

VISION

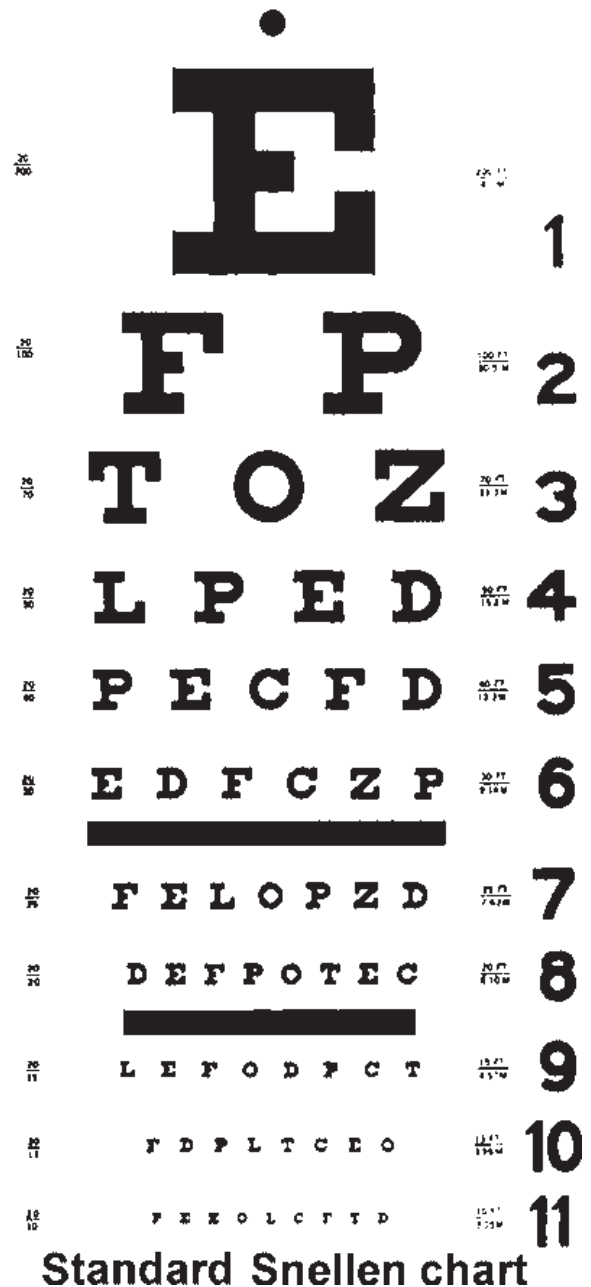
Visual Acuity Testing

Introduction:

In clinical practice, testing of visual function takes many forms. From testing visual acuity (standardized letters on a wall chart) to color testing to visual fields, each method has specific goals and yields specific information.

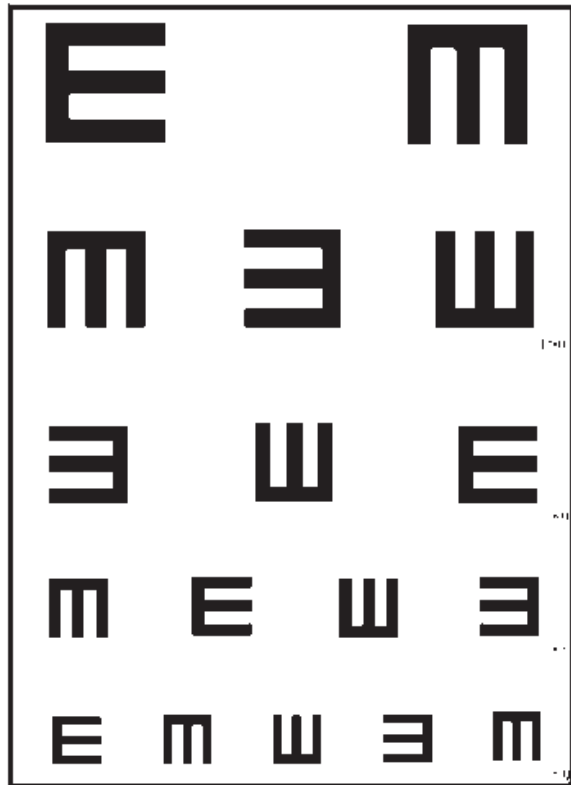
The most common form of visual function testing is to check visual acuity, the ability to tell an object from its background. For adults, this test is commonly done with black letters on a white background, but it can be lines, dots, or images of any shape.

For clinical acuity testing, each eye is tested alone, with the opposite eye covered. The patient is asked to read a chart of letters of gradually decreasing size. The lowest size letter which is recognizable is deemed the visual acuity. For standardization, a fixed distance is used (which varies with each chart system). USA standards are measured from 20 feet away. A standard person sees 20/20, while a patient with poorer vision may see only 20/40. These numbers refer to the size of the letters, and the size of the retina that the corresponding image covers. Therefore, a person seeing 20/20 can recognize letters twice as small as a person whose best acuity is 20/40. Another way to think of it is that the 20/40 person would have to get twice as close to a letter to see it as clearly as a 20/20 person. In reality, it is more realistic to turn it around: if a 20/40 person sees a letter on a chart when standing 20 feet away, the 20/20 person could walk back to 40 feet away and still recognize the letter. This system is based on the sizes of the letters and the distance the eye is from the chart. The 20/20 letters subtend an arc of 5 minutes when viewed from 20 feet away. The 20/40 letters are twice as large, subtending an arc of 10 minutes when viewed from 20 feet.





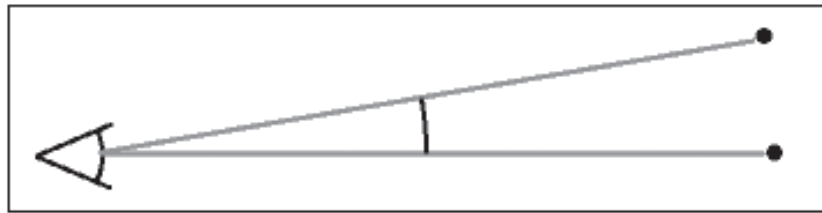
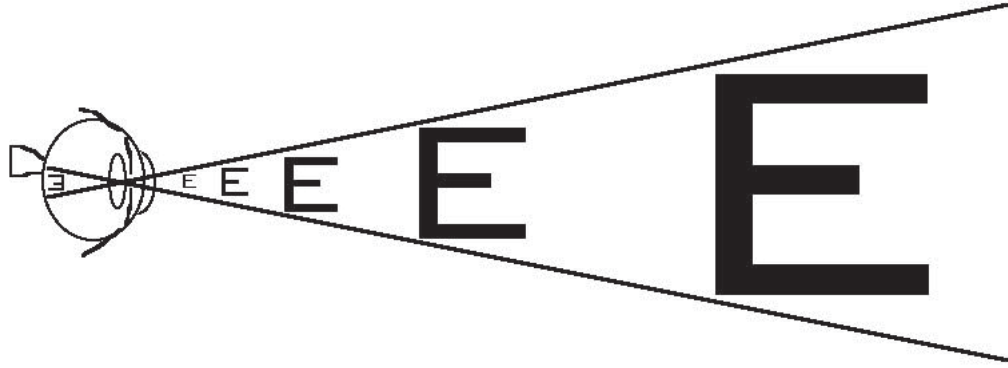
Picture chart



Tumbling E chart

These two types of charts can also be used with patients unable to name letters

Note that this is a direct relationship between viewing distance and arc subtended - for example, a 20/20 letter viewed from 40 feet away would subtend only half the arc it does at 20 feet away, and an eye seeing this letter at 40 feet would be said to see 20/10.



Let's do some examples.

<u>Patient</u>	<u>Standing at</u>	<u>Lowest line read</u>	<u>Acuity</u>
A	20 feet	20/40 line	20/40
B	40 feet	20/40	20/20
C	100 feet	20/100	20/20
D	40 feet	20/20	20/10
E	20 feet	20/10	20/10
F	100 feet	20/20	20/4

Visual acuity is only one part of our visual system. It doesn't measure color vision or contrast sensitivity or other visual functions, but is the most standardized portion of visual function testing. Note very closely: We are testing the absolute smallest-sized object the eye can discern, and therefore are involving only a VERY few photoreceptors in the center of the fovea (and therefore only cones, as you heard in earlier discussions in this module). A patient with complete loss of every single rod would still have 20/20 acuity if all of the cones were still intact.

Visual Field Testing

Testing the rest of the retina, which performs the function of peripheral vision, takes a much different test.

Clinically, there are two levels of testing of the peripheral retina: very gross and quick methods, good for Emergency Room use, and very detailed, objective, sensitive methods, good for monitoring diseases which cause slow loss of peripheral visual function.

Informal Visual Field Testing: Confrontation Methods

The quickest method of testing visual fields clinically is to compare the patient's function to your own (thus the word "confrontation.") In this method, patient and examiner each cover one of their eyes, the open eyes are aligned, and the examiner holds fingers up in various parts of the visual field. If the patient cannot see objects that the examiner clearly can, the patient's peripheral visual field may be damaged.

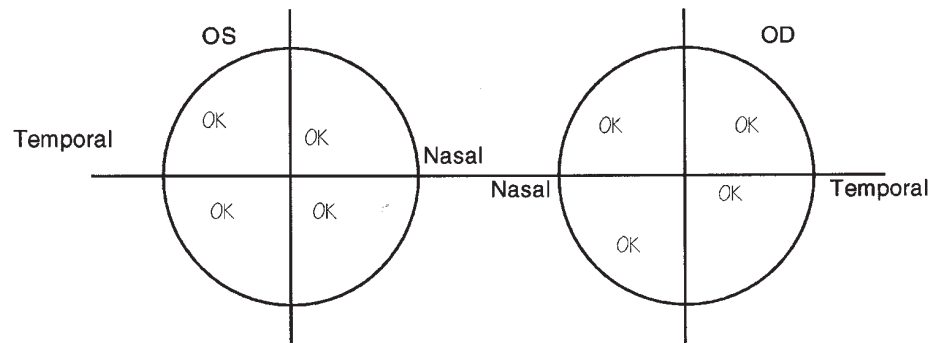
Due to all the variables involved (e.g. is the stimulus exactly the same distance from the patient as from the examiner, is the illumination exactly the same from each person's point of view, is the examiner's field completely normal, etc etc etc) the patient's field must be quite severely abnormal to be detected as such.

Results are noted as areas where the patient saw well, or missed answers, and are oriented AS THE PATIENT SEES THEM.

This method works well in ERs or for gross screening, but for detailed detection, there must be much better control over the lighting, distances, and stimuli. Machines to test the visual field provide this control.

NOTE: Throughout the module, the conventional abbreviation of "OS" for **left eye** and "OD" for **right eye** is used. [OS= oculus sinister, OD = oculus dexter]

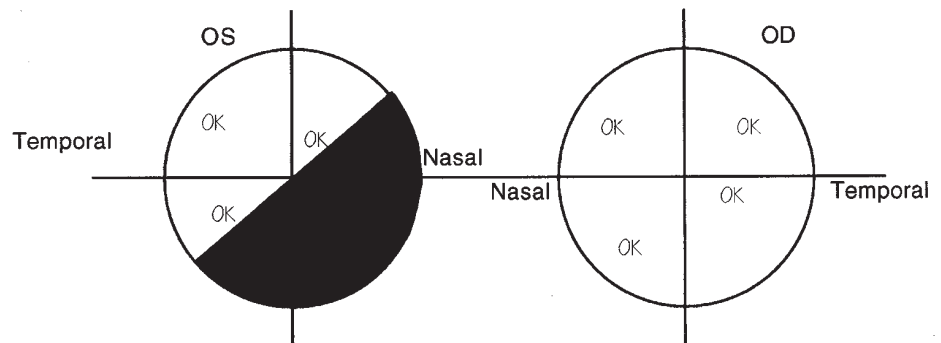
Confrontation Visual Field Testing: Documentation



Confrontational Visual Field Testing: Normal Patient.

One convention that is used to document confrontation field results is to draw the defects as though the patient were looking at the piece of paper on which the result is drawn. Often, circles are used, even though the fields themselves are not circular (patients can see more temporal visual field than nasal). The convention uses circles because they are simply easier to draw on the charts. The intersection of the two lines (and center of the circle) is the point of fixation (i.e. where the patient is looking). This patient sees well in all of the areas the examiner tested, and so no areas are blacked in. To show that all areas are tested, "OK" is written in each quadrant.

Confrontation Visual Field Testing: Example of Mapping of Visual Defect



Confrontational Visual Field Testing: Patient With Visual Defect.

Let's say you are mapping a patient's visual symptoms. The patient complains that s/he cannot see the lower-right portion of a page of paper if viewing it only with the left eye. The right eye sees normally. As you face the patient and test the left eye, the area which is not seen is to YOUR left-lower area, corresponding to the nose-side of the patient's view. You draw it into the chart as the PATIENT sees it, to his/her RIGHT and below.

Visual Field Testing Machines: Background

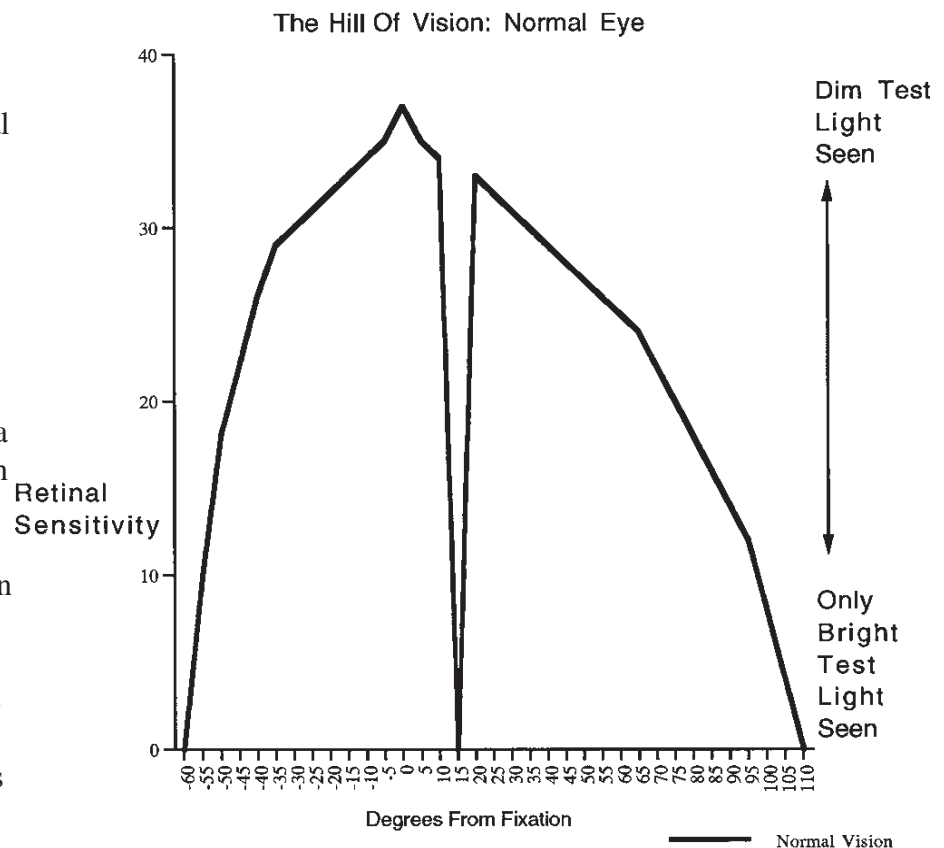
As described earlier, each rod and cone is connected to a network of ganglion cells in the retina. The output of each photoreceptor can affect one or several ganglion cells, and this information is then relayed to the visual cortex via the optic nerve, lateral geniculate body and optic radiations. You have learned that ganglion cells have circular receptive fields - areas of rods and cones to which they respond. These circular fields overlap heavily in the central retina, and overlap far less in the retinal periphery.

If all cells are working well, each and every visual stimulus is relayed to the visual cortex. But if any of the retinal cells are dysfunctional, not all stimuli will be processed and relayed. The method of objectively testing peripheral visual function is designed to create a geographic map of the areas which are working well and those areas of dysfunction.

The normal retina responds unequally to equal stimuli based on location. That is, the central retina has more cones, more ganglion cells, and is able to discern more attributes of the stimulus (e.g. color) than the visual periphery. **When adapted to photopic conditions** the central retina is also able to respond well to much dimmer white lights than can the periphery. The sensitivity peaks at the fovea and gradually diminishes towards the periphery. If you graph the sensitivity as a function of distance from the center, it looks somewhat like a hill with a gradual slope. The shape of the hill is the sum of the density and responsiveness of the photoreceptors and ganglion cells. The center of the retina is most responsive to light because there are more photoreceptors and ganglion cells in this area. As the density of photoreceptors falls off and the ganglion cell density decreases as you get farther away from the fovea, the sensitivity to the test lights decreases and the hill slopes down. Traquair called this the "hill of vision in a sea of darkness."

This is a very clinically useful image, and we often speak of visual field deficits with "steep edges" to describe a focal area of sensitivity which is markedly decreased from its immediate surroundings.

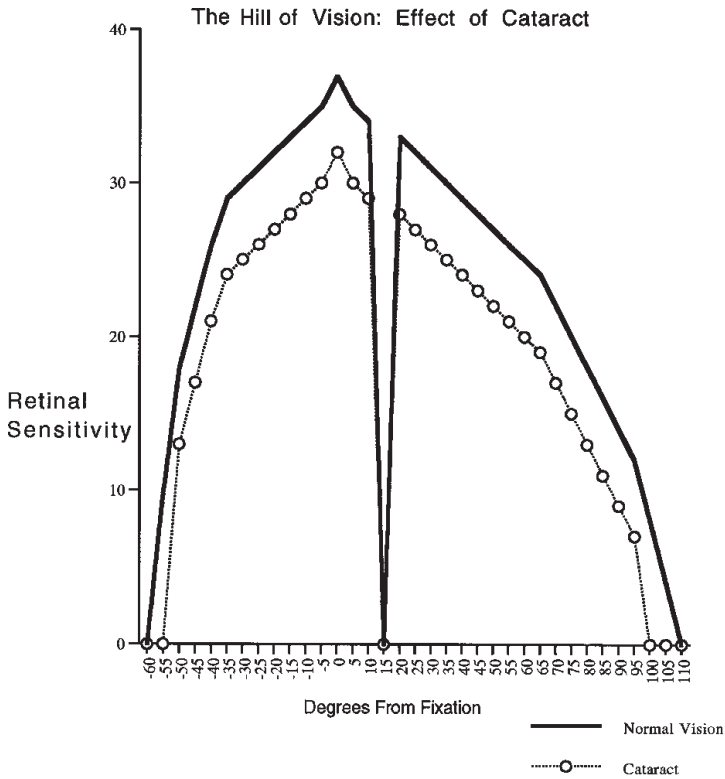
For illustration, there is a naturally-occurring example of a visual field "defect" in every eye. Notice in the diagram that there is a very deep, small-diameter "hole" in the hill of vision. This hole corresponds to the optic nerve. Since there are no photoreceptors in that area, the sensitivity to visual stimuli is zero. Since the area of retina immediately surrounding the nerve does contain photoreceptors and is fairly close to the macula, its sensitivity is very high. So this abrupt change of sensitivity results in a very steep, round, small hole.



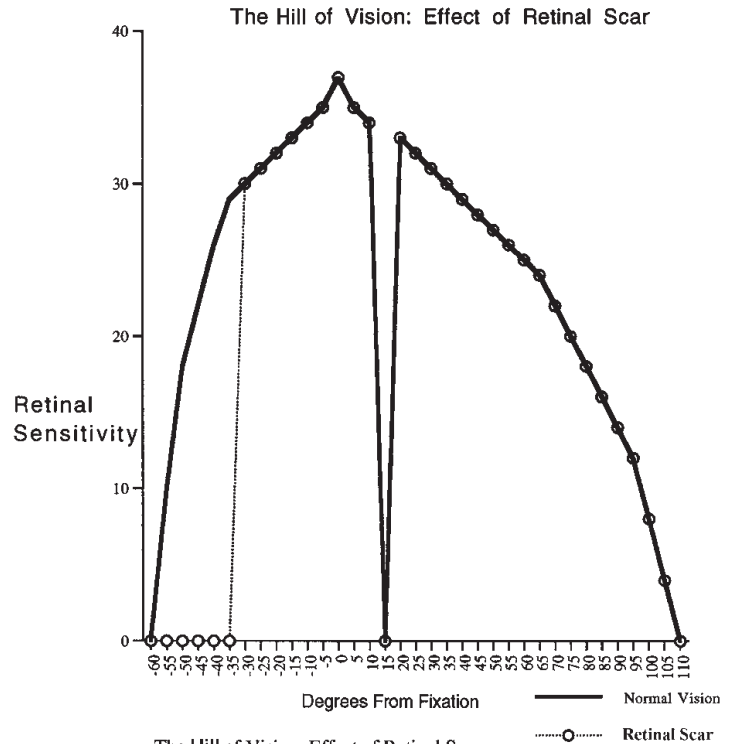
The Normal Hill of Vision.

Note that in the center of the hill, the retina can detect even the dimmest light. The notch at +15 degrees is the normal blind spot, corresponding to the optic disc, where there are no photoreceptors. The "point of fixation" (0 degrees on the graph) is the center of vision of the eye, corresponding to the retinal fovea.

The “hill” for most people is the same, fairly parabolic shape. You can imagine that the effect of a visual problem like cataract, (opacity of the lens), would affect the whole retina fairly diffusely, where a defect like a retinal scar from trauma would only affect a well-defined, small portion of the retina. In clinical practice, the hill of vision is affected similarly: cataract patients have a normal shape of the hill, but each and every point is slightly diminished (equally). Thus, it looks like the hill is sinking into the “sea of darkness.” The retinal-scar patient would have normal points everywhere but the defect, and so it would look like that patient had a hole similar to the optic nerve - steep-edged and very deep.



Hill of Vision: Effect of Cataract
In this diagram, I have decreased the sensitivity at each tested point by 5 deciBel units. The impression is of the hill maintaining its overall shape, but having sunk a little into the sea of darkness.

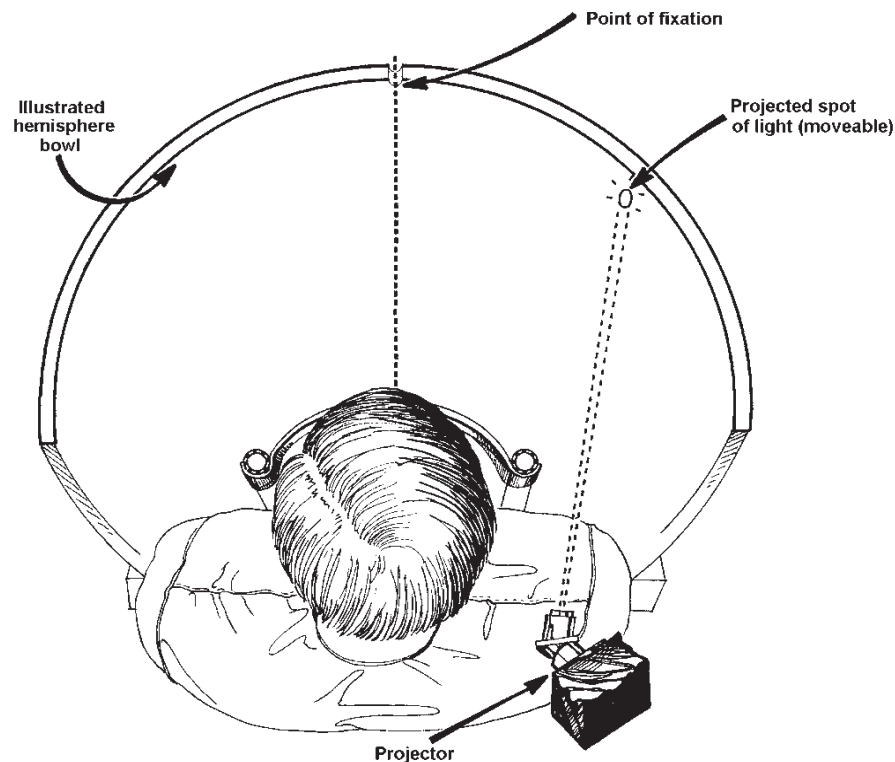


The Hill of Vision: Effect of Retinal Scar
In this diagram, I have left almost all of the hill to be normal height, but the focal area of retinal damage at the edge of the hill I have reduced to zero sensitivity. Notice how the edge is very steep, going from an area which sees normally to the scarred area that does not see at all. Clinically, the shape of the field damage is very helpful in determining the likely causes.

Visual Field Testing: Methods

We wish to create a map of visual sensitivity, to document geographically where the retina sees well and where it does not. We need a system of stimulus/response mechanism so that the exact same stimulus can be presented to various parts of the visual field and the patient can respond when the stimulus is seen. We also need a method of varying the stimulus intensity in order to map the various levels of sensitivity from the fovea to the periphery. We need to be careful to keep the test consistent from test to test, and to control for the distance from the eye that each stimulus appears. All visual field machines currently in use accomplish this by having a standard-sized hemispherical bowl, with a chin rest mounted so that the eye to be tested can be placed at the center of the hemisphere. The opposite eye is patched. The room lights are turned off, and the door closed, and a standard amount of diffuse light is used to dimly illuminate the entire surface of the bowl. This way, the background is kept constant from day to day and test to test. The amount of light is preset to a medium level **at which both rods and cones will respond**, so both central and peripheral visual function can be mapped at the same test.

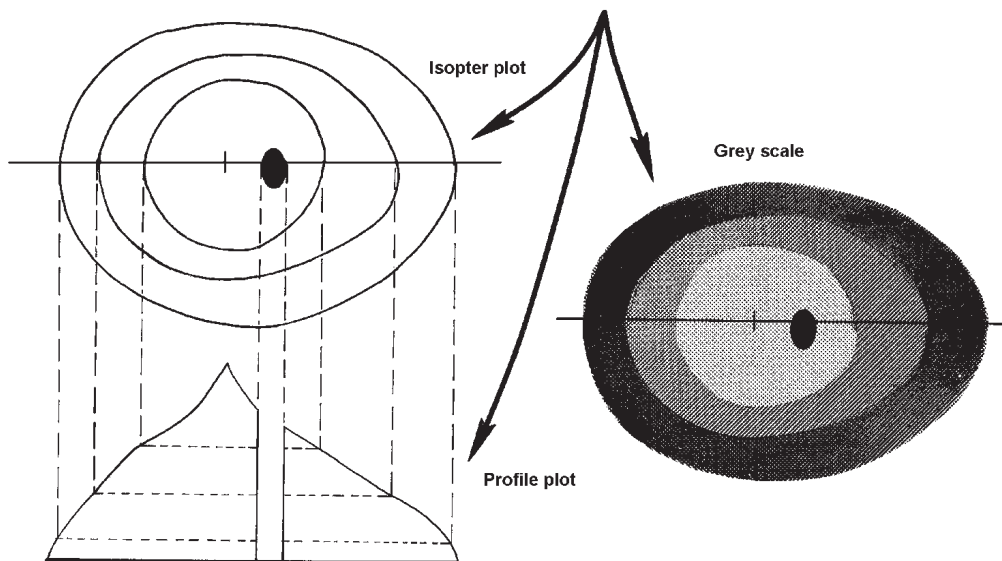
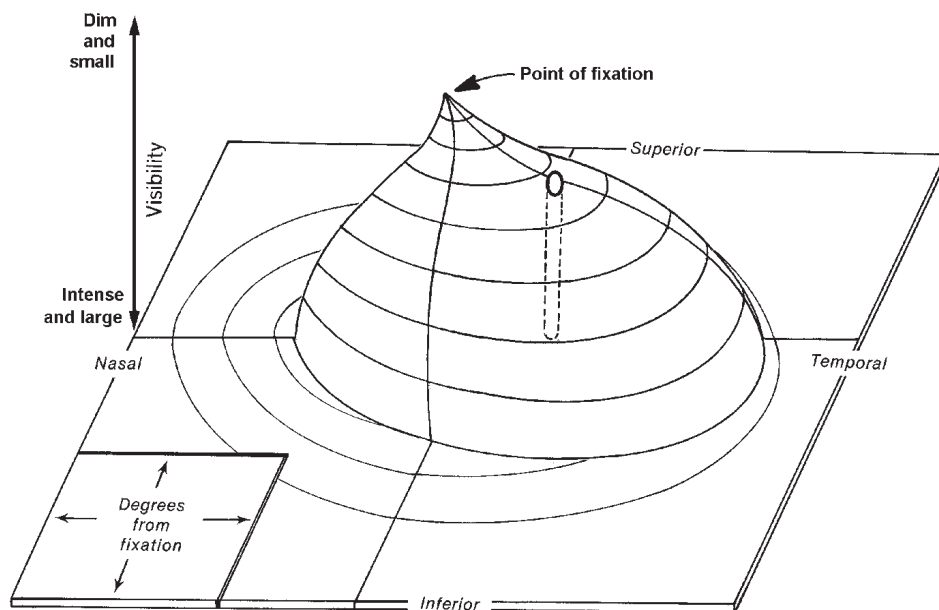
Now we need the stimulus - a light that is brighter than the surrounding bowl, such that the patient can detect the difference of the stimulus light as compared to the background. Since the hill of vision is not flat, there will be areas where the patient sees a given stimulus and areas where the patient does not. In order to map out these areas, we will need to vary the stimulus to find where the "edge" of detection actually is. There are two clinically useful ways of doing this: with lights that physically move across the threshold, or lights that cross the threshold by changing brightness.



This is the basis of the visual-field testing machine. The patient is asked to look straight ahead at all times, at the "point of fixation." The hemispherical bowl has a very low level of diffuse background lighting, and the center of the bowl is where the eye is placed. A projector behind the patient shines a spot of light briefly on the bowl, and the patient indicates s/he has detected the spot with a thumb switch. The operator of the test, whether human or computer, controls where the light is presented and keeps track of which lights are seen or missed by the patient.

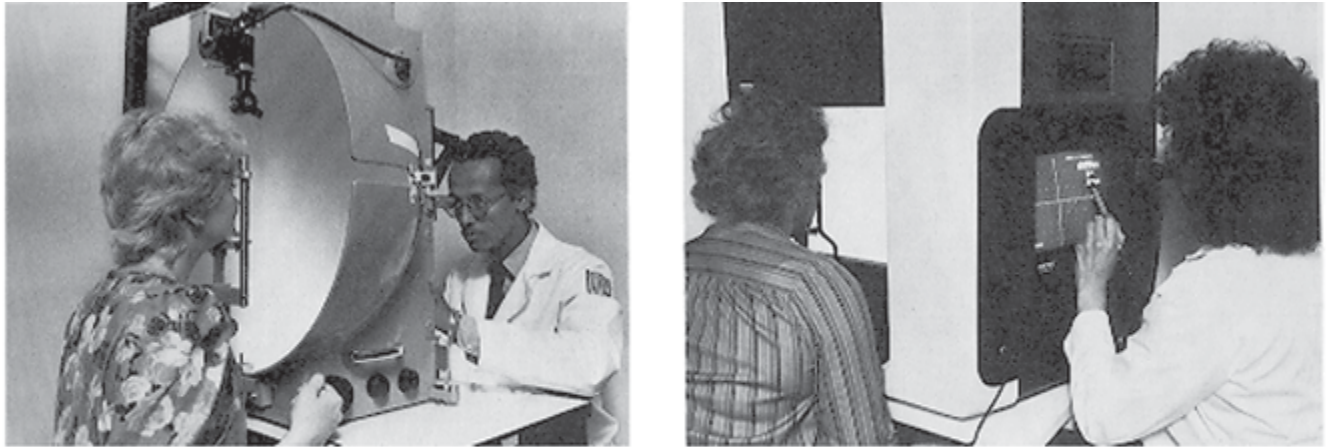
The older method, called Goldmann Visual Field testing, uses mobile light stimuli. While the patient is instructed to look straight ahead at the center of the bowl, a light of a defined size and brightness is moved from the periphery to the center of vision. The patient pushes a thumb button at the instant the moving light is detected in the peripheral vision. The technician marks the paper at the corresponding point, moves the light back out to the periphery, slides it over one clock hour, and begins the process again. To maintain our hill analogy, this is equivalent to taking a seaplane, starting in the sea of darkness, flying up to a defined height above the water, and flying into the hill at every point around the circular hill, and keeping track of how far towards the center of the hill you flew until you hit it. This results in a circle (in a normal field). You can also think of this as a topographic map contour line. By starting over with a different size or brightness stimulus (or flying at a different height) you can create a second circle of contour. Doing this with several stimuli will map out the surface - smaller, dimmer stimuli will create smaller circles, since the outer retina will be too insensitive to see them.

The other way to test the hill is to vary only the brightness, of fixed stimuli. If the moving-stimulus technique was flying horizontally until the hill was struck, this method is more like dropping a ball vertically onto the hill and measuring how soon the ball hits the hill. In this method, computers control the gradual brightening of the test lights until the patient sees the spot, and record the brightness of the dimmest light which is seen. In this way, a numeric map of sensitivity is created.

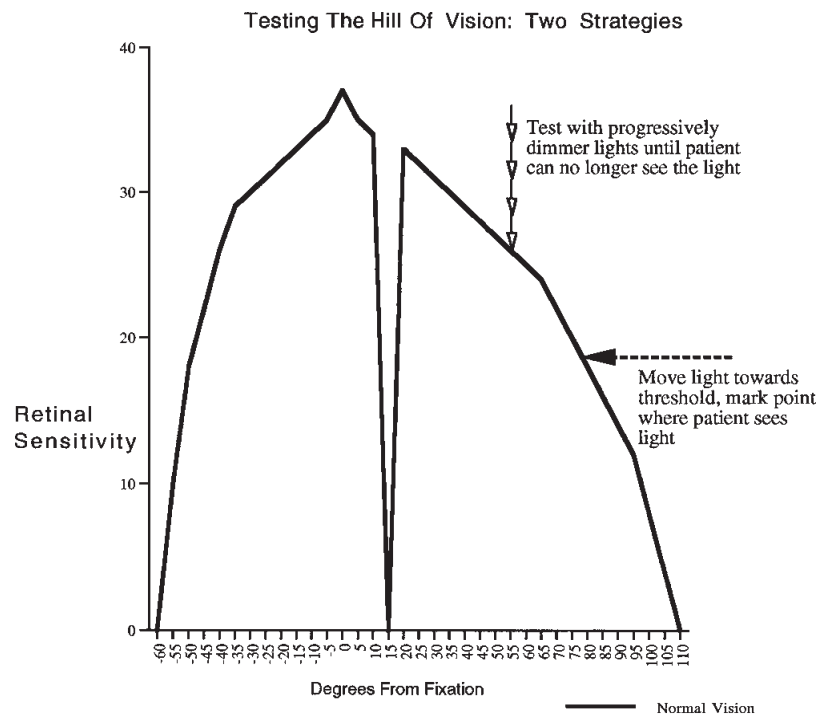


The normal "hill of vision" is seen in the upper diagram, and the two ways of representing the results on a flat sheet of paper are seen in the lower diagrams. The "isopter plot" shows where the patient saw each light as it was moved from the periphery to the center of vision. Each line represents a full 360° test with a different brightness or size of test dot. The inner circles are dim/small test dots that only the center of the retina is sensitive enough to detect. The grey-scale method is similar, but uses a different shading for each brightness of test dot. Areas shaded more darkly represent areas where only the brightest test dots were seen.

The computerized method is more objective - the lights are presented in the exact same order each time, there is no variability of how fast the object is moved by the technician, etc. It is used far more often clinically, and is graphically represented as a scale of grays that corresponds to how the patient sees: darker areas on the printout correspond to areas of poorer sensitivity.



These photos show the two field-test machines being used. The manual machine in the left photo is controlled by the technician. He is moving the lights manually and hand-marking the patient's responses on the recording paper. On the right, a technician is entering the patient data into the computer touch screen. Once the test is begun, the technician's only function is to monitor the patient's attention and fixation to the center of the bowl - the computer controls where and how bright the test spots are. Notice the thumb switch in the hand of the patient on the left.



The Normal Hill of Vision.

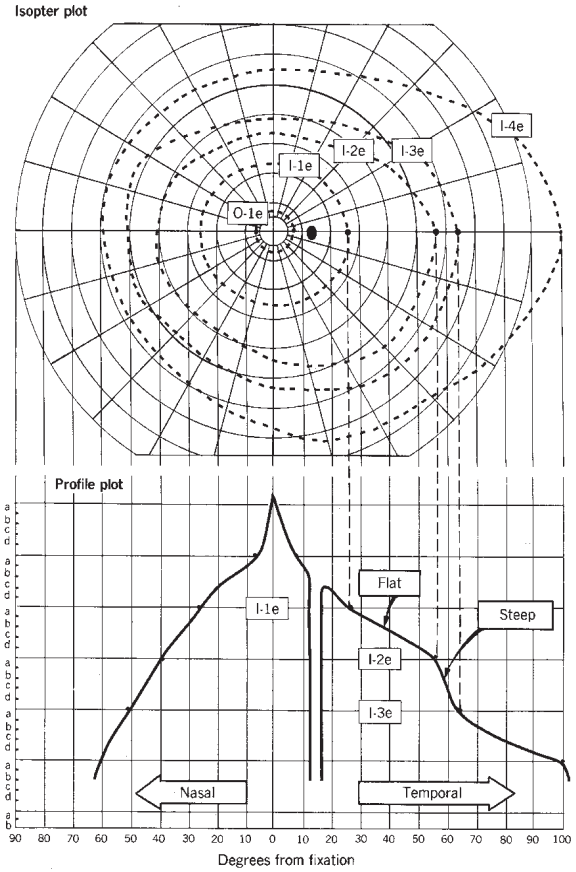
There are two ways to figure out the hill's shape and size- either keep the light in the same place, and progressively dim it until the patient can no longer see it (upper example) or keep the brightness the same, and move the light from an area where it cannot be seen until the patient sees it. Machine-driven tests use the first strategy and the human-driven method uses the moving-light strategy. Note that by changing the brightness of the moving-light system, you can intersect the hill at a different height. By using many different brightnesses of light, a smooth contour map can be made.

Here are more detailed examples of normal printouts of the field results. In the manual method at the right, the patient's response to each size and brightness of test light is connected by a line into a rough circle.

[Supplementary information: The first letter and second number refer to the size and brightness of the test spot, and the "e" refers to the neutral-density filter used.]

Notice that the temporal field is slightly more sloped and extends more into the periphery than the nasal field. This discrepancy is due almost entirely to the presence of the nose (in the nasal field).

In the figure below, the central grid of numbers is what the patient actually generated during the test. Each number corresponds to the retinal sensitivity (i.e. how dim the dimmest light was that the patient saw). A bigger number means a more sensitive retina. Note that the numbers for this normal patient are in the high 30s in the center and are in the lower 30s in the periphery.



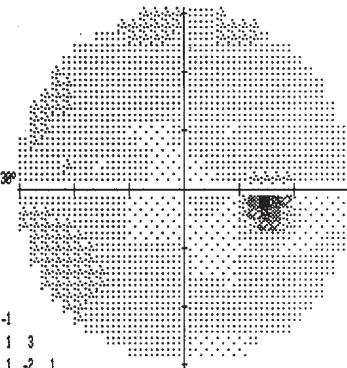
CENTRAL 30 - 2 THRESHOLD TEST

NAME
STIMULUS III, WHITE, BCKGND 31.5 ASB BLIND SPOT CHECK SIZE III
STRATEGY FULL THRESHOLD

BIRTHDATE 01-01-47 DATE 05-26-93
FIXATION TARGET CENTRAL ID 479174 TIME 00:23:50 AM
RX USED -4.00 DS +2.00 DCX 95 DEG PUPIL DIAMETER 4.5 MM VA 20/20

LOW PATIENT RELIABILITY

RIGHT	25	25	27	23						
AGE 46	27	27	27	26	28	30				
FIXATION LOSSES 5/24 xx	25	28	27	29	30	29	27	29		
FALSE POS ERRORS 1/15	25	28	28	31	31	28	31	30	28	28
FALSE NEG ERRORS 0/12	27	25	31	30	31	32	28	25	27	27
QUESTIONS ASKED 462	26	28	28	31	30	29	31	32	32	
FOVEA: 36 DB	26	28	28	31	30	29	31	32	32	
TEST TIME 14:28	22	24	28	31	31	32	31	30	32	30
HFA S/N 640-1489	23	24	29	26	31	31	28	30		
	27	26	27	28	30	30				



GLAUCOMA HEMIFIELD TEST (GHT)
WITHIN NORMAL LIMITS

1	1	4	0	0	0	3	-1										
1	0	0	-1	2	4	0	-1	-1	1	3							
-2	0	0	0	1	2	-1	2	-3	-1	-1	1	-2	1				
-1	0	-2	0	0	-3	1	1	-1	0	-1	-5	-1	-3	-2	-1	4	-3
0	-1	0	-2	-1	0	-3	-3	-2	-2	-2	-4	-2	-2	-3	-1	1	2
-1	-2	-3	-1	-2	-3	-1	2	2	-6	-6	-3	-2	-2	0	-1	1	0
-5	-5	-3	-1	-1	0	0	0	2	1	-6	-5	-2	-5	0	0	-2	0
-5	-4	-1	-5	0	0	-1	0	-2	-3	-3	-2	-1	0				
-1	-3	-2	-2	0	1	-2	1	2	2								
TOTAL	-1	1	3	3													
DEVIATION																	

PROBABILITY SYMBOLS
 :: P < 5%
 :: P < 2%
 :: P < 1%
 ■ P < 0.5%

MD -0.38 DB
 PSD 2.85 DB
 SF 1.31 DB
 CPSD 1.41 DB

For this PARTICULAR test, only the central 30° of field were tested. Compare that with the hill of vision at 30° in the upper diagram- the steep temporal slope has not been tested in this central-field test.

Note how the blind spot appears in the gray scale representation - a black spot at about 20° from fixation. An area where the retina was completely nonfunctional would have the same dark black appearance.

One huge advantage of the computer method of testing is that it is standardized and therefore allows comparison with other patients. The bottom four grids of this field show these comparisons, at every dot of the grid, and the lack of shading in the lower two graphs shows that this patient did NOT deviate from what was expected for his/her age group.

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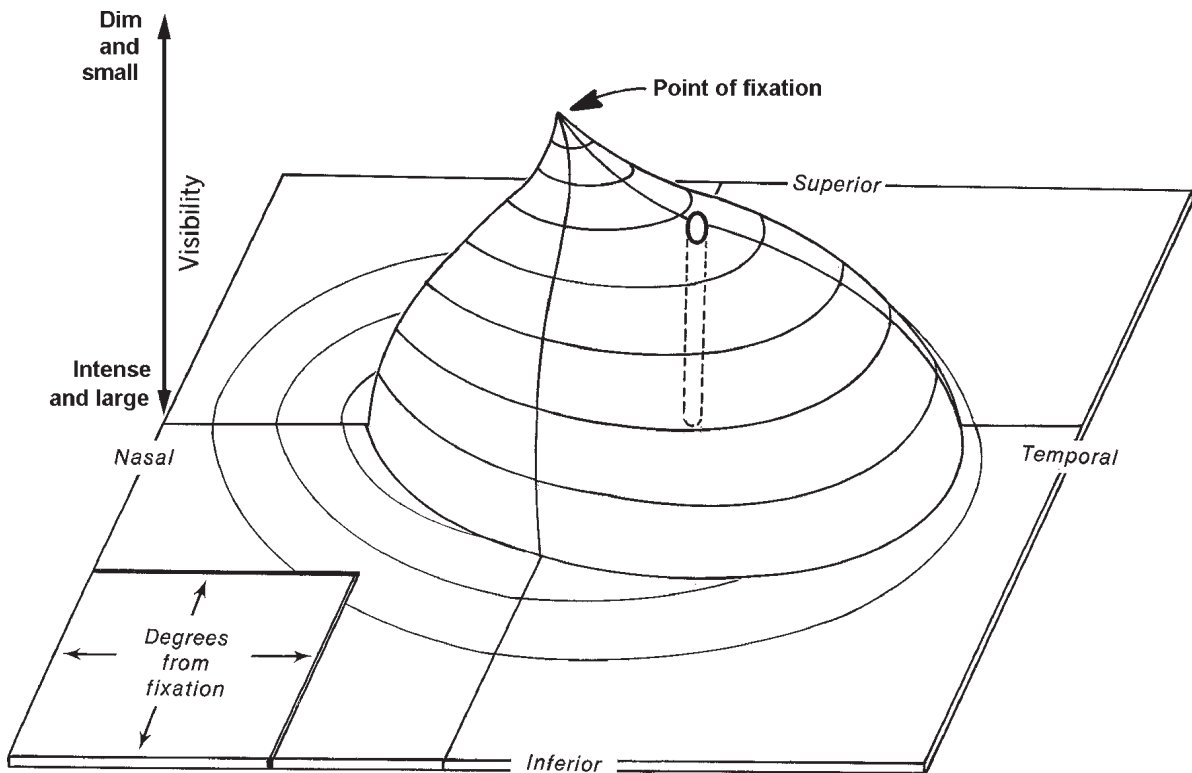
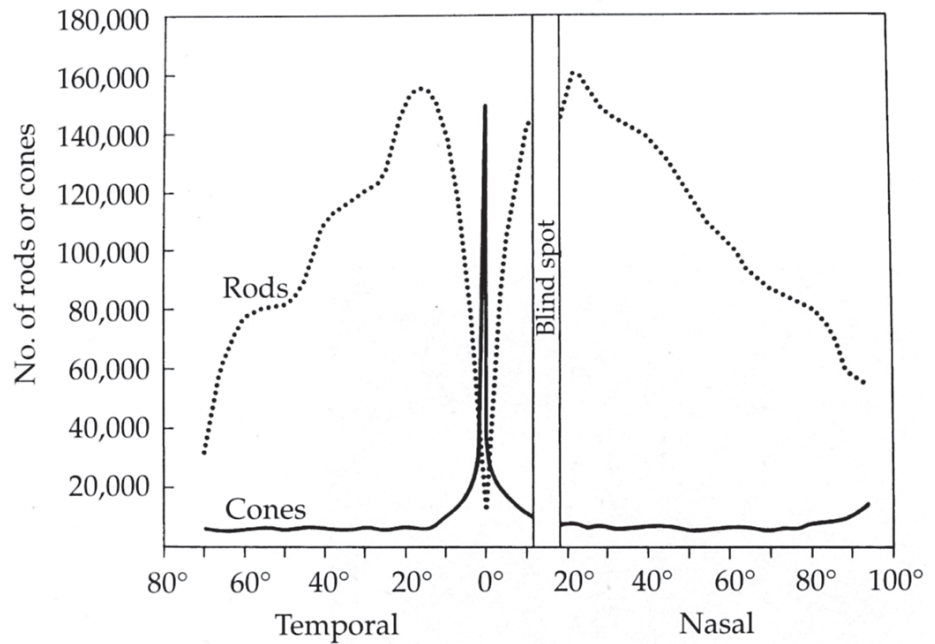
SYM										
ASB	.8	2.5	8	25	79	251	794	2512	7943	?
	.1	1	3.2	10	32	100	316	1000	3162	10000
DB	41	36	31	26	21	16	11	6	1	≤0
	50	40	35	30	25	20	15	10	5	

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The Hill of Vision: Correspondence with Rod and Cone Distribution

Recall the distribution of the individual rod and cone cells that was discussed earlier in the module. Remember that the cones predominate in the fovea, and the rods are very dense in the area surrounding the fovea.

Note the shape of the “hill of vision” as it compares to the photoreceptor density. The conditions for testing the visual field are called **mesopic** - which means somewhere **between** the photopic (high luminance) conditions under which the cones function best and the scotopic (low light) conditions under which the rods function best.



By testing the visual field in these mesopic conditions of **middle luminance**, we can get both photoreceptor types to function during the test. The hill of vision is what results. It isn't perfectly simple, because the sensitivity doesn't quite match the total density of photoreceptors, but it is as close to this as physiology can allow. The shape of the hill of vision is a combination of the receptor density and the response-sensitivities of the rods and cones to the test stimuli.

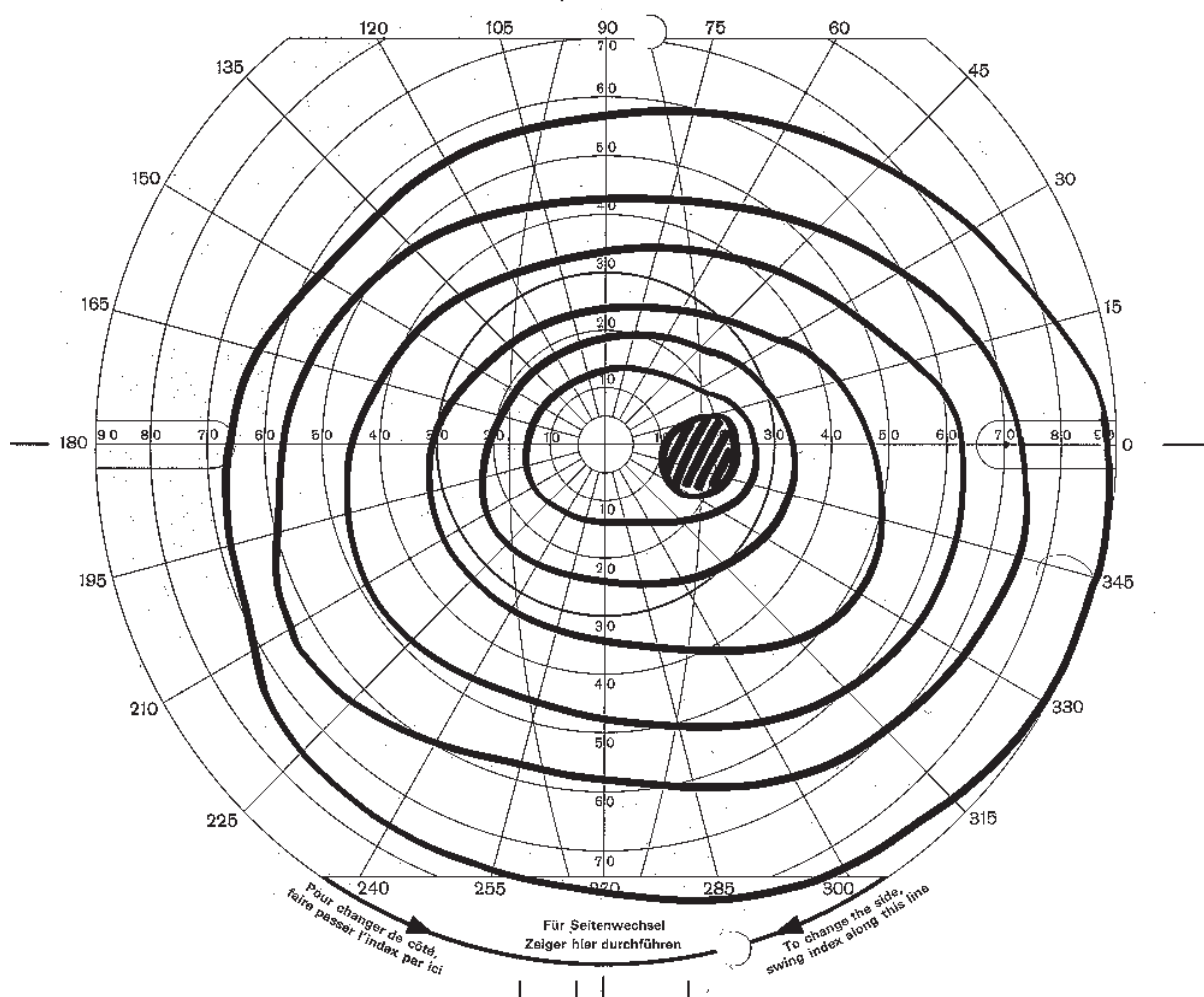
Visual Field: Example Results (Normal Patient)

These are visual fields from normal patients tested by both Goldmann and automated machines. Each circle on the Goldmann printout corresponds to a standardized size and brightness of stimulus, tested from the periphery towards the center on each of the radial lines (every 15°). The grid of numbers from the automated printout is the actual sensitivity numbers from the test, and the gray-scale printout has translated that same grid into a more pictorial format.

In these manners, a detailed measure of the visual function of each area of the retinas can be plotted. These tests are very commonly done in clinical situations. Often, they are repeated frequently and compared to prior results, to see if the patient is continuing to lose visual function, and to see if the treatment has been effective at stopping the process of loss. Remember how important the patient is to this test result- concentration and cooperation are necessary to good results, and sometimes patients cannot perform these tests very reliably.

In this diagram, I have drawn an imaginary (i.e. simplified) normal manual visual field of a right eye - the normal blind spot is in the temporal field (since the optic nerve is nasal to the fovea of central fixation) and all of the lines are roughly symmetric.

For the sake of clarity in later diagrams, I will include only a single test line of the 6 shown here.



Pupillary Light Reflex: Background

The pathways of photoreceptor - ganglion cell - nerve fiber layer - optic nerve - optic chiasm - lateral geniculate body - optic radiations - visual cortex have already been discussed. You already have a working personal knowledge of the pupillary light response, because you have noticed that pupils constrict when the eye is exposed to brighter light. How does that happen and how do we test that clinically to see if it is intact?

Pupillary Light Reflex: Pathway

Somehow, information about how much light is striking the retina(s) must be relayed back to the pupil muscles so that they can respond appropriately.

The pathway by which this happens is very similar to the visual pathway. It turns out that not all of the ganglion cell axons project onto the lateral geniculate nucleus. Some synapse on cells in the pretectum. The information is relayed from the pretectal nucleus to the Edinger-Westphal nucleus, and from there to the pupillary constrictor muscles by way of the third cranial nerve.

Let's go through this slowly, because there are several very important results of the details of the connections.

First, note that the input to each pretectal nucleus is **BILATERAL**. That's because the axons have already crossed at the chiasm. So the input that each of the pretectal nuclei receives is not input from one **EYE** but from one **visual FIELD** as was discussed earlier in this module.

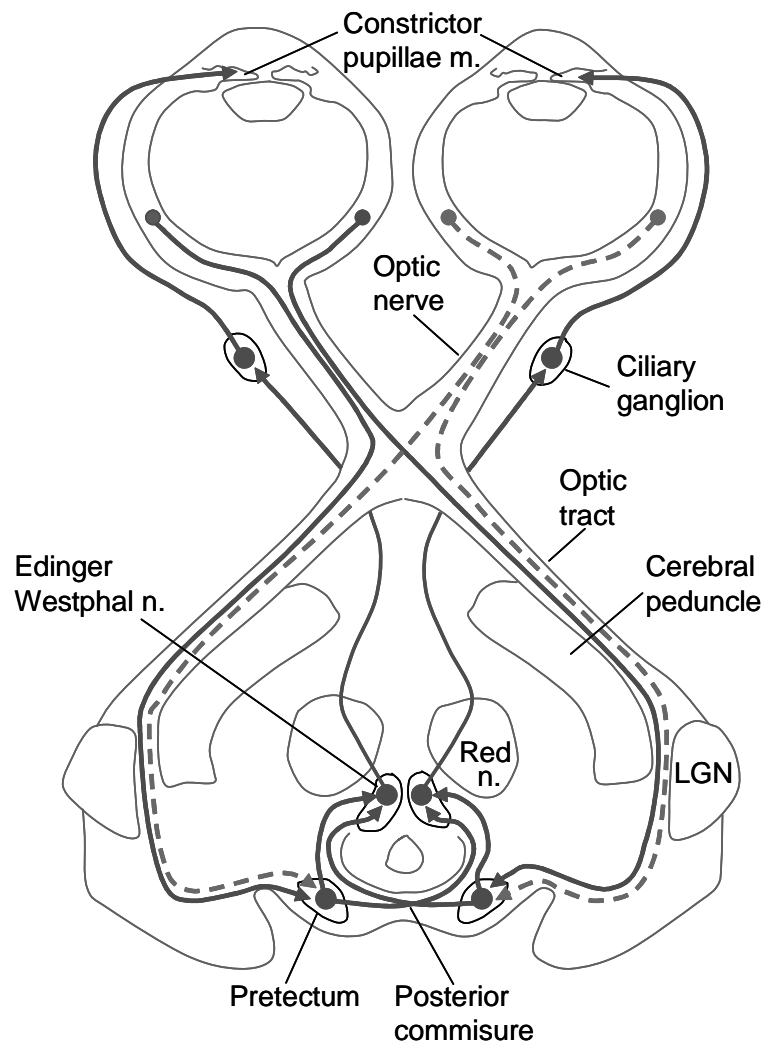
Second, note from the diagram that the two nuclei communicate with each other, sharing inputs.

Third, note that the output from each pretectal nucleus goes equally to **EACH** Edinger-Westphal nucleus.

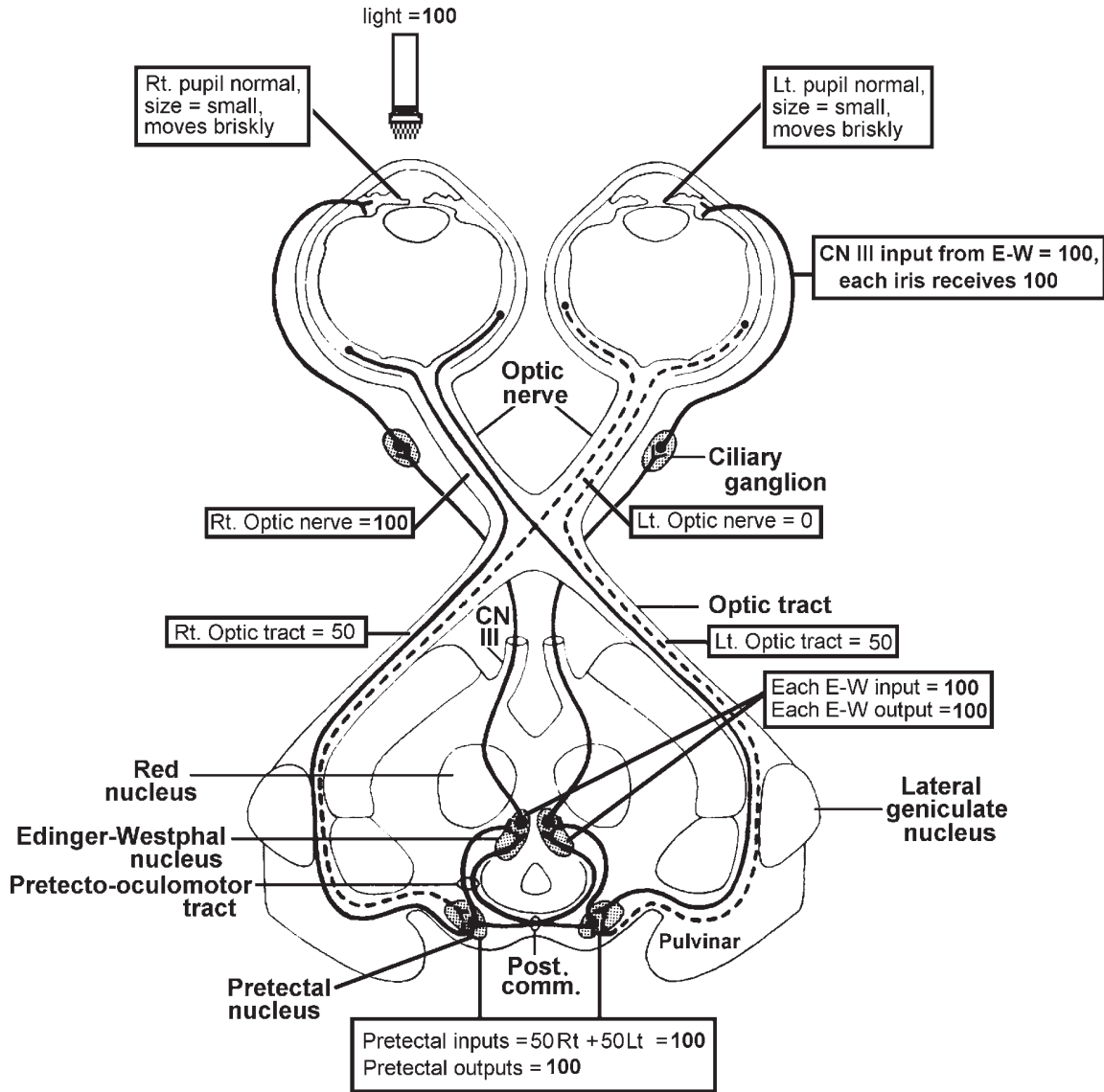
All of this crossed input means that the receive the **SUM** of the output from **BOTH** eyes at all times. You can think of this system as summation device. The entire output from both eyes is combined and projected equally onto the Edinger-Westphal nuclei.

The diagram above is the same one that you have seen earlier in this module. Follow the path of nerve impulses from each retina to the bilateral projections onto the pretectal nuclei, to the crossed bilateral inputs to the Edinger-Westphal nuclei, and then the paired (but uncrossed) outputs to each ciliary ganglion and on to the pupillary sphincter muscles. Notice that these fibers do **NOT** synapse in the Lateral Geniculate nucleus.

The convention of the right eye structures drawn on your left, as though you were under the system looking up at it, is traditional.

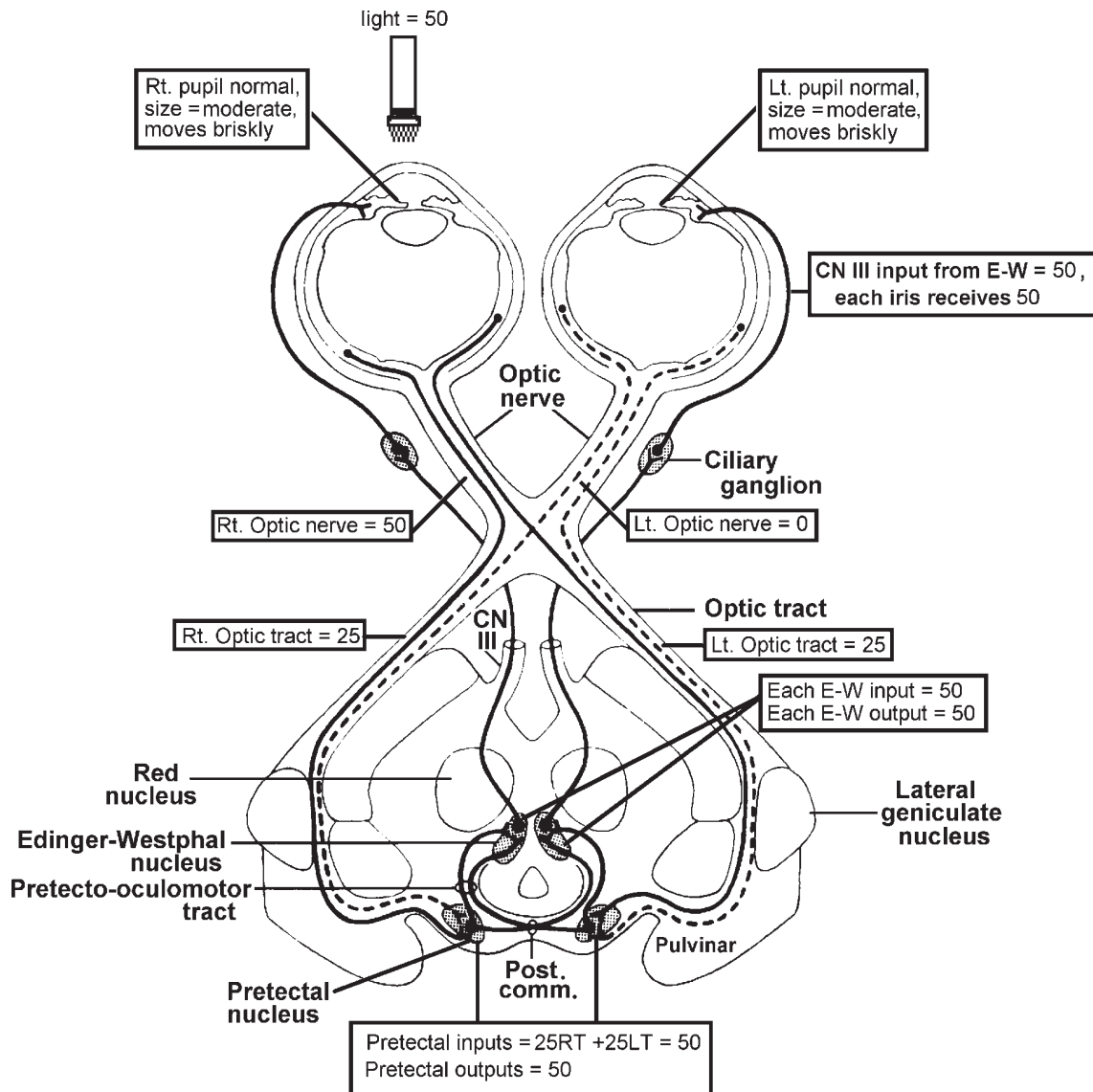


What this means in practical terms can be explained by examples, using an arbitrary unit of light intensity, let's say 100 units. If you shine 100 units of light onto the right eye, and the left eye is in darkness, each pretectal nucleus sees 100 units (because the information is shared), each pretectal nucleus sends 100 units of signal to each Edinger-Westphal, and the EW nuclei EACH send 100 units of pupillary constriction power to EACH pupil. So the pupils constrict 100 units worth of constriction and are EQUAL sized.



Normal situation: 100 units of light are transduced by the right eye into 100 units of optic nerve signal, the pretectal nucleus converts this signal into 100 units of signal to the Edinger-Westphal nucleus, the E-W nucleus responds with 100 units of constriction signal to the ciliary ganglion and on to the iris sphincter muscle. Both irides are small and symmetric.

If the source of light shining on the right eye is now decreased to 50 units, and the left eye remains in the dark, each pretectum receives 50 units of light, the EW nuclei receive 50 units of signal, and the pupil receives 50 units of constriction power, and each pupil DILATES, SYMMETRICALLY. Even though only the right eye light changed in intensity, BOTH pupils dilate symmetrically. Switching the light back to 100 units of brightness would cause both pupils to constrict again.

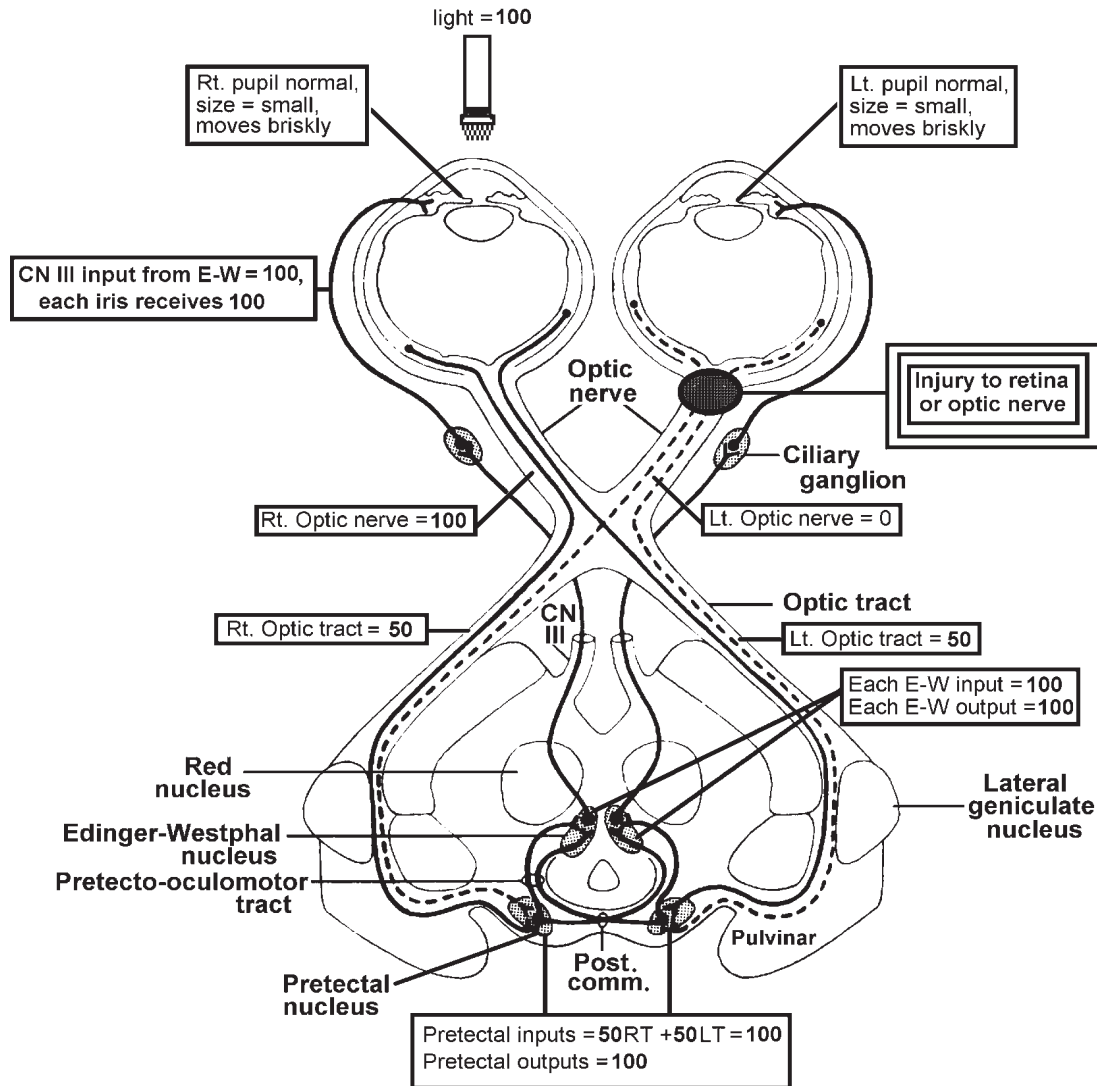


Normal situation: 50 units of light are transduced by the right eye into 50 units of optic nerve signal, the pretectal nucleus converts this signal into 50 units of signal to the Edinger-Westphal nucleus, the E-W nucleus responds with 50 units of constriction signal to the ciliary ganglion and on to the iris sphincter muscle. Both irides are moderate-sized and symmetric.

Let's now postulate that the left eye has some sort of damage. For ease of comparison, let's say that half of the neural output of the eye has been lost - it could be that half of the rods and cones have been lost, or that trauma has rendered half of the optic nerve fibers ischemic and nonfunctional, or a tumor is pressing on half of the nerve. The iris of both eyes is normal. For the example, the right eye optic nerve and retina are normal.

Let's look at the pupils in natural room light. Since the central "summary" system and the iris of each eye is normal, we see that the pupils are mid-sized and symmetric. Let's define this state as 0 units of brightness. Now, let's shine a light of 100 units of brightness on the right eye. The right retina and optic nerve are fully functional, and so they transduce 100 units of brightness into 100 units of neural signal, and send all 100 units of signal to the central summary system. The summary system receives 0 units of brightness from the left eye (still in the dark) and so the total signal reaching the summary system is 100 units. The system is undamaged in our example, and so sends 100 units of pupillary constriction signal to the pupils. Since this is more than before the light was turned on, both eyes symmetrically constrict, to a large degree (100 units of constriction).

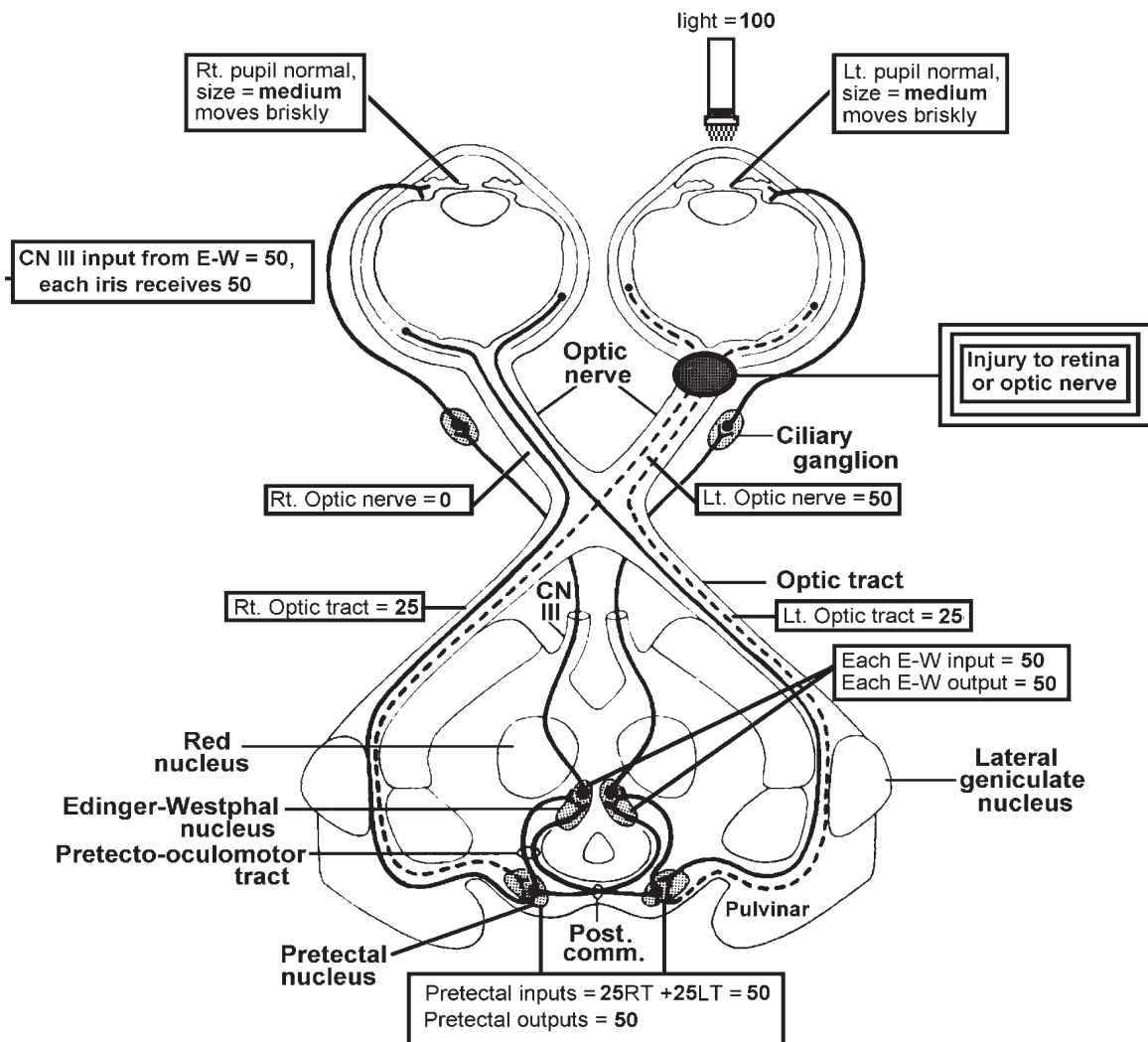
LEFT OPTIC NERVE DAMAGE, WITH OTHER CIRCUITRY NORMAL-
Light source on RIGHT Eye



Left Optic Nerve Damage situation : 100 units of light are transduced by the right eye into 100 units of optic nerve signal, the pretectal nucleus converts this signal into 100 units of signal to the Edinger-Westphal nucleus, the E-W nucleus responds with 100 units of constriction signal to the ciliary ganglion and on to the iris sphincter muscle. Each pupil is normal and responds to this stimulus with tight constriction. The pupils are symmetric and small.

But what if the light is moved to the left eye? We have said that the retina or optic nerve has been damaged such that only 50% of the usual cells are functional. So 100 units of brightness is transduced into 50 units of neural signal which is sent to the central summary area. The right eye is now back in relative darkness (arbitrarily denoted as 0 brightness units) and so the right eye output is now back to 0. The total signal sent to the central processing center is now 50. The center is working well, and so sends only 50 units of constriction power to the pupils. Compared to both eyes being in darkness, the pupils are constricted a little, but compared to when the light was shining on the right eye, the pupils are less constricted (or you may think of it as more dilated). That's because the best information the central processing system has is that the light is now half as bright.

LEFT OPTIC NERVE DAMAGE, WITH OTHER CIRCUITRY NORMAL-
Light source on LEFT Eye



Optic Nerve Damage situation : 100 units of light are transduced by the right eye into only 50 units of optic nerve signal, the pretectal nucleus converts this signal into 50 units of signal to the Edinger-Westphal nucleus, the E-W nucleus responds with 50 units of constriction signal to the ciliary ganglion and on to the iris sphincter muscle. Each pupil is normal and responds to this stimulus with moderate constriction. The pupils are symmetric.

Notice: This is exactly the same appearance as if the entire system were normal, but the light turned down to 50 units of brightness.

This would seem a pretty easy system to clinically test - lights and pupils are easily adjusted and measured. How do we clinically test this reflex loop?

Pupillary Light Reflex Test: Methods

A very important thing to know when discussing pupillary light reflexes is that no two people have the same pupillary diameter or the same response to light. In fact, it is easy to notice when examining a steady stream of patients in ophthalmology clinic, that older people have smaller pupils than younger people. So the amount of constriction isn't even constant for the same person over a lifetime. This makes it very hard to create a standardized test to see if a given eye is normally reactive.

But, there is one eye that should be able to serve as a reference for a given eye, an eye that has undergone all the same variables of aging, of interpersonal differences. The other eye is a perfect control for any given eye for which function is in question. Odds of having a tumor press on an exactly equal number of fibers from both eyes, (we'll discuss an exception to this later) or having trauma affect an exactly equal number of rods, cones, or optic nerve fibers is extremely low. So the "other", presumably uninjured (or less injured) eye can be used as a control. We simply compare one eye to the other. Because of this, the clinical pupillary light reflex test is called "relative." That is, one eye relative to the other (as opposed to one eye relative to a standard objective control).

The second part of the name of this test is "afferent." In this test, we presume that the efferent arm of the loop (the pretectum to EW nucleus to CN III to iris muscle) is intact.

{ Actually, as you will see, the first portion of this exam is done on each eye alone, to directly establish the integrity of the efferent arm }.

The third part of the name of this test is "pupillary," for obvious reasons.

The last word of the name is "defect", implying a positive (loss of normal reaction) finding.

Here's how it is done.

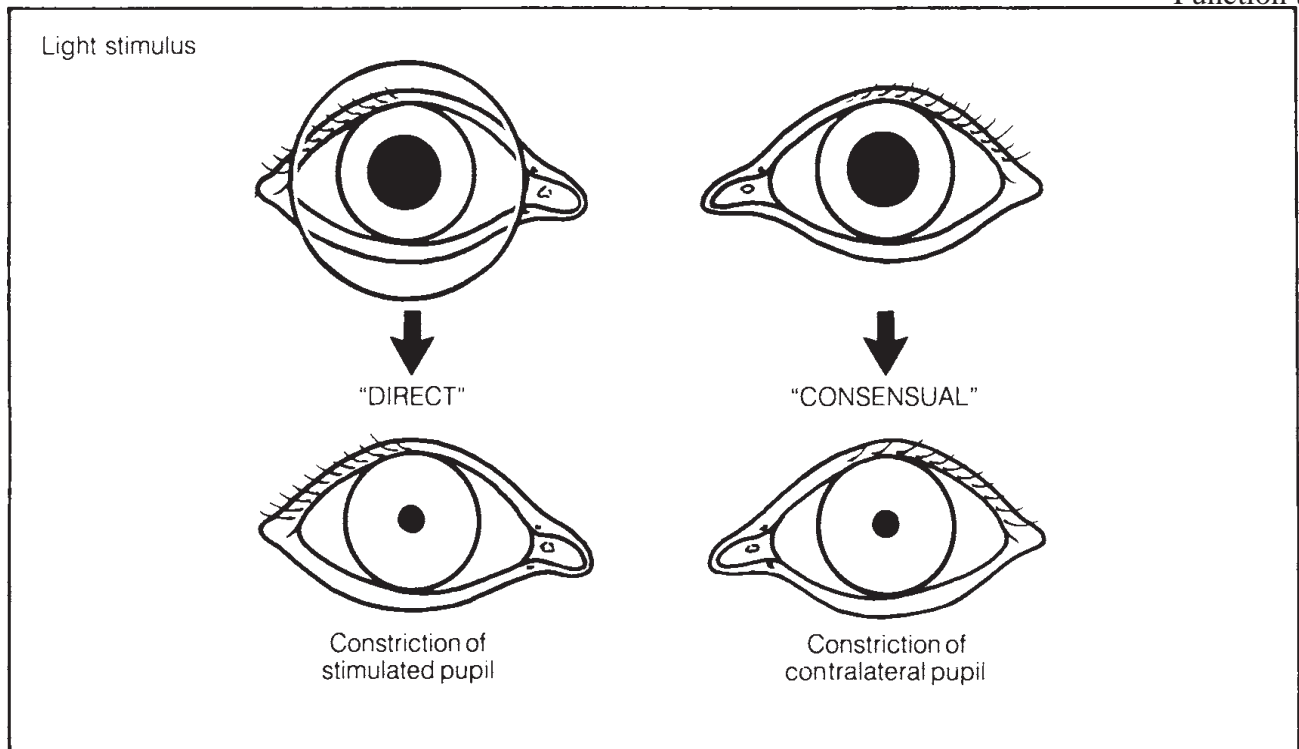
STEP ONE: Establish if the iris muscle is working - if the muscle is injured, testing for a normal pupillary reaction is futile.

METHOD: Watch right eye. Notice pupil size in darkness. Shine light on right eye. See if pupil constricts. If it does, then the loop is at least partly intact. Pupillary constriction does not rule out a lesion, because remember that in our example above, the left eye still constricted a little, even though half of the fibers were damaged. We still don't know if the right eye is injured, but we know that there will be at least some constriction to test in the next steps.

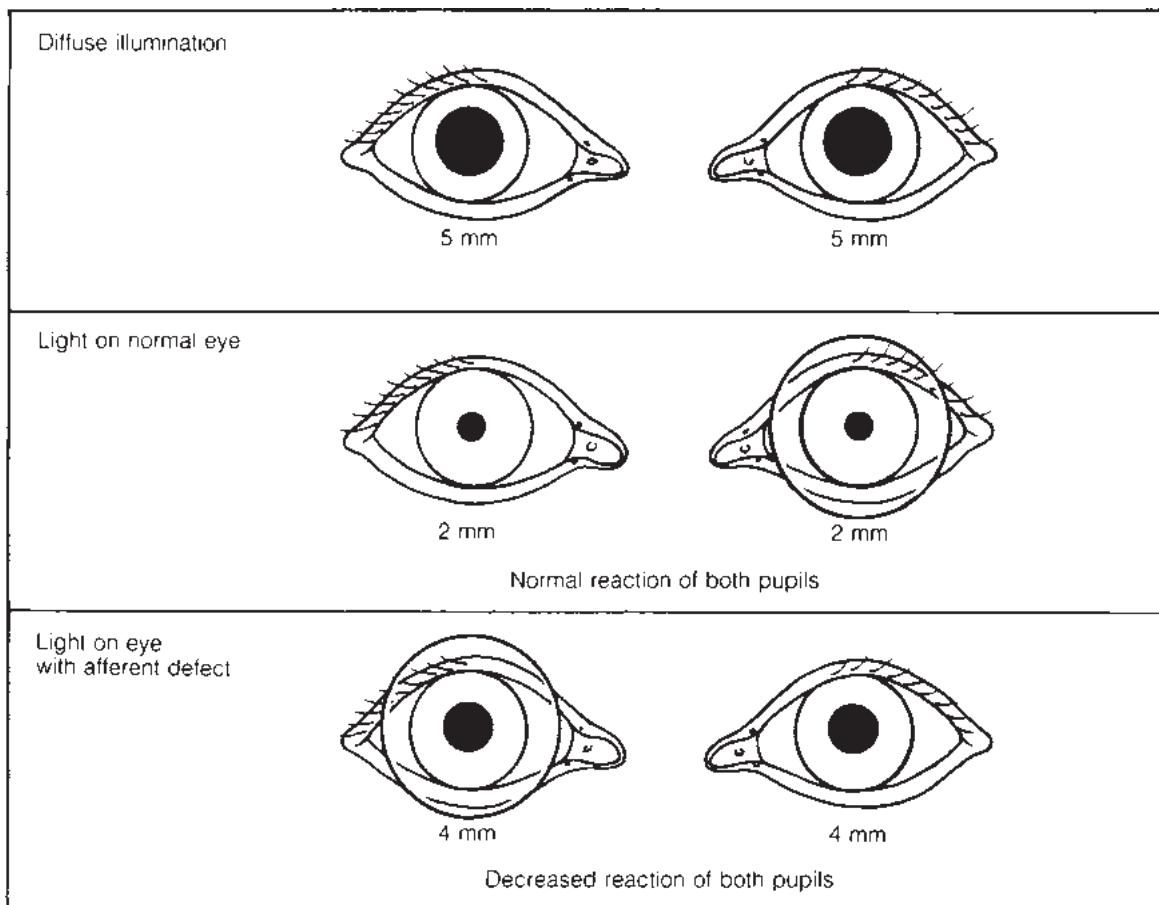
STEP TWO: Repeat for left eye, note if it constricts.

STEP THREE: Compare the two eyes to each other.

METHOD: Take the same light source. Start with the eyes in darkness (room lights down, the light source turned on but aiming over the patient's shoulder and not in either eye). Watch the right pupil. Move the light to the right eye - you should see constriction, as in step one. Now, keeping the light steady on the right eye, move your attention to the left eye, now in darkness. While watching the left eye, swing the light onto the left eye as smoothly and quickly as possible. What did the pupil do? Keeping the light steadily on the left eye, shift your attention to the right eye, and then quickly and smoothly swing the light back onto the right eye. What did the pupil do?

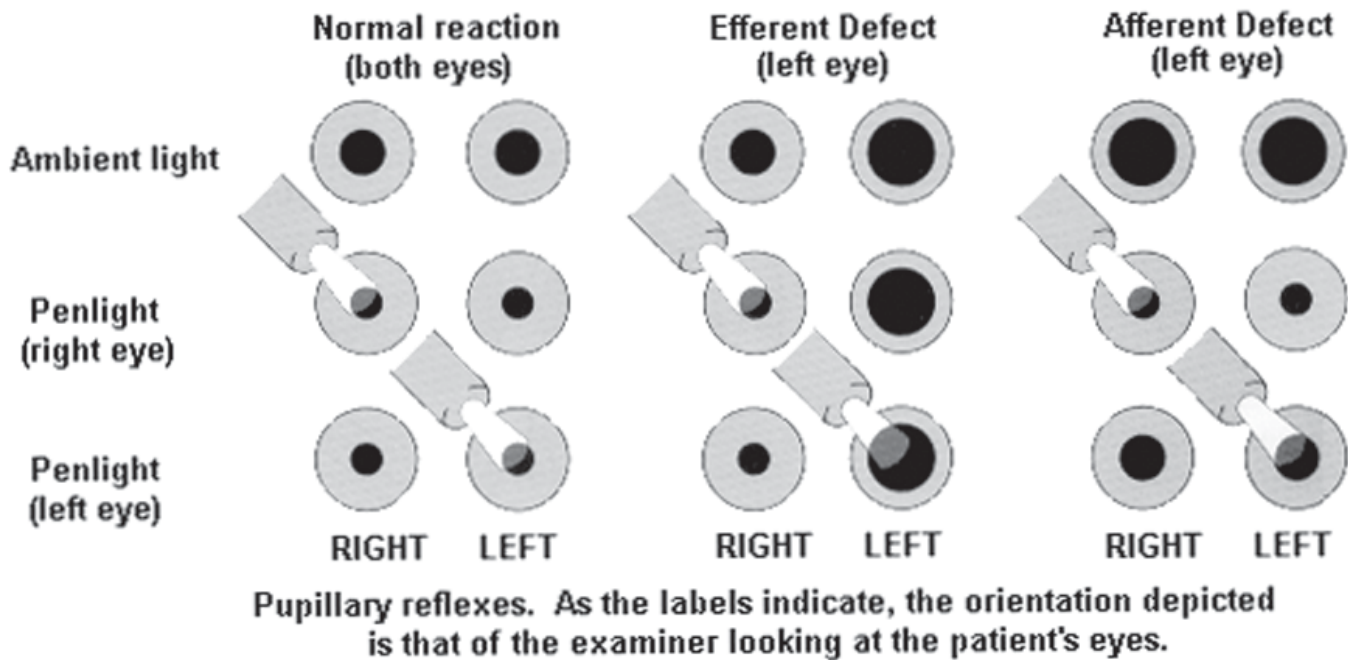


If there is no afferent-system damage, each pupil will be symmetric when the light is on either eye, and you will see **NO CHANGE** of pupil size as the light swings back and forth.



If you have afferent-system damage in one eye, you will have less signal generated when the light is on that eye. The brain will "think" that the light got dimmer, and **BOTH PUPILS WILL DILATE** slightly. When swinging the light back onto the good eye, the signal will go back up to normal, the brain will see more light signal, and **BOTH PUPILS WILL CONSTRICT** slightly.

STEP FOUR: If one pupil is nonfunctional due to efferent-system damage, you will see less/no constriction on that side. So can you still do this test? Sure. Remember that we have assumed that the efferent system is intact. Remember how many times the information crosses the midline - the output to each eye is **SYMMETRIC**. So if you gather no information about the eye with the efferent damage, where else can you look to see if that same eye has afferent-system damage? The **OTHER** eye, which is receiving identical efferent information.



METHOD: After establishing that one eye has efferent-system damage (e.g. the pupil is torn from blunt trauma), ignore that eye for the swinging-flashlight test. Keep looking at the eye with the working pupil, and if you see dilation in the good eye when the light is shining on the bad eye, that means that the nerve signal from the bad eye is less than the good eye.

DEFINITION: This test is called the Relative Afferent Pupillary Defect test. A positive test is when the pupils dilate symmetrically (when the light is on the bad eye) and constrict symmetrically (when the light is on the normal eye) when a light source is rapidly swung back and forth onto each eye.

This test is very commonly used in testing ocular function. If you don't understand this test, read this section again. You will use this test in emergency rooms as part of the universal evaluation of head injury patients. Be sure you understand it well.

Disease States: Visual Fields and Pupillary Reflex Tests

Now that you have seen the visual circuitry, and understand how the system is tested in clinical practice, let's go through several examples of clinical diseases and show how each disease affects these tests of visual function.

RETINAL DETACHMENT

DEMOGRAPHICS/INCIDENCE - **Retinal Detachment**

All ages are equally affected, with people who are nearsighted more likely to be affected. (Because, as explained earlier in this course, myopic eyes are of longer dimension and tissues are spread more thinly over the interior surface of the larger eye). Incidence is approximately 1 in 1,000 people/year.

CLINICAL PRESENTATION: SYMPTOMS - **Retinal Detachment**

As the retina pulls free from the retinal pigment epithelium, the physical traction on the retina irritates the nerves and results in spontaneous action potentials. These are interpreted by the brain as light. These patients see "flashing lights", often as a shower of sparks, in the area corresponding to the retina which is detaching (or detached and floating free in the vitreous cavity). In areas where the retina has detached, the patients experience a marked loss of vision. Since the tissue pulls free in a fairly defined area, they see this as a "shade". As the process involves more of the retina, the "shade" appears to be "pulling down" across the vision.

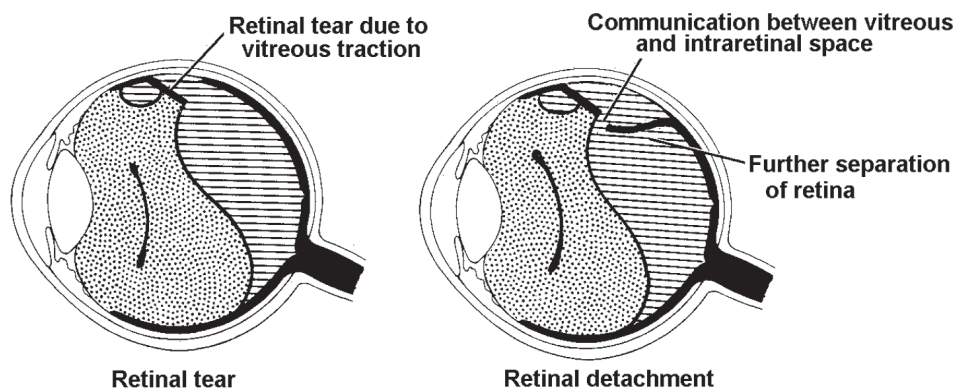
CLINICAL PRESENTATION: PHYSICAL EXAM - **Retinal Detachment**

When these eyes are examined, it is possible to detect that the retina is elevated, looks white from ischemia of the rods and cones, and as the eye moves during the exam, you can actually see the tissue floating in the vitreous fluid. There is no good analogy, but the closest thing I can describe is seaweed in gentle waves.

MECHANISM OF DAMAGE - **Retinal Detachment**

The retina is held to the retinal pigment epithelium by fairly little physical attachment. The interdigitation of the RPE cells and rods/cones is the largest force. If a thinned area of the retina should become a hole, fluid in the vitreous cavity can flow through the hole and force the rods/cones to separate from the RPE cells. Remember that the outer retina receives oxygen and nutrition from the choroid and RPE. If the retina detaches, the rods and cones are separated from the RPE and choroidal capillaries. The photoreceptors become ischemic and dysfunctional, and there develops a corresponding portion of the peripheral visual field which is lost (the "shade"). Even if this process is rapidly repaired surgically, the rods and cones rarely survive well, and the vision is usually worse in areas which have detached.

Areas which have not detached are usually completely unaffected and therefore normal. It is important to intervene quickly, in order to minimize the amount of permanent visual loss.



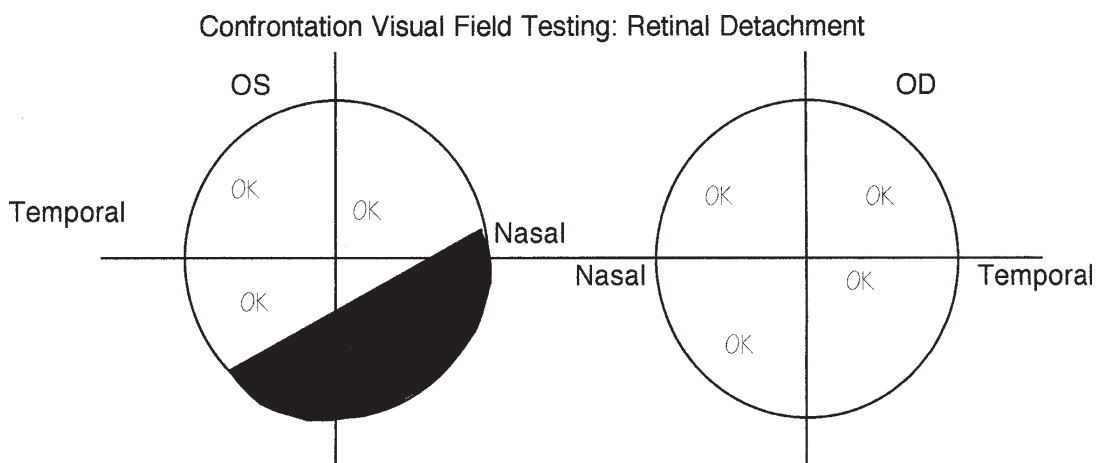
In the left drawing, gravity is pulling the vitreous to the bottom of the eye. This downward force is transmitted to the retina by the normal vitreo-retinal adhesions, and may cause a tear within the retinal layers. Since vitreous is partly free fluid, once the tear has occurred, vitreous fluid can flow into the tear, under the retina, and cause (or "allow") extension of the detached area. Most retinal tears are in the upper half of the retina, due to this effect of gravity.

VISUAL ACUITY - Retinal Detachment

If the central retina is uninvolved, the visual acuity is completely normal. If the central retina is detached, the vision may drop to the level of barely being able to count fingers held a few inches from the eye, or perhaps only able to detect if a bright light is on or off. (You'll see it written in charts as "Light Perception" or "LP").

CONFRONTATION VISUAL FIELD TESTING - Retinal Detachment

Testing visual function by holding fingers in the periphery of vision reveals marked loss of good vision in the areas corresponding to the retinal detachment. The patients **may** still be able to count the fingers in the detached retina areas, but they answer much more slowly and are clearly struggling to answer correctly. In undetached areas, the answers come quickly and accurately.

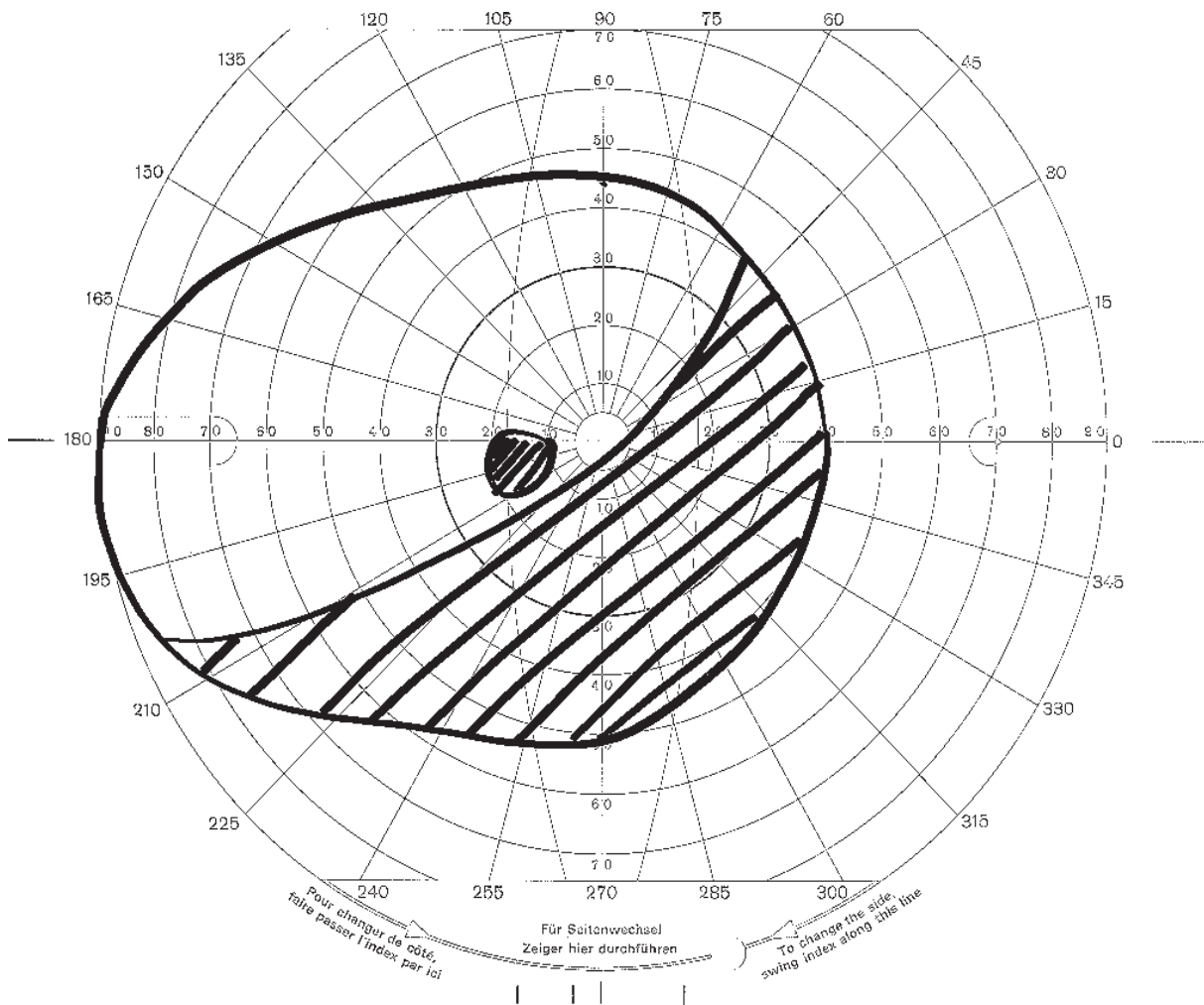


Confrontational Visual Field Testing: Retinal Detachment

In this patient, the superior-temporal portion of the retina in the left eye is detached, causing an infero-nasal loss of the visual field. The examiner has noted the decreased field with the darkened arc across this portion of the left eye field. Note well that the area lost does not strictly follow the vertical nor the horizontal meridia. Other disease states cause defects which DO follow these meridia.

FORMAL VISUAL FIELD TESTING - Retinal Detachment

Testing visual field function with field machines quantitates the lost vision with much more precision. The area of detached retina will always have markedly decreased visual field, and it may be relative (able to detect the brightest, largest light in areas formerly able to detect far dimmer lights) or absolute (able to detect NO light, no matter how bright it is made). Often, the edge is very sharp - in one zone, no lights are seen, and in the immediately adjacent zone, even the dimmest light is seen. Remember that this is called a “steep” defect.



In this hand-drawn field, I have included a single test-spot, and indicated that the blind spot is normal, and that the retina is detached in a shape that looks like a drop of water, with smoothly arcing edges. This is typical for a retinal detachment. Note especially well that the defect extends all the way to the periphery of vision, and that it crosses the vertical midline and horizontal midline. Other diseases that we will discuss later are famous for NOT crossing these midlines. If both midlines are crossed, a disease of the retina is almost always the cause. This will be clearer to you in a later section.

RELATIVE AFFERENT PUPILLARY DEFECT TEST - Retinal Detachment

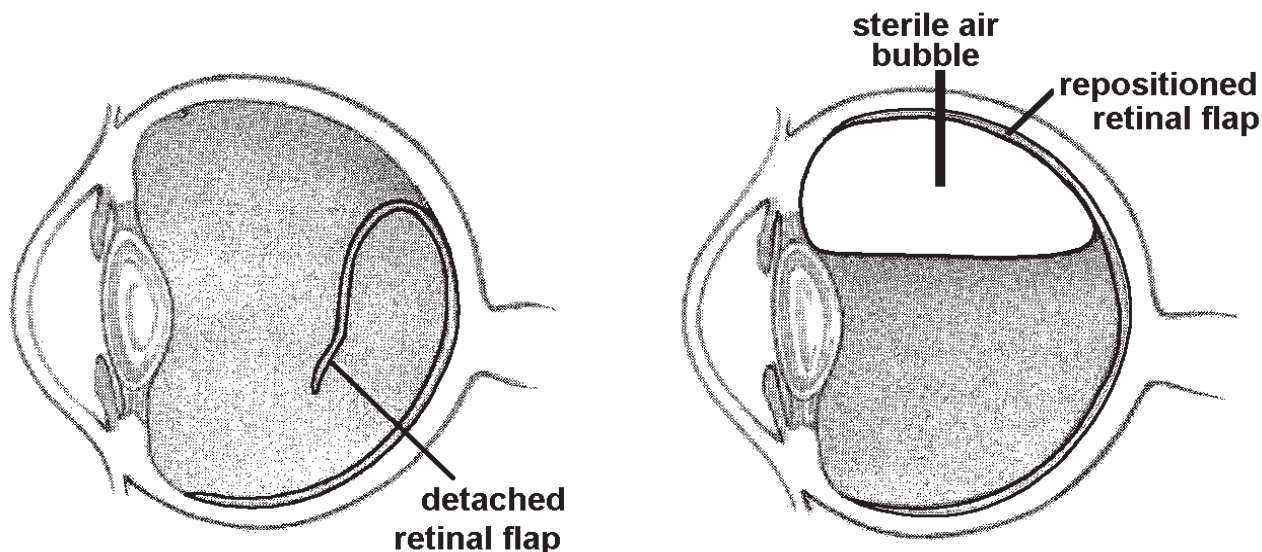
If a large enough area is affected, the amount of optic nerve output from a given amount of light will be reduced. We said that the afferent-defect test was fairly sensitive to detect a difference between the two eyes. Here's where the rubber hits the road - how much retinal detachment is the least that can be detected as a positive swinging-flashlight test?

It turns out that to be reliably interpreted as positive, HALF of the retina must be detached. Less than half results in a variably positive test, depending on PRECISELY where the light was shined, and exactly what proportion of the light fell on working/dysfunctional cells.

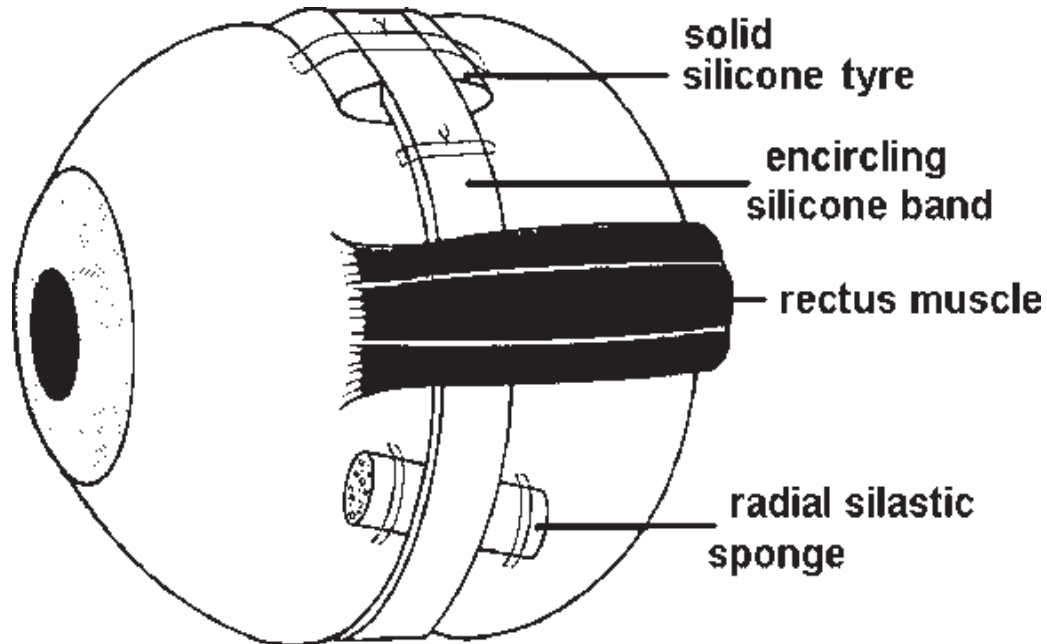
This patient, since less than half of the field was damaged, would have a NEGATIVE, or normal, swinging flashlight/relative afferent pupillary defect test.

TREATMENT - Retinal Detachment

The treatment of retinal detachment is urgent, and involves reapproximation of the two layers of RPE and retina. Two major approaches are taken: cut a hole in the sclera to drain the fluid that has seeped between the two layers: and seal the hole in the retina. Sealing the hole can be done by causing scarring with laser or freezing therapy. In order to keep the hole closed, the two layers need to be closely touching while the scarring occurs. Surgeons can do this by placing a bubble of gas in the eye to push the retina out to the sclera (below), or put a band of silicone tightly around the sclera to push it in to the retina (called a scleral buckle for its resemblance to tightening a belt buckle; opposite page.)



In this patient, the superior retinal break has caused a flap of retina to have detached. A bubble of sterile air has been placed within the vitreous cavity to push the retina back against the pigment epithelium, and then freezing therapy or laser spots are placed around the hole to keep fluid from passing through the hole under the retina.



If it is untenable to push the retina back out to the sclera/RPE, the sclera can be pushed in to meet the retina in the eye. A “buckle” of silicone material is placed around the entire eye and pulled tight until the sclera touches the retina. Then laser or freezing therapy is placed around the tear to keep fluid from leaking under the retina again. The buckle material is left in place indefinitely.

As you might guess, the patient’s glasses change significantly after this surgery, since the distance from the cornea to the retina is increased. (Quick question: Do you think this makes the patient more near or farsighted? If you aren’t sure, refer to earlier in the vision module.)

These treatments are usually done on the same day as the patient presents for evaluation, unless the fovea is already detached. In that case, there is little hope of preserving 20/20 vision, and these cases can be done early the next morning, in a more leisurely fashion, without causing worse visual outcome.

If the fovea is not detached, final acuity is unchanged from pre-detachment levels.

If the fovea is detached, it is rare to achieve a final vision better than 20/100.

OPTIC NERVE TRAUMA

DEMOGRAPHICS/INCIDENCE - **Optic nerve trauma**

Trauma to the optic nerve usually is associated with severe damage to the entire head/face region. The bony optic canal is well-fortified and only rarely is the optic nerve affected.

CLINICAL PRESENTATION: SYMPTOMS - **Optic nerve trauma**

Trauma to the optic nerve is usually quite symptomatic. Often, these patients have had significant, obvious craniofacial trauma. As such, they frequently are comatose in intensive care units for several days/weeks before regaining consciousness sufficiently to be able to document that their vision has been damaged. It is rare that we diagnose optic nerve trauma as an explanation of decreased vision in a routine outpatient clinic visit. The visual symptoms are usually significant enough that the patients come in emergently.

By whichever method the patient presents to the ophthalmologist for evaluation, the complaint is usually very generic: "My vision is bad."

CLINICAL PRESENTATION: PHYSICAL EXAM - **Optic nerve trauma**

The physical exam of these patients is usually most significant for the associated trauma to the face and head. The eye is very well protected in the cone of bone that makes up the orbit, and the force required to rupture or shear the optic nerve will usually cause zygomatic arch fractures, frontal sinus fracture or temporal bone fracture, and the corresponding deformation and lacerations are fairly evident.

In the eye itself, the blunt component of the trauma may:

- break iris blood vessels, causing blood to mix with aqueous humor ("hyphema"),
- cause tears in the iris sphincter muscle causing an irregular or immobile pupil,
- rupture the lens attachments to the ciliary body (the zonules), allowing the lens to fall loosely into the vitreous,
- cause a retinal detachment,
- cause bleeding under the retina in the choroid ("Choroidal rupture")
- cause the nerve to tear free from the eyeball at the lamina cribrosa ("optic nerve avulsion")

Outside of the eyeball itself, the damage within the bony eye socket may:

- cause blood to pool beneath the periosteum,
- rupture the orbital walls, connecting the ethmoid, maxillary or frontal sinus with the contents of the orbit ("blowout fracture")
- create sharp bone fragments capable of shearing the optic nerve, usually in the optic canal between the intracranial fossa and the orbital cone of muscle.

Direct physical exam of the entire eye, including testing of range of motion of the extraocular muscles, is very important in evaluating these cases. Ultrasound, CT and MRI are all very useful methods of determining amount of damage to the ocular structures.

MECHANISM OF DAMAGE - Optic nerve trauma

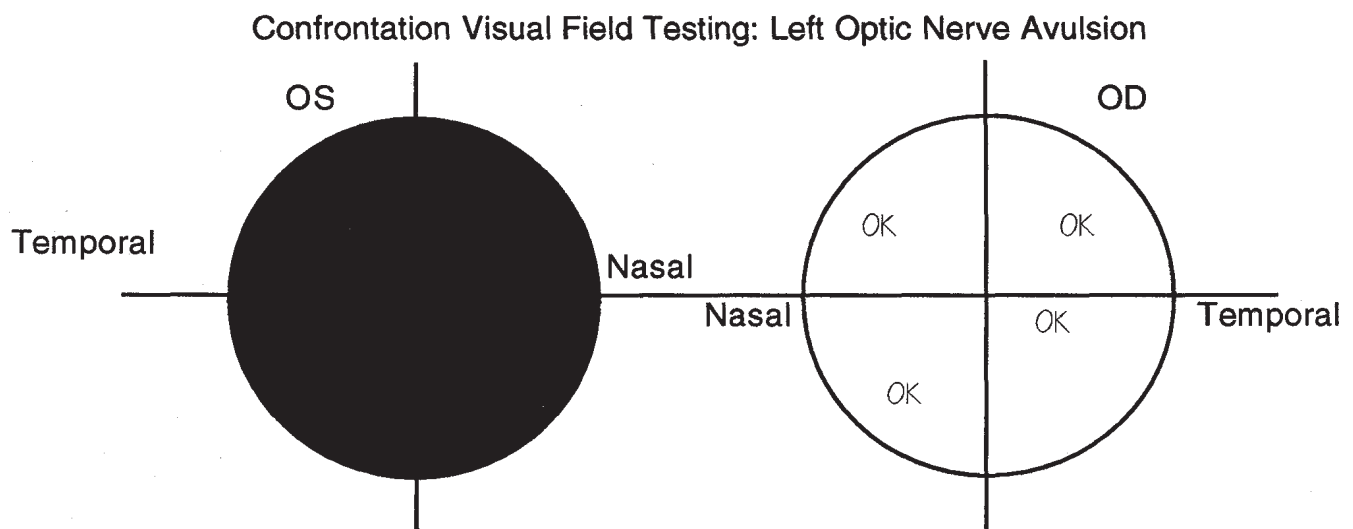
The mechanism of damage is either direct trauma, with tearing or laceration of the optic nerve sheath and nerve tissue within, or rupture of blood vessels within the optic nerve sheath. The sheath, like dura mater in the CNS, is fairly indistensible. Blood can collect, at systemic blood pressure, within the sheath, and compress the nerve tissue until axoplasmic flow ceases, causing death of the nerve cells.

VISUAL ACUITY - Optic nerve trauma

Effect on visual acuity ranges from blindness (complete avulsion or laceration through the entire nerve) to barely affected (if only minor compression of the nerve by nerve-sheath hematoma). It is usually decreased significantly.

CONFRONTATION VISUAL FIELD TESTING - Optic nerve trauma

Confrontation visual field testing is often abnormal, in areas which correspond to the areas of greatest nerve damage. It is usually fairly easy to clinically detect the asymmetry of function if the other eye is uninvolved in the injury. Remember, confrontation testing requires an alert patient, and it may be that this test cannot be performed until quite a while after the injury, as the patient recovers.

**Confrontational Visual Field Testing: Left Optic Nerve Avulsion**

In this patient, the left optic nerve is avulsed, and the eye sees nothing. The right eye is unaffected, and sees normally.

FORMAL VISUAL FIELD TESTING - Optic nerve trauma

Like confrontation visual field testing, the results of machine-driven visual field testing are usually abnormal. There is a wide range of possible levels of abnormality, reflecting the wide range of damage which has occurred. Patient effort is required for this test also, and because the concentration level is so much greater for this test, it is sometimes not able to be done until long after the patient has recovered from the other injury. As such, it is not useful for initial diagnosis, but can be invaluable for long-term followup.

RELATIVE AFFERENT PUPILLARY DEFECT TEST - Optic nerve trauma

Let's assume for the purposes of this discussion that one optic nerve has been lacerated at the level of the optic canal, leaving NO damage to the third nerve, ciliary ganglion, iris, or retina. Only the nerve is damaged.

Recall that this test measures the output of one nerve compared to the other eye. If only one eye is damaged, and the other normal, this test will be blatantly positive. When the light is shining on the good eye, the amount of light sensed will be high, and the pupils will BOTH constrict tightly. When the light is swung to the side of the severed nerve, the pretectum senses NO light present, and both pupils widely dilate. This excursion from tight constriction to wide dilation can be striking.

TREATMENT - Optic nerve trauma

The treatment for optic nerve damage is to remove any cause of further progression (decompress the bony fragments away from the optic canal, drain any collection of blood, etc) and often to begin high-dose steroids to minimize the edema and further compression which may result. It is not possible to restore nerve function, since these nerves are part of the CNS and as such do not regenerate.

OPTIC NERVE (or Tract) COMPRESSION

DEMOGRAPHICS/INCIDENCE - **Optic nerve compression**

Incidence of optic nerve compression is extremely low - the scale is on the order of 1:100,000 per year.

CLINICAL PRESENTATION: SYMPTOMS - **Optic nerve compression**

There are two main causes of optic nerve compression: tumor and aneurysm. Tumors can begin in the optic nerve sheath, at the meninges of the optic nerve canal, or in the CNS. Aneurysms with visual consequences can arise from the anterior communicating artery portion of the Circle of Willis.

Presenting signs of slow growing tumors can be eye/orbital pain, difficulty with eye movements (due to the bulk of the tumor), or decreasing vision. Presenting signs of fast growing tumors can be protrusion of the eye (due to the bulk of the tumor), and pain. If the origin of the tumor is the pituitary gland, it is useful to ask about signs/symptoms of endocrine abnormality (diabetes, gigantism, enlargement of mammary tissue etc).

CLINICAL PRESENTATION: PHYSICAL EXAM - **Optic nerve compression**

The physical exam of the eye may be notable for decreased ocular motility, protrusion of the eye compared to the contralateral eye, unusual curvature of the eyelid(s), increased eye pressure, signs of the usually round eyeball being indented by pressure from behind, or congestion of blood vessels in the conjunctiva and scleral wall. Some patients have no external abnormalities at all, and come in only with “bad vision”.

MECHANISM OF DAMAGE - **Optic nerve compression**

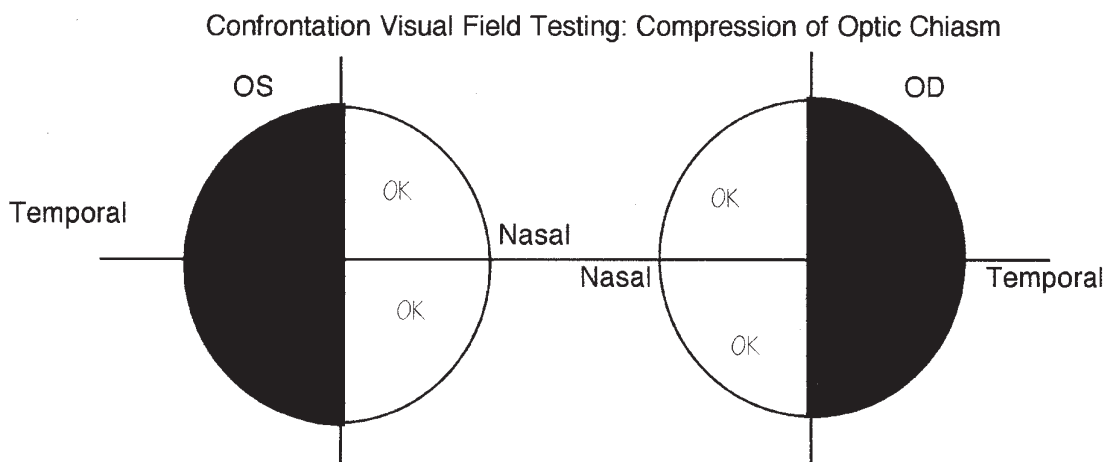
The mechanism of visual loss in optic nerve compression is impairment of both blood supply and axoplasmic flow. You have learned that nutrients and signal molecules flow from the cell body to the axonal dendrites, and back again. If the flow is obstructed, the cell nucleus atrophies and dies. Even if the problem is corrected, and the compression relieved, these cells do not regenerate. So long term compression causes atrophy of ganglion cells and optic nerve axons.

VISUAL ACUITY - **Optic nerve compression**

There are many sets of ganglion cell receptive fields in the fovea, and it takes quite a bit of damage to affect visual acuity. It is a late sign of compression, and not very sensitive at detecting subtle visual loss in chronic optic nerve compression cases.

CONFRONTATION VISUAL FIELD TESTING - **Optic nerve compression**

Confrontation visual field testing is much more sensitive at detecting compressive visual loss than acuity. It may show a defect in peripheral vision far sooner than acuity, but because of the coarseness of the stimulus (i.e. finger-sized), it is quite possible to miss subtle defects of vision if this is the only test used.



Confrontational Visual Field Testing: Bitemporal Hemianopia

In this patient, a pituitary tumor is pressing on the optic chiasm, causing the crossing fibers to be damaged. Since the nasal fibers cross, the temporal fields of both eyes are affected. Notice the strict adherence of the areas of loss to the vertical meridian. Any field loss with such strict adherence to the vertical meridian must be due to a disease process which is affecting the visual system at or behind the chiasm.

FORMAL VISUAL FIELD TESTING - Optic nerve compression

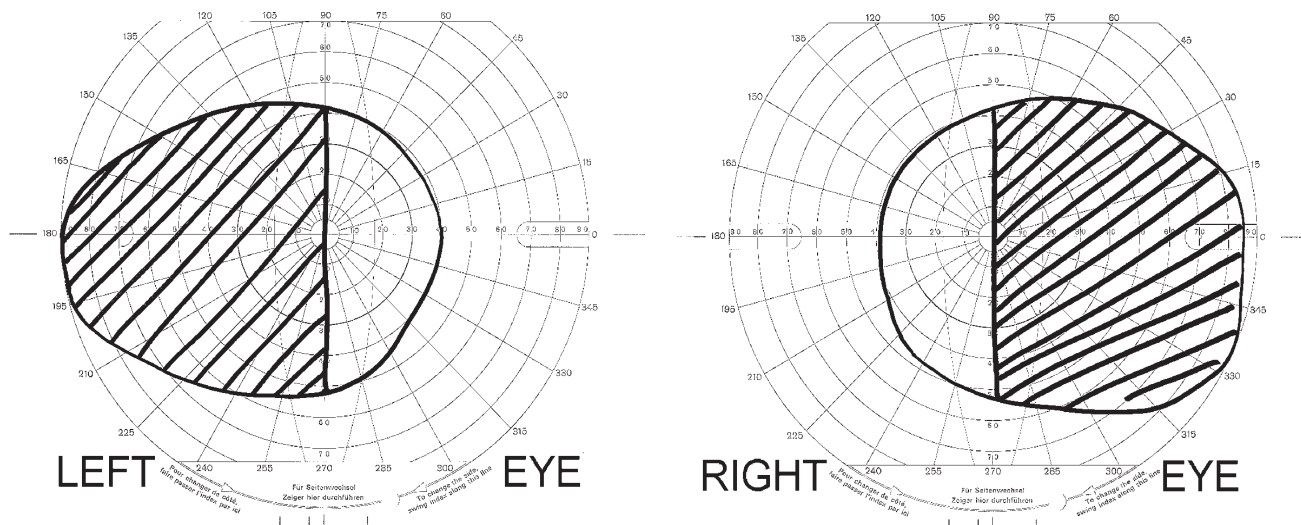
Machine-driven visual field testing is a very sensitive way to look for optic nerve compressive disease. The area of the nerve which is compressed can fairly easily be mapped, and the system is sensitive enough to detect a compression long before the patient will be affected by decreased acuity.

A classic visual presentation of nerve compression is pressure on the optic chiasm, where the fibers representing the nasal fibers (and hence temporal visual field) cross to join the fibers of the same visual field of the other eye, and to become the optic tract on its way to the lateral geniculate body. Because pituitary tumors and anterior communicating artery aneurysms can compress the same area, it is a classic finding.

Remember that the nasal fibers of each eye cross at the chiasm. If you were to cut all of these fibers, what would the visual field in each eye look like?

The nasal fibers “see” the temporal visual world of each eye. So if these fibers were lost, the temporal visual field of each eye would be blank. This is a classic presentation, and is called a bitemporal hemianopia (hemi = half, an= lack of, opia= vision) [Extra credit: some people still write this hemianopsia, others leave out the “s” without loss of meaning. You may see it either way, sort of like eucaryote and eukaryote]

This bitemporal hemianopia is classic for chiasmal compression, because there is no other place in the visual pathway for a single lesion to cause this result. And since pituitary tumors are not rare as endocrine tumors go, it is likely that you will see this kind of visual loss in one of your patients in your career.



This idealized manual visual field shows a single light-spot result, with normal nasal-field responses and NO LIGHT seen in the entire temporal field of both eyes. Note that the normal blind spots are hidden within the area of visual damage in the temporal field of both eyes

RELATIVE AFFERENT PUPILLARY DEFECT TEST - **Optic nerve compression**

This test can be tricky to predict in this disease process. It would seem that with half of the visual field gone in each eye, the pupils should respond only half as much, and this test should be positive, right?

Remember that the word Relative in the title indicates one EYE relative to the other EYE, and not relative to some external standard. Thus, if both eyes are affected equally, the damage is equal, and no relative difference is seen. It is COMMON to see a NORMAL swinging flashlight test and have COMPLETE bitemporal hemianopia. It is perhaps the only single-lesion disease process which may affect both eyes equally.

This disease process points up an important point of the method of the swinging-flashlight test - what if the examiner is sloppy, and doesn't keep the light directly in the center of each eye as it is swung back and forth? Let's say that the light is slightly to the right of center for EACH eye. In the left eye, that means that the temporal retina is getting more light than the nasal side, and the temporal fibers aren't crossing or being compressed, and so the light is "seen" as being pretty bright. Then the light swings over to the right side of the right eye's field, hitting more nasal retinal fibers than temporal, and since these fibers ARE being compromised, less signal gets to the pretectum, and the pupils dilate a little. This mimics a positive afferent defect test on the right side. Be very careful to keep the swinging flashlight in the exact center of the field of vision as you test each eye.

TREATMENT - **Optic nerve compression**

Treatment of compressive lesions can be active or passive. For optic nerve sheath tumors, for example, the treatment is often passive. Removal of these tumors so close to the nerve tissue can be very destructive to the surrounding normal tissue, and the patients can be blinded by the surgery. Since these tumors often are exceedingly slow-growing, they are often left alone and monitored with visual fields and imaging studies.

But pituitary tumors can be deadly, by way of massive upset of the endocrine system. Finding a pituitary tumor based on visual symptoms may catch these tumors in a fairly early stage when they can be irradiated, chemically treated, or physically removed/debulked.

Similarly, early detection of an aneurysm based on this kind of visual problem can allow surgical treatment or radiologic ablation before the aneurysm ruptures and causes serious neurologic deficit.

CORTICAL STROKE

DEMOGRAPHICS/INCIDENCE - Cortical stroke

“Stroke” indicates a sudden decrease in cortical function, due to alteration of blood flow to the cortex. The alteration can be a sudden decrease in flow, due to a blood clot or embolus (“ischemic stroke”) or due to rupture of a blood vessel with associated intracortical hemorrhage (“hemorrhagic stroke”). It is clinically difficult to separate which of the alterations has occurred, unless imaging studies are used to detect ischemic edema versus fresh intracortical blood.

The victims of stroke are often older patients with high blood pressure, diabetes, or high cholesterol.

CLINICAL PRESENTATION: SYMPTOMS - Cortical stroke

More than any of the other disease states we are discussing, there is a very wide range of clinical symptoms and areas of damage with stroke. The amount of bleeding or ischemia can be very small, causing a deficit that would go entirely unnoticed unless detailed visual field testing is done for some other reason, or massive, involving a whole side of the brain, with visual and systemic CNS effects.

Most times, if the stroke is small, and only visual cortex is involved, the stroke itself goes unnoticed by the patient.

But if the stroke is larger and involves a large portion of cerebral cortex, the patient will have symptoms that correspond to the damaged areas - loss of speech or memory, loss of coordination, strength or control of muscle groups, dizziness or imbalance, or unconsciousness.

As ophthalmologists, we are often asked to examine patients who have come for medical attention for non-visual symptoms. In the course of assessing the damage of the stroke, the primary care practitioner or neurologist asks the ophthalmologist to study the visual system for damage.

For purposes of a simple example for this course, I present a patient with a mild stroke, with only visual cortex involved, and only one portion of the calcarine fissure affected. Recall this diagram from Dr. Uhlrich’s presentation of the visual field effects of lesions in various locations. For this patient example, imagine that the lesion is in a small portion of the visual cortex.

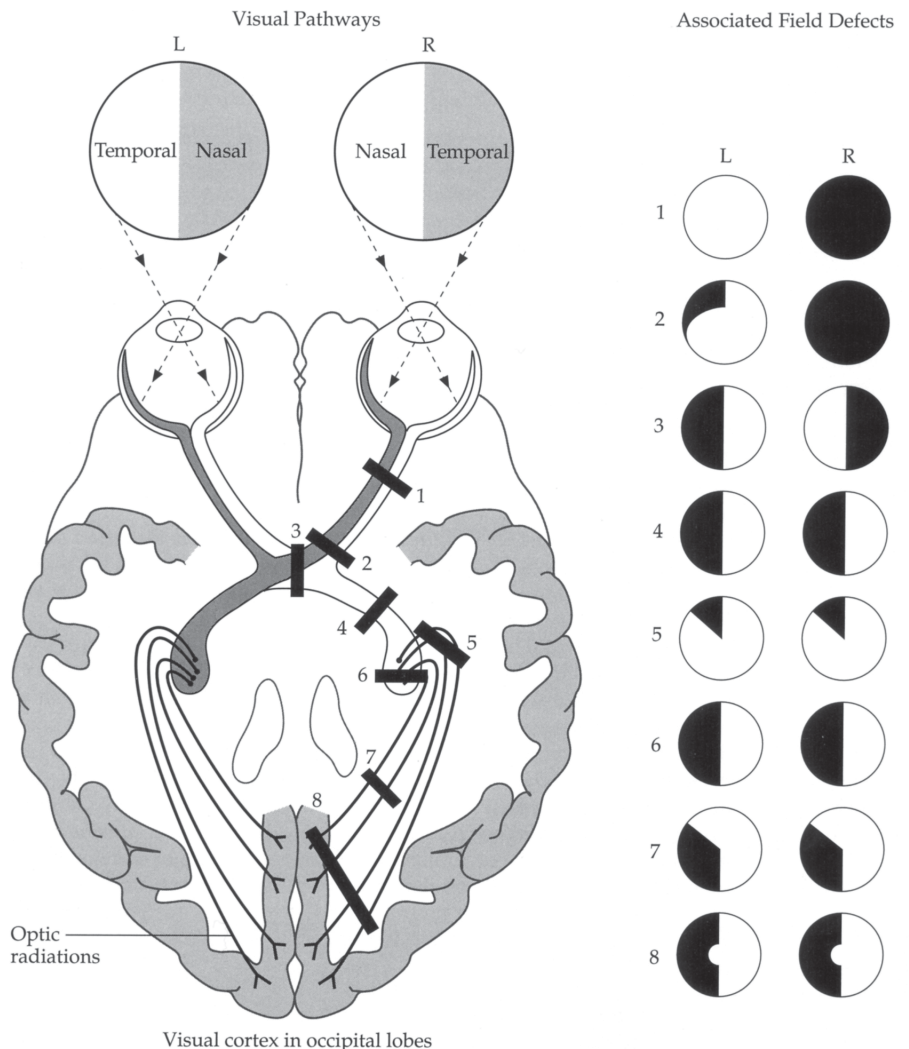


Illustration 7.3 Visual pathways with associated field defects. For the visual pathways, the view shown is a cross-section seen from above. Note that objects in the right half of the visual field form images in the left half of each retina and are transmitted to the left hemisphere. The numbers correspond to lesions in the visual pathways and to the field defects that result from these interruptions. For the purposes of the diagram, the fields shown reflect the effects of total interruption of the indicated structures. In actuality, partial impairment is more the rule than the exception. The key at the top of the facing page indicates the site of each lesion and the associated field defect.

CLINICAL PRESENTATION: PHYSICAL EXAM - Cortical stroke

For our example, the patient will have no other neurologic deficits. But if the patient has had damage to the temporal or parietal lobes or the basal ganglia, there may be symptoms of other cranial nerve dysfunction, memory or speech impairment, or unconsciousness. Remember the take-home message that the symptoms will be determined by the extent of the stroke location and that no two patients will present with exactly the same findings.

It is common for other physicians, acting as primary care givers, to ask for an ophthalmologic evaluation of stroke patients to “see what is left”, and fairly rare for these patients to present to us as the first indication that a stroke occurred.

On physical exam, there may be other signs of stroke, with alteration of speech, loss of cranial nerve function of CN VII-XII, etc.

On ophthalmic exam, there will be no sign of physical damage to the eyeballs themselves, and pupils will react well to light (see below). There may be subtle signs of vascular disease in the blood vessels in the retina, but usually no obvious sign of the bleeding or ischemia which is occurring in the visual cortex. Note that the ganglion cells are intact, since they synapse on the lateral geniculate nuclei and derive growth factors from these cells. No optic nerve atrophy is seen in cortical stroke.

Retinal blood vessel signs of vascular disease are small aneurysms of blood vessels, thickening of the muscular wall of the arteries (in response to the elevated systemic blood pressure), and in some cases, small hemorrhages of capillaries within the retinal surface.

MECHANISM OF DAMAGE - Cortical stroke

Other courses will describe the cascade of events that elevated blood pressure causes in the vascular system - for our purposes, it is enough to know that the primary causes of stroke are blood vessel rupture due to elevated blood pressure, or emboli from roughened internal vessel walls that flow downstream into the smaller-caliber arteries and clog them, denying blood to tissues. By either mechanism, the normal cycle of blood flow to capillaries and veins is interrupted, and the cortical cells become ischemic. An immediate response is edema, and late response is cell death. Cortical function is very metabolically demanding, and full function is lost very early in the process (within minutes).

VISUAL ACUITY - Cortical stroke

Visual acuity is often subtly compromised in strokes which involve the tip of the visual cortex. The cells of the foveas synapse in this area, in a very organized fashion. If this area is affected, visual response can be significantly lost. Because the blood flow to the two sides of the cortex responsible for the two fields of vision comes from two different vessels (i.e. the right and left posterior cerebral arteries) it is highly unlikely that BOTH sides will be equally affected.

The pattern that is seen, therefore, is that one HALF of foveal vision can be lost, and the other side be completely normal. With this normal half of foveal function, patients can do remarkably well on acuity tests.

EXTRA READING

The visual system is very good at pattern recognition - for example, as you read this sentence, your eyes are not stopping on every letter or every word as you scan the page. Groups of letters make shapes that we have come to “see” as words, without needing to “read” each letter therein. The brain simply “fills in” the rest. That’s why when you are proofreading your text, it is is easy to miss repeated words, especially small ones ones. Your brain has taken the shape of the word in context, supplied the meaning, and moved on, without you detecting the extra shape. This “visual completion” is evidenced by these patients who have lost half of their vision, split right down the foveal center. They are shown an “E” and “see” only the right half of the three horizontal bars, but know that the only letter which has such a right half is a capital E, and so “read” it as a full E. So their acuity is really very good, because they can fill in known shapes to complete a half-seen letter. They will only lose a line or two of the very smallest letters, despite having lost half of their “sight”. This does NOT mean that they can drive a car safely, however.

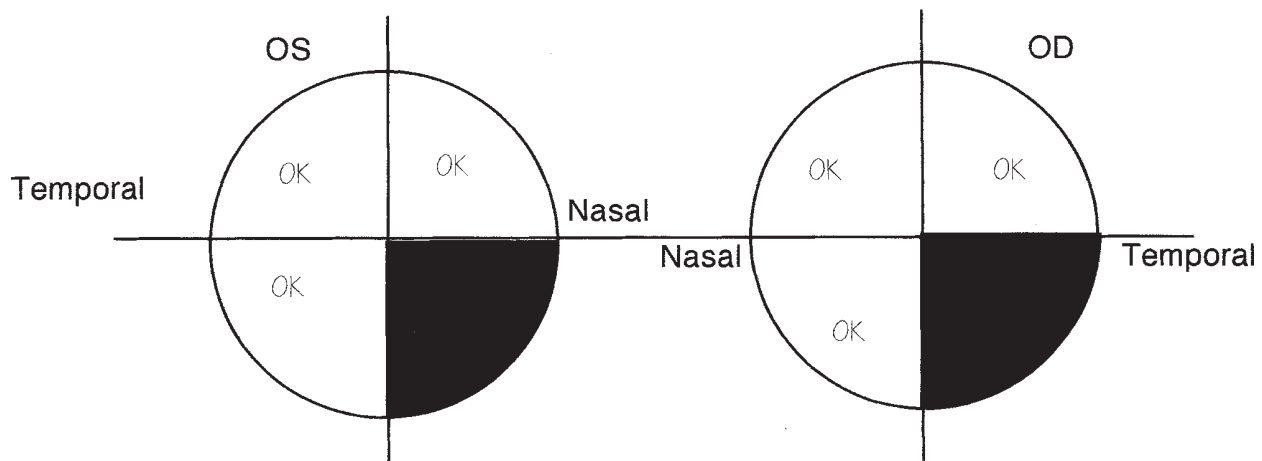
Summary: visual acuity will not be significantly affected.

CONFRONTATION VISUAL FIELD TESTING - Cortical stroke

This will often yield a great deal of information, since the area of lost vision will be fairly densely lost, and even coarse stimuli such as fingers will be missed in the damaged area.

Since the cortical stroke areas are beyond the chiasm, the lost vision will have an abrupt asymmetry at the vertical meridian. It is wise to check this vertical border above and below the visual center in many spots - if the stroke is small, the area which is abruptly abnormal may be hard to detect. But since many stroke patients are bedridden in the acute phase of the process, confrontation testing may be all that the examiner has with which to check visual function. Unfortunately, the patients must be able to respond that they have seen the fingers, so they must be conscious and alert. Fortunately, since many strokes affect verbal ability but leave other means of responses intact, the patients with verbal loss can still be tested. They can respond with hand

Confrontation Visual Field Testing: Cortical Stroke



Confrontational Visual Field Testing: Cortical Stroke

In this patient, a blood clot has stopped flow of blood to the left-side of the visual cortex, above the calcarine fissure. The opposite visual field is affected, so the lower-right field is lost. Since the damage is beyond the chiasm, strict adherence to the vertical meridian is seen.

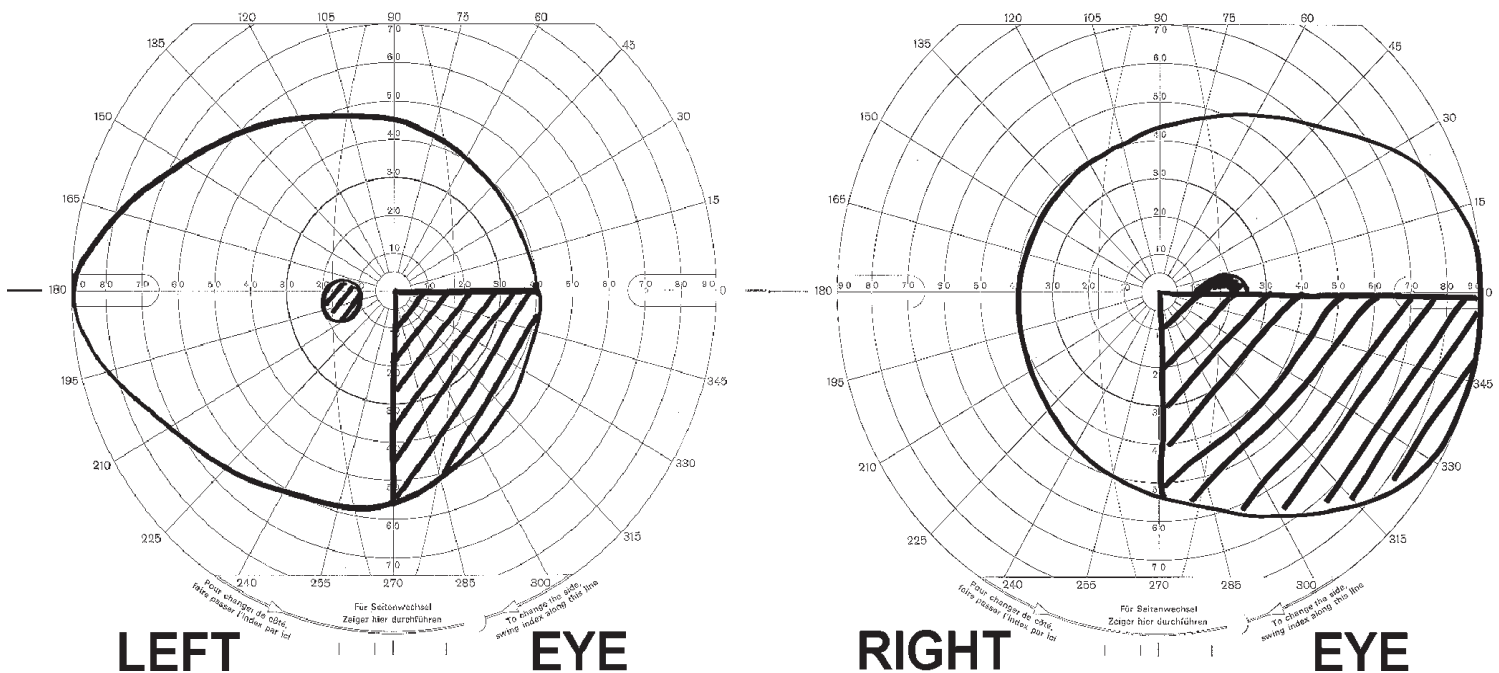
movements, can write answers, or in more severe cases, can blink once for every finger seen in a given area. It is an effective, but coarse, documentation of the stroke-affected area.

FORMAL VISUAL FIELD TESTING - Cortical stroke

Similar limitations of patient response apply as they do to confrontational visual field testing. Usually, motor skills are more important than verbal skills, as these machines typically use a thumb button as a means to indicate a positive patient response.

Within these limitations, machine-driven visual fields can map an area of loss with great precision, allowing the ophthalmologist to follow the patient carefully for any symptomless progression of the stroke area.

For both confrontational and formal field testing, the field defect seen is **HOMONYMOUS** - meaning the same left/right side of the field is lost in **BOTH** eyes. For example, if the entire right cerebral hemisphere were damaged with a massive stroke, all vision from the nerves on the right side of each eye would be lost. This means the right-sided nerves of the right eye (i.e. the temporal retina, nasal visual field) and the right-sided nerves of the left eye (i.e. the nasal retina, temporal visual field).



This idealized manual visual field shows complete loss of vision in the entire lower-right visual field of both eyes. Note that I have drawn complete adherence to both horizontal and vertical midlines, which is typical for occipital-cortical lesions. Note that since the nasal field of the left eye is affected and the temporal field of the right eye is affected, such that the blind spot in the left field appears normal, and half of the blind spot in the right eye is “buried” within the rest of the lost field.

CLINICAL PEARL OF WISDOM

NOTE EXTREMELY WELL how the field loss pattern will help you localize the lesion -

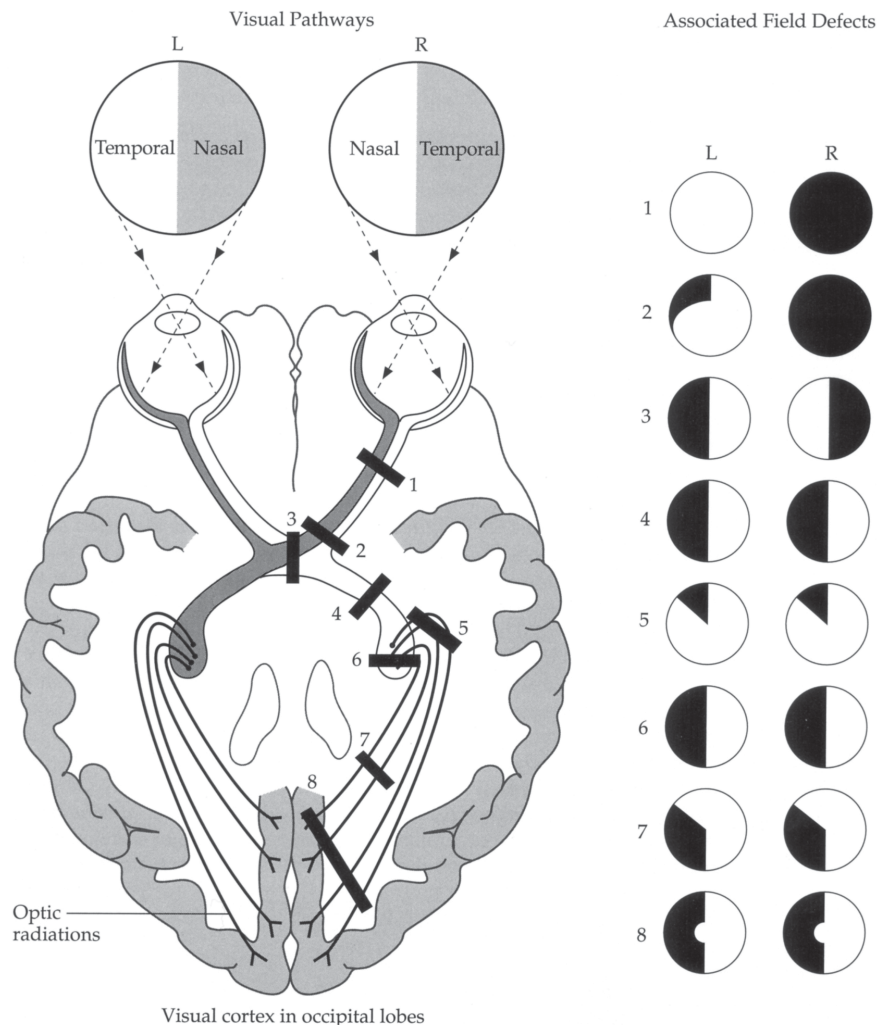
·if it is **HOMONYMOUS** (same left/right side in each eye) then **the lesion MUST be behind the chiasm**, for that is where the left/right organization of the fibers occurs).

·if it is **BITEMPORAL**, the **lesion must be at the optic chiasm**, for that is the only place that the nasal fibers/temporal field are close to each other.

·if it is in **ONE EYE ONLY**, the **lesion must be in front of the chiasm**, for that's where the fibers of the two eyes mingle and there is no place further towards the cortex where a single lesion can damage cells from one eye's field of view without simultaneously affecting the corresponding cells of the other's eye field.

NEVER answer somebody's question about "Where is the lesion" without looking carefully at the field of the OTHER eye.

Remember the diagram of the effect on the visual field of various locations of lesions? **Go back and review it now if this clinical pearl does not make sense to you.**



RELATIVE AFFERENT PUPILLARY DEFECT TEST - Cortical stroke

This test is tricky - you would presume that as long as you were careful to present the light to the exact SAME location in each visual field, that you would not get an afferent defect of note.

BUT - think more carefully - I submit to you that you will NEVER get an afferent pupillary defect in these people, no matter if you are somewhat careless or not.

Yikes. I said “never”, and I NEVER say that. How can I be so sure?

Remember a detail of how the pupillary light response was wired - fibers of the optic nerve SPLIT OFF from those going to the LGN, and instead synapsed on the pretectum.

Careful about that now - they split off, right? So it doesn't matter what happens to the visual cortex - for all the pretectum cares, it could be totally ischemic and the patient could be cortically blind - as long as those fibers from the retina to the optic nerve are intact, and those fibers from the pretectum to Edinger-Westphal to CN III to the ciliary ganglion to the pupillary sphincter are intact, there will be NO relative afferent pupillary defect.

Summary: In cortical stroke of any magnitude, there is NO RAPD.

TREATMENT - Cortical stroke

Stroke treatment is supportive - massive hemorrhages into the surrounding CSF or ventricles can be drained. Clots can be dissolved with thrombolytic agents (at the risk of inducing bleeding). Most important, blood pressure must be controlled and if the other cranial nerves are affected, the patient should be protected from aspiration of stomach content.

The most significant intervention to be made is to minimize the risk of further events by lowering blood pressure, instituting aspirin or coumadin therapy to avoid clots/emboli, and if an embolic source is presumed, to proceed with evaluation to find and rectify the source. Examples include: treatment of atrial fibrillation, replacement of a malfunctioning cardiac valve, coronary artery bypass grafting, etc.

Many patients regain function as the ischemia resolves, the edema abates, and the brain recruits other cells to assume the functions which were lost.

VERY OPTIONAL READING

MACULAR “SPARING”

By now, you have probably noticed the little central island of visual field that is maintained in a cortical visual pathway deficit. You have noticed that even though the ENTIRE half of the field is lost with a particular lesion, there is still a little circle of field that is left in the absolute center of the hemianopia. Why the heck is that there?

This effect is called macular sparing—the principle that when testing visual fields of people with a complete hemianopia, a central island in the macula is spared from being lost and appears really pretty well preserved. You know it should be a true hemianopia from all we have taught you, but it ain't.

Again, why the heck is that there?

This is a topic which is clinically IRRELEVANT, but is a cool headscratcher question. Here is what we teach the ophthalmology residents, as written by our two neuro-ophthalmologists, Dr. Richard Appen and Dr. Leonard Levin. I asked them to write down what they thought we should be teaching you. Here's what they wrote:

First, Dr. Richard Appen:

A striking feature of the cerebral vision pathways is the reliably consistent anatomic organization of the retina and the brain. The right half of the visual field of each eye is served by the left side of the retinas (temporal half of the left eye and nasal half of the right eye) and the left side of the brain. Similarly, the superior portion of the visual field acts on the inferior part of the retina, and that retinal location is represented by the inferior or caudal portion of the optic radiations, as in the temporal lobe. In the occipital cortex, where the optic radiations terminate, the most peripheral portion of the visual fields stimulates the most anterior part of the calcarine cortex, while the central part of the visual field (served by the macula of the retina) relates to the most posterior tip of the occipital cortex.

An absolute truism is that visual acuity is never affected when the visual field is damaged by a unilateral brain lesion that results in a homonymous hemianopia, regardless of how total is the homonymous hemianopia. Only half the macula and its cerebral connections are needed to provide 20/20 visual acuity.

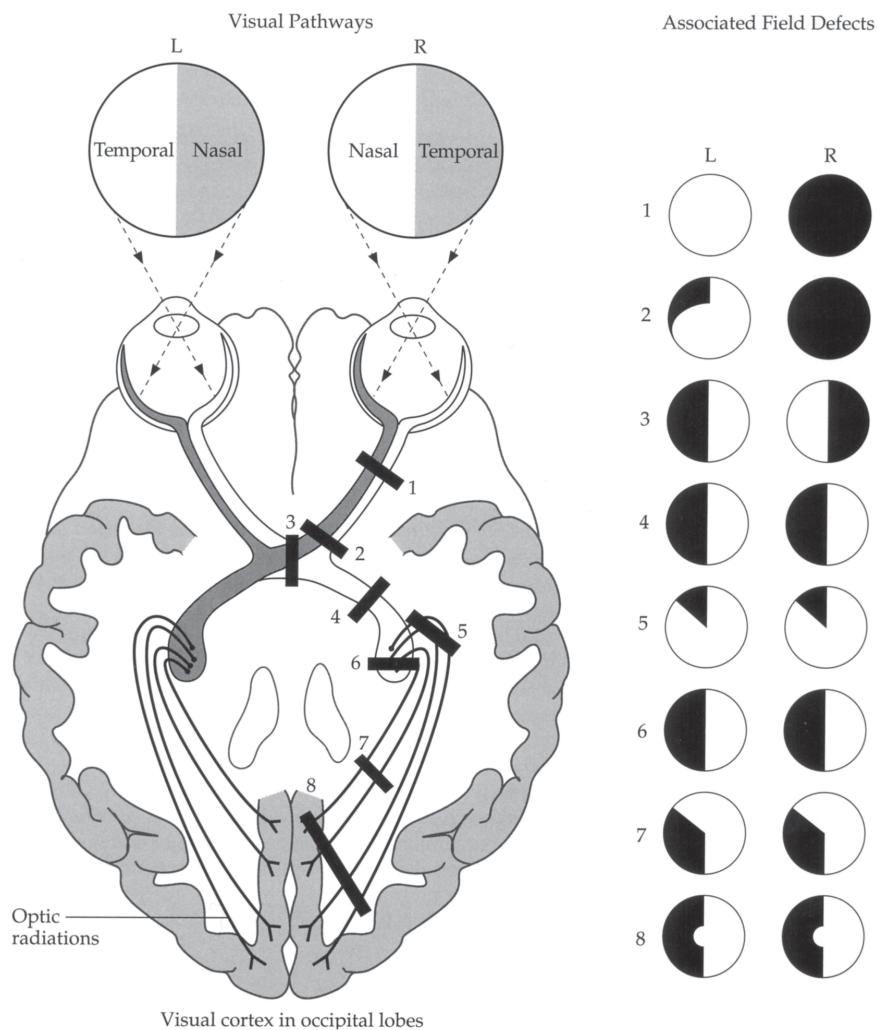
When the visual field is tested, if a patient has a unilateral cerebral lesion that results in damage to the vision pathways (the optic radiations), the result is that the patient has a homonymous hemianopia, i.e., a left parieto-occipital lesion causes inability to see the right half of the visual field of each eye, a right homonymous hemianopia (RHH). If the RHH is total, and the deficit has its boundary along the vertical line dividing the visual field into its right and left halves, the field defect is said to demonstrate “macular splitting.” If the field defect affects the peripheral portion of the right visual field, but does not involve its central portion, the field defect is said to show “macular

sparing.” Though this term has gained widespread popularity over the years, it is not a useful concept in localizing lesions. A common reason for its apparent presence is the occurrence of frequent eye movements by the patient when the visual field is being tested, such that some of the visual field defect near central fixation is not detected by the examiner because of the patient’s eye movements in looking toward the defective visual field. When true macula sparing is present, the finding does not clarify localization of a lesion to the temporal, parietal, or occipital lobe of the brain, but simply reflects the lack of involvement of the most posterior tip of the occipital cortex when there is an occipital lesion. If an occipital lesion predominantly involves the posterior tip of the cortex, the visual field defect will be adjacent to central fixation, and how much it extends peripherally will simply depend on how much the occipital lesion extends anteriorly.

Now, Dr. Leonard Levin:

Many patients with strokes of the occipital lobe still retain central vision. This is probably either due to imperfect fixation during testing, or more likely to the fact that the occipital lobe receives vascular supply from both the posterior and middle cerebral arteries. Thus, damage is usually incomplete. Although there is dual representation of the macula bilaterally in the occipital lobes, it is clinically minimal.

This is NOT something you need to know clinically. The other concepts we have taught you are far, far more important. It will not be tested. It is included only as a cool example of where Neurosciences meets clinical application. Enjoy.



Other Disease States

Introduction:

We have reviewed several diseases that affect the visual system at every step of the way from retinal to cortical processes. Although these processes are very useful as illustrations of how specific damage to each component of the visual system affects the clinical test of visual function, none of these processes is epidemiologically common. With the prior syndromes as background necessary to understand the visual system, let's now turn to diseases that have a very high prevalence in the United States. We'll use what we learned in the earlier discussion to understand how these processes affect the vision of a wide population of people who will be in your office someday. In decreasing order of prevalence, let's discuss cataract, age-related macular degeneration, and glaucoma.

Cataract

DEMOGRAPHICS/INCIDENCE - Cataract

Cataract is defined as ANY opacity of the natural "crystalline" lens. It can be diffuse or focal, peripheral or central, blinding or inconsequential.

Incidence: 100% if people live long enough. Incidence of visually significant cataract (i.e. effect on visual acuity on the eye charts or visual performance on daily activities such as driving and reading) is approximately 50% by age 70.

Cataract surgery is the number one surgical expense of Medicare patients in the US. 1.25 million cataract extractions were performed in the US in 1988.

Worldwide, it is the number one cause of blindness, and it is the number one cause of treatable blindness in the United States (defined as 20/200 or worse in the better of the two eyes).

CLINICAL PRESENTATION: SYMPTOMS - Cataract

This is an easy category - the presenting symptoms can be broken into two categories: decreased acuity and glare.

Patients with decreased acuity come in to clinic usually with a variant of two complaints: they can't see to read or they can't see detail well enough to drive - especially at night, or in areas where they are unfamiliar and they have to read the street signs. Both types of complaints are due either to the opacity of the lens blocking the light from getting to the retina, or more likely the irregularity (not opacity) of the refracting surface. Reading is commonly a symptom because it is a demanding visual task and people often do it for hours on end. Even though in clinic these patients can often look at the near cards and read 20/20, it is with such mental effort that they cannot maintain that effort for that long. Difficulty driving at night is an early symptom because at night, the pupil dilates, and if multiple opacities or irregularities are present, more become exposed as the pupil dilates. The most demanding visual situation is probably driving in unfamiliar areas - the visual landscape is constantly moving, the driver has many visual tasks to perform simultaneously in addition to seeing the road signs, and people are likely to be anxious and poorly concentrating if they feel uncertain while driving.

So these are the symptoms which bring patients to attention.

CLINICAL PRESENTATION: PHYSICAL EXAM - Cataract

On exam, the entire eye often appears totally normal with the exception of the lens. When viewed with the slit lamp microscope, we see the irregularities and opacities as reflections in what is usually an optically transparent tissue, and if we shine a light directly into the eye to create a “red eye” effect, we can see the opacities as black specks in the orange background.

Several types of cataract are defined - the most common is a generalized, uniform darkening of the center of the lens (mechanism discussed below). Focal areas of opacity and irregular refracting surface can be central (with marked visual effects) or peripheral (with little effect except in dim-light situations.)

MECHANISM OF DAMAGE - Cataract

The lens is an epithelial structure, with the cells facing internally. The cells continue to secrete stromal proteins throughout life, causing an increase of the lens size that becomes significant about age 60 to 70 years old. Eventually, the increased size leads to an accumulation of waste products in the core of the lens, and those proteins become opaque.

VISUAL ACUITY - Cataract

Visual acuity effects are wide-ranging. Early on, the diffuse density increases cause only refractive changes, and acuity with new refractions are still normal. Later on, as the density advances to the point where the visible color of the lens changes from grayish-clear to yellow to dark brown, the patient’s perception of color is decreased.

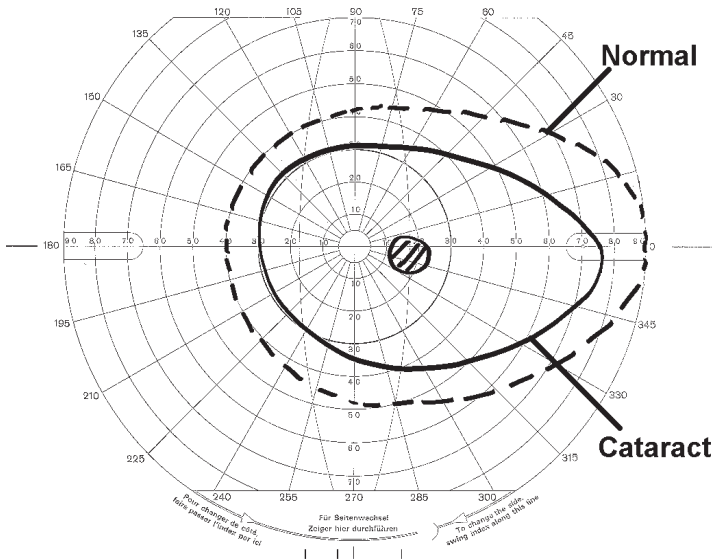
The focal lens changes have a markedly greater chance of significantly decreasing vision. By creating zones of opacity or differing refractive index, they have the effect of “smearing” the light by diffraction, causing a marked loss of acuity, especially when point-sources of bright light are present (classic e.g.: oncoming headlights). These are quickly intolerable to the patients, and commonly lead to surgery, because the attempt at changing glasses to improve the symptoms is unsuccessful.

CONFRONTATION VISUAL FIELD TESTING - Cataract

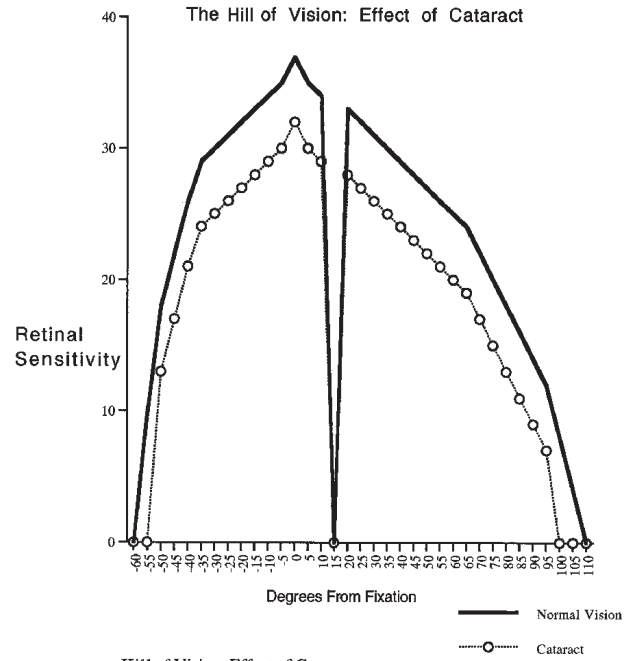
Even very dense cataracts cause no large confrontation visual field loss, because they are either diffuse or so focal as to be smaller than the test object.

FORMAL VISUAL FIELD TESTING - Cataract

No focal defects are seen on visual field testing, but since the lens is diffusing the test light over a larger area, it is more difficult for the retina to detect a subtle light. The entire “hill of vision” is therefore less sensitive.



In this idealized visual field, I have drawn a single test-spot line for a cataract patient and compared to a normal patient tested with the same spot. Most cataracts cause a fairly diffuse loss of visual function, and the "constriction" caused by these cataracts is common. I have emphasized the diffuse loss by showing that the shape of the test line is the same as the normal line, but uniformly less sensitive.



Hill of Vision: Effect of Cataract
In this diagram, I have decreased the sensitivity at each tested point by 5 deciBel units. The impression is of the hill maintaining its overall shape, but having sunk a little into the sea of darkness.

RELATIVE AFFERENT PUPILLARY DEFECT TEST - Cataract

Recall that the RAPD is a global, whole-retina function, and that about half of the nerve fibers have to be damaged in order to get a positive test. The cataract, with its diffuse effect, simply does NOT decrease the total amount of light striking the retina enough to generate an afferent pupillary defect.

TREATMENT - Cataract

In the most frequently performed surgery in the USA, the anterior capsule is partially removed, the lens material broken apart with an ultrasonic tip, and the lens material removed with fluid and suction.

Then, an implant made from inert plastic is inserted into the capsular remnant, with the refractive power of the implant having been calculated from the length of the eye and the amount of curvature of the cornea.

The incision is approximately 3 millimeters across (the corneal diameter is 11 millimeters) - some incisions do not require suture, and some surgeons do this surgery with only numbing drops for anesthesia, so that the patient can immediately see on the way home from the surgery.

When you get a chance (during your third year ophthalmology clerkship) WATCH this surgery.

Age-Related Macular Degeneration

DEMOGRAPHICS/INCIDENCE - ARMD

Funny thing about Age-Related Macular Degeneration. It's more prevalent in "chronologically gifted" age groups. Who would have thought?

ARMD is the leading cause of irreversible severe visual loss in white Americans over 50 years old. It is exceedingly rare in African Americans. Approximately 750,000 people over 65 years old have severe visual impairment due to ARMD.

Please note: Throughout the text we have used the word "fovea" to describe the central retinal area. Clinically, there is a similar term used to denote a slightly larger area, still in the central retina. Roughly, it corresponds to the area where cones outnumber rods, and is responsible for daytime/high-detail vision. It is this term, macula, which is clinically relevant, and so the name of this disease implies a degeneration of the central vision area of the retina.

CLINICAL PRESENTATION: SYMPTOMS - ARMD

Early forms of ARMD have absolutely NO symptoms. Only after the process includes loss of large clumps of rods and cones does the patient experience decreased vision. If there are any sensitive clinical tests which are positive for a given patient (e.g. decreased contrast sensitivity), there are usually other processes such as cataract which confound the test results.

CLINICAL PRESENTATION: PHYSICAL EXAM - ARMD

If there aren't any symptoms early, that must mean that making the diagnosis of ARMD is done on physical exam.

The patients in the age group that is at risk for ARMD are also at risk for cataract, and usually, the patient with ARMD also has at least a mild cataract. The cornea is usually clear, the lens slightly opaque, if only diffusely and uniformly, and the key finding is clumping of the pigment in the retinal pigment epithelium beneath the retina. Later stages may have a whitish scar if there has been leakage of blood.

MECHANISM OF DAMAGE - ARMD

This retinal pigment epithelium interleaves with the rod and cone outer segments, supplying oxygen and nutrients to the rods and cones by way of the choriocapillaris, and nutrients to the rods and cones. The pigment epithelium also metabolizes the outer segments which each photoreceptor sheds throughout the day, and regenerates the vitamin A isomers which transduce the photons to neuronal signals. Lifelong exposure to ultraviolet light leads to accumulated damage in the RPE cells, and they begin to die. The rods and cones then get less metabolic support, and function less well. As the RPE cells malfunction and/or die, the metabolic waste products from the photoreceptors accumulate faster than the remaining healthy RPE cells can metabolize them. The waste products accumulate beneath the RPE cells, on the basement membrane which separates the RPE cells from the choroidal capillaries. Although you can see these deposits very easily by clinical exam (they look like small yellow dust particles or clumps of yellowish gunk) they do not interfere with vision very much. The rods and cones can still function pretty well, even with this waste material present. Acuity may drop if the waste products build up in the exact center of the fovea, but usually not significantly (see next section). The process can occur anywhere in the retina, and there is a familial type where only the peripheral retina is affected, but usually, the process is worst in the

center of the retina (so it is called macular degeneration as opposed to retinal degeneration). It's not known why the macula is the primary site, but speculation centers on the principle that the macula is by far the most metabolically active area and that wastes build up more here simply because more wastes are produced here. In severe cases, there may be an area where no RPE cells are left - the choroidal capillaries then invade the photoreceptor layer and cause bleeding and scarring.

VISUAL ACUITY - ARMD

Effect of ARMD on visual acuity is very minimal early in the disease, where only clumps of pigment and waste product called drusen are present. Contrast sensitivity may be mildly decreased, but overall acuity rarely drops below 20/25 or 20/30.

In late stages, scar replaces photoreceptors, and since this occurs mostly in the central retina, visual acuity is markedly reduced. It is likely that vision will be worse than 20/200 (counting-fingers or hand-motions) but rarely will leave the patients totally blind to all light stimuli.

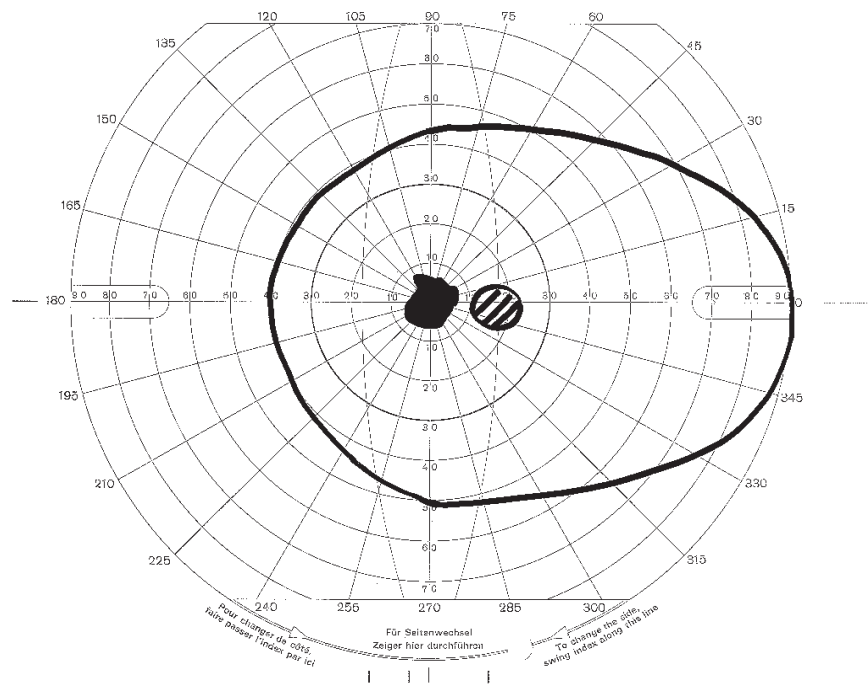
CONFRONTATION VISUAL FIELD TESTING - ARMD

The early stages of ARMD will be far too subtle to detect with confrontation visual fields.

Late stages will usually be easily detectable, for the patient will have a significant defect in the central areas of vision.

FORMAL VISUAL FIELD TESTING - ARMD

Machine-driven visual field testing can reveal subtle, generalized loss of function early in the disease process, but clinically this is of no use, for these patients invariably have a cataract which could easily be causing the same level of loss. If a patient has a generalized 10% loss of sensitivity, it is impossible to attribute an accurate proportion to the cataract and to the ARMD. Knowing how much each contributes to the whole loss is a matter of speculation unless the cataract becomes mature enough to undergo removal, and then whatever portion of deficiency remains is due to ARMD.



In this idealized visual field, I have drawn a single test-spot line, showing that the periphery of the retina is normal. In addition to the normal blind spot, I have shown a visual loss of the central vision, in an irregular shape that crosses all of the midlines. Note that the loss is absolute - the retina is destroyed in this region and will not be able to respond to any brightness of light. Recall from the retinal detachment field discussion that if you see the lesion crossing all 4 midlines that the disease process must be in front of the optic nerve and in front of the chiasm - therefore the retina is the most likely area to be the cause.

RELATIVE AFFERENT PUPILLARY DEFECT TEST - ARMD

Recall that we stated that it takes loss of function of approximately half of the photoreceptors to result in a detectable RAPD. In ARMD, it is rare to see anything but the central retina affected, and usually far less than 50%. So it is VERY RARE that Macular Degeneration will cause an afferent pupillary defect.

TREATMENT - ARMD

There is no way to replace the retinal pigment epithelial cells, or to reverse the decades of ultraviolet light exposure, and the layer cannot be transplanted (though people have recently tried). So once the process has begun, there is no way to reverse it.

Therefore, studies have been done to see if progression can be slowed or halted. The Age-Related Eye Disease Study followed 3000 patients for 8 to 10 years, giving some of them placebo, other groups getting multivitamins at 100% of the RDA, and some got higher doses of vitamins and minerals. Though all of the known vitamins and minerals were included, particular attention was being paid to the antioxidant group (vitamins A,C,E; minerals zinc and selenium) with the thought that the likely mechanism of damage of UV light is to create free radicals which then oxidize nucleic acids. The study concluded in 2002 and showed that only the most advanced eyes had any benefit from the anti-oxidant treatment and the benefit was very small.

Glaucoma

DEMOGRAPHICS/INCIDENCE - **Glaucoma**

Glaucoma is the second-most common cause of blindness in the US, and the most common cause of blindness among African-Americans. Roughly 5 million people in the US have the disease, and studies suggest that only half of them are aware of it. Of whites, between 1 and 3% of people between 60 and 80 years old have glaucoma.

CLINICAL PRESENTATION: SYMPTOMS - **Glaucoma**

Usually, NONE. (A very important take-home message.)

CLINICAL PRESENTATION: PHYSICAL EXAM - **Glaucoma**

Physical exam of the glaucomatous eye is usually normal in all respects except an abnormal appearance of the optic nerve. (see below)

MECHANISM OF DAMAGE - **Glaucoma**

Glaucoma results from elevated intraocular pressure. Remember from Dr. Uhlrich's section earlier that the ciliary body is a ring of very vascular tissue immediately behind the iris. The ciliary body secretes aqueous humor, a fluid very much like extracellular fluid, but higher in nutrients. This fluid bathes the lens and inner cornea, providing nutrition to these avascular but metabolically active tissues. The pressure in the eye is determined by the ratio of how much fluid is created as opposed to how much exits the eye. The normal cycle of fluid flow is: production at the ciliary body, with secretion into the space between the iris and the lens, flow through the pupil into the anterior chamber between the iris and the cornea, and then exit from the eye at the ring of porous tissue at the most peripheral part of the anterior iris, the trabecular meshwork. The fluid then joins the venous system and drains from the orbit with the orbital veins.

The ciliary body secretes fluid at a pretty constant rate, slowing during sleep, but continuing even if the pressure in the eye is very high. The trabecular meshwork controls the eye pressure by regulating how much fluid leaves the eye in a very active process.

In glaucoma, the amount of fluid being created is constant, and normal, but the meshwork cells function poorly and less fluid gets out. The increased pressure of the eye results in damage to the ganglion cells of the retina. The mechanism is unknown, but it appears that the longer ganglion cells, with more surface area of axons exposed to the increased pressure, are more sensitive to the elevated pressure. Gradual atrophy of these cells occurs, and there are gradually less and less axons joining together to form the optic nerve. This loss of tissue is seen clinically as a thinning of the optic disc rim, called "cupping." You will hear a lot more about this in your third year clinical clerkship in ophthalmology, for it is a major component of the annual exam of ocular health. Some amount of cupping is normal - less than 50% cupping is normal. Patients with more than 50% cupping are studied with visual field tests to detect glaucoma.

VISUAL ACUITY - Glaucoma

There are two reasons why visual acuity is NOT decreased in glaucoma, at least not until virtually ALL of the fibers are atrophied, very very late in the course of the disease.

First, the longer fibers are more sensitive to the compression caused by the increased pressure, and longer fibers go to the peripheral retina. The shortest fibers connect the fovea to the optic nerve, and so these fibers are more resistant to damage.

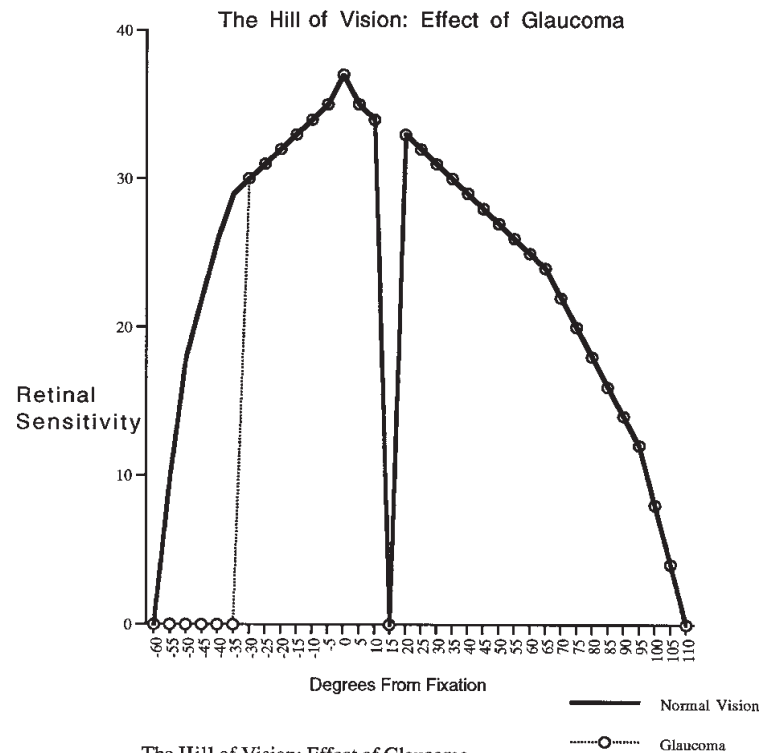
Second, recall from the earlier lectures that there are more ganglion cells serving the fovea than the periphery. Even if the central ganglion cells were equally damaged by the increased eye pressure, there are so many more of them that it would take a very long time before enough of them were lost to affect visual acuity function.

Summary: visual acuity is NOT affected until the final, final stage of glaucoma.

CONFRONTATION VISUAL FIELD TESTING - Glaucoma

Confrontation visual field testing is sometimes positive in glaucoma. Usually, the areas of decreased function form small “pockets” which are smaller than the finger-sized stimulus, and so a small glaucomatous defect can “hide” during this test and not be detected.

As the disease progresses, however, these pockets become larger and coalesce, and in late stages, confrontation visual field defects can be evident.



The Hill of Vision: Effect of Glaucoma

In this diagram, I have left almost all of the hill to be normal height, but the focal area of glaucoma damage at the edge of the hill I have reduced to zero sensitivity. In clinical practice, these defects usually are not so dramatic, but the effect of creating a "cliff" in the island is commonly seen. Remember that glaucoma damage begins in the periphery, in small focal areas, leaving the majority of the vision normal until very late in the disease process.

FORMAL VISUAL FIELD TESTING - Glaucoma

By far the most sensitive test for glaucoma is the machine-driven visual field test. The small pockets which are characteristic of glaucomatous visual loss can be mapped fairly easily, and more importantly, followed quantitatively over time. This gives the clinician an idea about whether the treatment to lower the eye pressure is being effective at stopping or slowing the progression.

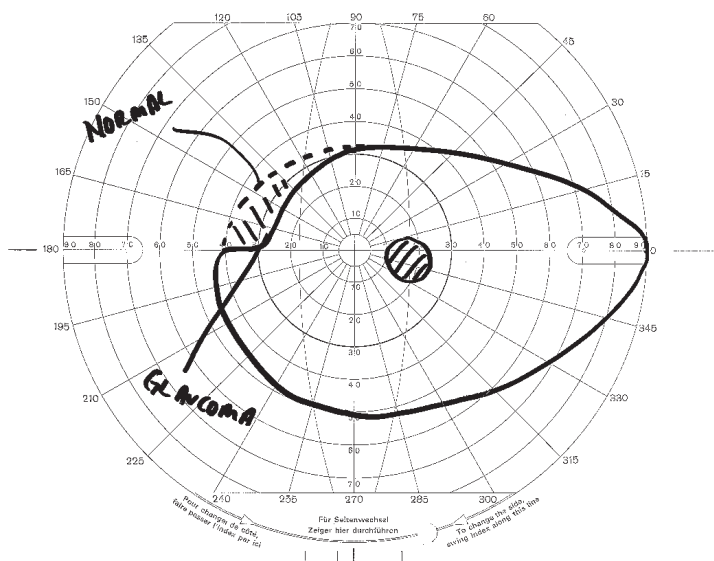
An example of the arc-like visual field defects that are seen in glaucoma are included below, as both the computerized and manual (Goldmann) varieties.

RELATIVE AFFERENT PUPILLARY DEFECT TEST - Glaucoma

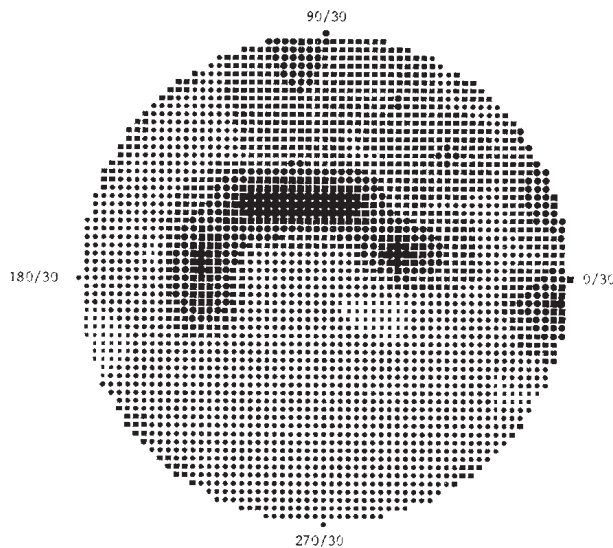
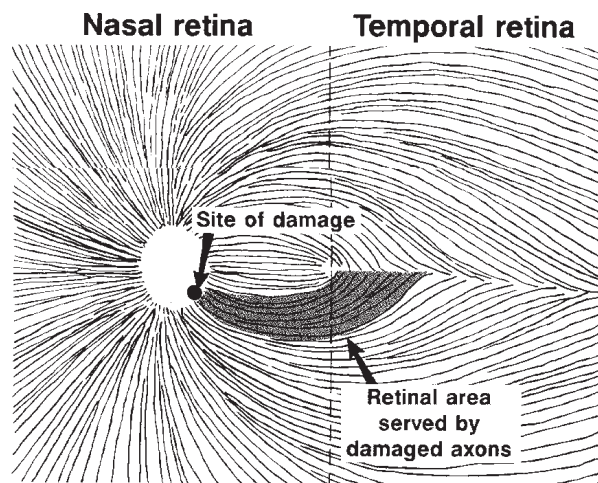
As we have mentioned *ad nauseum*, it takes loss of approximately half of the ganglion cells or photoreceptors to make an afferent pupillary defect become evident. This DOES happen in glaucoma, but only VERY LATE. Clinically, it is an unhelpful way to detect or follow glaucoma.

TREATMENT - Glaucoma

Glaucoma is a disease of increased eye pressure that leads to progressive, permanent loss of ganglion cells. Our best method of treatment is to decrease the eye pressure.

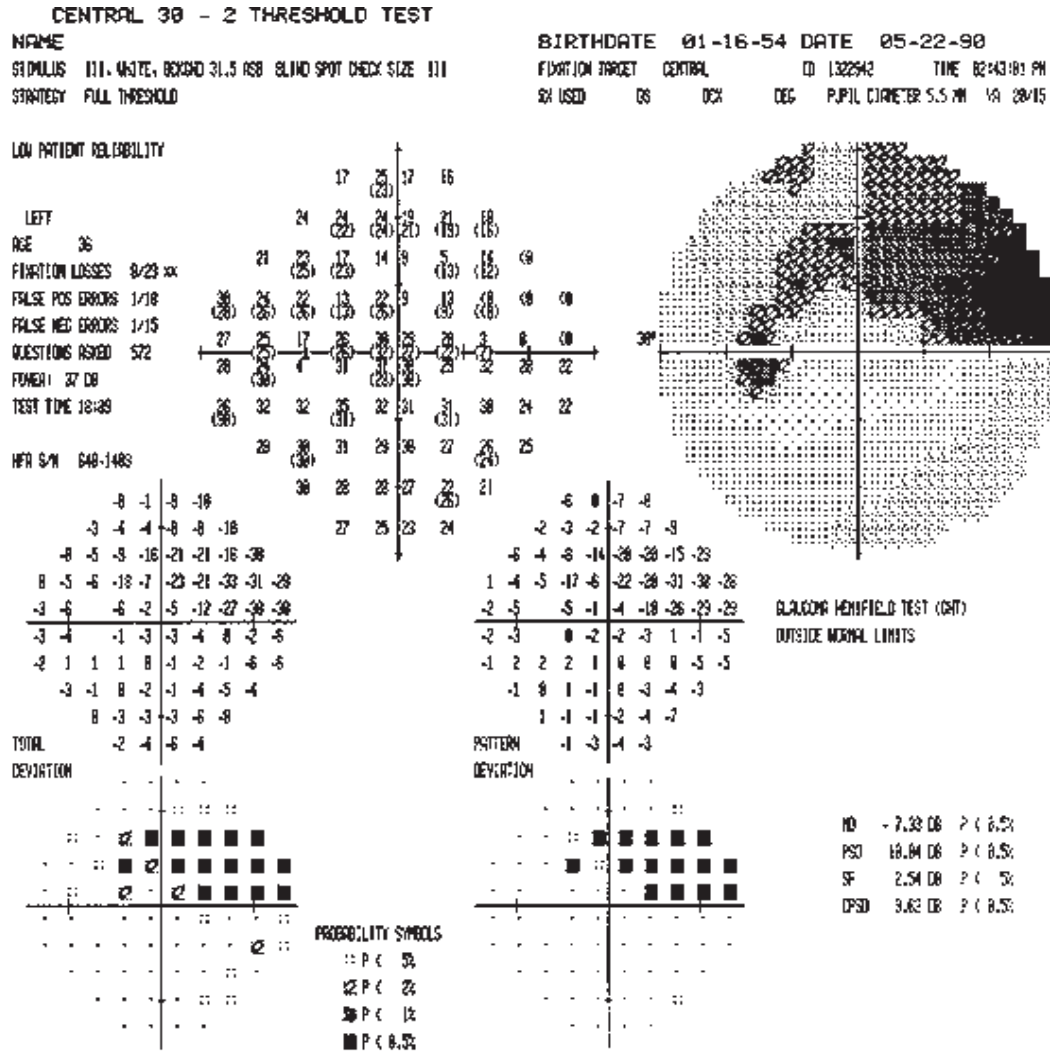


In this idealized visual field, I have drawn a single test-spot line which is normal in every direction except for the nasal field, where it is constricted superiorly. You know this is the nasal field because the blind spot is on the opposite side of the loss, and the blind spot is in the temporal field. Note very well how the loss STOPS AT THE HORIZONTAL MIDLINE. This is CHARACTERISTIC of GLAUCOMA or other optic nerve disorders. This abrupt change of normal to constricted field loss at the horizontal meridian looks like a staircase, and is often called a "nasal step."



—Arcuate Bjerrum scotoma. Glaucomatous damage to a nerve fiber bundle that contains axons from both inferonasal and inferotemporal retina resulted in the arcuate defect shown. An arcuate scotoma occurs in the area 10° to 20° from fixation. The scotoma often begins as a single area of relative loss, which then becomes larger, deeper, and multifocal. In its full form, an arcuate scotoma arches from the blind spot and ends at the nasal raphe, becoming wider and closer to fixation on the nasal side.

There are two ways to accomplish this: make the ciliary body produce less fluid, or assist the trabecular meshwork to drain the fluid at lower pressures. Medications and laser procedures exist which lower eye pressure by each method, and if these fail, it is possible to surgically connect the anterior chamber to the subconjunctival space, bypassing the meshwork altogether.



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SYM	[Symbol]	[Symbol]	[Symbol]	[Symbol]	[Symbol]	[Symbol]	[Symbol]	[Symbol]	[Symbol]
ASB	8	2.5	8	25	79	251	794	2512	7943
	.1	1	3.2	10	32	100	316	1000	3162
DB	41	36	31	26	21	16	11	6	1
	50	40	35	30	25	20	15	10	5

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In this automated field, you can see the loss in the nasal (i.e. opposite from the blind spot) field loss that stops abruptly at the horizontal meridian. It is closer to central vision than the manual-diagram result above. The gray scale diagram in the upper right shows the loss well, and you can see in the two lower grid diagrams that the amount of loss is definitely abnormal even when corrected for the patient's age. The Probability Symbols indicate that in every spot that is coded with a completely black square, less than 0.5% of normal people in this age group will have a result as poor as this person. The computer is trained to find these spots, and below the gray scale you can read that the "Glaucoma Hemifield Test" result is "Outside Normal Limits."

Visual Problem	Incidence	Symptoms	Physical Exam	Mechanism	Visual Acuity	RAPD
Retinal Detachment	more in myopes. (long eye)	flashing lights shade	"algae in gentle waves"	retina separates from RPE	if peripheral = OK if central = bad	positive/abnormal
Optic Nerve Trauma	low	"my vision is bad"	associated head-face trauma	tearing or laceration of nerve or sheath and blood vessels	from blindness to no loss	positive/abnormal
Optic Nerve Compression	low	tumor = pain in orbit, difficulty with eye movements, decreasing vision	see Symptoms	tumor, interrupt blood supply and axoplasmic flow of gang. cell axons	decrease in late stages	positive/abnormal if one eye affected; negative if at chiasm
Stroke	older people with hypertension	decreased cortical function	normal eye	cells in visual cortex are ischemic	subtle loss in central vision of contra. hemifield	negative/normal
Cataract	100% if live long enough	glare when driving at night	lens opacities	cont. prod. Stroma protein and accum. of waste products in core of lens	perception of color is affected; refraction corrects early prob.; later big loss esp. in bright lights	negative/normal
Age Related Macular Degeneration (ARMD)	leading cause of irreversible, severe vision loss	NONE	clumping of RPE beneath retina	ultraviolet damages RPE which affects metab. of rods and cones	early little loss, later major loss especially centrally	negative/normal (usually does not affect 50% of ganglion cells)
Glaucoma	second most cause of blindness (to ARMD)	NONE!!!	"cupping" of optic nerve	increase ocular pressure due to faulty removal of fluid from eye-production of fluid by ciliary body = normal	NORMAL until real late	negative/normal (usually does not affect 50% of ganglion cells)

Example Case:

Now that you know all the pieces of the visual system puzzle and have seen several example cases, let's go back to our example case in the first section and show you how much you have learned. New comments are in underlined italics.

PRESENTATION: EXAMPLE PATIENT

DU is a 38 year old woman with a known diagnosis of multiple sclerosis. She has had many attacks in the past in her motor and sensory systems, but no known attacks in her visual system. She presents to the eye clinic with an urgent problem: decreased vision.

SYMPTOMS:

CHIEF COMPLAINT:

- "My vision is bad to my right side."

SYMPTOM PURSUIT:

- First noticed this morning on awakening.

Sudden onset. Not cataract, not glaucoma, not tumor, could be ARMD, could be stroke, could be retinal detachment.

- No change from first notice till clinic exam (mid-afternoon)

• Vision on her right is "dim", not blurry, not blank. "Like looking through a very dense fog." Vision to the left side is normal, sharp, clear.

Segmental loss: not cataract, could be retinal detachment, could be glaucoma, could be stroke, could be chiasmal compression - compression would be bitemporal, not homonymous.

- Painless.

Doesn't narrow much down, except pain would rule IN something like acute angle closure glaucoma (more on that in your third year clerkship).

- First occurrence. Her pre-symptom vision was "20/20 in both eyes, without glasses."

So you know this really IS new.

- No double vision.

So ocular motility is normal, making processes which would damage the nuclei in the brainstem less likely.

• Vision was good before this episode - has had frequent visits to neurologists for her other MS care, visual system often evaluated in these visits and was always "normal."

Now you know it really is a change, not something gradual that she simply happened to notice acutely.

• Has covered each eye and tested vision monocularly - right side of vision in both eyes is lost, symmetrically.

This is the strongest localizing clue. If it is on the same visual side in each eye, you know the defect must be in an area where those fibers have gathered together in one place. Since the fibers are separated in the retina and optic nerve, come together in the chiasm, and segregate behind the chiasm, you now know that the defect must be posterior to the chiasm.

- No flashing lights. No "window -shade" progression of symptomatic area.

Lack of these symptoms pretty much rules out retinal detachment, especially in such an astute patient.

- No other neurologic symptoms, “and I would know if I had them.”

Gives you evidence this MS flareup is probably not multifocal, but not much information for severity or extent of the damage to the visual system.

- No head trauma.
- No new medications.
- No change of symptoms in any field of gaze (i.e. same “dim area” whether looking right, left, up, down, or straight ahead.)

Helps to rule out extraocular muscle imbalances, and tells you the defect is fixed in the system. That is, it probably isn't a huge intravitreal clot in each eye if it is this fixed even when the eyes move. (A clot which would exactly match size and location in each eye is pretty much impossible anyway.)

EXAMINATION:

- Visual Acuity: 20/20 right eye 20/20 left eye

Although she gets all the test letters correct, she has the sense that “some parts of the letters are missing to the right.”

Cortical fill-in is a powerful phenomenon, allowing good chart-reading ability even if a hemifield of vision is missing. This applies to left/right losses as well as superior/inferior field losses.

- Confrontation Visual Field testing:

Right gaze visual field of each eye symmetrically decreased to finger-counting stimulus testing. Defect does not cross vertical midline, but does cross horizontal midline of each eye's visual field.

Confirms her astute symptom description.

- Pupillary light reflexes:

5 mm in dark, both eyes

1 mm with flashlight, both eyes

Swinging flashlight test: no dilation of either pupil - no afferent defect.

Tells you that the defect does not involve the optic nerve, Edinger-Westphal, ciliary nerves, ciliary ganglion, or iris. Localizes the lesion to behind the chiasm.

Because her visual defects seem to be fairly “geographic”, you decide to map out exactly where she can and cannot see before you examine and dilate her eyes.

- VISUAL FIELD TESTS

Manual field testing shows marked constriction of her fields in both eyes. The right half of the field of each eye is nearly gone. There is perfect respect for the vertical meridian, and the left half of each eye's field is entirely normal.

Computerized field testing shows that the right half of each eye is markedly reduced - the test light has to be made much brighter than normal for her to see it. The left half of each eye's field is normal. There is respect for the vertical meridian.

In each testing method, there is a nearly perfect correspondence of the boundary of the loss between the right and left eyes. That is, the defect is homonymous.

Homonymosity (not a real word, mind you) indicates the lesion MUST be behind the chiasm. The manual method of field testing can test more of the visual periphery, but the computer method is better for detecting subtle losses, longitudinal changes (in time, as you follow patients for changes) and quantifying losses.

PHYSICAL EXAMINATION OF THE EYEBALLS

- Lids, lashes: Normal
- Conjunctiva: No inflammation or dilation of normal blood vessels.
- Corneas: Clear, smooth, intact. No explanation for decreased vision.
- Anterior chambers: negative “volcano sign.” Normal depth, no inflammation.
 - Irides: normal, round, all areas move briskly to constrict when light is shined in the pupils.
- Lenses: Clear, normal size and shape. No explanation for decreased vision.

AFTER DILATION OF PUPILS WITH EYE DROPS:

- Vitreous humor/cavities: Clear, normal. No blood, no inflammation.
- Retina:
 - All areas attached, “flat” to choroid. No tears, no tumors, normal curvature.
 - Blood vessels: normal caliber. No bleeding. No ischemia. No neovascularization.
 - Pigment in fovea and periphery normal. Color normal.
- Optic nerve:
 - No swelling.
 - Normal cupping.
 - No hemorrhages.
 - Margins sharp.
 - Nerve tissue normal color, no pallor.

This exam is completely normal and rules out any intraocular disease process, confirming your suspicion that the defect must be intracranial, behind the chiasm.

SUMMARY:

Right Homonymous visual field loss, respecting vertical midline, with completely normal physical exam, pupillary light reflex, and visual acuity.

There you go! 6 short lectures, and now you know all there is to know about ophthalmology. You have come a long way from when you first read this case a few days ago. Isn't it a shame you have to go through 3 more years of medical school and a 4 year residency? You know it all already! Congratulations!

GLOSSARY

Aberration Any degradation of an image by the processing system.

Accommodation Accomplished by active contraction of the ciliary body circular muscle. A thickening and increase of convexity of the lens in order to focus an external object more clearly on the retina. This increases the net refractive power of the eye, allowing it to become more myopic or less hyperopic.

Acuity (acu= sharpen) clarity of central vision. Often associated clinically with the more precise term “resolution acuity” indicating ability to resolve objects from one another (e.g. Snellen acuity).

Adaptation to dark Retinal response to changing external visual conditions. To reset the baseline of visual response to a darker environment, Vitamin A stores are metabolized to increase the “state of readiness” to single photons. By increasing the pool of active molecules, the retina is made more likely to respond to a single photon.

Allograft (allo = other in Greek) Transplantation of tissue from one animal to another of the same species. For example, corneal transplantation (see Penetrating Keratoplasty below) is an allograft.

Amblyopia (amblys, dull) The condition in which an eye (or both eyes) has impaired visual acuity without a structural ocular lesion, due to the failure of development of normal neural connections between the ganglion cells and the cortical cells. This connection network forms partly in utero and partly in the years between birth and age 6 to 7 years. Structurally, the retina, lateral geniculate body, optic radiations and cortical cells look normal. But the network of synaptic connections between the cells is poor, and this limits the acuity of the visual system.

Anterior Chamber The space between the posterior corneal surface and the iris surface, including the plane of the pupil. This space is filled with aqueous humor. A “Shallow AC” indicates that there is little fluid in the chamber, and that the separation of the iris from the cornea is a small distance.

Anterior Hyaloid (A.K.A. anterior vitreous face) Vitreous condensation to form an invisible membrane at the anterior extent of the vitreous body, adherent centrally to the posterior lens capsule, and peripherally adjacent but not adherent to the lens zonules and ciliary body processes. It is possible surgically to remove the lens completely, yet leave the hyaloid intact. This prevents vitreous from spilling into the anterior chamber after surgery. Optically transparent, this interface in no way affects the refractive power of the eye.

Aphakia Absence of the biologic crystalline lens. After cataract surgery, if a plastic lens has been placed, the eye is termed pseudophakic. Patients with crystalline lens intact are called Phakic. (Phakos is from Greek, meaning lentil or anything shaped like a lentil).

Aqueous humor Produced by the ciliary body processes, this clear fluid is found anterior to the anterior hyaloid face in the posterior chamber and into the anterior chamber, bounded by the posterior corneal endothelial surface. It provides nutrition to the avascular cornea and lens, aids in inflammatory processes, and its production-to-outflow ratio determines the intraocular pressure.

Aqueous veins Endothelial channels conducting aqueous flow from Schlemm’s canal to the orbital venous drainage system. Found within sclera in the 2 to 4 mm surrounding the corneal limbus.

Asteroid hyalosis A visually-benign condition of crystalline deposits suspended in the vitreous fluid. Light is reflected from the deposits, creating a bright starry image for the examiner, but the patient’s vision is little affected because the light which reaches the retina is relatively undistorted.

Astrocytes Supportive cells found in the brain, spinal cord, and into the optic nerve.

Astroglial cells Same as astrocytes.

Axon Structure connecting nerve cell bodies to their target neurons next in the transmission chain. In the optic nerve, the nerve fibers seen are axons of the ganglion cells, on their way from the inner retina to the Lateral Geniculate Nucleus for relay on to the visual cortex, or to the brainstem for control of pupillary diameter.

Bipolar cell Retinal interneurons which derive their name from their orientation of a single dendrite and single axon 180 degrees from each other across the cell body. They interact with photoreceptors, horizontal cells, and amacrine cells, and their output goes directly to the ganglion cells. Their function is to modify the photoreceptor cell inputs to allow sophisticated retinal integration of signal.

Blepharitis Meaning inflammation of the eyelid, blepharitis implies inflammation and subclinical infection of the Meibomian glands. The altered tear layer which results from the Meibomian inflammation may degrade image formation and/or cause discomfort if the cornea becomes dry in patches.

Bifocal Eyeglasses Bifocals, invented by Ben Franklin, have a small lens area ground in the lower half of the spectacle lens. This area is used if the patient looks down through it, usually at something close to the patient. The upper lens is used for seeing distant objects, and the lower lens used for reading. The lower lens is often called the “add”, short for “additional lens.” Patients with presbyopia usually benefit from bifocals. (See presbyopia).

Bowman’s layer Condensed corneal collagen, not a true membrane, on the anterior surface of the corneal stroma. It lies beneath the corneal epithelium and epithelial basement membrane, and if disrupted in trauma will result in scar formation.

Brightness adaptation The retinal “baseline” resets to increasing ambient brightness by quickly adjusting intraretinal calcium levels. This prevents “whiteout”, a condition where all receptors are firing in response to increased light levels, and the eye thus cannot distinguish shapes or forms until the baseline resets.

Canthus The area at each inner and outer corner of the eye fissure, where the upper and lower lid join at acute angles. (Derived from Greek canthos, which surprisingly has no other root than “corner of the eye”)

Cataract Derived from Greek katarrhakies, meaning waterfall or downrushing, a loss of transparency of the lens of the eye or its capsule.

Chiasm Derived from the letter Chi, a crossed X, this is the decussation of optic nerve fibers in front of the tuber cinereum. Fibers originating in the temporal retina do not cross, and fibers originating in the nasal retina cross to be interpreted in the cortex of the opposite lobe. The function of this chiasm is to bring information from the SAME VISUAL AREA of both eyes to the SAME CORTICAL CELLS.

Choriocapillaris The layer of choroidal capillaries under the Retinal Pigment Epithelium. By diffusion, this layer provides nourishment to the rods and cones of the retina as well as to the RPE.

Choroid (Greek, membrane) The vascular layer between the sclera and retinal pigment epithelium. The choroid supplies more blood flow per unit volume than any other area of the body with the notable exception of the kidney. It acts as a heat sink for excess light energy and to nourish the metabolically active retina.

Chromatic aberrations The difference in focus of different wavelengths of light from an original coherent white-light source. In essence, this is due to the difference of refractive power of a surface based on wavelength of the light being refracted. Typically, blue light is bent more than red light and may come to focus at a point “in front of” the point of focus of red light.

Chromatophores Pigments contained in cone cells which absorb light of selective wavelengths. This selectivity allows color perception.

Cilia Eyelashes.

Ciliary arteries Vessels supplying the vascular flow to the non-retinal structures of the eye. (Retinal blood comes largely from the central retinal artery).

Ciliary body A very vascular ring of tissue immediately posterior to the iris. It encircles the lens, supporting it by connecting strands called zonules, It is responsible for nourishing the avascular lens and cornea by production of aqueous humor.

Ciliary flush Dilation of the veins which drain the iris and ciliary body, due to inflammation of the eye (**iridocyclitis**). Ciliary flush, since it is venous in origin, has a blue-purple tint when the corneal limbal area is examined clinically, and is to be distinguished from the bright red appearance of superficial inflammation of the eye, called conjunctivitis. **Conjunctivitis** usually involved infection and steroid treatment is often contraindicated, while ciliary flush indicates intraocular inflammation and requires more detailed intraocular examination to elucidate the cause and often requires intensive steroid treatment.

Ciliary ganglion (cilium = eyelid in Latin) Synapse of parasympathetic fibers in the inferotemporal portion of the posterior orbit, outside of the sclera surface of the globe. Named for its wispy hairlike appearance on gross exam. Often difficult to identify in gross anatomic dissection.

Ciliary processes Fingerlike projections of the ciliary body, present to increase the surface area of the ciliary body, to allow the secretory function necessary to create adequate flow of aqueous humor.

Conjunctiva A thin, vascular mucus membrane lining the sclera of the globe and the inner eyelid surface. Its purpose is to manage airborne pathogens, and provide lubrication necessary to allow eye movement relative to the lid.

Conjunctivitis Viral or bacterial infection of the conjunctiva. See ciliary flush above for an important clinical distinction.

Consensual reflex The reaction of BOTH pupils to light sensed only in ONE eye. Mediated by ganglion cell fibers which synapse in the Edinger-Westphal nucleus.

Contrast The difference in quality or quantity of two surfaces. In optics, usually a difference of luminance (i.e. number of photons reflected from the objects).

Contrast sensitivity The ability of the eye to detect difference of contrast between two objects. Mediated largely by intraretinal processing.

Convergence Latin con + vergo; to incline together. In optics, the direction of visual lines toward a single point, In ocular motility, the inward deviation of the eyes relative to each other, necessary to view an object closer than 20 feet to the observer with both eyes simultaneously.

Converging lenses Optical lenses capable of causing light of parallel beams (from optical infinity) to converge to a single point somewhere past the lens.

Cornea Multilayered structure of specialized scleral collagen, avascular, optically clear, in the central anterior 12 mm diameter of the eye.

Corneal abrasion Loss through physical or chemical trauma of the superficial layers of corneal epithelium. May or may not include the basement membrane of the epithelium, may or may not include Bowman's layer of corneal stroma.

Corneal ulceration Corneal infection with loss of stroma. Usually follows an abrasion.

Cortical cataracts Cataract type where the opacification includes the outer portion of the lens stroma, the lens cortex. Often wedge shaped, intermediate in effects on visual acuity and color perception (between posterior subcapsular cataract and nuclear sclerosis cataract).

Crystalline lens The normal proteinaceous lens of the eye. Differentiated from the plastic lens often implanted at time of cataract extraction.

Depth of field The range in front and behind the PRECISE plane of focus of an image that the retina cannot detect the lack of focus. A large depth of field means that the eye can move a great distance

away from the exact plane and still “see” the image in focus. Shallow depth of field means the eye detects the image degradation with little movement away from the focal plane. Essentially, this is a measure of how tightly the image is converging at the point of focus - if the “cone” of convergence is wide, the depth of field is very shallow. If the rays are just-barely-converging and nearly parallel, the eye will have a wide range of movement without detecting the small change of focus.

Dermatochalasis The gradual separation over time of lid skin from its adherence to underlying lid structures, specifically the orbicularis oculi muscle. Loss of these sites allows skin to bag, often hanging over the lid margin into the field of view of the patient. A normal aging change.

Descemet’s membrane The basement membrane of corneal endothelium, the innermost layer of the cornea. Relatively impervious to water flow. Helps to keep cornea dehydrated to maintain its optical clarity.

Diabetic retinopathy Vascular changes caused by weakened vessel walls due to diabetes mellitus. Characterized by dilated irregular veins, small aneurysms, vascular leakage, etc. If severe, can block off capillary flow entirely, leading to ischemia of the retina. Late stages include growth of fragile new blood vessels attempting to relieve the ischemia. Breakage of these new blood vessels causes multiple damaging events in the retina and optic nerve.

Diopter An arbitrary measure of lens focus power: 1 diopter means light originating from optical infinity will focus 1 meter behind the lens. 2 diopters = will focus 1/2 meter behind the lens, etc.

Dry Eye Loss of any of the 3 layers of tears. If the corneal epithelium becomes exposed to air, it becomes very sensitive to light and air. Sensed by patients as decreased vision (loss of smooth air:tear interface which has the most refracting power of any optical interface in the eye) and soreness, burning and redness.

Emmetropia The perfect balance of refracting power of the eye’s optical elements and the distance of the retina from the cornea - resulting in images from optical infinity coming to focus on the precise plane of the retina, without needing accommodation by the patient.

Equator An anatomic descriptor of the center belt of the eye, cut in an A-P plane. This plane is parallel to the plane of the iris, posterior to the lens. Envision the optic nerve leaving the eye as the south pole, and the tip of the central cornea as the north pole. The equator is a ring of sclera in the middle of the eye, falling behind the iris-lens plane, in the mid-vitreous.

Ethmoid A bone in the nasal portion of each bony orbit.

Extorsion The ability of the eye to spin within the orbit. The axis about which torsional movement occurs is a line connecting central cornea, central pupil, central lens, and ending in the central retina. In Extorsion, if you look at the eye at rest and make note of a scleral blood vessel at the 12 o’clock area, extorsional movement would make the vessel move to the LATERAL clock hour. For a right eye, this would mean to 11 o’clock. For the left eye, extorsion would move the vessel to the 1 o’clock position. (The clock is defined from the point of view of the examiner as she looks at the eye from the front). Torsional eye movements are caused by contractions of the oblique muscles.

Extraocular motility The movement of the globes relative to the orbits, caused by the extraocular muscles.

Flicker sensitivity The ability of the eye to detect that a light source is not continuous : i.e. flashing. Rods see flicker poorly, being designed to pick up low levels of light and therefore using the principle of summation: see below. Cones detect flicker well, being able to rapidly fire and reset.

Fluorescein A molecule which absorbs light of all frequencies and is able to emit light of a defined frequency. Useful in two ocular situations: It binds to corneal epithelial basement membranes if the epithelial cells are gone, helping us detect corneal abrasions. It is also useful to inject into the venous system of the arm, watching for its appearance in the arteries and veins of the eye - telling us where blood flow is interrupted, leaking, or adequate (see diabetic retinopathy above).

Fovea The center of the retina, where the finest visual acuity is possible. Many alterations are present here as compared with retina elsewhere - see text. Strict definitions are possible, but most clinicians use macula and fovea interchangeably.

Foveola Anatomically, the area at the dead center of the retina, less than 500 microns across, where there are no supporting cell nuclei, only cones, and the best possible vision. Clinically, it means the ABSOLUTE center of the retina and field of view.

Ganglion cell The relay cell of the retina, the ganglion cell receives input from photoreceptors, amacrine, and bipolar cells, and passes the signal onto the LGN and pretectum by way of the optic nerve.

Glare A sensation caused by brightness within the visual field that is sufficiently greater than the luminance to which the eyes are adapted; results in annoyance, discomfort, or loss of visual performance or visibility. Common sources are street lights, sunlight, or auto headlights. One of the main visual disturbances caused by cataract.

Glaucoma A disease characterized by loss of ganglion cells from the retina, resulting in visual function deficits and characteristic loss of optic nerve tissue volume ("cupping" of the optic nerve). Often associated with malfunction of the rate of drainage of aqueous humor from the eye, due to poor function of the trabecular meshwork apparatus.

Goblet cells Cells in the conjunctiva which produce proteinaceous mucus, the innermost layer of the tear film.

Graves' Disease Also called Thyroid Orbitopathy, a disease related to thyroid dysfunction. Antibodies are created which attach to the extraocular muscles, causing them to swell with inflammation. This swelling results in malfunction of the extraocular muscle, leading to poor movement of the eye, or even proptosis of the eye. (**Proptosis** means the eye protrudes from the bony orbit more than normal for that person.)

Horizontal cell Cells in the retina which interconnect rods, cones and bipolar cells. Their function is to provide intraretinal processing of the original light signal.

Horner's Syndrome Resulting from interruption of the sympathetic innervation of the orbital structures at any point in their chain, Horner's Syndrome consists of Ptosis (loss of innervation of Müller's muscle in the upper eyelid), Miosis (loss of sympathetic innervation of the pupil - unopposed parasympathetic innervation results in marked pupillary constriction) and anhidrosis (loss of innervation of the sweat glands of the face and forehead).

Hue discrimination The ability to detect difference in WAVELENGTH of two light sources.

Hyaluronic acid A mucopolysaccharide made up of alternating residues of hyalobiunoric acid, forming a gelatinous material in tissue spaces. In the eye, found in the vitreous cavity.

Hypermetropia A visual condition of "farsightedness"; that is, the patient sees better at a distance than at near if uncorrected by spectacles. This condition is usually caused by the combination of a normal cornea and lens, but an eye which is shorter than normal. Thus, images come to focus behind the retina. Patients who are farsighted can see well in distance if they can accommodate enough to "pull" the image focal point anteriorly onto the retina. If they can accommodate even more, they may be able to see near objects as well without spectacles. Usually, this process strains the accommodative system to the maximum, and these patients often need reading glasses or bifocals.

Hyperopia Shortened form of the word hypermetropia.

Implant In our context, a plastic lens inserted into the eye at the conclusion of cataract removal surgery, to replace the focusing power of the removed crystalline lens. It is left in place for the remainder of the patient's life. Power of the implant is calculated from two numbers: the refractive power of the cornea, and the length of the eye.

- Index of Refraction** The relative velocity of light in a medium compared to its velocity in air.
- Induced myopia** The nuclear cataract development process usually increases the index of refraction of the cataractous nucleus. As the index increases, the lens gains refractive power, and the image is focused further in front of the retina. This is similar to garden-variety myopia, where the lens is normal but the eye is too long and the image comes in to focus somewhere in the vitreous cavity. When the patient requires myopic glasses to see clearly due to cataract, it is called induced myopia.
- Inferior oblique** A muscle connecting the lacrimal sac fossa to the inferior/lateral/posterior sclera. When it contracts, the eye EXtorts, ELEvates, and ABducts.
- Inferior rectus** A muscle connecting the posterior muscle cone to the inferior sclera. When it contracts, the eye depresses.
- Inner segment** That portion of the photoreceptor cell which is closest to the vitreous cavity. Most metabolic activity occurs here, and you may consider it the “cell body” to make an analogy to neurons.
- Interneuron** That group of cells in the retina which interpose between rods /cones and ganglion cells. Responsible for “sophisticating” the output signal of the photoreceptors, this group consists of bipolar, horizontal and amacrine cells.
- Intorsion** The movement of the 12 o’clock portion of the eye towards the nose of the patient. For the patient’s right eye, this is a clockwise rotation, for a left eye it is a counterclockwise rotation.
- Intraocular lens** Differentiated from the “Natural” crystalline lens that vertebrates are born with, this term refers to the plastic lens inserted at the conclusion of cataract removal surgery. (See implant.)
- Iridocyclitis** Inflammation of the iris or ciliary body, whether as part of an infection in the eye, or due to autoimmune phenomena. It is manifested by fibrin seen on exam of the eye, and by floating white blood cells in the aqueous humor which can be seen on slit lamp biomicroscopy. **Irido** refers to the iris, and **cyclitis** refers to the ciliary body. Amazing amounts of material can be released by this process. **Ciliary flush** is a finding associated with this process.
- Iris** (Greek = rainbow) The anterior division of the vascular tunic of the eye; a disk-like diaphragm, perforated in the center (the pupil), attached at its outer margin to the ciliary body; it is composed of stroma and a double layer of pigmented epithelium from which are derived the sphincter and dilator muscles of the pupil. Its major optical function is to control the amount of light which enters the eye. It receives sympathetic (dilating) and parasympathetic (constricting) innervation.
- Iritis** (See iridocyclitis above)
- Keratocytes** Cells within the cornea responsible for the maintenance of the stromal collagen. Derived from fibroblasts.
- Keratoplasty** The surgical removal of portion of the cornea, with the replacement from another source. Commonly, this is from human cadaver donors. Purpose is to replace damaged cornea with a clear substitute. The avascular nature of the cornea allows allograft transplantation with great success.
- Lacrimal apparatus** The lacrimal apparatus includes the lacrimal gland, creating the middle (aqueous) layer of tear film from its site in the superolateral orbit, and the lacrimal drainage system, including the puncta in the medial portion of each lid, the canaliculus connecting the puncta with bilateral lacrimal sacs, and extending into the nasal drainage openings.
- Lacrimal gland** This gland sits beneath the superolateral orbital rim, and has several ducts to connect it to the tear film in the palpebral conjunctiva. It is innervated by CN VII, and can increase its output of aqueous tears by many fold (i.e. crying).
- Lacrimal puncta** Small openings in the medial aspect of all four eyelids, allowing tear flow out of the eye and into the lacrimal drainage system (and on into the nose). See text for description of action during blinking.

Lamina cribrosa A specialized area of sclera containing perforations, through which pass the fibers of the optic nerve. It serves to separate CSF surrounding the optic nerve and CNS from vitreous humor, and to separate intraocular pressure from intracranial pressure.

Lateral geniculate nucleus The thalamic relay station for signals originating in the ganglion cells of the retina to proceed to the cells carrying the information to the visual cortex.

Lateral rectus The extraocular muscle connecting the muscle cone to the lateral scleral wall of the eye: contraction of this muscle results in ABduction of the eye.

Lateral retinal integration The interconnection of interneurons allows sophisticated processing of the original visual signal. By this connection system, information about color, presence and orientation of edges, contrast differences, etc. can be sent as already-processed signals in the optic nerve. This processing which occurs before the signal leaves the eye is called lateral retinal integration. It implies that input from adjacent rods and cones is somehow associated and analyzed rather than being simply relayed to the cortex.

Luminance The amount of light reflected from an object.

Lye chemical burn Any basic chemical splashed into the eye can have devastating effects: since the chemical saponifies tissue, it can penetrate deeply and continue causing damage long after initial exposure. Cornea, sclera and even ciliary body and iris can all be “melted” by strong base exposure.

Lysozyme Muramidase: capable of lysing components of bacterial cell walls. Found in tears, a natural defense against ocular infection.

Medial rectus The extraocular muscle connecting the muscle cone to the medial scleral wall of the eye: contraction of this muscle results in ADduction of the eye.

Meibomian gland A sebaceous gland found at the base of the eyelash. Producing the air-most (superficial) layer of the tear film, it is instrumental in protecting the underlying layers from evaporation while the eye is open. Normally colonized by skin flora, if these bacteria overrun the normal balance, inflammation can result, causing edema and closure of the gland outflow. This edema, resultant erythema of the eyelid and disruption the tear film is called **blepharitis**.

Meridian Defined as a clock hour of the sclera based on the visual axis. That is, the pivot point of the “hands” of the clock is a point on the central cornea. The meridian is defined by the point of view of the observer, such that a lesion in the superotemporal quadrant of the left eye is described as being at “1:30 o’clock”.

Miosis Constriction of the pupil. Caused by parasympathetic input to the eye.

Müller’s muscle A small, sympathetically innervated muscle in the upper eyelid. Its normal function results in approximately 2 mm of eyelid “lift.” Interruption of sympathetic tone (**Horner’s syndrome**) results in slight drop of the involved lid, or **ptosis**.

Muscle Cone The origin of all of the rectus muscles of each eye. A shared tendinous insertion of the muscles to the apex of the bony orbit. The only extraocular muscle not originating here is the inferior oblique, which originates in the lacrimal sac area of the nasal bony orbit.

Mydriasis Dilation of the pupil, caused by sympathetic innervation of the dilator muscle of the iris.

Myopia Nearsightedness; usually due to the increased length of these eyes. The cornea is usually normal, the lens is usually normal, but in myopic eyes, the overall length of the eye is longer than normal. This results in the images focusing in FRONT of the retina, within the vitreous somewhere. Myopic people can usually see objects close to them without glasses. Exactly how close the objects have to be to be in focus varies with the degree of myopia.

Nerve fiber layer The innermost layer of the retina (i.e. the layer closest to the vitreous), this layer consists of the axons of the ganglion cells on their way to the optic nerve, chiasm, and LGN or pretectum.

Nuclear sclerosis The gradual increase in density of the center of the crystalline lens with age. The lens is avascular, with nutrients and wastes diffusing in and out of the center of the lens. As the epithelial cells of the lens in the lens periphery continue to slowly lay down lens fibers throughout life, the diffusion distance increases. At some point, the central proteins undergo compaction and oxidation, increasing the refractive index of the lens (“Hardening”). This sclerosis makes the patient more nearsighted (or less farsighted) and is seen clinically as a darkening of the normally clear lens.

Optic nerve The collection of ganglion cell axons as they course from the lamina cribrosa to the optic chiasm to the LGN. Blood supply is from branches of the internal carotid. The outer wall of the nerve is dura mater, contiguous with the dura mater of the CNS/brain.

Orbicularis oculi The muscle of the eyelid which inserts at both canthi (medial and lateral of each lid). Controlled by CN VII (facial), contraction of this muscle causes the two lid margins to approximate (eye closes/blinks).

Orbital apex The opening in the superomedial orbit, through which most of the important nerves/vessels pass from the cranial vault to the orbit. The apex is where the muscle cone attaches, and thus indirectly is where each of the rectus muscles and superior oblique originates.

Orbital axis That line connecting the orbital apex with the center of the orbit at its anteriormost plane. Since the medial walls of the two orbits (ethmoid bones) are roughly parallel and the lateral walls of each orbit (zygomatic bones) are 90 degrees apart, the orbital axes split that angle, and are thus 45 degrees apart. The orbital axis is thus 22.5 degrees different from the visual axis of the eyeballs.

Orbital cellulitis Infection and inflammation of the tissues behind the aponeurosis of the eyelid muscle. Infection within the orbit can quickly follow dura into the CNS. Clinical distinction between preseptal cellulitis (i.e. limited to ONLY the eyelid anteriorly) and orbital cellulitis means the difference between watchful waiting & oral antibiotics or admission to the hospital for intravenous antibiotics and likely surgical debridement with drain placement. Clinical distinction is based on CT or MRI scanning, and careful assessment of ocular function.

Orbital septum The posterior fascia of the orbicularis oculi muscle, this membrane extends from the medial canthus to the lateral canthus to the upper and lower mid-orbital bones. It is a barrier to blood flow (black eyes), lymph flow, and infection. See **orbital cellulitis**.

Orbital veins

Outer segment

Photoreceptors Rods and cones. Those cells of the retina which are capable of the **phototransduction cascade**.

Phototransduction cascade The series of biochemical events which transduces light energy (photons) into neural impulses (ions). See the text for the full description.

Pilocarpine A parasympathomimetic, causing **miosis** and **accommodation**.

Posterior chamber That space between the anterior hyaloid face (peripherally) the anterior lens capsule (centrally) and the posterior iris surface.

Posterior ciliary arteries

Posterior ciliary nerves

Posterior subcapsular cataract That type of lenticular opacity which is found on the back internal surface of the lens itself. This type of cataract is often associated with toxic chemicals or blunt ocular trauma, and because it is usually centrally located, affects vision very early in its process.

Color perception is usually normal, but **glare** is almost always a debilitating symptom.

Presbyopia (“old vision”) Continuous creation of lens fibers throughout adult life results in a large round lens incapable of changing shape to allow **accommodation**. These presbyopic patients require reading glasses or bifocals to provide good refraction of images originating closer than 20 feet.

Preseptal cellulitis See **orbital cellulitis**.

Pretectal complex That area in the pretectum of the brainstem which receives information from the ganglion cells of the retina, and processes this information into a signal which controls the diameter of the pupils.

Proptosis The forward displacement of the eyeball due to some mass effect between the posterior surface of the sclera and the bony orbital wall. The most common cause of proptosis in adults, one eye alone or both eyes, is thyroid orbitopathy (**Graves' Disease**). The clinical findings are a protuberant eye, with the eyelids being spread further apart by the eye, creating a chronic "startled look."

Pseudophakia The presence of an intraocular **implant** after cataract removal surgery.

Puncta The opening at the medial margin of both upper and lower eyelids. The first portion of the **lacrimal drainage system**.

Red eye Conjunctival vessel dilation ("injection") in response to several possible etiologies: ocular irritation, conjunctival infection, intraocular inflammation, extremely elevated intraocular pressure.

Refraction Physically, the bending of the light ray as it passes from a medium of one refractive index to another of a different index. See text for diagram. Clinically, the process of placing lenses in front of an eye to best determine the spectacles needed to give the patient good vision.

Resting potential That level of depolarization or hyperpolarization maintained when a nerve is not being stimulated.

Retinotopic mapping The principle that the organization of nerve impulses is continued from close proximity in the retina to close proximity in the optic nerve, to close proximity in the LGN, to close proximity in the visual cortex.

Rhodopsin "visual purple": a red thermolabile protein, MW 40 KD, found in the external segments of the rods of the retina. It is bleached by the action of light, which converts it to opsin and all-trans-retinal and is restored in the dark by the retinal pigment epithelial cells.

Rods The photosensitive, outward-directed process of a rhodopsin-containing cell in the external layer of the retina. Containing one photopigment which responds to all wavelengths of incident light, rods provide only "black and white" information to the visual cortex, and function best in low-light situations.

Schlemm's canal Endothelial-cell-lined drainage ring contained within the corneal limbus, connecting the trabecular meshwork with the scleral aqueous veins. It communicates aqueous humor from the eye to the orbital venous network.

Sclera (From Greek Skleros = hard) a fibrous tunic forming the outer envelope of the eye. The "white of the eye".

Secondary cataract Modern cataract surgery removes the central 2/3 of the anterior capsule of the lens, removes the lens stroma, and replaces the crystalline lens with a plastic implant, placed within the remaining capsular tissue. If this capsule continues to make small amounts of lens stroma, or undergoes fibroblastic transformation due to the physical presence of the plastic, that scar which is produced interferes with the patient's vision. Since the visual effect can be nearly identical to that of the original cataract, this opacification is called a secondary cataract. Usually, simple incision with a laser will restore a clear visual axis and good vision.

Slit lamp biomicroscope A microscope with a coaxial pivoting light system, allowing a slit beam to be projected on the tissue on which the objectives are focused. The advantage of the slit over diffuse illumination is to better allow determination of depth in the tissue.

Spherical aberration Monochromatic aberration occurring in refraction at a spherical surface in which the paraxial and peripheral rays focus along the axis at different points.

Spoke That part of a bike wheel connecting the hub to the rim. Alternatively, that “wedge-of-pie” shaped opacity of lens cortex associated with cortical cataracts. It is unknown if the opaque tissue has truly precipitated, or just altered interactions with surrounding proteins, but the result is an optically apparent wedge shaped phenomenon.

Summation That process by which single photons can trigger a response in a rod which without summation would not respond to a single photon. The rod responds a little to each photon, and maintains that small response for a fairly long time. If another photon hits that rod before it has reset to its baseline, it hyperpolarizes a little more. If another photon hits, the rod then fires a full signal. In this manner, very faint signals result in rods firing. Without summation, each photon would merely hit an already-reset rod, and not have enough effect by itself to stimulate firing of the rod. Cones have little summation, and rods have a great deal.

Superior cervical ganglion Sympathetic chain synapse in the cervical neck, where neurons involved in pupillary diameter pass on the way from the spinal cord to the eye. Interruption of this input results in **Horner’s syndrome**.

Superior oblique An extraocular muscle, originating in the apical muscle cone of the orbit, and passing through a bony pulley (the trochlea) at the anterior superior orbital rim. It inserts on the postero-lateral sclera. Contraction of this muscle pulls the superior area of the eye in towards the nose (intorsion) and depresses the eye downward. It is innervated by the fourth cranial nerve.

Superior rectus An extraocular muscle originating in the apical orbital muscle cone, and inserting into the superior sclera of the eye. Contraction of the muscle, controlled by the third cranial nerve, pulls the eye upwards.

Tarsus A condensed-collagen plate within both upper and lower eyelids, serving as a structural support element, giving the lids solidity, and as a point of attachment of the muscles which open the eyelid fissure. Foreign bodies often become lodged in the edges of the tarsus as the soft conjunctival surfaces fold over the firm tarsal edge.

Tear film Three layers of fluid covering the cornea and conjunctiva. The interface between the liquid tear layers and air provides the biggest refractive interface of the ocular system.

Temporal contrast sensitivity The ability of the visual system to detect change in a visual stimulus. That is, if I flicker a light faster and faster, when does it appear to be a single image (not flashing) to you? Temporal in this context means time, not the side of the head.

Thyroid orbitopathy AKA Graves’ Disease. Swelling of the extraocular muscles in response to autoimmune-mediated antibodies related to thyroid disease. This swelling often interferes with the muscle function, and can be pronounced enough to cause protrusion of the globe beyond the orbital walls. (**Proptosis**).

Trabecular meshwork Cells and pores that resemble sponge tissue, in the base of the anterior iris insertion. This meshwork “filters” fluid volume from the anterior chamber, and deposits that fluid in **Schlemm’s canal** for transfer ultimately to the orbital venous drainage system. The process of “filtration” is really bulk fluid movement in a manner sort of like pinocytosis on a huge scale. It is an active process, If the cells or beams malfunction to the point that more fluid is being created by the **ciliary body** than filtered by the meshwork, the pressure of the eye increases and **glaucoma** may develop.

Visual acuity Although specifically broken down in to multiple types such as vernier, minimum resolvable, resolution, spatial, stereoscopic, visibility etc etc etc acuities, for the purpose of this course, visual acuity means Snellen Acuity - that system of various sized letters which the patient is asked to identify from a specified distance from the chart. In essence, the smallest letter the patient can detect, specified as a ratio with the “average” patient response.

Visual axis That imaginary line connecting the center of the cornea, pupil, lens, and retina (macula or fovea). In gross terms, the direction the eye is “pointing” towards the object of visual regard.

Vitreous hemorrhage Bleeding from either the ciliary body or retina into the vitreous cavity. Surprisingly little effect on visual acuity unless the hemorrhage is quite dense. Blood cells disperse evenly throughout the vitreous cavity to create a haze. Usually, the image of the retina to the examiner is much worse than the image of the world is to the patient.

Vitreous humor (vitreous: glassy) Hyaluronic acid and water, in solution, in the space between the retinal surface and the back of the lens and ciliary body.

Vortex veins Veins draining the choroidal circulation from the inner sclera surface, to the orbital veins. Seen ophthalmoscopically as whorl-like reddish spokes at the mid-retina in the 1:30, 4:30, 7:30 and 10:30 meridia.

Xerophthalmia (xeros = dry) Chronic lack of Vitamin A, such that the mucus-secreting goblet cells of the conjunctiva are unable to produce any of the innermost layer of the three-layer tear film. Without this layer to provide electrostatic anchors, the watery aqueous middle layer and superficial oily layer from the Meibomian glands do not coat the conjunctiva well and chronic dry eye results. Xerophthalmia is a major cause of blindness in developing countries, if the dryness is marked enough to cause/allow corneal scarring.

Zonules Threadlike protein strands connecting the equatorial lens capsule to the ciliary body. They originate in the ciliary processes, and radiate in a crisscrossing fashion in to the peripheral edge of the lens capsule.

Ophthalmology Web Sites Worth Visiting:

General:

www.eyenet.org (American Academy of Ophth — has great links)
www.nei.nih.gov (National Eye Institute - also good links)
<http://vision.arc.nasa.gov/VisionScience/VisionScience.html>

Academic Sites:

www.biostat.wisc.edu/ophth/eye.htm (University of Wisconsin Dept of Ophthalmology)
webeye.ophth.uiowa.edu/ (University of Iowa Dept of Ophthalmology)
wings.buffalo.edu/medicine/oph/assoc.html (SUNY-Buffalo Dept of Ophthalmology)
<http://netope.harvard.edu:80/meei/> (Harvard — Massachusetts Eye & Ear Infirmary)

Journals of Ophthalmic Interest:

www.meei.harvard.edu/meei/DJOhome.html (Digital Journal Of Ophthalmology)

GENERAL TEXTBOOKS

Basic and Clinical Science Coursebooks

Section 11 (Retina and Vitreous), Section 10 (Glaucoma), Section 2 (Fundamentals and Principles of Ophthalmology)

Basic Ophthalmology for Medical Students and Primary Care Residents, F G Berson MD

The Physician's Guide to Eye Care, JD Trobe MD

Atlas of Clinical Ophthalmology, DJ Spalton, RA Hitchings, PA Hunter

Essentials of Ophthalmology, GB Bartley, TJ Liesegang

General Ophthalmology, DG Vaughn, T Asbury, P Riordan-Eva

Principles of Neural Science, ER Kandel, JH Schwartz, TM Jessell

SPECIALIZED MONOGRAPHS AND TEXTBOOKS

Clinical Symposia (Ciba Geigy)

Volume 43 Number 4 1991 (Diagnosis and Management of Glaucoma)

Volume 42 Number 4 1990 (Management of Cataracts)

Diagnosis and Therapy of the Glaucomas; HD Hoskins MD and MA Kass MD, Mosby, St. Louis, MO, 1989.

Focal Points: Clinical Retinal Function Testing, (Volume IX, Module 1), S Sunness MD, American Academy of Ophthalmology, San Francisco CA, 1991.

Clinical Tests of Vision, L Friesen, Raven Press, NY NY, 1990. (UW Library Number WW 145 F917t 1990)