Episodic Disorders of Vision

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Introduction.

Of all our senses, vision is most commonly associated with patient distress, if not overt alarm, when abruptly compromised.

Despite patient concern, a rare, or rarely recognizable, clinical entity is perhaps of less significance to the physician than to the patient. When the broad spectrum of episodic disturbance of vision is closely examined, it becomes apparent that these visual symptoms are indeed of common occurrence in the population at large.

Surprisingly, despite their ultimate effect on the eye, a majority of the clinical entities that produce episodic disturbance of vision are neurologic (Fig 1).

Episodic is defined as being ". . . made up of separate, loosely connected episodes."* In turn, an episode is ". . . a usually brief unit of action . . . an occurrence or connected series of occurrences and developments which may be viewed as distinctive and apart, although part of a larger or more comprehensive series."*

In binocular man, episodic visual change has two essential parameters of basic diagnostic significance: Time and laterality.

In terms of time, an episode has both duration and frequency. Although both characteristics are of diagnostic value, duration is the one most frequently used in discussing disorders of vision. Episodes may vary in duration from seconds to as long as months (Fig 1).

A no less important element is the laterality of the visual event. Laterality is obviously restricted to right or left, a fact, however, which does not detract from its localizing value. Apart from laterality, yet closely related, is a further obvious fact: The eyes may be episodically involved independently or simultaneously. This too has great localizing value.

The entities that produce episodic visual disturbance will be considered in sequence of timing, beginning with those of brief duration and progressing to those of prolonged duration. Within each specific time unit the importance of laterality will be considered.

Visual Disturbances Lasting Seconds.

Obscurations. An obscuration is a brief alteration of vision. It may occur monocularly, but generally it is binocular. It usually lasts from 5 to 10 seconds, but it may continue for up to 30 seconds (Fig 2). Obscurations are usually frequent, occurring numerous times weekly if not daily. They may con-

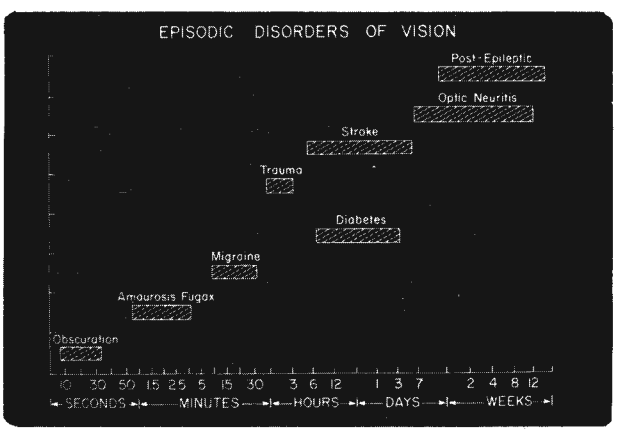

Fig 1—Graphic display of the time durations of all episodic disorders of vision.

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* Webster's *Third New International Dictionary*. Springfield, G & C Merriam Co, 1971.

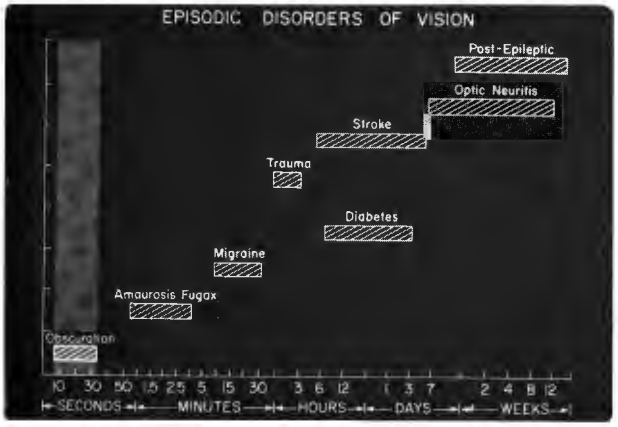

Fig 2—Graphic display of the time duration range of obscurations.

tinue over extended periods, their ultimate duration depending upon the course of the inciting illness. They seem to be infrequently brought on by posture or activity and are classically unpredictable.

A variety of terms are used in patient descriptions of obscurations. A popular description is that of ". . . a graying out of vision."[1] They are also called blackouts, fade outs, and simply blurring. They have been compared to fog, mist, and opaque curtains.

Obscurations are usually isolated events, unassociated with other neurologic or visual symptoms. Because of their very brief durations, they are less alarming than other episodic disturbances of vision.

Two causes of obscurations are presently recognized: Papilledema and vertebro-basilar insufficiency.[1,2] The differentiation of these two sources is simple and rests almost entirely upon the presence or absence of papilledema.

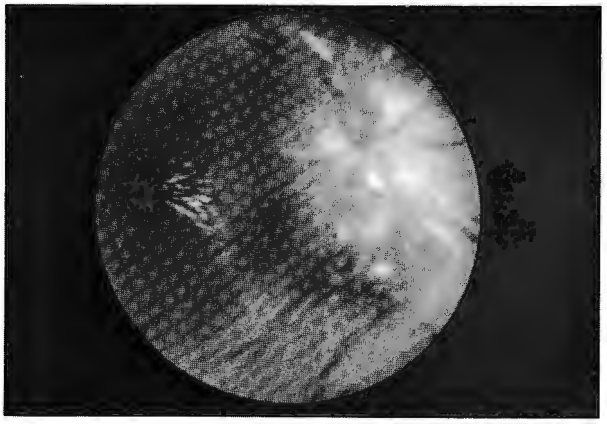

Fig 3—Example of severe papilledema resulting in obscurations.

When obscurations result from papilledema, the optic disc changes are nearly always severe (Fig 3). The obscurations of papilledema may be monocular or binocular. Their origin is presumed to be ischemia of the optic disc, secondary to the increased intracranial pressure. It is common to find additional signs and symptoms of increased intracranial pressure in patients presenting with obscurations of papilledema. These include headache, nausea and vomiting, and often diplopia. The incidence of focal neurologic signs is much less common and when present ominously suggests a mass lesion. Most patients in this group are young, reflecting the possibly single most frequent etiology of the increased intracranial pressure—pseudotumor cerebri.

Patients presenting with obscurations as a manifestation of vertebro-basilar insufficiency are, not surprisingly, much older. Here, obscurations are always bilateral and imply bilateral ischemia of the occipital cortex. Obscurations may be the only manifestation of transient ischemia in the vertebro-basilar arterial distribution, or they may be one of many. Other manifestations include dizziness, diplopia, facial paresthesias, dysarthria, weakness, and ataxia. These symptoms most commonly occur independently. Although they may presage a stroke in the vertebro-basilar arterial distribution, this relationship is unpredictable. Patients with obscurations of vertebro-basilar origin never have papilledema.

Obscurations per se are merely of diagnostic value and do not require treatment. Although papilledema may progress to blindness and optic atrophy the obscurations themselves do not reflect what the ultimate visual status will be.

Treatment should be directed at the etiology of the obscurations. In those associated with papilledema, a mass lesion must be ruled out or pseudotumor cerebri confirmed. Appropriate and specific therapy may then be initiated. If the obscuration is of vertebro-basilar origin, one must frequently evaluate the propriety of anticoagulation. In both circumstances appropriate therapy usually results in symptomatic resolution.

Visual Disturbances Lasting Minutes.

In differentiating the causes of episodic disturbances of vision lasting for minutes, discrete duration and laterality are of paramount importance.

Amaurosis Fugax. When the transient loss of vision occurs monocularly it is popularly referred to as amaurosis fugax—fleeting blindness.[3-8] This clini-

cal event is characteristically of abrupt onset; it seldom evolves in a progressive fashion. Its duration may be variable, lasting from one to several minutes, but the mode seems to be from one to three minutes (Fig 4). It is often difficult to establish the time sequence as concretely as one would like, since patients frequently lose the perspective and scale of time. The degree of visual loss varies from complete to only moderate blurring, and altitudinal visual loss is not infrequent. A sensation of color may accompany the visual loss, but photopsia or scintillations are rare. The resolution of the visual disturbance is generally as abrupt as its onset and usually complete without residual loss of acuity or field.

The episodes may recur many times per day or be separated by days, weeks, or months. They typically occur in isolation without associated neurologic symptoms, although additional transient signs of internal carotid ischemia such as hemiparesis, sensory change, and aphasia may occur independently.

The overwhelming majority of patients presenting with amaurosis fugax will harbor extracranial internal carotid artery disease of atherosclerotic origin with precise localization to the common carotid bifurcation (Fig 5). Although amaurosis fugax may be "the hallmark of carotid insufficiency," a wide variety of other sources exists. These include increased intraocular pressure; that is, glaucoma, arteritis of the branches of the ophthalmic artery; giant cell or temporal arteritis, alteration of numerous elements of the blood, red cells and proteins particularly, and thromboembolic events of cardiac origin such as mural thrombi, valvular vegetation of infectious or noninfectious origin, and tumor emboli.

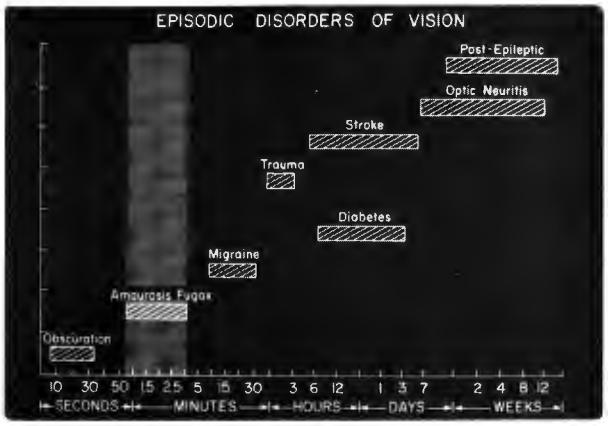

Fig 4—Graphic display of the time duration range of amaurosis fugax.

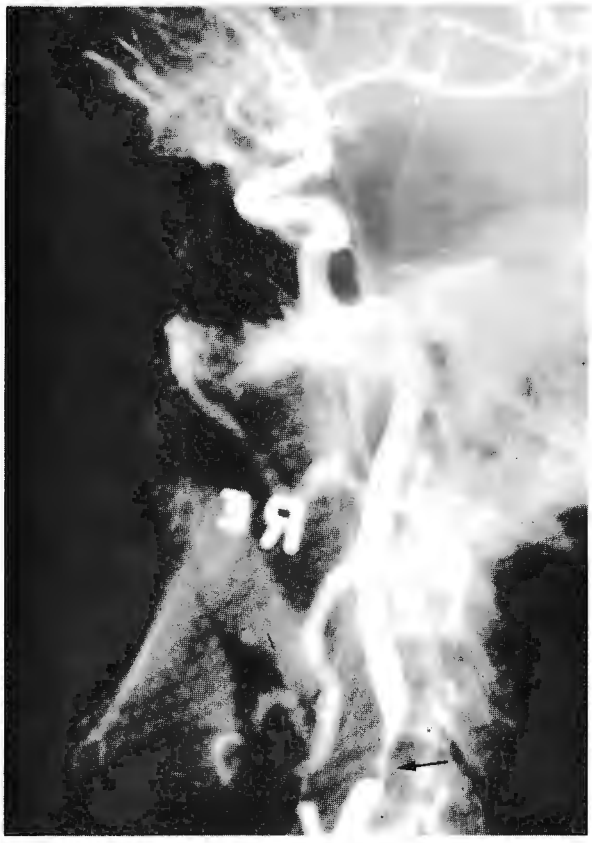

Fig 5—Lateral angiographic demonstration of extracranial internal carotid atherosclerotic disease (*arrow*) productive of amaurosis fugax.

As by far the most common etiology of amaurosis fugax is atherosclerotic disease of the carotid bifurcation, it is appropriate to direct diagnostic consideration to this focus.

The presence of a focal bruit over the carotid bifurcation at the angle of the jaw has been found to be strong evidence of disease. A bruit, however, may not be present even with significant disease and occasionally it may be falsely localizing with the significant changes occurring in the contralateral carotid. Asymptomatic bruits, although evidence of some degree of disease, should rarely be pursued diagnostically.

Changes in the carotid pulsation in the neck are seldom helpful. Often the external carotid overlies the internal carotid and masks changes in the latter vessel's pulsation.

Single or multiple bright refractile cholesterol emboli found in the ipsilateral retinal arterioles are

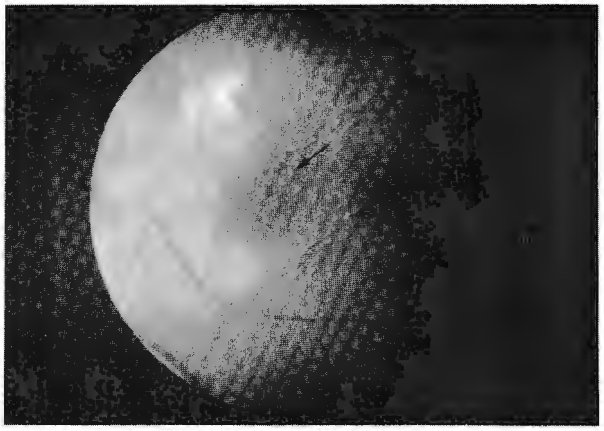

Fig 6—Fundus photograph of cholesterol emboli (*arrows*).

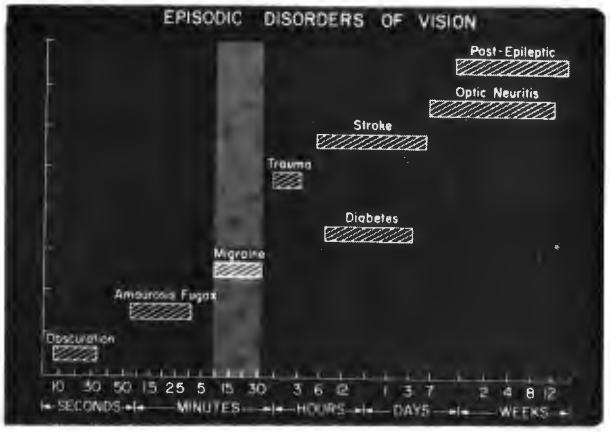

Fig 8—Graphic display of the time duration range of migraine.

strongly indicative of a carotid origin for amaurosis fugax.[9,10] Less apparent whitish platelet plugs may also be seen. These emboli often lodge in bifurcations of vessels distal from the optic disc, thus requiring pupillary dilatation and careful funduscopic examination for identification (Fig 6).

The use of ophthalmodynamometry, Doppler flow studies, and thermography provide additional pieces of incriminating evidence. It remains, however, for carotid angiography to confirm the presence, extent, and significance of bifurcation atherosclerotic changes (See Fig 5).

The identification of the other less common causes of amaurosis fugax is seldom difficult if their possibility is recognized.

Treatment should be directed at the cause of the amaurosis fugax. A significant incidence of stroke in the internal carotid artery distribution including the retina follows premonitory amaurosis fugax (Fig 7). In carotid bifurcation atherosclerotic disease we at the Medical College of Virginia have favored endarterectomy when appropriate. The primary obstacle to endarterectomy has been significant symptomatic cardiac disease. In this situation, anticoagulation may be advisable.

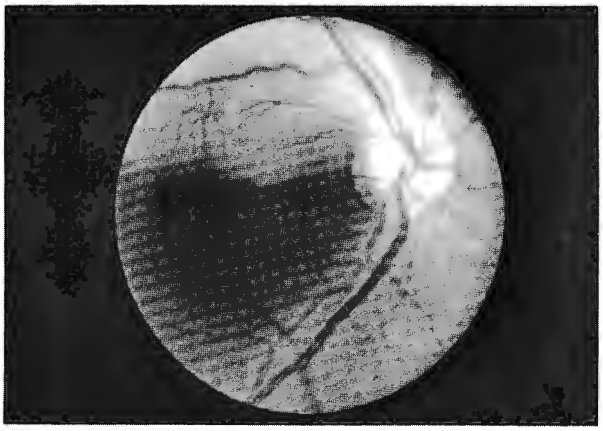

Fig 7—Fundus photograph of cloudy retinal edema (*arrow*) secondary to a central retinal artery branch occlusion with infarction.

Migraine. Typical amaurosis fugax which consistently lasts from 10 to 30 minutes should suggest the likelihood of a migrainous origin (Fig 8).

The recognition of visual phenomena as the forerunner of migraine dates to antiquity[11]; the variety of these visual hallucinations is remarkable.[12,13] It is also clear that the visual aura may constitute the sole manifestation of a migraine attack.[14] If indeed the visual aura is never followed by the hemicranial, vascular headache, nausea, and diffuse autonomic disturbance, one may have difficulty in recognizing its migrainous character. That this specific circumstance mimics amaurosis fugax seems well established.[14] Certain features of the clinical presentation are useful in identifying these admittedly uncommon cases of migraine.

Typically, the event recurs in an identical fashion over many years. It commonly begins under the age of 20. The expected hereditary aspect of migraine is frequently absent and the relationship of the visual event to the headache may become so inconstant as to appear to have no connection—so-called dissociated migraine. In some cases, headache may never occur. The most important feature, however, remains the duration. Amaurosis fugax as a manifestation of complicated migraine almost always lasts

between 10 and 30 minutes. It is comm
presentation to resolve as the patient ac
the 20's and 30's and be replaced by r
migraine.

Vertebro-basilar Insufficiency. Whe
loss of vision of minutes in duration occu
the source most commonly is in the vert
arterial distribution.[2] Although amauro
internal carotid origin conceivably might
erally and synchronously, this must ind
Obscurations, the momentary visual loss
onds due to vertebro-basilar insufficienc
discussed above. More commonly, ho
sient visual loss secondary to ischemia
tebro-basilar distribution lasts one to fi
With the increase in duration of the vi
ment, patient concern is, understanda
cantly increased. This in itself may acc
greater frequency with which these visu
reach the physician's attention. The e
visual loss is variable from a simple per
of central vision to total blackout.

In common with patients presenting
rations of vertebro-basilar origin, this p
tends to be elderly. The association o
symptoms of brain stem origin occurring
pendently or synchronously is to be anti
would again expect historical evidence
dizziness, vertigo, diplopia, dysarthria, a
and bilateral extremities paresthesias as w
ness.

The implied risk of stroke in the vert
arterial distribution in this patient group
those manifested simply by obscuratio
eration of anticoagulation is appropriat
few highly selected patients presenting a
history of symptom precipitation by nec
or position and confirmed by angiogr
surgery directed at decompression of c
vertebral arteries in the cervical spine ma
ered (Fig 9). This patient population is t
nificantly younger than the average pati
tebro-basilar insufficiency, and frequently
trauma is clearly evident.

Basilar Artery Migraine. In a mann
amaurosis fugax, when transient bilater
pairment occurs with a duration of fr
minutes, a migrainous etiology becomes
(See Fig 8).

Basilar artery migraine has been wel
and adequately described for some tim

ig 9—Subtracted lateral view of a vertebral angiogram demonrating arterial compromise (*arrow*) by osteoarthritis in the cercal spine, which resulted in visual loss.

ıarked tendency to occur in a recognizable pattern. t is most common in adolescent girls. Visual symptoms are the most common initial event with the isual disturbance varying from total loss of vision to lurring of central vision. Positive visual manifestaons characteristic of migraine may be totally absent. ıssociated vertigo, ataxia, dysarthria, and sensory aresthesias are common. Characteristically, a severe hrobbing occipital headache often accompanied by ausea and vomiting follows. As with the migrainous maurosis fugax, however, headache may not occur.

Generally the clinical pattern is such that doubts f its migrainous origin do not arise. Once again here is a tendency for these attacks to be replaced by ıore classic migraine as the years pass.

Visual Disturbance Lasting Hours.

Episodic disturbance of vision lasting hours eems to be less often recognized if not actually less

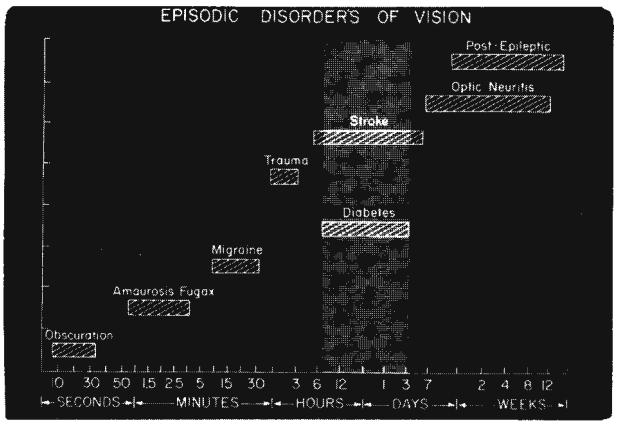

Fig 10—Graphic display of the time duration range of episodic visual loss secondary to uncontrolled diabetes mellitus.

common. Some of the sources of visual symptoms measured in hours are extensions of previously discussed entities. Some, however, are entirely new and distinct.

Diabetes Mellitus. The incidence of episodic visual disturbance lasting hours in diabetes mellitus is unclear (Fig 10). It is quite likely that early diagnosis and improved drug control has significantly reduced the magnitude of these visual symptoms if not their incidence.

Sudden bilateral blurring of vision is well known to occur in patients with diabetes mellitus.[16,17] It has on occasion been the initial symptom leading directly to the diagnosis in a previously unrecognized case. More commonly it occurs in established diabetic patients; its basis is known to be abrupt refractive changes in the eye of either myopic or hypermetropic nature. The refractive change seems rather clearly related to blood sugar levels; myopia occurring in the presence of hyperglycemia and hypermetropia with hypoglycemia. It may, in fact, be useful in alerting the physician to ineffective diabetic control.

The visual disturbance is described by the patient as blurring with impairment of central vision. In all circumstances it can be corrected by appropriate refraction. At the bedside the use of a pinhole may prove the refractive nature of the visual change to the satisfaction of the physician.

The precise mechanism by which the blood sugar level effects the abrupt refractive change is not entirely clear. It is postulated, however, to be the result of secondary changes of lens hydration.

The course of visual blurring in poorly controlled diabetics is transitory, lasting variable periods, usually measured in hours to a few days. It is almost invariably reversible. Occasionally cataracts may develop abruptly in this group of patients.[17]

Therapy of this episodic visual disturbance should be directed at more effective diabetic control rather than refractive correction by virtue of its transitory nature.

Post-traumatic Blindness. A second transient visual disturbance typically lasting hours which although probably uncommon is often unrecognized is post-traumatic or concussion blindness (Fig 11).

This transient bilateral visual loss seems clearly of occipital cortical origin. It is also quite certainly causally related to preceding head trauma. Children appear to be much more susceptible to post-traumatic blindness than adults.[18,19(pp2384–2385)] The degree of trauma necessary for the manifestation of this syndrome is markedly less in children where relatively minor head injury, usually occipital in nature, and unassociated with unconsciousness or skull fracture, has resulted in post-traumatic blindness. In adults the injury is frequently much more severe with unconsciousness and skull fracture the rule.

The visual disturbance is commonly severe with early total loss not unusual. The course of recovery is similar in both children and adults; there is generally progressive return of vision to normal over a period of from one to three hours. Permanent residual disturbance may or may not occur and generally reflects the magnitude of the occipital injury. The currently accepted mechanism of visual loss is felt to be occi-

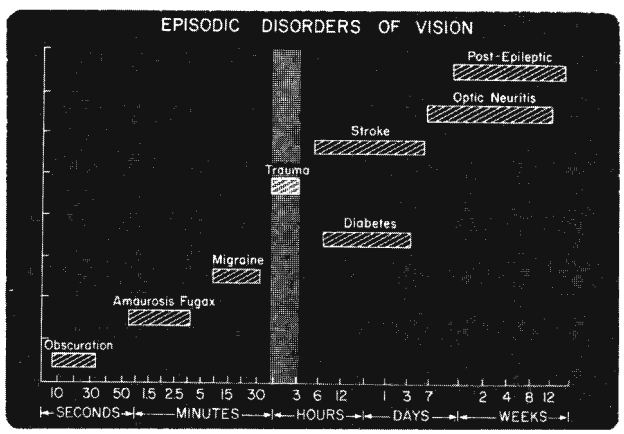

Fig 11—Graphic display of the time duration range of post-traumatic visual loss.

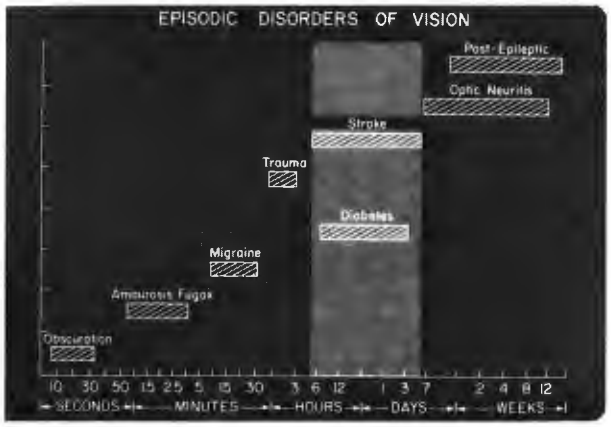

Fig 12—Graphic display of the time duration range of episodic visual loss secondary to stroke.

pital lobe-visual cortex concussion. Treatment is usually expectant requiring only observation.

Stroke. Final consideration in visual disturbances lasting hours should be given to those episodic disorders of vision previously discussed where the transient ischemia, whether localized to retinal vessels, internal carotid artery, or vertebro-basilar system, whether of atherosclerotic, systemic, or migrainous origin, ceases to be transient and results in more prolonged visual deficit (Fig 12).

An arbitrary point has been established at 24 hours which is used to separate transient ischemic attacks from strokes. As with most other artificial guidelines, much variation occurs in clinical practice. We therefore regularly classify as strokes events which last from 12 to 24 hours to three and more days.

Common to the majority of these events is a history of preceding episodes of transient visual disorder. These include obscurations,[2] amaurosis fugax,[8] and transient cortical visual loss,[3] as well as classical migrainous events of either monocular or binocular character.[12] Not infrequently, although resolution may be nearly complete, residual loss of visual acuity or field may remain, emphasizing that a stroke has occurred.

Retinal strokes basically consist of central retinal artery and central retinal artery branch occlusion.[9] The latter is obviously the most frequent retinal stroke to resolve.

The onset is classically abrupt with complete monocular visual loss. Characteristic of these events is funduscopic evidence of retinal ischemia. This usually is greyish retinal edema stopping short of the classic cherry-red spot of irreversible central retinal artery occlusion (See Fig 7). Recovery often begins within an hour or so and evolves over several hours. Residual visual acuity changes may vary from slight to severe and visual field changes are characterized most commonly by altitudinal or sector field loss.

Treatment should be directed toward the usually neglected internal carotid bifurcation disease (See Fig 5). Prompt therapy may avoid a tragic repetition resulting in permanent blindness.

Total occlusion of the cervical internal carotid artery at its bifurcation with the external carotid may result in a wide variety of clinical manifestations. On the one hand, a devastating hemisphere infarct with hemiplegia and aphasia may occur; on the other, the patient may note only the transfer of his odd, neck bruit from one side to the other and cessation of the bothersome episodes of transient monocular blindness. Between these two extremes, an episode of prolonged visual impairment of hours' to days' duration may occur. The clinical picture may so resemble that described for retinal strokes as to defy separation.

Of significant value in placing the inciting event in the internal carotid artery is the reversed bruit and an ipsilateral Horner's syndrome (Fig 13).

It is quite common for the focal bruit of internal carotid bifurcation disease to cease with occlusion of the artery. It is likewise frequent for a focal "increased flow bruit" to be either accentuated or become initially manifest over the contralateral internal carotid bifurcation. At the same time, in about 15% of internal carotid occlusions, edema of the arterial

Fig 13—Right-sided miosis and ptosis of Horner's syndrome secondary to internal carotid artery occlusion.

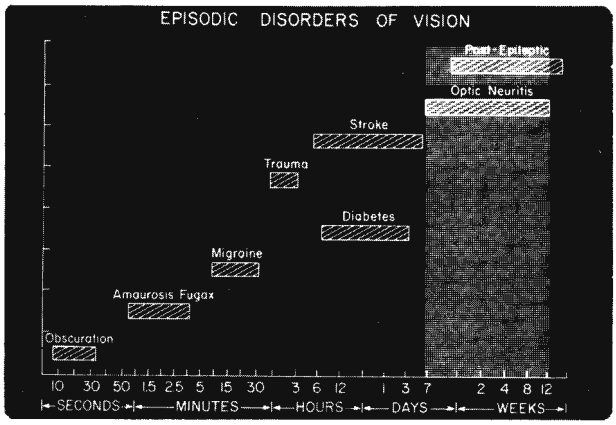

Fig 14—Graphic display of the time duration range of optic neuritis.

wall involves the vasa vasorum of the carotid sympathetic plexus resulting in an ipsilateral Horner's syndrome characterized by miosis and ptosis without anhidrosis (See Fig 13). The other clinical hallmarks, as with retinal stroke, may consist of funduscopically visible retinal edema, variable visual acuity loss, and field defects of altitudinal or sector character.

Diagnostic confirmation is angiographic. Unfortunately, therapy is ineffective, emphasizing the need of early clinical recognition of internal carotid arterial disease.

Strokes in the vertebro-basilar arterial distribution regularly result in prolonged visual loss lasting hours to days. Restitution of some visual function, however, is the rule. The association of additional neurologic deficit of brain stem and cerebellar origin is highly variable.

The onset of the bilateral visual loss is similar to a transient ischemic attack, which commonly has preceded the stroke by days to months. On this occasion, however, the vision does not improve promptly and blindness remains. Characteristically, vision begins to return in hours to a few days. In this time period the patient may experience a variety of visual phenomena including either crude or complex visual hallucinations and visual distortion.

Visual acuity not infrequently achieves remarkable recovery. Residual visual field defects of homonymous character, either hemianopic or quadrantinopic are frequent. It is not unusual for the patient to experience enduring visual agnosias of diverse nature including color-naming defects, and agnosias for faces, places, and even food.

Treatment again should be directed at prevention of repetitive strokes with anticoagulation, the most popular if not the most effective therapy.

The occurrence of permanent residual defects of vision following typical complicated migraine involving retinal, carotid, or basilar arterial distributions is well established, although rare.[12] The clinical circumstance is readily recognized by the presence of a long history of preceding typical repetitive episodes of visual disturbance suggesting a migrainous basis. The mechanism whereby a benign migrainous event is transformed into a permanent stroke is unclear. Therapy has been ineffective.

Visual Disturbance Lasting Days to Weeks.

A discussion of episodic visual disturbance lasting days to weeks becomes essentially a discussion of optic neuritis (Fig 14). There are unusual exceptions to this dictum which will be discussed subsequently.

Optic Neuritis. Optic neuritis is a misleading term. Its implication of an infectious or inflammatory basis of optic nerve disease is rarely accurate. Although bacterial and viral disease of the central nervous system and orbital contents occasionally results in optic nerve dysfunction, the clinical picture does not correspond in most circumstances with the general definition of optic neuritis. The systemic symptomatology, clear evidence of inflammatory orbital or central nervous system (CNS) disease as well as a more fulminant, frequently irreversible optic nerve injury, generally serve to distinguish this truly infectious optic neuritis from the more common clinical picture.

The clinical definition commonly used for optic neuritis is that of rapidly developing blurring of vision with or without pain about the eye or on movement of the eye.[20,21] It may occur either monocularly or binocularly. There is a tendency for binocular involvement to be most frequent in children.[22] The degree of visual loss is variable; however, loss of central acuity greater than 20/200 is the rule. The eye is normal on examination. Visual field evaluation should demonstrate field loss, most commonly in the form of a central scotoma. The funduscopic examination may be normal or reveal disc swelling and elevation with or without hemorrhages and exudates. Disc pallor is rare and if present should suggest previous bouts of optic neuritis whether or not a confirmatory history is available. Optic neuritis most commonly occurs in isolation without associated neurologic symptoms or signs.

The episode of optic nerve dysfunction tends to last from a few weeks to a few months. Total resolution is not uncommon, particularly from an acuity standpoint, but mild residual defects are not infrequent. Further episodes occur in a minority of cases, but may do so on multiple occasions.

The age of onset of optic neuritis varies tremendously from the pre-teens to the 70's and, rarely, 80's.[23] The median age in most studies has been about 30.

Optic neuritis has been characteristically classified as either typical or atypical. The typical patient is a young patient generally in the 20's, fulfilling rather closely the criteria already presented, with monocular involvement. The atypical patient generally falls outside the 20- to 40-year age group, and develops visual loss in either an abrupt fashion or progressively at a slower pace. Visual acuity is either insignificantly involved or severly impaired. Commonly, the episode of visual disturbance fails to remit or does so very incompletely in the expected period of days to weeks. Associated symptoms and signs of neurologic or medical disease are distinctly more frequent.

Within that group of patients with optic neuritis characterized as typical is a second subdivision. This is based upon the presence or absence funduscopically of disc edema and elevation. In the absence of disc changes, the optic neuritis is termed retrobulbar neuritis. In its presence, the optic neuritis becomes papillitis. This designation has little if any implication with regard to etiology or prognosis.[20]

If a specific etiology is not identified, it is common to assume that the optic neuritis is a harbinger of multiple sclerosis. Sufficient long-term studies are available to place the association of optic neuritis and multiple sclerosis in true perspective. It is now relatively clear that only one in six patients with optic neuritis can be expected to develop typical multiple sclerosis in the future.[20,21] This incidence of about 15% is significantly lower than prior incidence rates frequently placed nearer 50%.[24,25] Armed with this reduced statistical probability the physician can refrain from a discussion of multiple sclerosis and the psychological impact it carries with a much clearer conscience.

The potential etiology of remaining cases of optic neuritis is legion and virtually reads as an index of medical and neurologic disease.[23] Not surprisingly, however, a very substantial number of cases of typical optic neuritis remain of so-called idiopathic origin.

The identification of those cases of optic neuritis for which a firm etiology can be achieved is frequently linked to a more atypical presentation: binocular instead of monocular involvement; a young or elderly patient; and, perhaps most importantly, the association of additional medical or neurologic signs or symptoms.

The treatment of optic neuritis, typical or atypical, has been, and remains, an area of controversy. Obviously if a specific inciting etiology can be identified, therapy should be based on the diagnosis. In those cases classified as idiopathic, as well as those suspected to be premonitory of multiple sclerosis, it has been common to use adrenal corticosteroids; their efficacy remains controversial and many competent physicians do not use them. When adrenal corticosteroids are used, it is common to begin with a high dose, from 60 to 100 mg daily, and follow a rapidly tapering course over two to three weeks. Little if any difference in efficacy has been detected with the preparation used or the mode of administration. Prednisone, an inexpensive preparation taken orally, a simple painless mode of administration, is not an unreasonable approach if one wishes to treat a given patient.

A diagnostic pitfall occurs in a group of the atypical optic neuritis cases based upon their occasional response to adrenal corticosteroid therapy. Cases of optic neuritis ultimately found to be caused by sarcoidosis, meningioma, pituitary adenoma, and carcinomatous leptomeningitis may occasionally improve dramatically with steroids. This has led to diagnostic errors. In nearly every instance the optic neuritis has been atypical. Thus atypical features in optic neuritis should suggest diagnostic caution.

The typical course of optic neuritis has been one of resolution with or without a residual defect of variable nature and degree in from a few days to many weeks. Treatment with adrenal corticosteroids has been believed capable of hastening resolution but probably incapable of improving final function.

Post-epileptic Blindness. One final cause of episodic visual disturbance lasting from days to weeks is postictal amaurosis (Fig 15). Although this particular clinical entity occurs rarely, its occurrence can cause extreme consternation if the etiologic mechanism or association is unrecognized.

Postictal amaurosis of cortical origin has been repeatedly observed in young children and infants.[19(pp127–129),26] The convulsive episode has typically been violent. The blindness may be associated

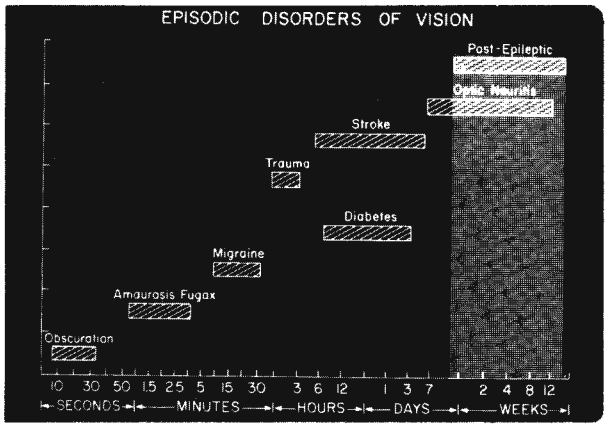

Fig 15—Graphic display of the time duration range of post-epileptic blindness.

with aphasia, deafness, or hemiplegia. Visual loss has typically lasted from days to weeks with slow, progressive recovery. The mechanism of its production remains unclear. No satisfactory therapy has been identified.

Summary.

A diverse and seemingly unrelated group of diseases has been integrated on the basis of their episodic disturbance of vision. The parameters of laterality, but more importantly the duration of individual episodes, have been used to direct diagnostic consideration. Many of these clinical entities are commonly encountered in practice. Their recognition and care are dependent upon prompt diagnosis based upon characteristic clinical signs and symptoms.

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