the afferent nerves, as is shown by the so-called "cross circulation" experiment. In this experiment the neck vessels of two dogs are joined in such a way that the head of each is supplied from the carotid arteries of the

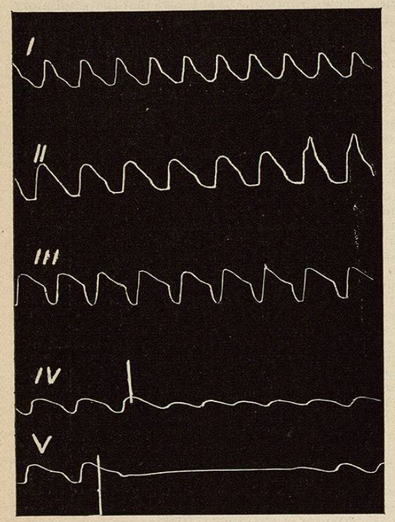

FIG. 4109.—Illustrates the Action of the Vagus on Respiration. Taken by the writer with an apparatus like that shown in Fig. 4107. *I*, Normal breathing of rabbit under ether; *II*, both vagi cut, respiration deeper and slower; *III*, cerebral hemispheres removed also; *IV*, weak stimulation of vagus opposite vertical line showing shallowing and quickening; *V*, stronger stimulation showing complete inhibition. Note: Downstrokes, inspiration; upstrokes, expiration.

other. The result is that the body of No. 1 and the brain of No. 2 receive the same blood and *vice versa*. If now the respiratory interchange of No. 1 be interfered with, the blood supplying his body will become venous, but his brain will continue to receive arterial blood from the other dog and his breathing will be unaffected. Dog No. 2, however, whose brain receives venous blood, will become dyspnœic, although the rest of his body is receiving good arterial blood.

Certain substances, other than carbonic acid, are produced in the muscles during activity, substances which also increase the activity of the respiratory centre. It has been shown that various acid substances have this effect, and the unknown substances formed in muscle are probably acid in nature.

The so-called automatic activity of the respiratory centre is believed to depend on a stimulus received from the blood, but it is yet undetermined whether the most important factor in this stimulus is a deficiency of oxygen, the presence of carbonic acid, or the action of the acid products of metabolism, although much can be said in favor of the last named.

The Influence of Afferent Nerves.—There is only one pair of nerves which have a tonic influence on the respiratory centre, as shown by a change in respiration when they are cut. These are the pneumogastrics.

On cutting one vagus (pneumogastric) the breathing becomes slightly deeper and slower for a time. Later the effect may pass off and the breathing become normal again. If both vagi are cut, the deepening and slowing of the breathing is more pronounced and the effect is more lasting.

The effects of stimulating the central end of one vagus are differently described by different writers. Nearly all agree that weak stimulation frequently produces shallowing and quickening, so that the breathing becomes more or less like what it was before the nerves were cut. With stronger stimulation various results are obtained according to the way in which the experiment is carried out, being influenced by the employment of anæsthetics and to some extent by the kind of stimulus used. This being the case, some writers believe that the pneumogastrics carry impulses which stimulate the respiratory centre to increased activity: and others, among whom is the writer, believe that it carries principally inhibitory impulses.

The impulses which normally ascend the vagus, exercising a constant inhibitory influence on the respiratory centre, are dependent upon the lung being distended, for Loewy has shown that opening the pleural cavity on one side so as to allow the lung to collapse has exactly the same effect on respiration as cutting the vagus on that side.

By rapidly inflating the lungs with a bellows the breathing may be entirely inhibited, the condition known as *apnœa* being produced. The result follows, no matter whether air be used for inflation, or some neutral gas, such as hydrogen. If air be drawn out of the lungs, diminishing their distention, very deep inspirations result. Hering and Breuer, who obtained these results, and also Head, who repeated their experiments, endeavor to explain them on the assumption that there are two kinds of fibres in the vagus, of which one set brought into action by distention of the lung favors expiration, and another set acting when the lung is collapsed favors inspiration. Their results are far more simply explained in the light of Loewy's work, by saying that the moderate distention of the lung normally present causes weak inhibitory impulses to ascend the vagus, which control the respiratory centre; increased distention gives rise to stronger impulses, which inhibit it altogether and produce a condition of apnœa; while during collapse of the lung the centre acts more powerfully in the absence of the usual inhibitory impulses.

In considering the effect of stimulating the fibres of the vagus directly we must remember that besides the respiratory fibres proper we can have passing up the vagus impulses of general sensibility and of pain, and these may cause changes in respiration through the sensorium like any other afferent nerve. The more completely we prevent the animal from feeling pain, the less likely are we to get pain effects and the more certain to see the direct action of the vagus on the respiratory centre. If an animal be experimented on without being completely narcotized, artificial stimulation of the vagus may produce almost any imaginable effect on the breathing, either inspiratory spasm (gasp), expiratory spasm (cry), or inhibition. If, on the contrary, the animal be well anæsthetized or decerebrated pain effects are eliminated and pure inhibition is nearly always seen, as shown by shallowing and quickening with weak stimulation, passing gradually, as the stimulus is increased in strength, into a state of complete inhibition or standstill in a position intermediate between inspiration and expiration (see Fig. 4109).

Even in unnarcotized animals the ascending constant current which stimulates without causing pain has almost always an inhibitory effect.

The other afferent nerves have no tonic action on the centre, for cutting any of them does not produce any change in the breathing. In special emergencies any afferent nerve may carry impulses that modify the action of the centre. If the nasal mucous membrane (fifth cranial nerve) be stimulated we get a *sneeze*, consisting of a gradual inspiration followed by a sudden spasmodic expiration through the nose. If the *glossopharyngeal* nerve be stimulated, as in swallowing, we get inhibition of respiration, which prevents food being drawn into the

larynx. Certain kinds of stimuli applied to the mucous membrane of the pharynx and tonsils cause the complex act of vomiting in which the muscles of respiration play a part. Stimulation of the *laryngeal* nerves causes in some cases mere slowing of the breathing; but if the stimulus be strong, we see inhibition of inspiration and expiratory spasm or cough. Stimulation of the *splanchnics* inhibits respiration. Stimulation of the optic or auditory is said to increase inspiratory activity. Stimulation of other sensory nerves, such as the sciatic, produces in many cases one or more deep inspirations with weak stimulation, and a strong expiration or cry if the stimulation is strong; but the results are by no means uniform.

The Influence of the Posterior Corpora Quadrigemina.—Removal of the brain in front of the posterior corpora quadrigemina has little effect on the breathing; but separation of these from the medulla has an effect just like bilateral section of the vagi; that is, the respiration becomes deep and slow. If the vagi be cut as well and the animal has been protected from excessive hemorrhage the respiration becomes still deeper and very infrequent. Usually in this case inspiration and expiration are separated from each other by long pauses. Restoration of the respiration, to about its normal character, may be effected by a well-chosen stimulus applied either to one of the vagi or to the corpora quadrigemina.

Cheyne-Stokes Breathing.—If the upper part of the medulla itself be injured the breathing is sometimes seen to take on a periodic character; that is to say, the respirations occur in groups of three, four, five, or more, of which the first respiration in each group is the deepest (Markwald), and the groups are separated by intervals in which respiration is in abeyance. A similar kind of respiration is seen when a blood extravasation presses on the region of the alæ cinereæ near the respiratory centre. These facts are interesting in connection with the so-called Cheyne-Stokes breathing occurring in various diseases of the brain, heart, and kidneys, which bears certain resemblances to this experimentally induced periodic breathing (see article on *Dyspnœa*).

The Conditions under which the Centre Acts.—A rational interpretation of all the facts given above would seem to be as follows:

1. The respiratory centre is situated in the medulla, in the lower part of the floor of the fourth ventricle.
2. It receives a constant stimulus from the blood.
3. Acting alone it would expend all its energy by responding at long intervals with a very great respiratory effort.
4. It receives inhibitory impulses from the posterior corpora quadrigemina, and by the vagi nerves from the lungs which control its action and convert the deep infrequent respiratory acts into the shallower and consequently more frequent ones that we know as normal breathing.
5. In special cases the centre may be influenced by impulses reaching it by other nerves.

The Efferent Nerves are the phrenics to the diaphragm, the intercostals, and the motor nerves to the other muscles of respiration. If the spinal cord be injured above the first dorsal vertebra the intercostal nerves and muscles are cut off from the centre and thoracic respiration ceases. If the injury be as high as the fourth or fifth cervical vertebra the phrenic nerves and diaphragm are also cut off from the centre and death ensues.

For the effect of breathing air at various pressures and air containing impurities see articles on *Air*, *Aërotherapeutics*, and *Caisson Disease*. *William S. Morrow.*

REFERENCES.—In preparing this article use has been made of the books of Schaefer, Mills, Foster, "American Text-Book" (Reichert), Hall, Halliburton, Jeffrey Bell, Böhm and Davidoff, Quain's "Anatomy," Hermann's "Handbook" (Rosenthal), Langendorff's "Physiologische Graphik"; also of numerous journal articles and data from experiments performed by myself. Special acknowledgment must be made of assistance received from two papers by Max Lewandowsky in Du Bois-Reymond's Archives for 1896 on "Die Regulierung der Athmung."

RETINA, DISEASES OF.—The retina is seldom affected by disease which is limited to itself alone, or even to the eye alone, but most often lesions of the retina are part of a general disease and are frequently of assistance in the diagnosis of the latter. The diseases most apt to produce serious retinal complications are, diseases of the kidneys, syphilis, diabetes, septicæmia, and leukæmia. Among ocular affections choroiditis and optic neuritis almost always lead to retinal changes, the former because of the close anatomical relationship of the choroid and retina, and the latter on account of the interference of the retinal blood supply produced by the swelling of the disc. Both choroiditis and optic neuritis, however, are usually in turn dependent upon some general disease.

VASCULAR DISTURBANCES OF THE RETINA.—*Pulsation of the retinal veins* on the disc is frequently seen under normal conditions, and can readily be produced by a slight pressure of the finger upon the eye. It is particularly associated with increased intra-ocular tension from any cause, and hence is common in glaucoma. No better explanation of the phenomenon than that of Donders has been advanced. According to Donders it is due to the changes in arterial tension being communicated to the veins through the vitreous humor. True transmitted venous pulsation has been seen in valvular heart disease, arteriosclerosis, and anæmia, but never under normal conditions.

Pulsation of the retinal arteries is always pathological, and indicates either an increase in intra-ocular tension or decrease in the arterial pressure. It may occur in glaucoma, anæmia, syncope, senile arteriosclerosis, aneurism of the arch of the aorta, aortic insufficiency, and Basedow's disease. It may also result from compression of the central artery by tumors of the nerve or orbit.

Hyperæmia of the retina may be either arterial or venous in nature. The general redness of the fundus depends to such a great extent upon the degree of pigmentation of the choroid, and the tortuosity of the vessels varies so much under normal conditions, that it is impossible to diagnose retinal hyperæmia unless the disc is also reddened. *Arterial hyperæmia* manifests itself by distention and tortuosity of the arteries which sometimes lie in antero-posterior planes so that they project toward the observer. It may result from eye strain due to improper illumination or errors of refraction, irritation of the eye from any cause, such as the presence of a foreign body on the cornea, and from keratitis, choroiditis, and iritis. It is common in meningitis, and may be noted in Basedow's disease, plethora, and neurasthenia. Strangely enough, it may result from excessive loss of blood or from chlorosis, and in the former case it may be so intense as to give rise to retinal hemorrhages. *Venous hyperæmia* is characterized by dilatation and tortuosity of the veins, which appear darker than normal, and is always associated with hyperæmia of the disc. It is not infrequently accompanied by retinal hemorrhages. The arteries may show no change, or they may be narrowed owing to the same obstruction which is producing stasis in the veins. In general, venous hyperæmia is due to some hindrance to the outflow of venous blood from the eye, as, for instance, to compression of the central vein in optic neuritis or glaucoma. Sometimes the obstruction lies in the orbit, as in cases of tenonitis and orbital cellulitis, or even in the cranial cavity, as in intracranial tumors, thrombosis of the cavernous sinus, and meningitis. Rarely it is a part of a general venous stasis due to valvular heart disease. A few cases of particularly exaggerated venous congestion have been seen associated with congenital heart disease, the condition then being spoken of as *cyanosis retinæ*.

Thrombosis of the central vein of the retina, which produces the highest grade of venous hyperæmia, is very rare. It usually is monocular and occurs in patients affected with general arteriosclerosis, and hence most often between the ages of sixty to seventy, but sometimes it occurs as the result of orbital cellulitis. The affection comes on suddenly without prodromal symptoms, and though vision is much diminished, blindness

is not produced at once. In marked cases the retinal veins are greatly distended and tortuous, the arteries are attenuated, and the fundus is covered with hemorrhages which are largest and most numerous around the disc. The disc itself is suffused with blood, and there is apt to be a small hemorrhage in the centre of the macula. In less marked cases the thrombosis may involve only a branch of the central vein, the disturbances then being confined to a limited portion of the retina. The intra-ocular tension is not increased. Ultimately the thrombus may break up, freeing the lumen, or organization may occur, producing permanent obstruction, though it would seem possible that even in the latter case canalization of the thrombus might take place so as to re-establish the circulation. Vision is not entirely destroyed for some time and may undergo marked temporary improvement, but relapses occur so that blindness is finally produced. If large extravasations of blood are poured into the vitreous body, as is sometimes the case, sight is early destroyed.

The *treatment* of hyperæmia of the retina must in every case be directed toward the cause, but the application of cold compresses to the eyes and the use of smoked glasses are often advisable. In thrombosis of the central vein treatment is of little avail, though strychnine has been advised, and potassium iodide and mercury may be given in the hope of hastening the absorption of the effused blood.

Anæmia of the Retina.—All degrees of this occur up to the complete cessation of the retinal circulation. Incomplete retinal anæmia may be either chronic or acute; in the former case it is usually dependent upon chronic general anæmia, either primary or secondary, and is not associated with any disturbance of vision, while in the latter it most often results from excessive loss of blood and frequently produces permanent blindness. It is rather remarkable that instead of anæmia, loss of blood may give rise to retinal hyperæmia and hemorrhages. Incomplete retinal anæmia is also an accompaniment and no doubt often the cause of retinal atrophy, and is constantly seen as the result of optic atrophy. The retinal changes seen in albuminuric retinitis are also in all probability dependent upon the anæmia resulting from sclerosis of the retinal vessels, and the impairment of vision in acute glaucoma is thought to be due to anæmia from pressure. Ophthalmoscopic examination in incomplete anæmia shows constricted arteries, dark veins, pallor of the disc, and sometimes arterial pulsation. As a matter of fact, however, unless the changes are quite marked, the condition is apt to be overlooked.

Ischæmia, or complete anæmia of the retina, is usually due to obstruction of the central artery, and may be the result of embolism, primary thrombosis, spasm of the muscle walls of the artery, hemorrhage into the optic sheath, direct injury to the artery within the nerve, or to pressure exerted upon the artery by a neoplasm. It was considered one of the earliest and most positive signs of death, but recent observations have shown that it cannot always be relied upon.

Embolism of the central artery is of very rare occurrence, probably more so than is generally believed, many cases diagnosed as such being due to some other cause. This view has recently been urged by H. H. Thompson, who states that the few anatomical examinations that have been made are unsatisfactory, and suggests that the majority of the cases of so-called embolism are due to spasm of the central artery. Embolism is said to be more common in men than in women and to be almost always unilateral, occurring more frequently in the left eye. The diagnosis of embolism is practically always made when in addition to ischæmia of the retina there is reason to suspect the presence of emboli in the circulation, as in cases of endocarditis. The embolus may be carried to a branch of the central artery, the anæmia then involving only a portion of the retina. Sometimes the macular region alone escapes in this way. If the embolus is infected, suppurative panophthalmitis results. *Thrombosis of the central artery* has been diagnosed in a few cases, but usually upon insufficient evidence. In one case, however, Haab has recently demonstrated the process of thrombosis by means of serial sections. *Hemorrhage into the sheath of the optic nerve* has never been demonstrated by an anatomical examination to be a primary cause of obstruction in the central artery, but it occurs as a result of trauma to the eye or from a hemorrhage at the base of the brain, forcing its way along the nerve. (Plate L., Fig. 2.)

Spasm of the Muscle Walls of the Central Artery.—In migraine attacks of temporary blindness not infrequently occur, and naturally enough have been attributed to spasm of the central artery, especially so since the inhalation of amyl nitrite gives such prompt relief. Wagenmann observed one of these attacks ophthalmoscopically, and saw the retina become markedly anæmic and then return to its normal condition within an interval of about ten minutes. Six months later a similar attack occurred in this case, and resulted in permanent blindness with the ophthalmoscopic picture ordinarily considered characteristic of embolism. Quinine in large doses may also cause constriction of the retinal arteries, which may be seen with the ophthalmoscope. There is great impairment of vision together with contraction of the visual field. In some cases there is complete blindness, and a cherry-red spot has even been seen in the macula. Central vision is ultimately recovered, sometimes, however, only after many months, but the peripheral field is apt to remain contracted. Some recent investigators have maintained that the impairment of vision in such cases is due to the direct action of the drug upon the ganglion cells of the retina, but the ophthalmoscopic findings certainly point strongly to spasm of the arteries as the primary factor.

In ischæmia of the retina, no matter what the cause of the shutting off of the blood supply, the changes produced are always much the same. The disc is pale; the arteries are so much reduced in calibre that they can be followed only for a short distance, and interrupted columns of blood may be seen in them. If the case is seen early, a to-and-fro motion of the blood may be observed, such as is seen in the vessels of a frog's web when the circulation is beginning to stop. The veins are also contracted, but to a much less degree, and pulsation may still be brought out in them by pressure on the globe. The retina soon becomes opaque, first near the vessels, the opacity being most marked around the macular region. But the most striking as well as the most characteristic feature of retinal ischæmia is a cherry-red spot about one-third the diameter of the disc, which makes its appearance in the centre of the macula. This spot is not invariably present, however. The explanation usually given for its occurrence, which was first offered by von Graefe, is that it is due to the dark and congested choroid showing through the thin fovea, the general white opacity of the rest of the retina, especially the surrounding macula, rendering it unduly conspicuous. A later theory is that the spot is due to a hemorrhage, and a recent observation leads the writer to believe that this view is correct. A short time ago, at the Massachusetts Charitable Eye and Ear Infirmary, there was enucleated and submitted to the writer for examination an old glaucomatous eye upon which an optico-ciliary neurectomy had been performed ten days previously, the optic nerve and with it the artery and vein being severed close to the globe. It will readily be seen that such an eye presented an exceptional opportunity for the anatomical study of retinal ischæmia, since eyes affected by embolism, etc., ordinarily offer no indication for enucleation, or at least not sufficiently early to be of much value from the standpoint of pathological anatomy. The eye had undergone no outward change as the result of the first operation, but it was removed on account of an unsightly squint. On macroscopic examination there presented itself the typical sharply defined red spot in the centre of the macula, and on microscopic examination this proved to be a hemorrhage. The extravasation of blood was limited to the macula, and there was no hemorrhagic infiltration of the

EXPLANATION OF
PLATE L.

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Fig. 1.—Hemorrhages into the Retina; Retinal Apoplexy. (From Noyes: "Diseases of the Eye," Wood's Library of Standard Authors, 1881.)

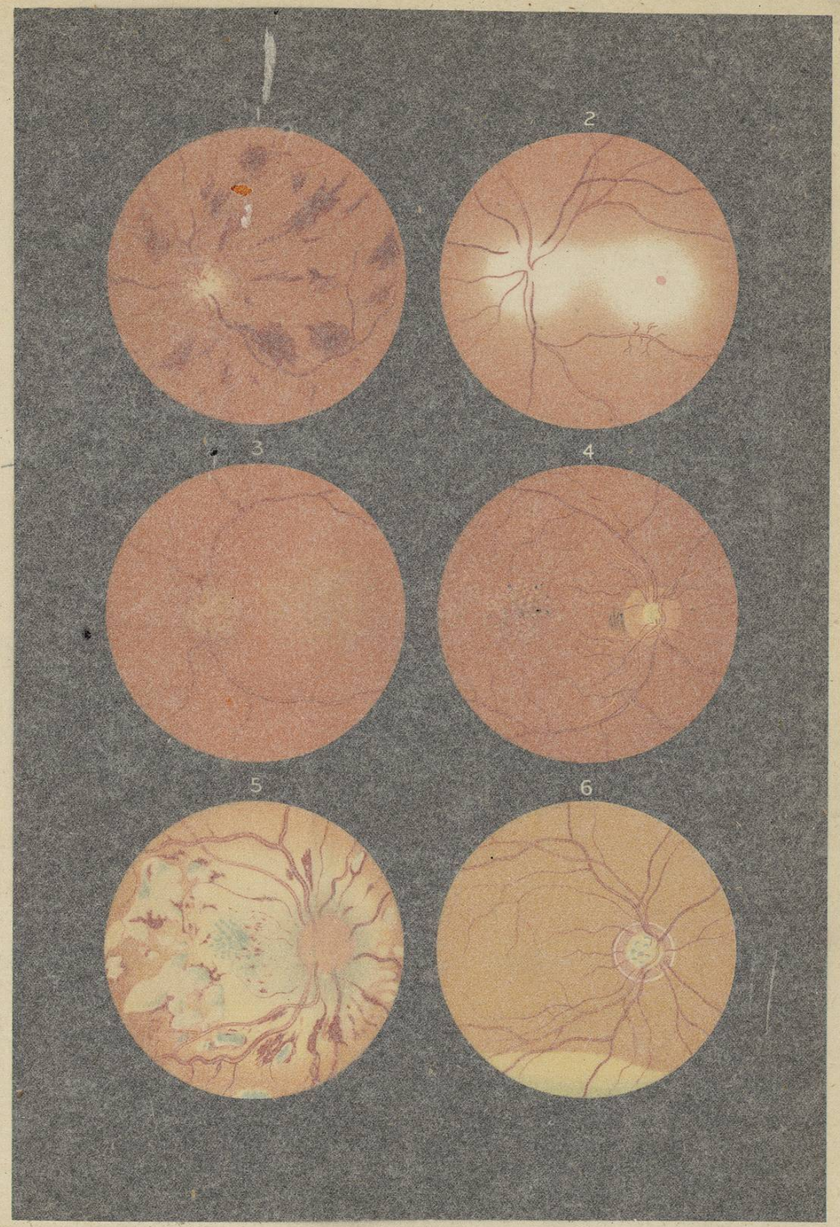
Fig. 2.—Ischæmia of the Retina (due to Embolism of the Central Artery?). (From Noyes: *Op. cit.*)

Fig. 3.—Serous Retinitis. (From Noyes: *Op. cit.*)

Fig. 4.—Albuminuric Retinitis. (From Noyes: "Diseases of the Eye," 1890.)

Fig. 5.—Albuminuric Retinitis at an Advanced Stage. (From Stellwag's "Diseases of the Eye," Translation of Hackley and Roosa, 1868.)

Fig. 6.—Separation of the Retina. (From Stellwag.)



Diseases of the Retina.

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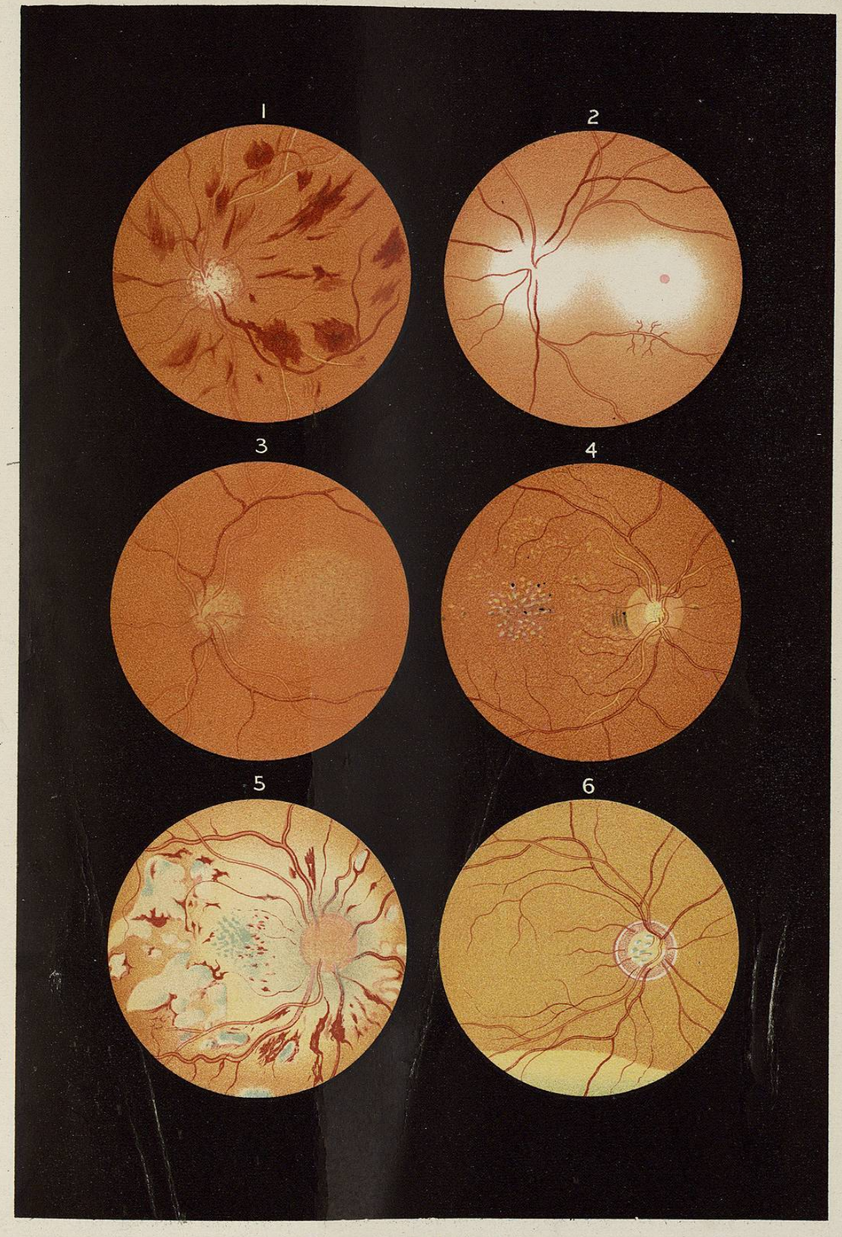

Diseases of the Retina.

choroid behind it, a fact which showed that the blood did not come from the choroidal vessels. The retina was almost completely necrotic, especially in its inner layers, and there were marked proliferation and migration of the cells of the pigment layer. In almost all of the retinal vessels the red blood corpuscles stained very feebly, but in the hemorrhage itself, and in a few of the vessels near the macula, the blood was well preserved, thus indicating that the source of the hemorrhage was the cilio-retinal vessels. The fact that the hemorrhage was comparatively fresh, and that there were no other retinal hemorrhages, went to show that it was not the result of the glaucoma but that it was dependent upon the cutting off of the circulation in the central artery.

In a number of cases of supposed embolism the circulation after a time has been seen to return. This has possibly been due to the breaking up of an embolus, or more likely to the establishment of a collateral circulation through the cilio-retinal vessels, but on the other hand it certainly supports the view that the cases in which it occurred were really due to spasm. Sometimes the direction of the circulation is reversed. The return of the circulation gives rise to numerous hemorrhages, most of them in the macular region, and no doubt due to the injury to the vessels produced by the cessation of the flow of blood. The final picture is that of atrophy of the retina and optic nerve.

As a result of ischæmia of the retina, vision is almost instantaneously lost. Occasionally a part of the visual field may remain intact for a while, but later it also becomes blind. It is said that in some instances the macula is sufficiently well nourished by the cilio-retinal vessels to prevent impairment of central vision. If the circulation returns quickly, as in migraine, vision is completely restored, but in total embolism it is almost always permanently destroyed.

Treatment of Retinal Anæmia.—In the simple variety of retinal anæmia dependent upon general anæmia, treatment appropriate to the latter must be adopted. If the anæmia is very marked, lowering the head at intervals during the day may be practised. The acute retinal anæmia resulting from excessive loss of blood should be combated by saline infusions and general supportive treatment. Inhalations of amyl nitrite are of great value in spasm of the central artery associated with migraine. In embolism, paracentesis of the cornea, iridectomy, and massage of the cornea with the purpose of dislodging the embolus, have been recommended.

Retinal Hemorrhages.—Aside from trauma, which is of course a frequent cause, hemorrhages into the retina may occur as the result of a general disease, less commonly as the result of disease confined to the eye alone, or they may occur occasionally in young people without any assignable cause. As already noted, hyperæmia of the retina frequently gives rise to them, especially the venous hyperæmia resulting from thrombosis of the central vein, optic neuritis, or neuroretinitis. When they are the predominant feature in the latter, the condition is usually designated by the term *hemorrhagic retinitis*. Venous hyperæmia due to pressure on the central vein at the disc is also probably the cause of the retinal hemorrhages which sometimes occur in primary glaucoma, and the intense hyperæmia resulting from suffocation is also likely to produce them. Their occurrence as an after-effect of embolism has already been referred to. The retinal hemorrhages that occur as the result of general disease are dependent either upon alterations in the retinal vessels, associated in most cases also with high arterial pressure, or upon changes in the character of the blood itself. Thus they are seen with comparative frequency in general arteriosclerosis, Bright's disease, gout, diabetes, anæmia, leukæmia, purpura, and scurvy. When such predisposing factors are present the immediate cause is often violent exertion, such as coughing or straining at stool. In arteriosclerosis they may appear quite suddenly in great numbers, just as in cerebral apoplexy, and in such cases the condition is, in fact, often spoken of as retinal apoplexy. Retinal hemorrhages also occur as the result of menstrual disturbances, sometimes as one of the manifestations of vicarious menstruation. They frequently occur in new-born infants as the result of excessive pressure on the head during delivery, but they are quickly absorbed without leaving any macroscopic changes. In this way no doubt many obscure cases of congenital amblyopia are produced. Among other general causes may be mentioned poisons, especially lead and phosphorus, jaundice, pregnancy and parturition, malarial fever, septicæmia, and pyæmia. (Plate L., Fig. 1.)

Retinal hemorrhages vary greatly in regard to size, shape, number, and position. When few in number they are usually situated not far from the disc or near the macula. They occur in any of the retinal layers that contain blood-vessels, but most frequently in the nerve-fibre layer where they assume a characteristic striated "flame-like" shape, due to the fact that the blood insinuates itself in between the nerve fibres. Owing to the radial arrangement of the fibres around the disc, hemorrhages in its neighborhood tend to take the form of red radii. The extravasations of blood may break through the retina into the vitreous humor producing vitreous opacities, or they may force their way between the retina and choroid. Sometimes the blood collects beneath the hyaloid membrane, forming the *subhyaloid hemorrhage*. This most frequently occurs in the macular region, appearing as a sharply defined dark red disc. When recent, retinal hemorrhages are bright red in color, but they soon become darker, often almost black. The blood is absorbed rapidly, especially when the hemorrhage is small, but white spots in many cases are left to mark their sites. The white spots are due to necrosis of the retinal tissue and may contain fat globules and cholesterin crystals. They may finally disappear or they may remain permanently, not infrequently becoming more or less pigmented, owing to the migration of cells of the pigment layer into them. It is said that the pigment striæ, known from their resemblance to obliterated vessels as *angioid streaks in the retina*, are due to metamorphosis of retinal hemorrhages. Extravasations that have broken into the vitreous humor but that still remain attached to the retina, sometimes undergo organization and become converted into connective tissue. It is in this way that *retinitis proliferans* is thought to arise.

The disturbance of vision produced by retinal hemorrhages obviously depends upon their size and position, and of course is particularly great when one of the hemorrhages occupies the macula. Sometimes there is metamorphopsia, less often photopsiæ. Even small hemorrhages may produce a temporary clouding of vision if they break through the retina into the vitreous humor. The prognosis is favorable when they are small and the tendency to relapses can be successfully overcome. It is particularly unfavorable when they occur in connection with a general retinitis. The subhyaloid hemorrhages which occur at the macula are usually absorbed and vision is completely restored. When the hemorrhages are dependent upon a general disease the treatment must necessarily be directed chiefly toward the latter, but rest in bed, the application of cool compresses to the eyes, and the administration of mercurial inunctions or potassium iodide to favor the absorption of the effused blood, are usually indicated. Leeching, purging, and the production of diaphoresis by pilocarpine are also advised.

Phlebectasia retinæ is a name given to a rare condition in which the retinal veins show dilatations and constrictions, sometimes producing a decided beaded appearance. In some cases it is probably due to vaso-motor disturbances. Schöbl describes a case in which the condition was also present in the conjunctival veins and was evidently dependent upon suppression of the menses, the phenomenon disappearing when the menstrual flow was re-established.

Retinal aneurisms are of very rare occurrence, but they have been seen in the living subject and also in enucle-