and dry skin—conditions found only in the early stages of acute febrile diseases in robust or plethoric patients—in the beginning of pneumonia, pleurisy, rheumatism, etc.; in the hypertrophic stage of cardiac disease it may be needed, but not often; in typhoid, septic, and other adynamic febrile conditions it should never be given, nor as an emetic.

In overdoses the vomiting, purging, and prostration are best combated by opium and stimulants, with a strictly enforced recumbent position.

ADMINISTRATION.—The dose of the substance itself is about a decigram (gr. iss.), repeated rather frequently and in increasing doses until the pulse is affected. During the administration the patient should be carefully watched. Once an hour is a good interval for the first few doses. The following good preparations are official: The fluid extract (*Extractum Veratri Viridis Fluidum*, U. S. P.), and a tincture (*Tinctura Veratri Viridis*, U. S. P.), strength 40 per cent., dose ♏ ij. to x.

W. P. Bolles.

HELLEBORE, BLACK.—HELLEBORUS NIGER. *Christmas Rose.* The dried rhizome of *Helleborus niger* L. (fam. *Ranunculaceæ*). This is a low perennial herb of Southern Europe, mostly in the mountains, and largely cultivated for its winter bloom.

The drug much resembles cimicifuga or black cohosh, but is rather smaller, blacker, and more tortuous, with fewer and broader wood-wedges in the rhizomes, and those of the roots less conspicuously stellate. It contains the two poisonous glucosides helleborin and helleborein, and has been used as a drastic. In overdoses it is an emetico-cathartic poison. It is now little used. The dose is 0.5 to 1.5 gm. (gr. viij.–xx.).

The rhizome of *H. viridis* L. or green hellebore (but not to be confused with *veratrum viride*, also so called), is very similar, and even more highly acrid and poisonous.

Henry H. Rusby.

HELLEBORE. (TOXICOLOGICAL.)—Green Hellebore (Veratrum viride), *American hellebore, Indian poke*, and White Hellebore (Veratrum album), the European species, owe their poisonous properties to several alkaloids which have been examined by a number of investigators. The results are not entirely accordant, and what is doubtless the same substance has received different names from the different writers. The researches of Wright and Luff and of Salzberger are of particular value. Wright and Luff found in veratrum viride, jervin, cevadin (crystalline veratrin), pseudojervin, with traces of rubijervin and veratralbin. Salzberger found in V. album, jervin, the pseudojervin and rubijervin of Wright and Luff, and two other bases, protoveratrin and protoveratridin, but no veratrin.

The official veratrin, obtained from sabadilla, and which melts at about 147° C., is a mixture of two isomeric alkaloids—the crystalline veratrin or cevadin of Wright, which melts at 205° C., and amorphous veratridin, melting at 143–148° C. Crystalline veratrin or cevadin crystallizes with difficulty, is readily soluble in alcohol, ether, and chloroform, and insoluble in cold water. It has a bitter, disagreeable taste and excites violent sneezing. It combines with acids to form salts, which are generally non-crystalline. The reactions for the official alkaloid and crystalline veratrin are substantially the same. Sulfuric acid gives a series of colors—yellow, orange red, carmine red. If heated, the latter appears at once. Hydrochloric acid produces no color in the cold, but on heating a fine red is obtained which is quite permanent. Sulfuric acid with sugar or with furfural gives a yellow, changing to an indistinct green, dark blue, and finally to violet. Nitric acid gives a transient rose pink; on evaporation and moistening with alcoholic potash a brown color is obtained.

Physiological Test: A minute quantity of veratrin injected hypodermically in a frog causes vomiting, slowing of heart beat, and contractions of the muscles, suggesting the tetanic convulsions of strychnine.

Jervin forms white crystals which melt at 238° C., and have a bitter, acrid taste. The alkaloid is non-sternutatory. It is readily soluble in alcohol and chloroform, less soluble in ether, and insoluble in cold water. Sulfuric acid gives a yellow, changing to greenish-yellow and greenish-brown with green at edges. On heating, a mahogany-brown appears at once. Sulfuric acid with sugar or with furfural gives a brownish color, changing to a deep blue and then to an indistinct violet. Hydrochloric acid gives no color in the cold; on heating, a yellow is obtained. Nitric acid, followed by alcoholic potash, gives the same reaction as does veratrin.

Experiments upon animals show that both hellebores first excite, then paralyze the sensory nerves of the skin and of the mucous membranes of the nose and alimentary tract. Both respiration and the action of the heart are first accelerated, then retarded, and finally paralyzed. The symptoms, which usually appear soon after taking, are burning in the mouth extending to the stomach, difficulty of swallowing, intense nausea, violent vomiting, and later purging, usually accompanied by tenesmus. Great prostration follows, headache and giddiness are frequently present, the pupils are slightly dilated, the pulse is feeble, respiration is difficult and sometimes fails entirely. Death may occur from heart failure or from exhaustion caused by vomiting.

Several homicidal cases have occurred in Europe, notably in France, but the only cases reported in this country are accidental. Thirty-six drops of the tincture of veratrum viride, taken in divided doses, caused the death of a child of one and one-half years. A teaspoonful of the tincture, seventy-five drops of Tilden's fluid extract, sixty drops of Norwood's tincture in divided doses, and an unknown quantity of the tincture, taken by mistake for whiskey, have caused the death of adults. Recovery has followed the taking of much larger quantities. On several occasions, a teaspoonful of the fluid extract (H. C. Wood) and a tumblerful of the tincture have been taken without fatal results. One-half ounce of powdered white hellebore has been taken with recovery. No death from veratrin is on record. The different preparations are evidently of greatly varying strength. One sixteenth of a grain nearly caused death, while recovery has occurred after taking three grains in a liniment and thirty grains of the crude alkaloid.

The treatment of a case of poisoning will vary with the nature of the symptoms. Vomiting, so commonly present, will have emptied the stomach; if it has not occurred, it should be induced until the object is reached, then restrained. Warm drinks and external warmth are used to raise the temperature and opiates to control pain. Alcohol and aromatic spirits of ammonia are useful in meeting the extreme prostration. The latter precipitates the insoluble alkaloids and thus retards absorption. Artificial respiration may be required.

Post-mortem examination discloses the intestinal tract usually congested and the viscera filled with blood, as occurs with many other poisons. There is an absence of characteristic appearances.

For the recovery of the poison, the contents of the stomach, or the tissues, after suitable comminution, are digested on the water bath with alcohol and acetic acid for some time, filtered, and the alcohol is removed by evaporation. The residue is extracted with water, filtered, and shaken with ether to remove fatty matters and extractives. It is then shaken with chloroform, rendered alkaline with sodium hydroxid, and after repeated shaking the chloroform layer is separated and evaporated. It is generally advisable to repeat this process to remove impurities, and on evaporation the veratrin will be found sufficiently pure for the application of the tests.

Brouardel isolated from a corpse of eighteen months, a ptomaïn which was extracted from alkaline solution with ether. It gave a violet color when heated with sulfuric acid and a cherry red on boiling with hydrochloric acid. This suggests the importance of confirming the chemical tests by the physiological test.

Curtis C. Howard.

HEMERALOPIA AND NYCTALOPIA, respectively, day-vision=*night-blindness*, and night-vision=*day blindness*, from ἡμέρα, day, νύξ, night, respectively, and ὤψ, eye, are names used by the older medical writers in opposite senses, to the great confusion of the literature of the subject. Following Hippocrates, Aristotle, and Galen, although in opposition to the usage of later Greek authors,[1] nyctalopia is a condition in which vision is comparatively good at night, or in a very feeble light, but is defective in strong daylight; and, conversely, hemeralopia (used, in contradistinction to nyctalopia, in a single passage in Galen[2]) is a condition in which vision is acute by daylight, but falls off disproportionately at night. If certain recorded observations are to be accepted as trustworthy, it would seem necessary to admit that hemeralopia may occur under two types, the one marked by a quasi-diurnal fluctuation in the perceptive power of the retina, the other directly dependent on changes in illumination. Thus, it has been stated that the acuteness of vision increases from early dawn to the middle of the day, and diminishes as the sun declines toward evening,[3] falling to its minimum as twilight deepens into night, or, according to some writers, not until midnight;[4] that the blindness is less marked in the early dawn than in twilight,[5] and that in aggravated cases the flame of a candle, the stars, and even the moon are either totally obscured or are seen as through a thick smoke or fog (Nachtnebel[6]). As a rule, however, luminous bodies are seen distinctly, while objects at a little distance from the lamp appear enveloped in deep gloom, so that the light of the full moon may be insufficient to enable the hemeralope to see his way.[7] By concentrating the light of a powerful lamp upon the book, the hemeralope may be able to read large print; in other cases reading by artificial light is impossible.[8] That the falling off in vision at night is closely related to the defective illumination is proved by the fact that it occurs in the daytime on entering a dark room, although with some persons, as it would seem, in a lesser degree than at night.[9] Experimental tests of the vision of hemeralopes, by varying degrees of illumination, have shown that it begins to fail under nearly the same conditions as with normal-seeing persons, but that on further diminishing the light the falling off in vision, as measured by the size of the test objects which can be discerned, is much more rapid.[10] The hemeralope requires also a relatively long time to attain his maximum of visual acuteness on going from full daylight into a darkened room;[11] on the other hand, some hemeralopes suffer from dazzling of the eyes on first going out into the sunlight.[12] The condition would appear, therefore, to be essentially one of dulled perceptive power (*torpor retinæ*), which may, in certain cases, be conjoined with some degree of retinal irritability. This view is, moreover, in accord with the seemingly well-attested fact that the same combination of causes may give rise to hemeralopia in certain persons, and to more or less distinctly marked nyctalopic symptoms in others.[13]

Hemeralopia is an almost constant symptom in certain affections of the retina, notably in retinal degeneration with stellate deposits of pigment (*retinitis pigmentosa*), in syphilitic retinitis, and in incipient detachment of the retina; and it is then associated with particular limitations of the visual field, characteristic of the special retinal disease, and very often also with marked falling off in the acuteness of vision in full daylight (see *Retina, Diseases of*). Rarely it is congenital, and it may then be an early symptom of pigmented retina, or perhaps of retinal degeneration in which the usually characteristic pigmentation may be absent.[14]

Idiopathic hemeralopia has been oftenest observed as an acute epidemic affection attacking large numbers of persons living under nearly identical abnormal conditions. De Sauvages mentions such an epidemic as having broken out among the soldiers in several garrison towns bordering on one of the smaller rivers in the south of France, not far from Montpellier.[15] Other extensive epidemics have been observed, occuring almost always in large bodies of men crowded together under unfavorable hygienic conditions conjoined with excessive exposure to the direct influence of strong sunlight. Thus soldiers in garrison, going habitually from crowded, and often very dark, quarters in casemates to drill for hours together on confined and unsheltered parade-grounds, seamen and marines on tropical stations, prisoners employed in stone-breaking or other outdoor work in courtyards enclosed by high whitewashed walls, also children in great public orphan-houses, have been especially subject to these visitations, while the officers, whose duties ordinarily involve much less exposure, and who are better nourished and lodged, also the inhabitants of garrison towns, have generally escaped. Hemeralopia has been described as endemic in certain localities in the East and West Indies, in Brazil, on rice plantations in China, in several provinces of France, in the countries bordering on the Mediterranean, in Podolia toward the end of winter, and also in midsummer among the harvesters of both sexes;[16] also in the Russian provinces bordering on the Baltic, at the period of the very strict Lenten fasts of the Greek Church.[17] An outbreak of hemeralopia, associated with cases described as nyctalopia, is reported by Carron du Villards as having been observed by his father, in 1793, in the Piedmontese army while encamped at a high elevation on the Mount Cenis and Little St. Bernard passes.[18]

From very early times both hemeralopia and nyctalopia have been attributed to "redundancy of humors in the system." Celsus repeats in connection with night-blindness[19] the observation made by Hippocrates regarding nyctalopia,[20] that it does not occur in women whose menses are regular. De Sauvages lays stress on the exposure incident to guard-mounting by day and night in a humid and nebulous atmosphere.[21] Demours particularly mentions exposure to the night air.[22] Hemeralopia is called moon-blindness by sailors, and is attributed by them to a morbific influence emanating from that planet, especially affecting such persons as commit the imprudence of sleeping on deck. In Brazil it has been described as endemic among the negroes;[23] it has also been said to affect especially persons with darkly pigmented eyes,[24] an observation which was made by Aristotle in connection with nyctalopia.[25] Stellwag observed numerous cases of night-blindness occurring in an asylum in Vienna, but almost exclusively in two pavilions which were exposed to the light on three sides,[26] and he cites this instance in support of the opinion that sleeping with the face turned toward the window may be an exciting cause of the affection. On shipboard it is often associated with scurvy, and the same connection was observed in the war in the Crimea.[27] In certain epidemics pregnant women have been especially affected.[28] As a rule, whether in epidemics of night-blindness or in the conditions prevailing in localities in which it has been observed as endemic at certain seasons of the year, also in most of the sporadic cases which have been reported, two principal factors are to be distinguished, namely, impaired nutrition and long-continued exposure to strong sunlight, often intensified by reflection from large bodies of water, snow, or sand; and it appears most rational to regard the former as a predisposing, and the latter as the chief exciting, cause of the affection. Simulation has doubtless often played a part in swelling the number of supposed cases in outbreaks which have occurred in garrisons, on ships, in workhouses, etc., and this probability must be considered in judging of some of the remarkably prompt cures which have followed very diverse plans of treatment.

Of the objective signs of hemeralopia, that upon which the most stress has been laid by military and naval surgeons is a considerable dilatation of the pupils, generally most conspicuous at night; also the sluggish response of the pupils to changes of illumination. Dryness of the scleral conjunctiva with formation of scaly patches, a dulled appearance of the corneal epithelium, even xerosis of the conjunctiva and cornea, also conjunctival hyperæmia with lachrymation and photophobia, a staring expression of the eyes, with the eyelids widely separated,

have all been noticed, especially in endemic hemeralopia; but so far as organic changes have been remarked, they would seem to be connected rather with concomitant disturbances of nutrition than directly with the affection of vision.[29]

The duration of the affection in different cases, or as stated by different observers, has been variously reported as limited to a single day or to a few days, or as extending to several weeks or months. As a rule, recovery speedily follows any change which works an amelioration of the two conditions of impaired nutrition and undue exposure to strong light. Relapses are apt to occur on renewed exposure to conditions similar to those which have given rise to the first attack.[30]

The treatment of night-blindness, as also of day-blindness—in both of which conditions the name nyctalopia has been used with little or no discrimination—was, until well into the last century, chiefly depletive and derivative. Bleeding from the arm or from the angle of the eye,[31] cupping, purgation[32] by elaterium,[33] scammony,[34] etc.; the administration of emetics,[35] diuretics, and diaphoretics;[36] the application of blisters and other counter-irritants behind the ears[37] or to the back of the neck; errhines and masticatories;[38] baths, frictions, exercise, and gargles;[39] in extreme cases trepanning the skull;[40] various applications to the conjunctiva, especially of an ointment made from the juices exuding from roasting liver; also steaming the eyes with the vapor of water in which liver is boiling, to which is added the recommendation to eat the liver;[41] these are the therapeutic measures recommended by the earlier and later Greek and Roman authors, by the Arabian writers on medicine, and by their followers down to quite recent times. Seclusion from bright sunlight and amelioration of diet must have contributed incidentally to the cure, but upon these points little or no stress was laid. In modern times a short confinement to a more or less perfectly darkened room,[42] attention to any concomitant pathological conditions, improvement of the nutrition, and, above all, the removal of the patient from the sphere of operation of the combined causes of the affection, have taken the place of the heroic plans of treatment formerly in vogue.

Nyctalopia, in the sense of seeing exceptionally clearly in comparative darkness, has been observed in the case of prisoners confined for a considerable period in dark dungeons;[43] and such persons are said also to suffer from dazzling of the sight for a longer or shorter time after being set at liberty, or, in some instances, to have continued permanently day-blind.[44] Physiological or pathological variations in the size and in the dilatability of the pupils may be relatively favorable to night-seeing or to day-seeing, according as the pupils are habitually larger or smaller than normal. In ametropia—whether myopia, hypermetropia, or astigmatism—the requisite conditions for distinct vision are generally best in a strong light, owing to the incidental contraction of the pupils and the consequent partial suppression of disturbing circles of confusion on the retinæ; and this may be also the case in turbidity of the vitreous or in diffuse clouding of the crystalline lens, in which conditions the penetrating power of strong light may be required for fairly good vision. On the other hand, in certain cases of circumscribed central opacity of the cornea or crystalline lens, vision may be comparatively good when the pupils are widely dilated, as at night, and very bad when they are strongly contracted, as in bright sunlight. Normally pigmented eyes, as a rule, best fulfil the conditions for clear and undazzled vision in strong sunlight, whereas the sight of persons with very light blue eyes, and notably of albinos, is generally best in moderate light. Retinal irritation following prolonged exposure to direct or reflected sunlight (snow-blindness), and especially reflex irritability of the retina dependent on irritation of the terminal ramifications of the fifth nerve in the cornea and iris, are marked by excessive and often painful contraction of the pupils under the influence of strong light, with consequent inability to see well in the daytime (photophobia). It is a curious fact that nyctalopia (day-blindness) is mentioned by medical authors several centuries before the definite recognition of hemeralopia (night-blindness), and it is still more remarkable that the earlier teachings regarding the etiology and treatment, and also the name, of the former came later to be applied to the latter affection. Excluding cases of photophobia and of central opacity of the cornea or crystalline lens, the conditions to which the name nyctalopia is in any degree applicable are reduced to certain retinal affections attended with irritability, and to the few imperfectly reported instances of day-blindness following long confinement in the dark. A few brief notices exist of day-blindness of an endemic or epidemic type,[45] but no recent instance of the kind has been reported.

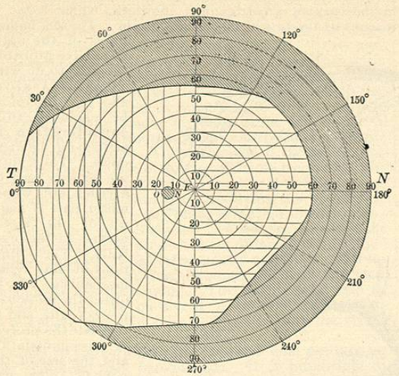
John Green.

[1] Aetius, Paulus Ægineta, Oribasius, et al.
[2] Galen: Εἰσαγωγή, 16.
[3] Stellwag: Die Ophthalmologie vom naturwissenschaftlichen Standpunkte, Buch iv., § 193, Freiburg im Breisgau, 1853.
[4] *Vide* Himly: Die Krankheiten und Missbildungen des menschlichen Auges, Band ii., S. 451, Berlin, 1843.
[5] *Vide* Stellwag: *Op. cit.*, iv., § 197.
[6] *Ibid.*, iv., § 193.
[7] *Vide* Leber: Graefe-Saemisch, Handbuch der gesammten Augenheilkunde, Band v., cap. viii., § 380, Leipzig, 1877.
[8] *Ibid.*, cf. Demours: Traité des Maladies des Yeux, t. i., p. 424, Paris, 1818.
[9] Stellwag: *Op. cit.*, iv., § 197.
[10] Förster: Ueber Hemeralopie, etc. C. Raymond: Giorn. d'Oft. ital., xii.; *ibid.*, xx.; Ann. di Ott., ii. (cited from Leber: *Op. cit.*).
[11] Förster: *Ibid.*
[12] Leber: *Op. cit.*
[13] Carron du Villards: Guide pratique pour l'Etude et le Traitement des Maladies des Yeux, t. ii., Paris, 1838.
[14] Leber: *Op. cit.*, § 89.
[15] De Sauvages: Nosologia Methodica, cl. vi., ord. i., iii., i., Amstelodami, 1768.
[16] *Vide* Himly: *Op. cit.*, ii., S. 453.
[17] Blessig, *vide* Leber: *Op. cit.*, §§ 384, 385.
[18] Carron du Villards: *Op. cit.*, t. ii., p. 487 sq.
[19] Celsus: De Medicina, vi., vi., 38.
[20] Hippocrates: Προρρητικόν, B′.
[21] De Sauvages: *Loc. cit.*
[22] Demours: *Op. cit.*, i., p. 425.
[23] *Vide* Leber: *Op. cit.*, § 385.
[24] Dubois (cited from Schmidt-Rimpler in Eulenburg's Real-Encyclopädie der gesammten Heilkunde, B. vi., Art. Hemeralopie).
[25] Aristotle: Περὶ ζῴων γενέσεως, E′., ed. Bekker, p. 170, 16, Berolini, 1831.
[26] Stellwag: *Op. cit.*, iv., § 198.
[27] Ophthalmic Hospital Reports, ii., pp. 35–43, London, July, 1859.
[28] *Vide* Mackenzie: A Practical Treatise on the Diseases of the Eye, xxiv., xii., fourth edition, London, 1854.
[29] *Ibid.* Cf. Leber: *Op. cit.*, § 386.
[30] *Vide* Himly: *Op. cit.*, ii., S. 452.
[31] Paulus Ægineta, iii., xxii.
[32] De Sauvages: *Loc. cit.*
[33] Hippocratic Treatise, Περὶ ὤψιος.
[34] Paulus Ægineta: *Loc. cit.*
[35] Demours: *Op. cit.*, p. 430.
[36] De Sauvages: *Loc. cit.*
[37] *Ibid.*
[38] Paulus Ægineta: *Loc. cit.*
[39] Celsus: *Loc. cit.*
[40] Hippocratic Treatise, Περὶ ὤψιος.
[41] *Ibid.* Celsus: *Loc. cit.* Paulus Ægineta: *Loc. cit.*, et al. Said to be even now a popular remedy in Italy and in other European countries, and still occasionally lauded by medical writers. Cod-liver oil has also been recommended as of especial therapeutic value.
[42] Netter (cited from Leber, *op. cit.*, § 386).
[43] Buffon, *vide* Himly: *Op. cit.*, ii., S. 449, note.
[44] *Vide* Larrey: Mémoires de Chirurgie Militaire et Campagnes.
[45] *Vide* Mackenzie: *Op. cit.*, xxiv., xiii.

HEMIANOPSIA is a condition of blindness limited to one-half of the visual field. *Hemiopia* is used as a synonym incorrectly, as it signifies vision (not blindness) in one-half of the visual field. This condition may be present in either the outer (temporal) or inner (nasal) half of the field of vision, and may affect one eye alone or both eyes. If both eyes are affected like-named halves of both may be involved—*e.g.*, the left half of both eyes, in which case the condition is called *homonymous hemianopsia*; or unlike-named halves of both eyes may be involved—*e.g.*, the left half of the left eye and the right half of the right eye, in which case the condition is known as *heteronymous hemianopsia*.

By means of Förster's perimeter the visual field of each eye has been determined (Fig. 2597). It is found to have an irregular outline, and to be divided into a large outer and a small inner portion by a line drawn vertically

through the point of fixation. This point corresponds with the macula lutea, and lies on the outer side of the entrance of the optic nerve. The two inner fields really overlap one another, so that the actual outline of the entire visual field of both eyes is that represented in Fig. 2598. It becomes evident from this figure that the loss of one nasal half of the visual field is not appreciated by any one unless the unaffected eye is closed. This fact is of importance, as it is evident that a nasal hemianopsia is a symptom which may not be noticed by a patient, and hence must be looked for by the physician. The unequal size of the two portions of each visual field is so marked that in an homonymous hemianopsia the patient usually ascribes the blindness wholly to a defect in the eye whose larger portion is affected, and a careful medical examination is necessary to establish the existence of a bilateral affection.



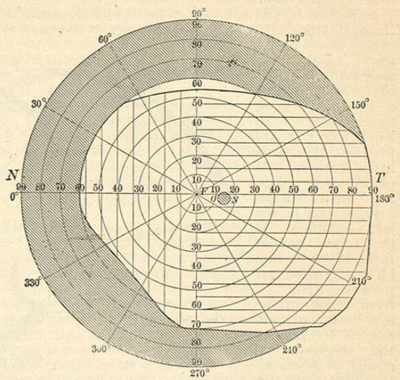


FIG. 2597.—Visual Field of Both Eyes, taken separately. The vertical lines represent the portion of the visual fields affected in left hemianopsia; the horizontal lines, in right hemianopsia.

To determine a patient's visual field, each eye is to be tested separately. The patient holds a card before one eye and looks the examiner in the eye with the other. Any object, preferably a white one, is then moved about in the visual field of the eye which is uncovered, and the power and extent of indirect vision is thus determined. If hemianopsia exists the patient will be unable to see the object when it is within the field of vision which is defective, or when it is carried beyond the vertical line passing through the fixation point toward the defective side. More accurate measurements may be made by means of a perimeter, but the method described is sufficient to establish the existence of blindness in one-half of the visual field. In no case of hemianopsia is direct vision at the fixation point affected.

Since the lens of the eye reverses the image of the object seen upon the retina, each half of the visual field corresponds to the opposite half of the retinal expansion. A hemianopsia therefore indicates a suspension of function in the half of the retina opposite to the defective visual field. Such a functional derangement of one-half of the retina is rendered possible by the origin, course, and distribution of the nerve fibres in the optic nerves, as seen in Fig. 2599. Each optic nerve is seen to pass from the eyeball back to the optic chiasm, and there to divide into two parts. One of these turns outward and joins the optic tract of the same side. The other crosses the median line in the chiasm, decussating with its fellow from the opposite optic nerve, and joins the optic tract of the opposite side.

The anatomical separation of the fibres from one optic nerve renders it possible for a lesion situated in the chiasm, or in the optic tract, to affect a portion of the fibres coming from one eye, and consequently a part of the retina; while a lesion affecting the optic nerve itself involves necessarily the entire extent of the retina, producing amaurosis, not hemianopsia. It is found that the portion of the retina lying to the outer side of the fixation point is joined with the fibres of the optic nerve which pass directly into the optic tract of the same side; while the inner portion of the retina sends its fibres across the median line in the decussation (Fig. 2599). It has been already shown that the nasal portion of the retinal expansion, corresponding to the temporal half of the visual field, is larger than the other portion. The number of fibres in the optic nerve which decussate is greater than the number which do not cross the median line. The ratio of non-decussating to decussating fibres in the optic

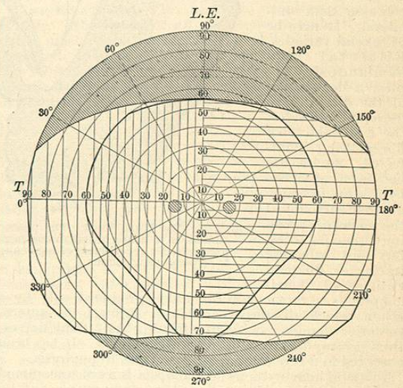


FIG. 2598.—Outline of the Entire Visual Field of Both Eyes.

nerve, in any animal, is directly proportionate to the extent of the visual field common to both eyes. Thus in the horse, or the rabbit, in whom there is no part of the visual field which is common to both eyes, the decussa-

tion of the optic fibres is total. In dogs, cats, monkeys, and man, in whom, to a less or greater degree, the eyes are directed in parallel lines, the decussation is partial. In a rabbit or horse hemianopsia is impossible. In the other animals named it is observed.

Unilateral hemianopsia is produced by lesions affecting the optic chiasm only. If the lesion lies on the outer side of the chiasm it produces nasal hemianopsia on the side of the lesion. Thus a tumor pressing upon point *N* in Fig. 2599 would produce blindness in the temporal half of the left retina, and hence nasal hemianopsia in the left visual field. If the tumor lies in front of or behind the chiasm, or presses upon it from above or below in such a manner as to involve the decussating strand of fibres either before or after they have crossed to the opposite side, it will cause a temporal hemianopsia (*T* in Fig. 2599). As it is impossible to determine whether the fibres are affected before or after decussation, no statement as to the side upon which the lesion lies can be made.

Bilateral hemianopsia is far more common than unilateral. It may be of several varieties. Bilateral nasal hemianopsia implies a destruction of the direct optic fibres on both sides, the decussating fibres being unaffected. Such a case has been observed by Knapp, in which a tumor surrounded the chiasm, pressing upon its sides, but not affecting its centre. Bilateral temporal hemianopsia is a rare condition, and is produced only by a lesion which divides the chiasm through its antero-posterior diameter without affecting its lateral halves, thus destroying both decussating strands. It has been observed in a few cases of tumor of the hypophysis.

Bilateral homonymous hemianopsia is a not uncommon condition, and may be produced by many causes, and by lesions situated in many various parts. This will be better understood after the course of the optic tracts has been followed to their terminations in the visual area of the cerebral cortex (Fig. 2599), since a lesion at any point in this course will produce the symptom named. Each optic tract, after leaving the chiasm, passes around the crus cerebri, lying directly upon the fibres which pass through the foot of the crus (pes pedunculi), and ends on the level of the tegmentum of the crus in the external geniculate body, in the pulvinar of the optic thalamus (*i.e.*, the eminence forming its posterior surface), and in the corpora quadrigemina anteriora, which latter it reaches by the brachium conjunctivum. The last-named fibres of the optic tract have probably nothing to do with conscious vision, and may therefore be excluded from consideration in studying hemianopsia. They form the sensory part of a reflex arc, whose motor part is made up of the motor nerves to the eyeballs. The functions of this reflex mechanism are to direct the motion of the eyeballs, and to regulate the process of accommodation and the size of the pupil. The primary visual centres are, therefore, the external geniculate body and optic thalamus. The fibres of the optic tract end in the cells of these ganglia, and from these cells new fibres arise which collect in a large tract and issue from the posterior external angle of the optic thalamus into the posterior third of the internal capsule. This visual tract turns upward and backward in the internal capsule, radiates into the centrum semiovale of the occipital lobe, and, passing around the outer border of the posterior horn of the lateral ventricle, terminates in the convolutions of the occipital cortex, including the cuneus. At no point in this course is any decussation found. The only decussation of fibres between the eye and the cortex is in the optic chiasm.[1]

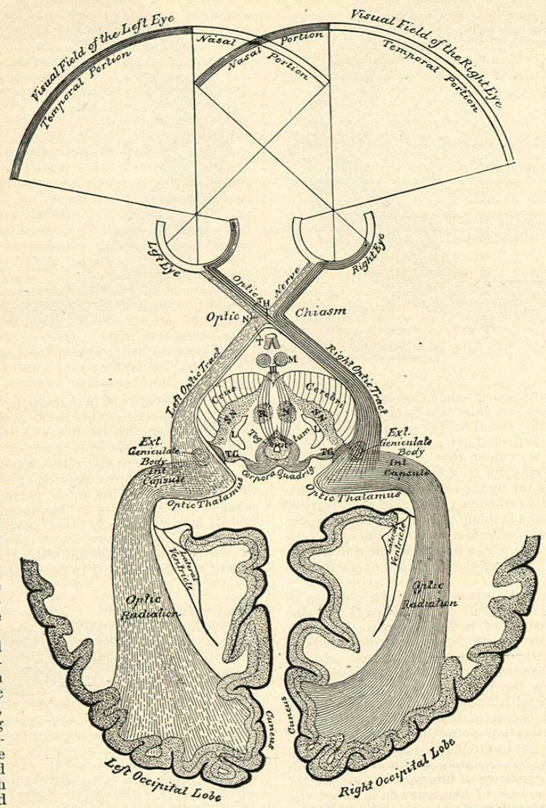


FIG. 2599.—The Visual Tract. The result of a lesion anywhere between the chiasm and cuneus is to cause homonymous hemianopsia. III. Third nerve; S. N., substantia nigra; R. N., red nucleus of tegmentum; L., lemniscus; T. G., fibres from optic tract to corpora quadrigemina.

Bilateral homonymous hemianopsia may be caused by a destructive lesion lying anywhere in the course of these fibres between the optic chiasm and the occipital cortex, or by a lesion in the cortex which destroys the perceptive centres in which the fibres end. Wherever the lesion, the character of the symptom will be the same. From the accompanying symptoms, it is in some cases possible to locate the lesion causing the hemianopsia. Thus, if the optic tract is involved as it curves around the crus cerebri, the same lesion which causes the hemianopsia will be likely to affect the motor tract in its passage through the pes, or to involve the third nerve in its exit from the pes. In this case hemianopsia and hemiplegia of one side, with oculo-motor paralysis of the other side, will be combined. Or, if the termination of the optic tract in the geniculate body and thalamus is involved the lesion, unless extremely limited, will affect the sensory tract from the opposite side of the body in its passage through the tegmentum of the crus, or in the posterior portion of the internal capsule; and in this case hemianæsthesia and hemiataxia will be associated with hemianopsia. Or, if the visual tract in its passage through the internal capsule is involved by disease in the basal ganglia or in the capsule, the proximity of both sensory and motor tracts in the capsule will render a combination of hemianopsia, hemianæsthesia, and hemiplegia quite probable. This is the seat of the lesion, and this is the combination of symptoms most frequently observed (see *Brain Diseases: Diagnosis of Local Lesions*).

Lesions in the centrum ovale involving the optic radiation may not produce any other symptom than that under discussion. If it is the left hemisphere, however, in which the disease is present, a condition of word-blindness (see *Aphasia*) is not infrequently associated with it; and in all the cases of word-blindness hitherto reported hemianopsia has been found. This is probably due to the destruction of association fibres between the occipital and temporal lobes which lie side by side with the visual tract.

Lesions in the cortex of the occipital lobe produce no other symptom than hemianopsia. It is impossible to limit the visual area of the brain to any one part of the lobe, since destruction of a portion of the convexity has produced the same result as destruction of its median surface.[2] Nor is it possible to project upon the cortex the retinal expansion, as has been attempted by Munk, since lesions in all parts of the occipital convolutions produce the same effect.

Lesions in the angular gyrus may produce hemianopsia, and it is well known that Ferrier has located in this convolution the visual centres. It is probable that such lesions have caused the symptom by affecting the visual tract as it passes beneath this gyrus in the centrum ovale. There is no reliable evidence that a unilateral lesion in the cortex can produce blindness of one eye alone.

Hemianopsia is, therefore, a local symptom of brain disease of great value in determining the situation of a lesion, when considered in connection with other symptoms. The symptom alone affords little evidence of the nature of the disease producing it, since it may be caused by any of the various forms of brain lesion (see *Brain*). It has been observed in cases of basilar meningitis, in tumors of the occipital lobe and basal ganglia, in hemorrhage and softening involving the internal capsule, and in embolism of the terminal branches of the posterior cerebral artery, and of the trunk and posterior branch of the middle cerebral artery, as well as in other rarer conditions.

The diagnosis of the symptom may be made by an examination such as has been described. The prognosis and treatment of it will depend entirely upon the nature of the disease producing it. *M. Allen Starr.*

[1] Von Monakow: Arch. f. Psychiatrie, xiv., 698–750; xvi., 151–200. Wernicke: Lehrbuch der Gehirnkrankheiten, Bd. I., S. 79–84.

[2] Compare cases of Haab with those of Fritsch and Westphal cited by Starr: Visual Area of the Brain, American Journal of the Medical Sciences, January, 1884; and these with Seguin's case, Journal of Mental and Nervous Disease, January, 1886. Full bibliographies are to be found in these articles.

HEMICRANIN is a mixture of phenacetin five parts, and one part each of caffeine and citric acid.
W. A. Bastedo.

HEMIDESMUS.—Hemidesmus Radix. *Indian Sarsaparilla.* "The dried root of *Hemidesmus indicus* R. Br." (B. P.) (Fam. *Asclepiadaceæ*). The plant is a slender, twining shrub, native of India. The description in the British Pharmacopœia, into which it is introduced apparently out of compliment to the Indian physicians, is as follows: "The root is long, rigid, nearly cylindrical, tortuous and longitudinally furrowed. It seldom exceeds ¼ of an inch (6 mm.) in thickness and is of a reddish-brown or dark-brown color. On one side of the root the cork is frequently separated from and raised above the cortex, and is transversely fissured. The transverse section exhibits numerous laticiferous cells in the cortex. The root has a fragrant odor and a somewhat sweet taste." A syrup, made from one ounce of hemidesmus to ten and one-half ounces of menstruum, is used in England.

The medicinal properties of hemidesmus are said to be those of sarsaparilla, in the stead of which it is used in British India; in Europe or in this country it is rarely used. Its composition has not been fairly studied, but it is safe to say that nothing physiologically very peculiar or active is contained in it. An odorous principle, perhaps cumarin, has been partially examined. The syrup in which it is prepared, like that of sarsaparilla, is scarcely more than a flavoring vehicle. *Henry H. Rusby.*

HEMIPLEGIA (ἡμισυς, "the half," and πλήσσω, "I strike"). Paralysis of one-half of the body.

Causes.—Hemiplegia, as ordinarily seen, is usually the final result of an apoplectic stroke, from cerebral hemorrhage, embolism, or thrombosis. The stroke is often attended by loss of consciousness and profound paralysis, but if the patient live the paralysis gradually lessens and leaves the final hemiplegic state. In some instances the paralysis is not ushered in in such stormy manner, but increases gradually for a day or two, either with or without loss of consciousness. The early symptoms are the expression of shock to a larger or smaller part of the brain, of pressure, and, sometimes, of inflammation of the tissues adjacent to the lesion, as well as of destruction of brain substance. The final symptoms are the expression of destruction and degeneration of brain tissue.

Hemiplegia may also come on slowly, caused by a brain tumor, brain abscess, chronic softening, meningitis, etc. In these instances the clinical picture may be complicated by a sudden paralysis from hemorrhage or thrombosis in the diseased area, or the like.

What has already been spoken of is organic hemiplegia, due to destruction or injury of the motor centres, or the cortico-muscular tract, that is, the tract of fibres carrying impulses from the motor centres to the muscles. But hemiplegia also occurs without palpable lesion. Notably this is true of hysterical hemiplegia.

Manifestations.—The paralytic manifestations are variable. Shortly after an apoplectic attack, if it be severe, most of the muscles on one side of the body are paralyzed. The arm and leg are entirely powerless. The muscles supplied by the lower branches of the seventh nerve, those of the cheek and mouth, are completely paralyzed. That side of the face is expressionless, and the mouth may be drawn toward the other side. But the muscles supplied by the upper branches of the seventh nerve—the orbicularis palpebrarum, occipito-frontalis, and corrugator supercilii—are as a rule but little affected. The tongue may remain motionless in the floor of the mouth. When protruded it deflects to the paralzyed side. The muscles of the chest are usually somewhat affected, and the breathing is less deep on the paralyzed side. The writer has found the latter symptom an aid to diagnosis of both the presence and the side of hemiplegia in cases in which the coma was so profound that it was impossible to elicit any sign of power on either side. On the other hand, some muscles almost invariably escape injury. These are the muscles supplied by the third, fourth, fifth, and sixth nerves—those of the eyeballs and of mastication—and the muscles concerned in swallowing and vocalization. The articulation is usually somewhat indistinct for a short period, or there is a loss of speech—aphasia. Aphasia occurs mostly with right hemiplegia (in right-handed individuals). But even indistinctness of speech, when there is no aphasia, is often more marked in right than in left hemiplegia.

The high degree of paralysis just described, though in