extent. The entire eye grounds should be searched for points that may help make a diagnosis. If we should fail to find the hemorrhages or white spots characteristic of the albuminuric form which will be described later, we have to notice whether patches can be found in the chorioid that suggest by their appearance a syphilitic, or a tuberculous origin.

Syphilitic optic neuritis can often be recognized from the presence of a large amount of edema which extends into the retina for the distance of two papillary diameters. Old or fresh patcnes in the chorioid, with deposits of pigment are frequently to be seen in the periphery. The retinal vessels show sclerotic changes, in consequence of which hemorrhages, arranged like the spokes in a wheel, and white spots are not uncommon.

In as much as a Wassermann test is made in every case of any question, it is rarely that the diagnosis of syphilitic optic neuritis will fail to be made.

As a rule it is not possible to recognize tuberculous optic neuritis as such from the ophthalmoscopic picture, unless tubercles are visible in the chorioid. It is met with most always in young people, and is to be diagnosed by the exclusion of every other cause that may produce an optic neuritis and the existence of tuberculous lesions elsewhere in the body. The prognosis is bad as regards life in the cases in which a tubercle is located in the optic nerve.

Albuminuric and diabetic optic neuritis can often be distinguished by the early appearance of hemorrhages and patches of degeneration in the retina. The pathology in

the retina usually retires that of the papilla to the background, and we shall learn more of it a little later.

Arteriosclerotic optic neuritis is characterized by a rather sluggish course and more or less distinct arteriosclerotic changes in the retina. To make a diagnosis of this type of optic neuritis one must have observed the morbid process from the start, being fairly sure that the picture represents the acme of the disease; otherwise the same picture could be brought about by some other cause.

An optic neuritis due to inflammation of the middle ear usually exhibits only engorgement and hyperemia, the vessels are changed only a little, and there is little edema. It has been observed that the inflammatory symptoms augment considerably after the opening of an abscess, for example, but this has no unfavorable influence on the prognosis.

The forms of optic neuritis that are caused by abscess in the orbit and empyemas of the accessory sinuses show a marked contrast to the ologenous in that the disturbance of vision, a central scolomata which is often very large, may be quite considerable at a time when scarcely anything wrong can be seen on the optic nerve. In many cases of abscess of the orbit a thrombosis of the retinal vessels is produced, which can be recognized by the deep black color of the columns of blood and the absence of the pressure pulse.

Inflammation may attack any part of the optic nerve between the brain and eye, causing a papilledema or choked disk.

There is as yet no universally accepted theory as to the nature of the origin of choked disk; it is still uncertain whether it is caused purely by engorgement, or by inflammation.

Considering the course of choked disc, the arteries are first seen to become small and to be provided with broad reflex stripes upon the papilla. The large venous trunks are much broadened, tortuous, of a dark red color, and are destitute of pulsation. The smaller veins become more distinct because of their greater fullness. The vessels in general, but particularly the veins, appear to be bent and broken on the other side of the margin of the papilla; a large number of vessels often become visible on the papilla itself, and give it a reddish gray tone. The papilla forms a marked elevation with a precipitous descent to the retina, and exhibits an increasing opacity with radiating lines, which covers its margins, extends out beyond them, and is bordered by a gray edge. The excavation may exist for a while, or only a part of the papilla may be affected. In its further course the elevation and swelling of the papilla increases, the retina in its immediate neighborhood becoming more and more opaque, and consequently gives the impression that the papilla has become broader. The arteries appear to be drawn and more contracted than at first, while the veins, beginning with pale, pointed ends, show a deep, dark red color, have diameters that vary a great deal according to the depth at which they are situated, and run a tortuous course thru the retina.

Frequently the vessels are hidden, or obscured, for a distance by a gray opacity, and hemorrhages are often found arranged in radial striae, usually in the retina at the margin of the papilla, as well as here and there in the latter itself. Fine, brilliant white lines, ordinarily arranged radially, on and also outside of the papilla, or small, brilliant white spots, which appear at a very early period, are chiefly to be observed in children or young people. These lines and spots often extend beyond the margin of the papilla and maintain such an extent and grouping that the retina may present the same condition as in albuminuric retinitis.

The papilla gradually loses its reddish tone of color, which is replaced by a white, or yellowish white opacity, inclining to gray, but its margins remain obscured and the swelling continues to be plainly demonstrable. The onset of these changes usners in the so-called atrophic stage of choked disc, in which the protrusion of the papilla subsides. The swelling and the opacity do not undergo complete involution, the arteries remain small, the veins engorged.

In a case where choked disc is found, one of the most important questions one would ask is, "What etiological conclusions can be drawn from the ophthalmoscopic picture of a choked disc?"

Unilateral choked disc occurs in affections of the orbit, such as tumor, abscess, and gumma, and in diseases of the accessory sinuses. Abscesses in the middle fossa of the skull may protrude into the orbit.

Bilateral choked disc occurs in all conditions of the brain that reduce the amount of space in the cranial cavity. Chief among these are all kinds of tumors of the brain, including not only the true tumors, but also cysticerci, aneurysms, gummata, and tubercles (about 70 to 80%). Choked disc is absent in only from 5 to 10% of the cases of tumor of the brain, and these are mainly tumors of the frontal brain and of the hypophysis. The farther back the tumor lies the more certain is a choked disc to appear. A very rapid onset of visual disturbance, with a high degree of choked disc and severe pains in the back of the head, is indicative of a gumma in the cerebellum; a choked disc with disturbance of auditory and facial nerves, of a tumor in the angle between the cerebellum and the pons.

The cause of choked disc next in importance to tumor of the brain is serous meningitis, or hydrocephalous internus. None of the other causes, such as abscess of the brain, sinus thrombosis, are of equal consequence.

Finally, a choked disc may be caused by an obstruction to the outflow of the venous blood into the cavernous sinus.

It should be said that albuminuric and arteriosclerotic optic neuritis may present the picture of choked disc, in consequence of the engorgement that takes place at the same time. Therefore, the urine should be analysed and a Wassermann test made in every case.

The vision may remain normal a long time, but in the atrophic stage the vision gradually disappears, fleeting at-

tacks of blindness being present from the first.

Hemorrhages in the papilla may be seen in optic neuritis, or choked disc, or when the vessels of the retina are sclerotic. Sometimes they result from injury.

The demonstration of hemorrhages, when they are not of traumatic origin, is of great diagnostic importance. For example, this immediately decides the question in a doubtful case of optic neuritis or pseudoneuritis in favor of the former.

PATHOLOGY OF THE RETINA AND CHORIOID

Because of the profound relationship between the chorioid and retina, it will be advisable to consider their diseases in close connection with each other.

The outer layers of the retina are dependent for their nutrition upon the chorioid, so that when the latter suffers, the former is always envolved secondarily. Primary affection of the retina may occur without involvement of the chorioid; primary affections of the chorioid invariably involve the retina secondarily.

The extremely sensitive tissue of the retina, with its very small capillaries, reacts with great ease to any disturbance of circulation, and likewise any change in the composition of blood or tissue juice leaves its trace in the retina. Often the very first signs of general disease are made visible in the retina because it is so very sensitive.

It is unfortunate that the manifestations in the eye of various constitutional diseases are remarkably alike, so that it is only in rare cases that the exact etiological diagnosis can be made from the ophthalmoscopic picture alone, it usually has to be learned from the results of general examination. Even though it is not always possible to make the etiological diagnosis from the ophthalmoscopic examination, yet this much can be learned, that a general disease is present in all cases in which fresh changes are found in the form of white or black spots, opacities or hemorrhages. In such a case it is our im-

perative duty to submit the body to a very thorough examination, paying particular attention to the urine.

The most important change in the retina is hemorrhage, and when high myopia, glaucoma, and injuries can be excluded, they are always a sign of a general disease. They form a signal of warning; there is something out of order in the organism.

The first cause of hemorrhage to be mentioned is an injury, which need not be trauma directly to the eye, but may be severe body injury, such as compression of the thorax.

The most important cause is arteriosclerosis, and in these cases they are of very great prognostic value because, in at least 50%, they are forerunners of hemorrhage into the brain.

Diabetes and nephritis are very important causes both of little, stippled hemorrhages, and of large lake-like ones; both forms usually appear at the same time with white spots, or disease of the optic nerve. The etiology is always to be borne in mind when the hemorrhages are isolated.

Syphilis is likewise one of the principal causes of retinal hemorrhages, but other manifestations are usually present.

All the remaining causes are much less frequent. First among them is diseases of the blood such as chloroses, pernicious anemia, and leukemia. In well-marked cases of leukemia, the orange tone of the fundus and the great breadth of the vessels are diagnostic.

Hemorrhages are seen in the acute infectious diseases at times and should be regarded seriously.

Another very important type of pathology in the retina

is white spots which may be due to connective tissue, to proliferation of glia, to varicose thickening of the layer of nerve fibers, to fatty degeneration, to edema, to fibrinous or serous exudates, to deposits of calcareous matter, or to hyaline degeneration.

White spots in the retina must not be confused with medullated nerve fibers, which are quite superficial, in harmony with their anatomical development, and thus partly cover the vessels of the retina. They radiate from the papilla and show a more or less distinct fibrillation. Medullated nerve fibers form a congenital anomaly and are therefore of no clinical importance.

A third important change found in the retina is diffuse opacity. Here edema is the most common cause, and is found in almost all severe diseases of the retina. Another, very much rarer, form of diffuse opacity of the retina is due to an infiltration with white blood corpuscles, being met with in syphilis and leukemia. Another cause is "flat" detachment of the retina which may be due to injury, nephritis, arteriosclerosis, or syphilis.

In diseases which involve the chorioid pigmentation and depigmentation are the most important diagnostic signs.

The pigment of the chorioid, which lies in spaces between the vessels of this membrane, may last very long in spite of serious chorioidal disease. An abnormal heaping of pigment takes place at the same time wherever pigment is destroyed.

Almost all diseases of the chorioid are symptomatic of general diseases, with the exception of those due to traumatism

and some conditions that are congenital. Therefore, a very thorough examination is indicated in all such cases, just as in retinitis. The practitioner may be able to see black spots with the ophthalmoscope, but may not perceive or interpret the minuter differences, on account of his lack of special practice. So, if he should find black or white spots present in the fundus,, a thorough examination must be made of the organism, which is not to be confined to the ordinary physical and chemical methods alone, but in which the tuberculin and Wassermann tests are to be made.

If sclerosed vessels are present in the chorioid, the probably cause of the disease is either syphilis, arteriosclerosis, or nephritis; if no such vessels are present, the probably cause is tuberculosis.

There are so many diseases of the retina and chorioid that it will be best, for our purpose, to select the more common conditions with which the practitioner will be confronted and discuss them each separately. We will give special attention to the outstanding diagnostic features to be seen in the fundus in these diseases.

Syphilis

Syphilis is one of the commonest causes of retinitis, but is usually a secondary retinitis, accompanying disease of the onorioid. It does occur as a primary retinitis, and in this form syphilitic endarteritis is a prominent sign. The retina is cloudy, particularly in the neighborhood of the disc, which may be hyperemic. White spots may be seen in the macular region,

and yellowish or white spots often bounded by pigment, at the periphery of the fundus. The vessels may be degenerated with whitish exudations along their course; hemorrhages are rare.

In acquired syphilis the disease usually occurs one to two years after infection; usually both eyes are envolved, but not with simultaneous onset. Rarely the macular area alone is affected, snowing a grey or yellow deposit, or numerous small yellow spots and dots of pigment.

Retinitis is not uncommon in congenital syphilis. Such patients often show a dusty or peppery discrete pigmentation of the retina at the periphery, associated with a tigroid condition of the fundus in this situation. It is only distinguishable from the normal appearance by a greater aggregation of pigment. There may be thickly strewn black and white spots, like a mixture of salt and pepper. A proliferation of connective tissue often takes place.

The sight of white spots, as described above, should always arouse the suspicion of syphilis, and a positive diagnosis made by use of the Wassermann, history, and general physical examination.

Renal Disease

Ophthalmoscopic examination has come to be very useful in early diagnosis of kidney disease, as the retinal changes are often the first evidence that something is wrong. Though the degree of retinitis present bears no fixed relationship to the nature or severity of the renal mischief, yet in all cases its presence is of grave significance.

Diminution in visual activity is commonly the only symptom and may lead to the discovery of renal disease. Generally the history of severe headaches can be elicited, and the blood pressure is quite high. The condition is almost always bilateral, and very rarely causes blindness.

In this type of pathology known as Albuminuric Neuro-Retinitis, the opthalmoscopic picture is almost pathognomonic, being simulated only in some cases of intra-cranial tumor. In addition to the general signs of retinitis -- haziness of the retina and disc, hyperemia and hemorrhages -- the distinguishing feature is the presence of brilliant white spots and patches in the retina. The earlier deposits are cloudy, with soft edges ("cotton-wool" patches); the latter brighter, more sharply defined and punctuate. The disc is surrounded by large white patches or by a continuous "snow bank." Around the macula are smaller dots or round patches, also silvery white. Radiating from the fovea are spokes of white dots or fine lines, forming a star-shaped figure which is extremely characteristic. The beautiful picture of the stellate figure in the retina is so impressed on the minds of beginners that they expect to find it in every case of this disease, and yet it is met with only exceptionally; ordinarily we see only single white spots. In the typical cases, the vessels generally show degenerative changes. In some cases, especially in the albuminuric retinitis of pregnancy, a flat detachment of the retina occurs, almost certainly due to the retina being raised from the chorioid by exudates. Unlike most detachments of the retina, these fre-

quently disappear, the exudates being absorbed.

Albuminuric retinitis occurs in about 1/3 of cases of nephritis, and may occur in all forms of nephritis, including scarlatinae, and puerperal, in the greatest number of cases the disease is chronic interstitial nephritis, and it is very rare in acute nephritis.

In cases of nephritis of pregnancy, where retinal changes are found, we should lay great importance upon this finding. The earlier the onset of retinitis, the worse the prognosis, but fortunately it generally occurs in the last trimester. The artificial induction of abortion is indicated, and usually has a prompt beneficial effect; vision however, is usually permanently impaired, the degree depending upon the duration of the retinitis. The "cotton wool" patches clear up first, the glistening macular spots more slowly. Partial optic atrophy and slight retinal changes, such as white or pigmented spots at the macula, follow. The disease does not always occur at the first pregnancy; but may, after one attack, recur at subsequent pregnancies, though by no means always. The patient should, however, be warned of the danger of such a thing happening. The excellent pre-natal care provided mothers by modern obstetricians calls for routine examination of the urine. which would detect any albumin present, also routine checking of the blood pressure. The complaint of visual disturbance. such as spots before the eyes, or dimness of vision, is looked upon with a great deal of suspicion, and should serve as a warning signal to look out for complications.

Diabetic Retinitis

Retinitis as a complication of diabetes is relatively rare. but does occur in the late stages, and in elderly people. It no doubt is often missed, because of the peripheral position of the lesions and the opacities of the lens. It is generally, but not always, bilateral. Irregularly scattered small, bright spots around the macular region are the commonest manifestation. The snowy patches and stellate arrangement at the macula are usually absent, but it must be remembered that albuminuria is a frequent concomitant of the late stages of diabetes, and all the characteristics of albuminuric retinitis may occur. The white spots may coalesce into larger plaques with crenated edges, which indicate their mode of formation. Punctuate hemorrhages are freely scattered over the fundus; they are more often round and deeply seated than linear and superficial, as in renal retinitis. The vessels and optic disc are generally normal, as well as the remainder of the retina.

The prognosis depends upon the severity of the constitutional condition; 60 per cent. live more than two years.

With the routine examination of the urine in every case, it is seldom a case of diabetes is "picked up" by examination of the eye grounds; however, such a thing has happened.

Leukemic Retinitis

In leukemic retinitis the ophthalmoscopic picture is characteristic. The fundus is pale and orange-colored. The veins are dilated and tortuous, often with white lines along them, and are bright red, not dark; the arteries are small and pale

yellowish red. Very typical are white spots surrounded by a red rim; they consist of leucocytes surrounded by red corpuscles. These are present only rarely, and are also found in pernicious anemia. In every doubtful case where the above condition may be in question, a differential blood count should be done for further diagnostic aid.

Arteriosclerosis

Degenerative changes in the retinal vessels may be the first evidence of arteriosclerosis, and particularly of diseases of the cerebral vessels, pointing to danger of cerebral hemorrhage, and indicating lines of treatment which may prolong life. Undue tortuosity of the vessels is of no significance unless accompanied by other abnormalities, such as irregularity in size and breadth of the arteries, so that stretches of the vessels are much constricted alternating with normal or somewhat dilated portions. These changes in the lumen are due to endothelial proliferation in the intima. The normal light reflex from the vessel walls is often unusually bright and broad, especially in vessels at some distance from the disc ("silverwire" arteries). Under normal conditions it is possible to see a vein thru an artery at a point of crossing; in arteriosclerosis the artery loses its translucency so that the vein is obscured. Moreover, the artery exerts an abnormal pressure on the veins so that the blood flow is obstructed; the vein seems therefore to stop at the crossing and is more distended on the distal side than on the side toward the disc. Sometimes the vein appears to be pushed aside by the crossing artery; in

severe cases the vein, whether crossing above or below the artery, is diverted so that it crosses at right angles, the shortest possible route. The veins may exhibit a beaded appearance with alternate constrictions and dilatations. More pronounced changes make the walls of the vessels visible, so that the blood column, often narrowed, is bounded by white lines, the thickened fibrous walls; this may affect both arteries and veins, usually only individual vessels, in a portion of their course.

The changes indicated lead to increased permeability of the walls, and increased internal pressure, due to general disease, supplements this defect, and causes undue extravasation of lymph, and even hemorrhages. Edema of the retina thus arises, manifesting itself as a greyish opacity around the disc, or in spots along the course of the vessels. Hemorrhages occur as linear striated extravasations along the vessels, or as round spots scattered over the fundus.

These changes occur most frequently in elderly people and are seldom entirely absent in the aged. They are frequently associated with high blood pressure, and always indicate the necessity for exhaustive examination of the circulatory and excretory systems.

The prognosis as regards life in retinal vascular disease is decidedly better than in cases of renal retinitis, though the patient may die suddenly from cerebral hemorrhage or thrombosis.

Tubercle of the Chorioid.

Tubercle of the chorioid occurs in acute or miliary and chronic forms. Miliary tubercles are found in the late stages of acute miliary tuberculosis, especially tuberculous meningitis. Occasionally they may be seen before there is any evidence of meningitis or generalized tubercle. Opnthalmoscopically they appear as round, pale yellow spots, most frequently observed in the neighborhood of the disc, though any part of the chorioid may be attacked. Generally only three or four spots are seen, but as many as sixty or seventy have been found. They vary in size from pin-point specks to 1 or 2 mm. in diameter. They usually project slightly so as to raise the retina. They afford most important diagnostic evidence of tubercle in cases of meningitis and obscure general disease.

Often-times the history and symptoms of tuberculosis are not clear-cut enough for diagnosis. The use of the ophthalmoscope would again be a helpful aid in the making of a diagnosis.

CONCLUSIONS

We have discussed many of the conditions which may be present in the eye grounds which assist the physician in making a diagnosis.

The majority of the older practitioners depend on the ophthalmologist when an opinion on eye grounds is desired; however, many of the younger men just out of college are very good with the ophthalmoscope, although they, too, are practitioners.

If we who are new in the profession can learn to use the ophthalmoscope well enough to recognize a pathological condition, when we look into a patient's eye, then we will benefit both ourselves, and our patient. We will at least give the patient a confidence in us that we are able to recognize a pathological condition when we see it, even though its complexity must be more completely interpreted by a specialist.

BIBLIOGRAPHY

- Adam, C., Ophthalmoscopic Diagnosis, New York City, Ribman Co., 1931.
- Alt, Adolph, Ophthalmology for the Gen. Practitioner, St. Louis, J. H. Chambers & Co., 40-42, 1893.
- Bedell, A. J., Significance of Chorioidal Hemorrhages, Arch. of Ophth., 8: 186-208, August 1932.
- Bedell, Arthur J., The Significance of Some Changes in the Vessels of the Fundus, Arch. of Ophth., 4: 695-719, Nov. 1930.
- Browne, Edgar A., How To Use the Ophthalmoscope, Philadelphia, Henry C. Lea, 63-70, 1877.
- Buffum, F. H., Diseases of the Eye, Chicago, Gross and Delbridge, 34-40, 1884.
- Churchman, V. T., The Ophthalmoscope and the General Practitioner, West. Va. Med. J. 28: 15-19, Jan. 1932.
- Deaver, J. B., Surgical Anatomy, Philadelphia, P. Blakiston's Son & Co., 1901.
- Fick, A. E., Diseases of the Eye and Ophthalmoscopy, Philadelphia, P. Blakiston, Son & Co., 1896.
- Gager, Leslie T., Hypertensive Retinal Disease, Arch. of Ophth., 2: 305-315.
- Gilman, Coit Daniel, The New International Encyclopedia, New York, Dodd, Mead & Co., Vol. XV, 1904.
- Haab, O. Dr., Ophthalmoscopy, New York, William Wood & Co., 16-18, 1895.

Hartridge, Gustavus, The Ophthalmoscope, II Ed., London,

J. & A. Churchill, 1894.

Head, Gustavus, The Eye, Ear, Nose & Throat, Chicago,

The Year Book Publishers, Vol. III, 1910.

Jabez, Hogg, The Ophthalmoscope, London, John Churchill, 5-6. 1858.

Jackson, Edward, Refraction & Diseases of the Eye,

Philadelphia, W. B. Saunders & Co., 175-179, 1901. Lange, Fritz, Hemorrhages in the Fundi In Hypertension,

Arch. of Ophth., 2: 551-554.

Mann, A. William, Grouped Pigmentation of the Retina, Arch. of Ophth., 8: 66-71, July 1932.

Osler, William Sir, The Principles and Practice of Medicine,

New York, D. Appleton & Co., 1056-1060, 1930.

Parsons, J. H., Diseases of the Eye, VI Ed., New York,

The Macmillan Co., 1931.

Rolfe, Floyd, Retinal Changes in Hypertension and in Renal Disease, Arch. of Ophth., 6: 433-444, Sept. 1931.

Sage, Earl C., Obstetrical Notes For Juniors, Omaha Nebraska, Uni. of Nebr. Col. of Med., 7-8, 1930.

Stough, John T., Choking of the Optic Discs in Diseases Other

Than Tumor of the Brain, Arch. of Ophth., 8: 821-830.

Wilmer, W. H., Pierce, H. F., Friedenwald, J. S., The Light

Streaks on Retinal Blood Vessels, Arch. of Ophth.,

9: 368-380, March 1933.