CORRECTABLE EYE DISEASES

ACHROMATOPSIA

Achromatopsia (**ACHM**) is the inability to see color. Although the term may refer to acquired disorders such as color agnosia and cerebral achromatopsia, it typically refers to an autosomal recessive congenital color vision disorder, also called rod monochromacy and total congenital color blindness. Individuals with the congenital form of this disorder show complete absence of cone cell activity via electroretinography. There are at least four genetic causes of congenital ACHM, two of which are cyclic nucleotide-gated ion channels (ACHM2/ACHM3), a third the cone photoreceptor transducin (*GNAT2*, ACHM4), and the last unknown.

Classification

- Acquired achromatopsia (Cerebral achromatopsia)
- Congenital/inherited achromatopsia
    - Complete/typical achromatopsia (Rod monochromacy)
    - Incomplete/atypical achromatopsia

Signs and symptoms

Complete Achromatopsia

Aside from a complete inability to discriminate colors, individuals with complete achromatopsia have a number of other ophthalmologic aberrations. Included among these aberrations are greatly decreased visual acuity (<0.2 or 20/100), nystagmus, and severe photophobia. Furthermore, while the fundus is completely normal, there is no photopic ERG response. Rod cell function is normal.

Incomplete Achromatopsia

In general, symptoms of incomplete achromatopsia are similar to those of complete achromatopsia except in a diminished form. Individuals with incomplete achromatopsia have reduced visual acuity with or without nystagmus or photophobia. Furthermore, These individuals show only partial impairment of cone cell function but again have retained rod cell function. Visual acuity and stability in this case improves during first 6-7 years of life.

Cause

Acquired Achromatopsia

Cerebral achromatopsia is a form of acquired color blindness that is caused by damage to the cerebral cortex of the brain, rather than abnormalities in the cells of the eye's retina.

Congenital Achromatopsia

The known causes of the congenital forms of achromatopsia are all due to malfunction of the retinal phototransduction pathway. Specifically, this form of ACHM seems to result from the inability of cone cells to properly respond to light input by hyperpolarizing. Known genetic causes of this are mutations in the cone cell cyclic nucleotide-gated ion channels *CNGA3* (ACHM2) and *CNGB3* (ACHM3) as well as the cone cell transducin, *GNAT2* (ACHM4).

1. **Cerebral achromatopsia** is a type of color-blindness that is caused by damage to the cerebral cortex of the brain, rather than abnormalities in the cells of the eye's retina.

Cerebral achromatopsia differs from other forms of color blindness in subtle but important ways. It is a consequence of cortical damage in lingual and fusiform gyri, near the base of the brain (ventro-medial occipital lobe). This damage is almost always the result of injury or illness. Because of the location of the tissues whose damage is associated with cerebral achromatopsia, and the requirement that it be damaged in both hemispheres of the brain, complete cerebral achromatopsia is very rare.

Patients with cerebral achromatopsia deny having any experience of color when asked and fail standard clinical assessments like the Farnsworth-Munsell 100-hue test (a test of color ordering with no naming requirements). Patients may often not notice their loss of color vision and merely describe the world they see as being "drab".

The critical difference between cerebral achromatopsics and people with other forms of color blindness is that cerebral achromatopsics retain the ability to perceive chromatic borders. For example, they see a red square on a green background effortlessly even when the red and green are equally bright. There are various ways of ruling out a role for unintended luminance differences in accounting for this, e.g. random luminance masking. It even appears that cerebral achromatopsics can discriminate contrasts on the basis of color direction, but they can't use these contrasts to compare the color of surfaces that do not adjoin directly. It has been suggested that cerebral achromatopsia might best be seen as a failure in specific color-constancy mechanisms.

Cerebral achromatopsia illustrates the way in which chomatic information can be used to achieve many goals, only one of which is the perception of surface color, and that those different goals may be met by distinct pathways within the visual system.

http://en.wikipedia.org/wiki/Achromatopsia

ASTHENOPIA -EYESTRAIN

Asthenopia or eye strain is an ophthalmological condition that manifests itself through nonspecific symptoms such as fatigue, red eyes, eye strain, pain in or around the eyes, blurred vision, headache and occasional double vision. Symptoms often occur after reading, computer work, or other activities that involve tedious visual tasks.

When concentrating on a visually intense task, such as continuously focusing on a book or computer monitor, the inner eye muscles may tighten, which can cause the eyes to get irritated, dry, and uncomfortable. Giving the eyes a chance to focus on a distant object at least

once an hour usually alleviates the problem. Small font sizes exacerbate the strain if they cause unconscious squinting or straining to focus.

On a computer, a CRT with a low refresh rate (less than 70 Hz) can cause similar problems because of the flickering image. Aging CRTs also often go slightly out of focus, and this can also cause eye strain. LCDs do not go out of focus and are less susceptable to visible flicker. Higher refresh rates and larger font sizes are worthwhile when addressing eyestrain problems.

Causes

Sometimes, asthenopia can be due to specific visual problems, such as uncorrected refraction errors or binocular vision problems like accommodative insufficiency or heterophoria.

http://en.wikipedia.org/wiki/Asthenopia

ASTIGMATISM

Astigmatism means that the cornea is oval like a football instead of spherical like a basketball. Most astigmatic corneas have two curves – a steeper curve and a flatter curve. This causes light to focus on more than one point in the eye, resulting in blurred vision at distance or near. Astigmatism often occurs along with nearsightedness or farsightedness.

Signs and Symptoms

Blurred vision (near and distance)

Detection and Diagnosis

Astigmatism can be detected and measured with corneal topography, keratometry, vision testing and refraction.

Treatment

Astigmatism can be corrected with glasses, contacts, or surgically. The most common surgeries used to correct astigmatism are astigmatic keratotomy (procedures that involve placing a microscopic incision on the eye) and LASIK. The objective of these procedures is to reshape the cornea so it becomes more spherical or uniformly curved.

http://www.stlukeseye.com/conditions/Astigmatism.asp

BLEPHARITIS

Blepharitis is inflammation of the eyelids. It is characterized by inflammation of the eyelid margins. Blepharitis usually causes redness of the eyes and itching and irritation of the eyelids in both eyes. Its appearance is often confused with conjunctivitis and due to its recurring nature it is the most common cause of "recurrent conjunctivitis" in older people. It is also often treated as "dry eye" by patients due to the gritty sensation it may give the eyes - although lubricating drops do little to improve the condition.

There are two types:

1. Anterior blepharitis affects the front of the eyelids near the eyelashes. The causes are seborrheic dermatitis (similar to dandruff) and occasional infection by Staphylococcus bacteria.
2. Posterior blepharitis affects the back of the eyelids, the part that makes contact with the eyes. This is caused by the oil glands present in this region. It is by far, the most common type of blepharitis

Staphylococcal blepharitis

Staphlycoccal blepharitis is a type of external eye inflammation. As with dandruff, it is usually asymptomatic until the disease progresses. As it progresses, the sufferer begins to notice a foreign body sensation, matting of the lashes, and burning. Usually, the primary care physician will prescribe topical antibiotics for staphylococcal blepharitis. Unfortunately this is not an effective treatment.

This ailment can sometimes lead to a chalazion or a stye.

Seborrheic blepharitis

Seborrheic blepharitis, the most common type of blepharitis, is usually one part of the spectrum of seborrheic dermatitis seborrhea which involves the scalp, lashes, eyebrows, nasolabial folds and ears. Treatment is best accomplished by a dermatologist.

Posterior blepharitis or Rosacea associated blepharitis

The most common type of blepharitis is often found in people with a rosacea skin type. The oil glands in the lid (Meiobian glands) secrete a modified oil which leads to inflammation at the gland openings which are found at the edge of the lid.

Treatment and management

The single most important treatment principle is a daily routine of lid margin hygiene as described below. Such a routine needs to be convenient enough to be continued lifelong to avoid relapses as blepharitis is a lifelong condition.

A typical lid margin hygiene routine consists of 3 steps:

1. Softening of lid margin debris and oils: Apply a warm wet compress to the lids - such as a washcloth with hot water - for about 2 minutes.

2. Mechanical removal of lid margin debris: At end of shower routine, wash your face with a wash cloth. Use facial soap or non-burning baby shampoo (make sure to dilute the soap solution 1/10 with water first). Gently and repeatedly rub along the lid margins while eyes are closed.
3. Antibiotic reduction of lid margin bacteria (at the discretion of your physician): After lid margin cleaning, spread small amount of prescription antibiotic ophthalmic ointment with finger tip along lid fissure while eyes closed. Use prior to bed time as opposed to in the morning to avoid blurry vision.

The following guide is very common but is more challenging to perform by visually disabled or frail patients as it requires good motor skills and a mirror. Compared to above it does not bear any advantages:

1. Apply hot compresses to both eyes for 5 minutes once to twice per day.
2. After hot compresses, in front of a mirror, use a moist Q-tip soaked in a cup of water with a drop of baby shampoo. Rub along the lid margins while tilting the lid outward with the other hand.
3. In front of mirror, place small drop of antibiotic ophthalmic ointment (e.g. erythromycin) in lower conjunctival sack while pulling lid away from eye with other hand.

Often the above is advised together with mild massage to mechanically empty glands located at the lid margin (Meiobian glands, Zeiss glands, Moll glands).

Depending on the degree of inflammation of the lid margin, a combination of topical antibiotic and steroid drops or ointments can be prescribed to provide instant relief. However, this harbors significant risks such as increased intraocular pressure and posterior subcapsular cataract formation. Since cataract formation is irreversible and even intraocular hypertension might be (harboring the risk of glaucoma with permanent visual loss), both need to be checked for monthly. Steroid-induced cataracts and ocular hypertension can affect all ages.

If acne rosacea coexists, treatment should be focused on this skin disorder as the underlying cause together with the above lid margin hygiene routine. Typically, 100 mg doxycycline by mouth twice per day is prescribed for four to six weeks which can be tapered to 50 mg once daily for several years. Some physicians use a lower starting dose. Patients are instructed to continue use for at least two months before symptoms improve significantly. Contrary to common belief, use of tetracycline-type antibiotics is not primarily to treat bacterial infection but rather to inhibit matrix metalloproteinases resulting in thinning of oil gland secretions and change of the characteristic prominent capillary pattern.

Dermatologists treat blepharitis similarly to seborrheic dermatitis by using safe topical anti-inflammatory medication like sulfacetamide or brief courses of a mild topical steroid. Although anti-fungals like ketoconazole (Nizoral) are commonly prescribed for seborrheic dermatitis, dermatologists and optometrists usually do not prescribe anti-fungals for seborrheic blepharitis.

If these conventional treatments for blepharitis do not bring relief, patients should consider allergy testing and ocular antihistamines. Allergic responses to dust mite feces and other allergens can cause lid inflammation, ocular irritation, and dry eyes. Prescription optical antihistamines like Patanol, Optivar, Elestat, and over the counter optical antihistamines like

Zaditor are very safe and can bring almost immediate relief to patients whose lid inflammation is caused by allergies.

http://en.wikipedia.org/wiki/Blepharitis

BUPHTHALMOS

Buphthalmos is a congenital condition of the eye. An abnormally narrow angle between the cornea and iris blocks the outflow of aqueous humor, which leads to an increased intraocular pressure and a characteristic bulging enlargement of the eyeball. Other clinical signs include excessive tearing and cupping of the optic disk, which may the first sign to develop.

Treatment

Treatment involves surgically opening a hole in the iris to allow flow of aqueous humor, thus relieving the pressure in the posterior chamber of the eye. Early treatment is critical for preserving vision.

http://en.wikipedia.org/wiki/Buphthalmos

CATARACT

A **cataract** is an opacity that develops in the crystalline lens of the eye or in its envelope. Early on in the development of age-related cataract the power of the crystalline lens may be increased, causing near-sightedness (myopia), and the gradual yellowing and opacification of the lens may reduce the perception of blue colours. Cataracts typically progress slowly to cause vision loss and are potentially blinding if untreated. Moreover, with time the cataract cortex liquefies to form a milky white fluid in a **Morgagnian Cataract**, and can cause severe inflammation if the lens capsule ruptures and leaks. Untreated, the cataract can cause phacomorphic glaucoma. Very advanced cataracts with weak zonules are liable to dislocation anteriorly or posteriorly. Such spontaneous posterior dislocations (akin to the historical surgical procedure of couching) in ancient times were regarded as a blessing from the heavens, because it restored some perception of light in the bilaterally affected patients.

Causes

Cataracts develop from a variety of reasons, including long-term ultraviolet exposure, exposure to radiation, secondary effects of diseases such as diabetes, hypertension and advanced age; they are usually a result of denaturation of lens proteins. Genetic factors are often a cause of congenital cataracts and positive family history may also play a role in predisposing someone to cataracts at an earlier age, a phenomenon of "anticipation" in pre-senile cataracts. Cataracts may also be produced by eye injury or physical trauma. Cataracts may be partial or complete, stationary or progressive, hard or soft.

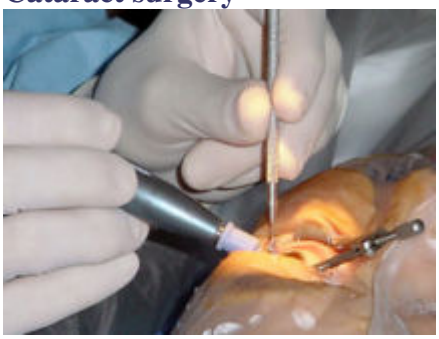
Some drugs can induce cataract development, such as Corticosteroids and Ezetimibe

There are various types of cataract, e.g. nuclear, cortical, mature, hypermature. Cataracts are also classified by their location, e.g. posterior (classically due to steroid use) and anterior (common (senile) cataract related to aging).

Epidemiology

Cataracts are the leading cause of blindness in the world.

Cataract surgery



Cataract surgery, using a temporal approach phacoemulsification probe (in right hand) and "chopper" (in left hand) being done under operating microscope at a Navy medical center

The most effective and common treatment is to surgically remove the cloudy lens. There are two types of surgery that can be used to remove cataracts: extra-capsular (extracapsular cataract extraction, or ECCE) and intra-capsular (intracapsular cataract extraction, or ICCE).

Extra-capsular (ECCE) surgery consists of removing the lens but leaving the majority of the lens capsule intact. High frequency sound waves (phacoemulsification) are sometimes used to break up the lens before extraction.

Intra-capsular (ICCE) surgery involves removing the entire lens of the eye, including the lens capsule, but it is rarely performed in modern practice. In either extra-capsular surgery or intra-capsular surgery, the cataractous lens is removed and replaced with a plastic lens (an intraocular lens implant) which stays in the eye permanently.

Cataract operations are usually performed using a local anaesthetic and the patient is allowed to go home the same day. Recent improvements in intraocular technology now allow cataract patients to choose a multifocal lens to create a visual environment in which they are less dependent on glasses. Under some medical systems multifocal lenses cost extra. Traditional intraocular lenses are monofocal.

Complications after cataract surgery, including endophthalmitis, posterior capsular opacification and retinal detachment, are possible.

In ICCE there is the issue of the Jack in the box phenomenon where the patient has to wear aphakic glasses - alternatives include contact lenses but these can prove to be high maintenance, particularly in dusty areas.

Prevention

Although cataracts have no scientifically proven prevention, it is sometimes said that wearing ultraviolet-protecting sunglasses may slow the development of cataracts. Regular intake of antioxidants (such as vitamin A, C and E) is theoretically helpful, but taking them as a supplement has been shown to have no benefit.

http://en.wikipedia.org/wiki/Cataract

CENTRAL SEROUS CHORIORETINOPATHY-(CSCR)

Central serous chorioretinopathy (CSCR) is a problem that affects the macula (central portion of the retina). The exact cause is not understood. CSCR occurs when a small break forms in the pigment layer of the retina. Fluid from the layer of blood vessels that lie underneath the retina seeps up through the break, causing a small detachment to form under the retina.

This problem is somewhat similar to a water blister that forms on the skin. The process is similar to CSCR: fluid collects beneath the skin's surface, causing the layers of skin to separate.

CSCR affects men more often then women and usually occurs between the ages of 25 and 50. Stress is thought to be linked to this problem. CSCR typically resolves spontaneously, but it can recur. In some cases, it may lead to moderate but permanent loss of central vision.

Signs and Symptoms

- Blurred central vision
- Wavy, distorted vision
- Central blind spot

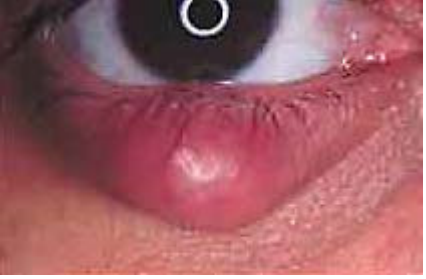
Detection and Diagnosis

Usually the doctor can diagnose CSCR with an exam of the retina using ophthalmoscopy. In most cases fluorescein angiography is used to gather additional information about the extent and severity of the problem.

Treatment

Most patients with CSCR do not require treatment. The fluid usually absorbs gradually over a period of months. Occasionally, steroid and non-steroidal anti-inflammatory eye drops are prescribed. In cases where visual recovery is delayed, laser treatment may be required to seal the leak and help the vision improve.

http://www.stlukeseye.com/conditions/CSCR.asp



CHALAZION

A chalazion (stye) is a small lump in the eyelid caused by obstruction of an oil producing or meibomian gland. Chalazia may occur in the upper or lower lids, causing redness, swelling and soreness in some cases.

Signs and Symptoms

- Raised, swollen bump on the upper or lower eye lid
- Often red
- May be tender and sore

Diagnosis

Patients often request an examination after an episode of pain and swelling of the lid. The doctor can make the diagnosis during a simple examination of the eyelids.

Treatment

In the early stages, chalazia may be treated at home with the repeated use of warm compresses for 15 - 20 minutes followed by several minutes of light lid massage. This helps to reduce the swelling and makes the lid more comfortable. However, if the chalazion does not diminish or recurs, medical attention may be necessary. This may include draining the chalazion along with the use of antibiotic and anti-inflammatory medications.

http://www.stlukeseye.com/Conditions/Chalazion.asp

CHEMICAL BURNS

Quick reactions can make the difference between sight and blindness

It can happen in the blink of an eye. While pouring liquid drain cleaner down a sink, some of the chemical splashes up in your face, hitting you squarely in the eye. Chemical injuries don't just happen in the workplace. Most homes have dozens of everyday products that pose tremendous danger to vision if they contact the eye.

The severity of the injury is related to whether the chemical is alkali or

Alkali- based chemicals
• Lime (cement, plaster, whitewash) • Drain cleaners • Lye

- Metal polishes
- Ammonia
- Oven cleaners

Acid-based chemicals

- Swimming pool acid (muriatic acid)
- Battery (sulfuric) acid

acid-based. Alkali chemicals are more destructive then acidic chemicals because of their ability to adhere to the eye and penetrate tissues. However, acid burns may be compounded by glass injuries caused by an explosion.

Often, the difference between a serious but treatable injury and losing vision is a matter of understanding a few principles of ocular first aid.

Emergency care

After chemical exposure, the first step is to immediately (within seconds) begin flushing the eye with water. If the accident occurs in an industrial setting, special irrigating facilities should be available. If the injury happens at home, begin flushing the eye with water right away, call for help immediately and contact your local ophthalmologist.

The easiest way to irrigate at home is for the patient

First aid at home

- Help the patient hold his or her head over a sink
- Gently hold the lids apart with a cotton swab or dry cloth
- Pour water over the eye, making sure to rinse inside the eyelids

to hold his or her head over a sink while the helper continuously pours water over the eye with a glass or cup. It is important to gently hold the lids apart while irrigating in order to rinse underneath the lids and wash away as much of the chemical as possible. Using a dry cloth is helpful because the lids are difficult to hold back when they are wet. Continue flushing the eye for approximately 20 minutes.

Call your ophthalmologist

Secondary care at the ophthalmologist's office

If possible, bring the chemical used at the time of the accident to the doctor's office. The type of chemical, concentration, and key ingredients may give the doctor valuable information needed for treatment. The doctor may continue irrigation to insure that the chemical is diluted as much as possible. The eye will be carefully examined under magnification to determine the extent of the injury and whether there are any foreign particles imbedded in the eye.

An ounce of prevention...

Taking care to prevent chemical injuries is the best first aid. Follow these simple steps to reduce your risk:

- Follow package directions and warnings before using chemicals
- When using chemicals, always wear safety glasses
- Never put your face over a drain after applying chemicals

The chance of regaining useful vision following a chemical accident is dependent on the nature and type of injury. However, knowing how to initiate treatment at home greatly increases the odds of recovery and saving vision.

http://www.stlukeseye.com/conditions/ChemicalBurn.asp

COGAN'S DYSTROPHY

Cogan's Dystrophy (Map-Dot-Fingerprint Dystrophy)

Cogan's Dystrophy is a disease that affects the cornea. It is commonly called Map-Dot-Fingerprint Dystrophy because of microscopic dot and fingerprint-like patterns that form within the layers of the cornea.

The cornea is comprised of five layers. Cogan's affects the superficial cornea layer called the epithelium. The epithelium's bottom, or basement layer of cells becomes thickened and uneven. This weakens the bond between the cells and sometimes causes the epithelium to become loosened and slough off in areas. This problem is called corneal erosion.

Even though this disease is commonly known as a dystrophy (a term that describes genetic diseases), Cogan's is not necessarily an inherited problem. It often affects both eyes and is typically diagnosed after the age of 30. Cogan's usually becomes progressively worse with age.

Signs and Symptoms

Some patients with Cogan's dystrophy have no symptoms at all. The symptoms among patients may may vary widely in severity and include:

- Light sensitivity
- Glare
- Fluctuating vision
- Blurred vision
- Irregular astigmatism (uneven corneal surface)
- Mild to extreme irritation and discomfort that is worse in the morning

Detection and Diagnosis

The doctor examines the layers of the cornea with a slit lamp microscope. In some cases, corneal topography may be needed to evaluate and monitor astigmatism resulting from the

disease.

Treatment

The treatment for Cogan's is dependent on the severity of the problem. The first step is to lubricate the cornea with artificial tears to keep the surface smooth and comfortable. Lubricating ointments are recommended at bedtime so the eyes are more comfortable in the morning. Salt solution drops or ointments such as sodium chloride are often prescribed to reduce swelling and improve vision. Gas permeable contacts are occasionally fit for patients with irregular astigmatism to create a smooth, even corneal surface and improve vision.

For patients with recurrent corneal erosion, soft, bandage contact lenses may be used to keep the eye comfortable and allow the cornea to heal. In some cases, laser treatment may beneficial. The surgeon removes the epithelium with an Excimer laser, creating a regular, smooth surface. The epithelium quickly regenerates, usually within a matter of days, forming a better bond with the underlying cell layer.

http://www.stlukeseye.com/conditions/CogansDystrophy.asp

COGAN SYNDROME

Cogan syndrome is a rare disorder characterized by recurrent inflammation of the front of the eye (the cornea) and often fever, fatigue, and weight loss, episodes of dizziness, and hearing loss. It can lead to deafness or blindness if untreated. The classic form of the disease was first described by D.G. Cogan in 1945.

Symptoms of Cogan's Syndrome include a decrease in vision (typically in one eye) and either a decrease in hearing or a "ringing" sensation in the same ear. Treatment includes steroids (most commonly prednisone) to address inflammation.

http://en.wikipedia.org/wiki/Cogan_syndrome

COLOR BLINDNESS

Color blindness may be a hereditary condition or caused by disease of the optic nerve or retina. Acquired color vision problems only affect the eye with the disease and may become progressively worse over time. Patients with a color vision defect caused by disease usually have trouble discriminating blues and yellows.

Inherited color blindness is most common, affects both eyes, and does not worsen over time. This type is found in about 8% of males and 0.4% of females. These color problems are linked to the X chromosome and are almost always passed from a mother to her son.

Color blindness may be partial (affecting only some colors), or complete (affecting all colors). Complete color blindness is very rare. Those who are completely color blind often have other serious eye problems as well.

Photoreceptors called cones allow us to appreciate color. These are concentrated in the very center of the retina and contain three photosensitive pigments: red, green and blue. Those with defective color vision have a deficiency or absence in one or more of these pigments. Those with normal color vision are referred to as trichromats. People with a deficiency in one of the pigments are called anomalous trichromats (the most common type of color vision problem.) A dichromat has a complete absence in one cone pigment.

Signs and Symptoms

The symptoms of color blindness are dependent on several factors, such as whether the problem is congenital, acquired, partial, or complete.

- Difficulty distinguishing reds and greens (most common)
- Difficulty distinguishing blues and greens (less common)

The symptoms of more serious inherited color vision problems and some types acquired problems may include:

- Objects appear as various shades of gray (this occurs with complete color blindness and is very rare)
- Reduced vision
- Nystagmus

Detection and Diagnosis

Color vision deficiency is most commonly detected with special colored charts called the Ishihara Test Plates. On each plate is a number composed of colored dots. While holding the chart under good lighting, the patient is asked to identify the number. Once the color defect is identified, more detailed color vision tests may be performed.

Treatment

There is no treatment or cure for color blindness. Those with mild color deficiencies learn to associate colors with certain objects and are usually able to identify color as everyone else. However, they are unable to appreciate color in the same way as those with normal color vision.

http://www.stlukeseye.com/conditions/ColorBlindness.asp

COMPUTER VISION SYNDROME

Computer vision syndrome (CVS) is a term that describes eye-related problems and the other symptoms caused by prolonged computer use. As our dependence on computers

continues to grow, an increasing number of people are seeking medical attention for eye strain and irritation, along with back, neck, shoulder, and wrist soreness.

Magnified view of a printed letter

These problems are more noticeable with computer tasks than other near work because letters on the screen are formed by tiny dots called pixels, rather than a solid image. This causes the eye to work a bit harder to keep the images in focus.

There is no scientific evidence that computer screens are harmful to the eyes. A common myth is that too eye strain caused by reading and close work is damaging to the eyes. This is not true; however, those who work at computers often experience many frustrating symptoms.

http://www.stlukeseye.com/Conditions/ComputerVisionSyndrome.asp

Magnified view of a letter on a computer screen

CONE-ROD DYSTROPHY

The rod and cone photoreceptor cells are necesssary for good vision. They help us see different things. While RODS are good at seeing things that are moving, CONES help us see things that are stil. Rods arenecessary to see things in black and white but Cones are neccessary for seeing in color. In this type of eye disesase, the person's photoreceptor cells may not work from childhood, or may lose their ability to function over time. Gradual loss of night vision and gradual loss of peripheral vision are the symptoms of this disease. The cause is not known and as of today there are no cures for the disease and the vision loss can not be prevented

CONJUNCTIVITIS

Conjunctivitis commonly called "**Pink Eye**" and "**Red Eye**" in the UK, and "**Madras Eye**" in India is an inflammation of the conjunctiva (the outermost layer of the eye and the inner surface of the eyelids), most commonly due to an allergic reaction or an infection (usually bacterial, or viral).

Variants

Blepharoconjunctivitis is the combination of conjunctivitis with blepharitis (inflammation of the eyelids).

Keratoconjunctivitis is the combination of conjunctivitis and keratitis (corneal inflammation).

Episcleritis is an inflammatory condition that produces a similar appearance to conjunctivitis, but without discharge or tearing.

Causes

Some forms of conjunctivitis are extremely contagious while others are not. It all depends on the etiology.

Diagnosis

Symptoms

Eyes with conjunctivitis

Redness, irritation and watering of the eyes are symptoms common to all forms of conjunctivitis. Itch and the closing of the throat is variable.

Acute *allergic conjunctivitis* is typically itchy. Sometimes distressingly so, and the patient often complains of some lid swelling. Chronic allergy often causes just itch or irritation, and often much frustration because the absence of redness or discharge can lead to accusations of hypochondria.

Viral conjunctivitis is often associated with an infection of the upper respiratory tract, a common cold, and/or a sore throat. Its symptoms include watery discharge and variable itch. The infection usually begins with one eye, but may spread easily to the fellow eye.

Bacterial conjunctivitis due to the common pyogenic (pus-producing) bacteria causes marked grittiness/irritation and a stringy, opaque, grey or yellowish mucopurulent discharge (*gowl*, *goop*, "gunk", "eye crust", *sleep*, or other regional names) that may cause the lids to stick together (*matting*), especially after sleeping. Another symptom that could be caused by Bacterial Conjunctivitis is severe crusting of the infected eye and the surrounding skin. However discharge is not essential to the diagnosis, contrary to popular belief. Many other bacteria (e.g., *Chlamydia*, *Moraxella*) can cause a non-exudative but very persistent conjunctivitis without much redness. The gritty and/or scratchy feeling is sometimes localised enough for patients to insist they must have a foreign body in the eye. The more acute pyogenic infections can be painful. Like viral conjunctivitis, it usually affects only one eye but may spread easily to the other eye. However, it is dormant in the eye for three days until patient shows signs of symptoms.

Irritant or *toxic conjunctivitis* is irritable or painful when the infected eye is pointed far down or far up. Discharge and itch are usually absent. This is the only group in which severe pain may occur.

Signs

An eye with bacterial conjunctivitis.

Infection (redness) of the conjunctiva on one or both eyes should be apparent, but may be quite mild. Except in obvious pyogenic or toxic/chemical conjunctivitis, a slit lamp (biomicroscope) is needed to have any confidence in the diagnosis. Examination of the tarsal conjunctiva is usually more diagnostic than the bulbar conjunctiva.

Allergic conjunctivitis shows pale watery swelling or edema of the conjunctiva and sometimes the whole eyelid, often with a ropy, *non-purulent* mucoid discharge. There is variable redness.

Viral conjunctivitis, commonly known as "pink eye", shows a fine diffuse pinkness of the conjunctiva which is easily mistaken for the 'ciliary infection' of iritis, but there are usually corroborative signs on biomicroscopy, particularly numerous lymphoid follicles on the tarsal conjunctiva, and sometimes a punctate keratitis.

Pyogenic *bacterial conjunctivitis* shows an opaque purulent discharge, a very red eye, and on biomicroscopy there are numerous white cells and desquamated epithelial cells seen in the 'tear gutter' along the lid margin. The tarsal conjunctiva is a velvety red and not particularly follicular. Non-pyogenic infections can show just mild injection and be difficult to diagnose. Scarring of the tarsal conjunctiva is occasionally seen in chronic infections, especially in trachoma.

Irritant or *toxic conjunctivitis* show primarily marked redness. If due to splash injury, it is often present only in the lower conjunctival sac. With some chemicals—above all with caustic alkalis such as sodium hydroxide—there may be necrosis of the conjunctiva with a deceptively white eye due to vascular closure, followed by sloughing of the dead epithelium. This is likely to be associated with slit-lamp evidence of anterior uveitis.

Differential diagnosis

Conjunctivitis symptoms and signs are relatively non-specific. Even after biomicroscopy, laboratory tests are often necessary if proof of aetiology is needed.

A purulent discharge strongly suggests bacterial cause, unless there is known exposure to toxins. Infection with *Neisseria gonorrhoeae* should be suspected if the discharge is particularly thick and copious.

A diffuse, less "injected" conjunctivitis (looking pink rather than red) suggests a viral cause, especially if numerous follicles are present on the lower tarsal conjunctiva on biomicroscopy.

Scarring of the tarsal conjunctiva suggests trachoma, especially if seen in endemic areas, if the scarring is linear (von Arlt's line), or if there is also corneal vascularisation.

Clinical tests for lagophthalmos, dry eye (Schirmer test) and unstable tear film may help distinguish the various types of dry eye.

Other symptoms including pain, blurring of vision and photophobia should not be prominent in conjunctivitis. Fluctuating blurring is common, due to tearing and mucoid discharge. Mild photophobia is common. However, if any of these symptoms are prominent, it is important to exclude other diseases such as glaucoma, uveitis, keratitis and even meningitis or caroticocavernous fistula.

Many people who have conjunctivitis have trouble opening their eyes in the morning because of the dried mucus on their eyelids. There is often excess mucus over the eye after sleeping for a long period of time.

Investigations

These are done infrequently because most cases of conjunctivitis are treated empirically and (eventually) successfully, but often only after running the gamut of the common possibilities.

Swabs for bacterial culture are necessary if the history & signs suggest bacterial conjunctivitis, but there is no response to topical antibiotics. Research studies indicate that many bacteria implicated in low-grade conjunctivitis are not detected by the usual culture methods of medical microbiology labs, so negative results are common. Viral culture may be appropriate in epidemic case clusters. Conjunctival scrapes for cytology can be useful in detecting chlamydial and fungal infections, allergy and dysplasia, but are rarely done because of the cost and the general lack of laboratory staff experienced in handling ocular specimens. Conjunctival incisional biopsy is occasionally done when granulomatous diseases (e.g., sarcoidosis) or dysplasia are suspected.

Treatment and management

Conjunctivitis sometimes requires medical attention. The appropriate treatment depends on the cause of the problem. For the allergic type, cool water constricts capillaries, and artificial tears sometimes relieve discomfort in mild cases. In more severe cases, non-steroidal anti-inflammatory medications and antihistamines may be prescribed. Some patients with persistent allergic conjunctivitis may also require topical steroid drops.

Bacterial conjunctivitis is usually treated with antibiotic eye drops or ointments that cover a broad range of bacteria (chloramphenicol or fusidic acid used in UK). However evidence suggests that this does not affect symptom severity and gains only modest reduction in duration from an average of 4.8 days (untreated controls) to 3.3 days for those given immediate antibiotics. Deferring antibiotics yields almost the same duration as those immediately starting treatment with 3.9 days duration, but with half the two-week clinic reattendance rate.

Although there is no cure for viral conjunctivitis, symptomatic relief may be achieved with warm compressesand artificial tears. For the worst cases, topical corticosteroid drops may be prescribed to reduce the discomfort from inflammation. However prolonged usage of corticosteroid drops increases the risk of side effects. Antibiotic drops may also be used for treatment of complementary infections. Patients are often advised to avoid touching their eyes or sharing towels and washcloths. Viral conjunctivitis usually resolves within 3 weeks. However in worst cases it may take over a month.

Conjunctivitis due to burns, toxic and chemical require careful wash-out with saline, especially beneath the lids, and may require topical steroids. The more acute chemical injuries are medical emergencies, particularly alkali burns, which can lead to severe scarring, and intraocular damage. Fortunately, such injuries are uncommon.

http://en.wikipedia.org/wiki/Conjunctivitis

CORNEAL ULCER

A **corneal ulcer** forms when the surface of the cornea is damaged or compromised. Ulcers may be sterile (no infecting organisms) or infectious. The term infiltrate is also commonly used along with ulcer. Infiltrate refers to an immune response causing an accumulation of cells or fluid in an area of the body where they don't normally belong.

Whether or not an ulcer is infectious is an important distinction for the physician to make and determines the course of treatment. Bacterial ulcers tend to be extremely painful and are typically associated with a break in the epithelium, the superficial layer of the cornea. In some cases, the inflammatory response involves the anterior chamber along with the cornea. Certain types of bacteria, such as *Pseudomonas,* are extremely aggressive and can cause severe damage and even blindness within 24-48 hours if left untreated.

Sterile infiltrates on the other hand, cause little if any pain. They are often found near the peripheral edge of the cornea and are not necessarily accompanied by a break in the epithelial layer of the cornea.

There are many causes of corneal ulcers. Contact lens wearers (especially soft) have an increased risk of ulcers if they do not adhere to strict regimens for the cleaning, handling, and disinfection of their lenses and cases. Soft contact lenses are designed to have very high water content and can easily absorb bacteria and infecting organisms if not cared for properly. Pseudomonas is a common cause of corneal ulcer seen in those who wear contacts.

Bacterial ulcers may be associated with diseases that compromise the corneal surface, creating a window of opportunity for organisms to infect the cornea. Patients with severely dry eyes, difficulty blinking, or are unable to care for themselves, are also at risk. Other causes of ulcers include: herpes simplex viral infections, inflammatory diseases, corneal abrasions or injuries, and other systemic diseases.

Signs and Symptoms

The symptoms associated with corneal ulcers depend on whether they are infectious or sterile, as well as the aggressiveness of the infecting organism.

- Red eye
- Severe pain (not in all cases)
- Tearing
- Discharge
- White spot on the cornea, that depending on the severity of the ulcer, may not be visible with the naked eye
- Light sensitivity

Detection and Diagnosis

Corneal ulcers are diagnosed with a careful examination using a slit lamp microscope. Special types of eye drops containing dye such as fluorescein may be instilled to highlight the ulcer, making it easier to detect.

If an infectious organism is suspected, the doctor may order a culture. After numbing the eye with topical eye drops, cells are gently scraped from the corneal surface and tested to determine the infecting organism.

Treatment

The course of treatment depends on whether the ulcer is sterile or infectious. Bacterial ulcers require aggressive treatment. In some cases, antibacterial eye drops are used every 15 minutes. Steroid medications are avoided in cases of infectious ulcers. Some patients with severe ulcers may require hospitalization for IV antibiotics and around-the-clock therapy. Sterile ulcers are typically treated by reducing the eye's inflammatory response with steroid drops, anti-inflammatory drops, and antibiotics.

http://www.stlukeseye.com/Conditions/CornealUlcer.asp

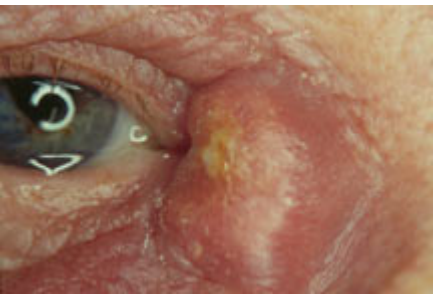
CYTOMEGALOVIRUS

The **cytomegalovirus (CMV)** is related to the herpes virus and is present in almost everyone. Normally, most people's immune systems are able to fight the virus, preventing it from causing problems in their bodies. However, when the immune system is suppressed because of disease (HIV), organ or bone marrow transplant, or chemotherapy, the CMV virus can cause damage and disease to the eye and the rest of the body.

CMV is the most common type of virus that infects those who are HIV positive. It affects the eye in about 30% of the cases by causing damage to the retina. This is called CMV retinitis. The likelihood of developing CMV retinitis increases as the CD_4 cell count decreases.

CMV retinitis may affect one eye at first, but usually progresses to both eyes and becomes worse as the patient's ability to fight infection decreases. The virus is sight threatening and usually requires the care and treatment of a vitreo-retinal surgeon. Patients with CMV

retinitis are at risk of retinal detachment, hemorrhages, and inflammation of the retina that can lead to permanent loss of vision and even blindness.



Signs and Symptoms

CMV retinitis usually causes symptoms, but not always. Patients with a condition that suppresses the immune system should watch for the following eye symptoms while under the care of a physician.

- Floaters (spots, bugs, spider webs)
- Light flashes
- Blind spots
- Blurred vision
- Obstructed areas of vision
- Sudden decrease of vision

Detection and Diagnosis

Most patients with CMV retinitis are referred for eye treatment by another physician. The vitreo-retinal surgeon diagnoses CMV retinitis by thoroughly examining the back of the eye using ophthalmoscopy. Fluorescein angiography may be needed to evaluate the circulatory system of the retina.

Treatment

When managing CMV retinitis, the doctor's goal is to slow the progression of the disease and to treat related eye problems. Anti-viral medications such as ganciclovir or foscarnet are often prescribed. These drugs can be administered orally, intravenously, injected directly into the eye or through an intravitreal implant.

http://www.stlukeseye.com/Conditions/CMV.asp

DACRYOCYSTITIS

Dacryocystitis is an infection of the tear sac that lies between the inner corner of the eyelids and the nose. It usually results from blockage of the duct that carries tears from the tear sac to the nose. The blocked duct harbors bacteria and becomes infected. Dacryocystitis may be acute (sudden onset) or chronic (frequently recurs). It may be related to a malformation of the tear duct, injury, eye infection, or trauma.

This problem is most common in infants because their tear ducts are often underdeveloped and clog easily. Babies often have recurrent episodes of infection; however, in most cases, the problem resolves as the child grows. In adults, the infection may originate from an injury or inflammation of the nasal passages. In many cases, however, the cause is unknown.

Signs and Symptoms

- Generally affects one eye
- Excessive tearing
- Tenderness, redness, and swelling
- Discharge
- Red, inflamed bump on the inner corner of the lower lid

Detection and Diagnosis

During the exam, the doctor will determine the extent of the blockage. Cultures may be taken of the discharge to identify the type of infection. The doctor will also determine whether the infection has affected the eye.

Treatment

The treatment for dacryocystitis is dependent on the person's age, whether the problem is chronic or acute, and the cause of the infection.

Infants are usually treated first by gently massaging the area between the eye and nose to help open the obstruction along with antibiotic drops or ointments for the infection. Surgery may be necessary to clear the obstruction if medical treatment is not effective and the problem persists over several months.

Before surgery, the doctor may treat the child with antibiotics to make sure the infection is cleared. The operation is performed under general anesthesia. The tear duct is gently probed to open the passage.

For adults, the doctor may clear the obstruction by irrigating the tear duct with saline. Surgery is sometimes necessary for adults if irrigation, or antibiotics fail to resolve the infection or if the infection becomes chronic. In these cases, dacryocystorhinostomy (DCR) is performed under general anesthesia to create a new passage for the tear flow.

http://www.stlukeseye.com/Conditions/Dacryocystitis.asp

DIPLOPIA

Diplopia, commonly known as **double vision**, is the simultaneous perception of two images of a single object. These images may be displaced horizontally, vertically, or diagonally (i.e. both vertically and horizontally) in relation to each other.

Binocular diplopia

Binocular diplopia is double vision arising as a result of the misalignment of the two eyes relative to each other, such as occurs in Esotropia or Exotropia. In such a case whilst the fovea of one eye is directed at the object of regard, the fovea of the other is directed elsewhere, and the image of the object of regard falls on an extra-foveal area of retina.

The brain calculates the 'visual direction' of an object based upon the position of its image relative to the fovea. Images falling on the fovea are seen as being directly ahead, whilst those falling on retina outside the fovea may be seen as above, below, right or left of straight ahead depending upon the area of retina stimulated. Thus, when the eyes are misaligned, the brain will perceive two images of one target object, as the target object simultaneously stimulates different, non-corresponding, retinal areas in either eye, thus producing double vision.

This correlation of particular areas of the retina in one eye with the same areas in the other is known as Retinal correspondence. This relationship also gives rise to an associated phenomenon of binocular diplopia, although one that is rarely noted by those experiencing diplopia: Because the fovea of one eye corresponds to the fovea of the other, images falling on the two foveas are 'projected' to the same point in space. Thus, when the eyes are misaligned, the brain will 'project' two different images in the same visual direction. This phenomenon is known as 'Confusion'.

Double vision is dangerous to survival, therefore, the brain naturally guards against its occurrence. In an attempt to avoid double vision, the brain can sometimes ignore the image from one eye; a process known as suppression. The ability to suppress is to be found particularly in childhood when the brain is still developing. Thus, those with childhood strabismus almost never complain of diplopia whilst adults who develop strabismus almost always do. Whilst this ability to suppress might seem a wholly positive adaptation to strabismus, in the developing child this can prevent the proper development of vision in the affected eye resulting in amblyopia. Some adults are also able to suppress their diplopia, but their suppression is rarely as deep or as effective and takes longer to establish. They are not at risk of permanently damaging their vision as a result though. It can appear sometimes, therefore, that diplopia disappears without medical intervention. However, in some cases the cause of the double vision may still be present.

Monocular diplopia

More rarely, diplopia can also occur when viewing with only one eye; this is called **monocular diplopia**, or, where the patient perceives more than two images, **monocular polyopia**. In this case, the differential diagnosis of multiple image perception includes a structural defect within the eye, a lesion in the anterior visual cortex (rarely cause diplopia, more commonly polyopia or palinopsia) or non-organic conditions.

Temporary diplopia

Temporary diplopia can also be caused by intoxication from alcohol or head injuries, such as concussion. If temporary double vision does not resolve quickly, one should see an eye doctor immediately. It can also be a side effect of the anti-epileptic drugs Phenytoin and Zonisamide, and the anti-convulsant drug Lamotrigine, as well as the hypnotic drug Zolpidem and the dissociative drug Ketamine.

Treatment for binocular diplopia

The appropriate treatment for binocular diplopia will depend upon the cause of the condition producing the symptoms. Efforts must first be made to identify and treat the underlying cause of the problem. Treatment options includes prism lenses and/or vision therapy and/or surgery, and also botulinum toxin can be used. On occasions, in certain conditions such as Oculomotor nerve palsy for example, it may be necessary to occlude one eye either temporarily or permanently. Daily wear of prism lenses is a passive compensatory treatment. Vision therapy is an active treatment which retrains the visual and vestibular systems (brain, eye muscles, and body). Vision therapy may eliminate the need for daily wear of prism lenses but is only suitable for a minority of those with diplopic symptoms.

Voluntary diplopia

Some people are able to consciously uncouple their eyes, inducing double vision on purpose. These people do not consider their double vision dangerous or harmful, and may even consider it enjoyable. It makes viewing stereograms much easier.

http://en.wikipedia.org/wiki/Diplopia

DRY EYE SYNDROME

Dry eye syndrome is one of the most common problems treated by eye physicians. Over ten million Americans suffer from dry eyes. It is usually caused by a problem with the quality of the tear film that lubricates the eyes.

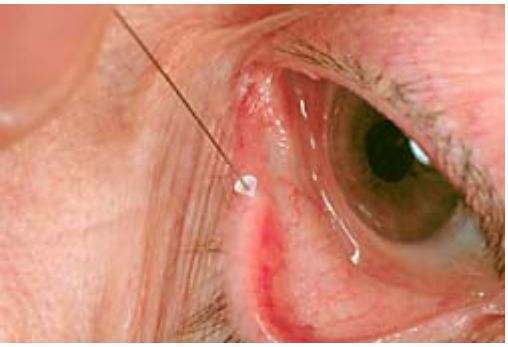
Tears are comprised of three layers. The mucus layer coats the cornea, the eye's clear outer window, forming a foundation so the tear film can adhere to the eye. The middle aqueous layer provides moisture and supplies oxygen and other important nutrients to the cornea. This layer is made of 98 percent water along with small amounts of salt, proteins and other compounds. The outer lipid layer is an oily film that seals the tear film on the eye and helps to prevent evaporation.

Tears are formed in several glands around the eye. The water layer is produced in the lacrimal gland, located under the upper eyelid. Several smaller glands in the lids make the oil and mucus layers. With each blink, the eyelids spread the tears over the eye. Excess tears flow into two tiny drainage ducts in the corner of the eye by the nose. These ducts lead to tiny canals that connect to the nasal passage. The connection between the tear ducts and the nasal passage is the reason that crying causes a runny nose.

In addition to lubricating the eye, tears are also produced as a reflex response to outside stimulus such as an injury or emotion. However, reflex tears do little to soothe a dry eye, which is why someone with watery eyes may still complain of irritation.

Dry eye syndrome has many causes. One of the most common reasons for dryness is simply the normal aging process. As we grow older, our bodies produce less oil – 60% less at age 65 then at age 18. This is more pronounced in women, who tend to have drier skin then men. The oil deficiency also affects the tear film. Without as much oil to seal the watery layer, the tear film evaporates much faster, leaving dry areas on the cornea.

Many other factors, such as hot, dry or windy climates, high altitudes, air-conditioning and cigarette smoke also cause dry eyes. Many people also find their eyes become irritated when reading or working on a computer. Stopping periodically to rest and blink keeps the eyes more comfortable.



Contact lens wearers may also suffer from dryness because the contacts absorb the tear film, causing proteins to form on the surface of the lens. Certain medications, thyroid conditions, vitamin A deficiency, and diseases such as Parkinson's and Sjogren's can also cause dryness. Women frequently experience problems with dry eyes as they enter menopause because of hormonal changes.

Symptoms

- Itching
- Burning
- Irritation
- Redness
- Blurred vision that improves with blinking
- Excessive tearing
- Increased discomfort after periods of reading, watching TV, or working on a computer

Detection and Diagnosis

There are several methods to test for dry eyes. The doctor will first determine the underlying cause by measuring the production, evaporation rate and quality of the tear film. Special drops that highlight problems that would be otherwise invisible are particularly helpful to diagnose the presence and extent of the dryness.

Treatment

A treatment option - BioTears™

When it comes to treating dry eyes, everyone's needs are a little different. Many find relief simply from using artificial tears on a regular basis. Some of these products are watery and alleviate the symptoms temporarily; others are thicker and adhere to the eye longer. Preservative-free tears are recommended because they are the most soothing and have fewer additives that could potentially irritate. Avoid products that whiten the eyes – they don't have adequate lubricating qualities and often make the problem worse.

Closing the opening of the tear drain in the eyelid with special inserts called punctal plugs is another option. This works like closing a sink drain with a stopper. These special plugs trap the tears on the eye, keeping it moist. This may be done on a temporary basis with a dissolvable collagen plug, or permanently with a silicone plug.

There are also simple lifestyle changes that can significantly improve irritation from dry eyes. For example, drinking eight to ten glasses of water each day keeps the body hydrated and flushes impurities. Make a conscious effort to blink frequently – especially when reading or watching television. Avoid rubbing the eyes. This only worsens the irritation.

Treating dry eye problems is important not only for comfort, but also for the health of the cornea.

http://www.stlukeseye.com/Conditions/DryEyeSyndrome.asp

ECTROPION

Patients with **ectropion** have a sagging lower eyelid that leaves the eye exposed and dry. It is caused by a lack of tone of the delicate muscles that hold the lid taut against the eye. Excessive tearing is a common with ectropion, but wiping the tears away only causes the lid to sag more. Ectropion is most common among people over the age of 60.

Signs and Symptoms

- Irritation
- Burning
- Gritty, sandy feeling
- Excessive tearing
- Red, irritated eyelid

Detection and Diagnosis

Ectropion can be diagnosed with a routine eye exam.

Treatment

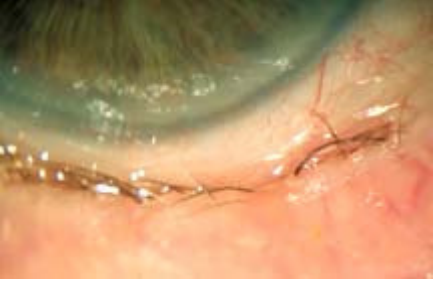
The irritation can be temporarily relieved with artificial tears and ointments to lubricate the eye; however, surgery to tighten the lid is usually necessary to correct this problem.

http://www.stlukeseye.com/Conditions/Ectropion.asp

ENTROPION

Entropion, an eyelid that turns inward, is a problem that typically affects the lower lid. It usually stems from a muscle spasm; however, it can also be caused by scarring from trauma or inflammation from certain diseases that involve the eyelids.

When the eyelid turns inward, the lashes rub against the eye, resulting in irritation, scratchiness, tearing and redness. Surgery is often required to correct the problem.



Signs and Symptoms

- Tearing
- Burning
- Irritation
- Sandy, gritty feeling
- Red eye

Detection and Diagnosis

Entropion can be detected during a routine eye exam. A slit lamp microscope is used to examine the effects of the in-turned eyelashes on the surface of the eye.

Treatment

The most effective treatment for entropion is surgery, although some patients find temporary relief by pulling the lower lid down with a piece of tape. Artificial tears are also helpful to ease the irritation caused from the lashes rubbing against the eye.

http://www.stlukeseye.com/Conditions/Entropion.asp

EPISCLERITIS

Episcleritis is an inflammatory condition of the connective tissue between the conjunctiva and sclera known as the episclera. The eye's red appearance makes it look similar to conjunctivitis, or pink eye, but there is no discharge or tearing. It usually has no apparent cause; however, it is sometimes associated with systemic inflammatory conditions such as arthritis, lupus, and inflammatory bowel disease. Rosacea, herpes simplex, gout, tuberculosis, and other diseases are also occasionally underlying causes.

Women are typically affected by episcleritis more frequently than men. It characteristically occurs in people who are in their 30's and 40's and is often a recurrent problem.

Signs and Symptoms

- Generalized or local redness
- Mild soreness or discomfort.

Detection and Diagnosis

Episcleritis is diagnosed with a slit lamp examination. The doctor will look for discharge, pain, and involvement of the underlying scleral to rule out other problems.

Treatment

Treatment for episcleritis is usually not needed. Chilled artificial tears can be used to soothe the eye and reduce mild inflammation. In more severe cases of episcleritis, mild steroids and anti-inflammatory medications are prescribed to reduce inflammation.

http://www.stlukeseye.com/Conditions/Episcleritis.asp

FLOATERS

Floaters are deposits of various size, shape, consistency, refractive index, and motility within the eye's vitreous humour, which is normally transparent. They may be of embryonic origin or acquired due to degenerative changes of the vitreous humour or retina. The perception of floaters is known as *myodesopsia*, or less commonly as *myiodeopsia*, *myiodesopsia*, or *myodeopsia*. Floaters appear as shadow-like shapes that appear alone or together with several others in one's field of vision. They may appear as spots, threads, or fragments of cobwebs, which float slowly before the sufferer's eyes. Since these objects exist within the eye itself, they are not optical illusions but are entoptic phenomena.

One specific type of floater is either called *Muscae volitantes* (from the Latin, meaning 'flying flies'), or *mouches volantes* (from the French), and consist of small spots, these are present in most people's eyes and are attributed to minute remnants of embryonic structures in the vitreous humour.

Floaters are suspended in the vitreous humour, the thick fluid or gel that fills the eye. Thus, they generally follow the rapid motions of the eye, while drifting slowly within the fluid. When they are first noticed, the natural reaction is to attempt to look directly at them. However, attempting to shift one's gaze toward them can be difficult since floaters follow the motion of the eye, remaining to the side of the direction of gaze. Floaters are, in fact, visible only because they do not remain perfectly fixed within the eye. Although the blood vessels of the eye also obstruct light, they are invisible under normal circumstances because they are fixed in location relative to the retina, and the brain "tunes out" stabilized images due to neural adaptation. This does not occur with floaters and they remain visible.

Floaters are particularly noticeable when looking at a blank surface or an open monochromal space, such as blue sky. Despite the name "floaters", many of these specks have a tendency to sink toward the bottom of the eyeball, in whichever way the eyeball is oriented; the supine position (looking up or lying back) tends to concentrate them near the fovea, which is the center of gaze, while the textureless and evenly lit sky forms an ideal background against which to view them. The brightness of the daytime sky also causes the eyes' pupils to contract, reducing aperture and increasing depth of field, which makes floaters less blurry and easier to see.

Floaters are not uncommon, and do not cause problems for most people; they represent one of the most common presentations to hospital eye services. A survey of optometrists in 2002 suggested that an average of 14 patients per month per optometrist presented with symptoms of floaters in the UK alone.However, floaters are more than a nuisance and a distraction to

those with severe cases, especially if the spots seem to constantly drift through the field of vision. The shapes are shadows projected onto the retina by tiny structures of protein or other cell debris discarded over the years and trapped in the vitreous humour. Floaters can even be seen when the eyes are closed on especially bright days, when sufficient light penetrates the eyelids to cast the shadows. It is not, however, only elderly people who suffer from floaters; they can certainly become a problem to younger people, especially if they are myopic. They are also common after cataract operations or after trauma. In some cases, floaters are congenital.

Floaters are able to catch and refract light in ways that somewhat blur vision temporarily until the floater moves to a different area. Often they trick the sufferer into thinking they see something out of the corner of their eye that really is not there. Most sufferers, with time, learn to ignore their floaters. For people with severe floaters it is nearly impossible to completely ignore the large masses that constantly stay within almost direct view. Some sufferers have noted a decrease in ability to concentrate while reading, watching television, walking outdoors, and driving, especially when tired.

Causes

There are various causes for the appearance of floaters, of which the most common are described here. Simply stated, any damage to the eye that causes material to enter the vitreous humour can result in floaters.

Vitreous syneresis

The most common cause of floaters is shrinkage of the vitreous humour: this gel-like substance consists of 99% water and 1% solid elements. The solid portion consists of a network of collagen and hyaluronic acid, with the latter retaining water molecules. Depolymerization of this network makes the hyaluronic acid release its trapped water, thereby liquefying the gel. The collagen breaks down into fibrils, which ultimately are the floaters that plague the patient. Floaters caused in this way tend to be few in number and of a linear form.

Posterior vitreous detachments and retinal detachments

In time, the liquefied vitreous body loses support and its framework contracts. This leads to posterior vitreous detachment, in which the vitreous body is released from the sensory retina. During this detachment, the shrinking vitreous can stimulate the retina mechanically, causing the patient to see random flashes across the visual field, sometimes referred to as "flashers." The ultimate release of the vitreous around the optic nerve head sometimes makes a large floater appear, usually in the shape of a ring ("Weiss ring"). As a complication, part of the retina might be torn off by the departing vitreous body, in a process known as retinal detachment. This will often leak blood into the vitreous, which is seen by the patient as a sudden appearance of numerous small dots, moving across the whole field of vision. Retinal detachment requires immediate medical attention, as it can easily cause blindness. Consequently, both the appearance of flashes and the sudden onset of numerous small floaters should be rapidly investigated by an ophthalmologist.

Regression of the hyaloid artery

The hyaloid artery, an artery running through the vitreous humour during the fetal stage of development, regresses in the third trimester of pregnancy. Its disintegration can sometimes leave cell matter.

Other common causes

Patients with retinal tears may experience floaters if red blood cells are released from leaky blood vessels, and those with a posterior uveitis or vitritis, as in toxoplasmosis, may experience multiple floaters and decreased vision due to the accumulation of white blood cells in the vitreous humour.

Other causes for floaters include cystoid macular edema and asteroid hyalosis. The latter is an anomaly of the vitreous humour, where by calcium clumps attach themselves to the collagen network. The bodies that are formed in this way move slightly with eye movement, but then return to their fixed position.

Tear film debris

Sometimes the appearance of floaters has to be attributed to dark specks in the tear film of the eye. Technically, these are not floaters, but they do look the same from the viewpoint of the patient. People with blepharitis or a dysfunctional meibomian gland are especially prone to this cause, but ocular allergies or even the wearing of contact lenses can cause the problem. To differentiate between material in the vitreous humour of the eye and debris in the tear film, one can look at the effect of blinking: debris in the tear film will move quickly with a blink, while floaters are largely unresponsive to it. Tear film debris is diagnosed by eliminating the possibility of true floaters and macular degeneration.

Diagnosis

Floaters are often readily observed by a doctor with the use of an ophthalmoscope or slit lamp. However, if the floater is a small piece of debris and near the retina they may not be able to observe it even if it appears large to the sufferer.

Increasing background illumination or using a pinhole to effectively decrease pupil diameter may allow a person to obtain a better view of his or her own floaters. The head may be tilted in such a way that one of the floaters drifts towards the central axis of the eye. In the sharpened image the fibrous elements are more conspicuous. (If the pinhole is kept moving slowly in small circles, the same technique evokes an interesting entoptic effect known as the vascular figure, which is a view of the blood vessels within one's own eye.)

Treatment

Normally, there is no treatment indicated.

- Vitrectomy may be successful in treating more severe cases; however, the procedure is typically not warranted in those with lesser symptoms due to the potential for complications as severe as blindness. Floaters may become less annoying as sufferers grow accustomed to them, even to the extent that they may no longer notice them.

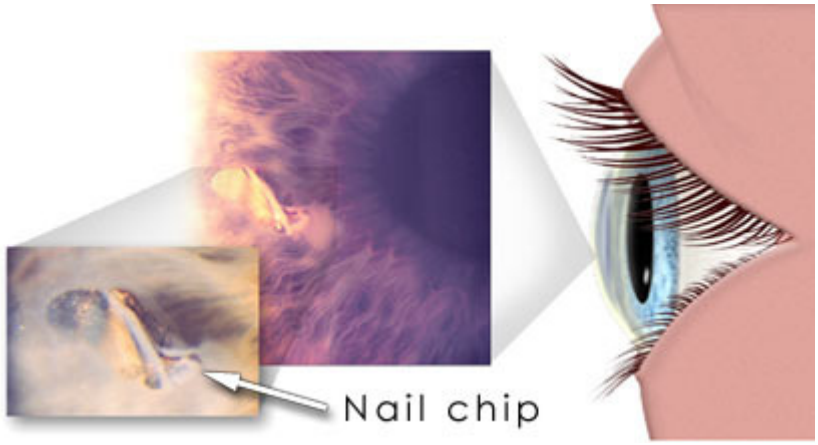
- There is also Sutureless Vitrectomy, as the standard vitrectomy involves cutting through the conjunctiva, or fleshy part of the front of the eye, and making openings in the pars plana area which require stitches at the end of the surgery, in the sutureless technique, small tubes or canulas or trochars are placed through the pars plana area and very tiny instruments are placed through these tubes. Once the surgery is complete, the tubes are removed and no stitches are needed. Only in certain cases can sutureless vitrectomy surgery be performed.

- Another treatment is laser vitreolysisIn this procedure a powerful laser (usually a Yttrium aluminium garnet laser) is focused onto the floater and in a quick burst vaporizes the structure into a less dense and not as noticeable consistency.

http://en.wikipedia.org/wiki/Floater

FOREIGN BODY

Anyone who has felt as if there was a grain of sand in his or her eye has probably had a foreign body. Foreign bodies might be superficial, or in more serious injuries, they may penetrate the eye. Fortunately, the cornea has such an incredible reflex tearing system that most superficial foreign bodies are naturally flushed out with our natural tears. But if the object is more deeply embedded, medical attention is required.



This photo-illustration shows a foreign body in the iris tissue. The patient was hammering a nail (without wearing eye protection) and was struck in the eye by a chip from the nail. Note how the nail chip tore the iris.

Signs and Symptoms

The symptoms of a foreign body may range from irritation to intense, excruciating pain. This is dependent on the location, material, and type of injury.

In rare situations where an object penetrates the eye, there may be few or no symptoms. If you have no symptoms, but suspect an object may have penetrated your eye, it's always best to seek medical attention. The entry point of an intraocular foreign body is sometimes nearly

invisible. Depending on their location, foreign bodies inside the eye may or may not cause pain or decreased vision.

- Mild to extreme irritation
- Scratching
- Burning
- Soreness
- Intense pain
- Redness
- Tearing
- Light sensitivity
- Decreased vision
- Difficulty opening the eye

Detection and Diagnosis

The evaluation includes vision testing along with careful examination of the surface of the eye with a slit lamp microscope. When a superficial foreign body is suspected, the upper lid should be gently turned up to check underneath for trapped particles. If the foreign body is difficult to see even with a microscope, the doctor may instill a drop of fluorescein dye to highlight the area.

An examination inside the eye with ophthalmoscopy may also be indicated depending on the severity of the injury.

Treatment

If a foreign object becomes embedded within the cornea, conjunctiva, or sclera, a medical professional must remove it. Attempting to remove it yourself is dangerous and could result in a permanent scar that could affect your vision.

Superficial foreign bodies are usually treated in the office. After numbing the eye with topical anesthetic, the particle is carefully removed using a microscope. Afterward, antibiotic medications are generally prescribed to prevent infection. In some cases, foreign bodies become trapped underneath the eyelid. It is extremely important to examine under the eyelid for any remnant particles.

Intraocular foreign bodies typically must be removed in the operating room using a microscope and special instruments designed for working inside the eye. These injuries are often vision threatening and should be treated quickly.

Wearing appropriate safety glasses is the best way to prevent this type of injury. Protecting the eyes is especially important when working with machinery that could cause chips of wood or metal to splinter, as well lawn equipment such as hedge and line trimmers.

If a particle of wood, glass, metal or any other foreign substance becomes trapped in your eye, here are some tips:

1. Do not touch or rub your eye! This can embed the object more deeply, making it more difficult to remove.

2. Keep your eye closed as much as possible. Blinking only increases the irritation.

3. Do not try to remove the object yourself. This is very dangerous and may make the problem worse.

4. Seek professional help immediately.

5. Tell your doctor what you were doing at the time of the injury, or what materials you may have been working with.

http://www.stlukeseye.com/conditions/ForeignBody.asp

GLIOMA

A **glioma** is a type of primary central nervous system (CNS) tumor that arises from glial cells. The most common site of involvement of gliomas is the brain, but gliomas can also affect the spinal cord or any other part of the CNS, such as the optic nerves.

By type of cell

Gliomas are named according to the specific type of cell they most closely resemble. The main types of gliomas are:

- Ependymomas — ependymal cells
- Astrocytomas — astrocytes
- Oligodendrogliomas — oligodendrocytes
- Mixed gliomas, such as oligoastrocytomas, contain cells from different types of glia.

By grade

Gliomas are further categorized according to their grade, which is determined by pathologic evaluation of the tumor.

- **Low-grade** gliomas are well-differentiated (not anaplastic); these are benign and portend a better prognosis for the patient.
- **High-grade** gliomas are undifferentiated or anaplastic; these are malignant and carry a worse prognosis.

Of numerous grading systems in use, the most common is the World Health Organization (WHO) grading system for astrocytoma. The WHO system assigns a grade from 1 to 4, with 1 being the least aggressive and 4 being the most aggressive. Various types of astrocytomas are given corresponding WHO grades.

WHO grading system for astrocytomas

- WHO Grade 1 — e.g., pilocytic astrocytoma
- WHO Grade 2 — e.g., diffuse or low-grade astrocytoma
- WHO Grade 3 — e.g., anaplastic (malignant) astrocytoma
- WHO Grade 4 — glioblastoma multiforme (most common glioma in adults)

The prognosis is the worst for grade 4 gliomas, with an average survival time of 12 months. Overall, few patients survive beyond 3 years.

By location

The gliomas can also be roughly classified according to their location:

- infratentorial : mostly in children (70%)
- supratentorial : mostly in adults (70%)

Symptoms

Symptoms of gliomas depend on which part of the central nervous system is affected. A brain glioma can cause headaches, nausea and vomiting, seizures, and cranial nerve disorders as a result of increased intracranial pressure. A glioma of the optic nerve can cause visual loss. Spinal cord gliomas can cause pain, weakness, or numbness in the extremities. Gliomas do not metastasize by the bloodstream, but they can spread via the cerebrospinal fluid and cause "drop metastases" to the spinal cord.

Pathology

High-grade gliomas are highly-vascular tumors and have a tendency to infiltrate. They have extensive areas of necrosis and hypoxia. Often tumor growth causes a breakdown of the blood-brain barrier in the vicinity of the tumor. As a rule, high-grade gliomas almost always grow back even after complete surgical excision.

On the other hand, low-grade gliomas grow slowly, often over many years, and can be followed without treatment unless they grow and cause symptoms.

Treatment

Standard therapy

Treatment for brain gliomas depends on the location and the grade. Often, treatment is a combined approach, using surgery, radiation therapy, and chemotherapy. The radiation therapy is in the form of external beam radiation or the stereotactic approach using radiosurgery. Spinal cord tumors can be treated by surgery and radiation. Temozolomide is a chemotherapeutic drug that is able to cross the blood-brain barrier effectively and is being used in therapy.

Refractory disease

For recurrent high-grade glioblastoma, recent studies have taken advantage of angiogenic blockers such as bevacizumab in combination with conventional chemotherapy, with encouraging results.

Experimental therapies

The use of oncolytic viruses or gene therapy using prodrug converting retroviruses and adenoviruses is being studied for the treatment of gliomas.

A small number of low-scale clinical studies have shown possible links between prescription of Carphedon and improvement in a number of encephalopathic conditions, including lesions of cerebral blood pathways and certain types of glioma.

American scientists are also studying the effects of *Leiurus quinquestriatus* scorpion (Israeli Yellow Scorpion) venom on glioma. They have successfully isolated the peptide chlorotoxin from the venom of the *L. quinquestriatus* scorpion by means of gel filtration chromatography. The peptide appears to target glioma-specific chloride ion channels within the cancerous glial cells of the brain, where it binds with a high affinity.

In 2006, German physicians reported on a dose-escalation study for the compound AP 12009 (a phosphorothioate antisense oligodeoxynucleotide specific for the mRNA of human transforming growth factor TGF-beta2) in patients with high-grade gliomas. At the time of the report, the median overall survival had not been obtained and the authors hinted at a potential cure.

As of 2006, additional research started within the past few years is ongoing. Some of the topics included in this research are:

- efficiency of variations in radiotherapy procedures
- drugs to stop the growth of tumors by preventing them to develop blood vessels
- efficiency of combinations of different treatments
- vaccination therapy.

In July 2007, Italian researchers reported they had found some preclinical evidence that the experimental cancer medicine "Ukrain" could have value in the treatment of gliomas.[5] Ukrain has been used in the therapy of several solid cancer tumours, but little is known about its effect on glioblastoma and, in general, about the molecular mechanisms responsible for its effects. After treatment with high dose Ukrain, the concentration of the so-called glial fibrillary acidic protein increased within the cancer cells. There was no effect on the so-called Connexin 43 protein. Ukrain-induced programmed cancer cell death (apoptosis) was 4.63 % in low concentration, 10.9 % at medium concentration and 28.9% in high concentration. The apoptosis was likely to be mediated by release of the substance Cytochrome C in the cell cytoplasm. Considered as a whole, these findings provide new information to complete the understanding of the mechanisms of Ukrain antitumor and chemopreventive effect, and support the possible potential of Ukrain for the therapy of brain tumors. There are also several case reports of good clinical efficacy on gliomas.

Although there have been individual cases of patients receiving an experimental treatment who still showed no signs of tumor 3 years or even more after the first diagnosis, often a new treatment for GBM will already be considered successful if it significantly increases the percentage of survivors after two years.

A cancer vaccine "Oncophage" is currently showing great promise in clinical trails, 2007.

http://en.wikipedia.org/wiki/Glioma

HEMANGIOMA

Before considering the hemangioma it is important to understand that there have been recent changes in the terminology used to define vascular anomalies (abnormal lumps made up of blood vessels). The term hemangioma was originally used to describe any vascular tumour both present around birth or appearing later in life. Mulliken et al separated these conditions into a family of self involuting tumours (growing lesions that eventually disappear) from the family of malformations (enlarged or abormal vessels present at birth and essentially permanent). The importance of this separation is that it allows us to differentiate early in life between lesions that will resolve versus those that are permanent. Examples of permanent malformations include Port-wine stains (capillary vascular malformation) and masses of abnormal swollen veins (venous malformations).

A **hemangioma** is a benign self involuting tumour of endothelial cells (the cells that line blood vessels) Haemangiomas of infancy They are connected to the circulatory system and filled with blood. The appearance depends on location. If they are on the surface of the skin they look like a ripe strawberry, if they are just under the skin they present as a bluish swelling. Sometimes they grow in internal organs such as the liver or larynx. In most cases, hemangiomas will disappear over time. They are formed either during gestation or most commonly they are not present at birth but appear during the first few weeks of life. They are often misdiagnosed, initially, as a scratch or bruise but the diagnosis becomes obvious with further growth. Typically at the earliest phase in a superficial lesion one will see a bluish red area with obvious blood vessels and surrounding pallor. Sometimes they present as a flat red or pink area. Hemangiomas are the most common childhood tumor occurring in approximately ten percent of Caucasians, and are less prevalent in other races. Females are three to five times more likely to have hemangiomas than males. They are also more common in twin pregnancies. Approximately eighty percent are located on the face and neck, with the next most prevalent location being the liver. Although hemangiomas are benign, some serious complications can occur. Hemangioma's never develope as an adult but one misunderstanding is that all hemangioma's go away by the age of 10 very few do not. Many people say that they are a vascular malformation but they are infact a hemangioma.

Causes

The cause of hemangioma is currently unknown; however, several studies have suggested the importance of estrogen signaling in hemangioma proliferation. In 2007, a paper from the Stanford Children's Surgical Laboratory revealed that localized soft tissue hypoxia coupled with increased circulating estrogen after birth may be the stimulus. There is also a hypothesis presented by researchers at Harvard and the University of Arkansas that maternal placenta embolizes to the fetal dermis during gestation resulting in hemangiomagenesis, yet Duke researchers conducted genetic analyses of small nucleotide polymorphisms in hemangioma tissue compared to the mother's DNA that contradicted this notion. More research is required in order to fully understand the explosive nature of hemangioma growth which will hopefully yield targeted therapeutics to treat its most complicated presentations.

Complications

The vast majority of hemangiomas are not associated with complications. Hemangiomas may break down on the surface to form ulcers. If the ulceration is deep, significant bleeding may rarely occur. Ulceration on the diaper area can be painful and problematic.

If a hemangioma develops in the larynx, breathing can be compromised. A hemangioma can grow and block one of the eyes, causing an occlusion amblyopia. Very rarely, extremely large hemangiomas can cause high-output heart failure due to the amount of blood that must be pumped to excess blood vessels. Lesions adjacent to bone can also cause erosion of the bone.

The most frequent complaints about hemangiomas, however, stem from psychosocial complications: the condition can affect a person's appearance and can provoke attention and malicious reactions from others. Particular problems occur if the lip or nose is involved, as distortion can be difficult to treat surgically.

Treatment

Most hemangiomas disappear without treatment, leaving minimal or no visible marks. Large hemangiomas can leave visible skin changes secondary to severe stretching of the skin or damage to surface texture. When hemangiomas interfere with vision, breathing, or threaten significant cosmetic injury, they are usually treated. The mainstay of treatment is oral corticosteroid therapy. Other drugs such as interferon or vincristine are sometimes considered if the corticosteroids do not work. If this fails, surgical removal often becomes necessary. Blockage of the airway will often require a tracheostomy to be performed (insertion of an external airway through the front of the neck into the trachea below the level of the obstruction). Smaller raised lesions are sometimes treated with injection of corticosteroid directly into the lesion. Pulsed dye laser can be useful for very early flat superficial lesions if they appear in cosmetically significant areas or for those lesions that leave residual surface blood vessels in the case of incomplete resolution. Unfortunately raised lesions or lesions under the skin do not respond to laser.

Ulceration will usually heal with topical medication and special dressings under medical supervision. Sometimes pulsed dye laser can be used to accelerate healing.

Prognosis

Hemangiomas go through three stages of development and decay:

1. In the **proliferation** stage, a hemangioma grows very quickly. This stage can last up to twelve months.
2. In the **rest** stage, there is very little change in a hemangioma's appearance. This usually lasts until the infant is one to two years old.
3. In the **involution** phase, a hemangioma finally begins to diminish in size. Fifty percent of lesions will have disappeared by age five with the vast majority gone by puberty.

http://en.wikipedia.org/wiki/Hemangioma

HERPES ZOSTER

Herpes zoster, commonly known as shingles, is caused by the same virus responsible for chicken pox. After the initial exposure, herpes zoster lies dormant in certain nerve fibers. It may become active as a result of many factors such as: aging, stress, suppression of the immune system, and certain medications.

Because of the layout of the nerves that herpes zoster resides in, it only affects one side of the body or face during an outbreak. It begins as a rash that lead to blisters and sores on the skin. When the nerve branch that supplies the eye is involved, the forehead, nose, and eyelids may also be affected. Sores on the nose are a key signal of possible eye involvement.

Herpes zoster can cause several problems with the eye and surrounding skin that may have long term effects. Inflammation and scarring of the cornea, along with conjunctivitis (inflammation of the conjunctiva) and iritis (inflammation of the iris) are typical problems that require treatment. In some cases, the retina and optic nerve are involved. Eye problems caused by severe or chronic outbreaks of herpes zoster may include: glaucoma, cataract, double vision, and scarring of the cornea and eyelids.

Many who experience this infection find it extremely painful. This acutely painful phase usually lasts several weeks; however, some continue to experience pain or neuralgia long after the outbreak has cleared. This is known as post-herpetic neuralgia.

Signs and Symptoms

Herpes zoster causes a wide range of problems affecting the skin and the eye. They range in severity depending on the extent of the outbreak. Some problems listed occur indirectly from the inflammation caused by the disease.

Problems affecting the body

- Flu-like symptoms (fever, headache, fatigue)
- Rash
- Red, sensitive, sore skin
- Blisters and sores on the skin
- Pain (may be burning or throbbing), itching and tingling

Problems affecting the eye

- Redness
- Light sensitivity
- Swollen eyelids
- Dry eyes
- Blurred vision (depending on how the eye is affected)
- Corneal inflammation that may lead to scarring
- Inflammation inside the eye and optic nerve
- Glaucoma
- Cataract
- Double vision
- Loss of sensation

Detection and Diagnosis

When the eye is affected, the doctor will perform a thorough examination with a slit lamp microscope and an ophthalmoscope. Visual acuity and intraocular pressure are also monitored. Signs of breakout on the face and body are noted.

Treatment

Herpes zoster is treated with anti-viral, pain and anti-inflammatory medications. Eye drops and ointments may be prescribed to treat ocular problems. In some cases, secondary conditions caused by herpes zoster may require surgery.

Those who are infected should avoid contact with people who may be more susceptible to contracting the disease such as: the elderly, children, pregnant women, or anyone with a compromised immune system.

http://www.stlukeseye.com/conditions/HerpesZoster.asp

HYPHEMA

Hyphema is a term used to describe bleeding in the anterior chamber (the space between the cornea and the iris) of the eye. It occurs when blood vessels in the iris bleed and leak into the clear aqueous fluid. Hyphemas are usually characterized by pooling of blood in the anterior chamber that may be visible to the naked eye. The red blood cells of very small hyphemas are visible only with magnification. Even the slightest amount of blood in the anterior chamber will cause decreased vision when mixed in the clear aqueous fluid.

Bleeding in the anterior chamber is most often caused by blunt trauma to the eye. It may also be associated with surgical procedures. Other causes include abnormal vessel growth in the eye and certain ocular tumors.

Signs and Symptoms

- Decreased vision (Depending on the amount of blood in the eye, vision may be reduced to only hand movements and light perception only)
- Pool of blood in the anterior chamber
- Elevated intraocular pressure (in some cases)

Detection and Diagnosis

It is very important for the doctor to determine the cause of the hyphema. If the hyphema is related to an ocular injury, any detail regarding the nature of the trauma is helpful. The doctor will assess visual acuity, measure intraocular pressure, and examine the eye with a slit lamp microscope and ophthalmoscope.

Treatment

The treatment is dependent on the cause and severity of the hyphema. Frequently, the blood is reabsorbed over a period of days to weeks. During this time, the doctor will carefully monitor the intraocular pressure for signs of the blood preventing normal flow of the aqueous through the eye's angle structures. If the eye pressure becomes elevated, eye drops may be prescribed to control it. The pupils are also evaluated to rule out damage to the iris.

In some cases, a procedure is performed to irrigate the blood from the anterior chamber to prevent secondary complications such as glaucoma and blood stains on the cornea.

Patients with significant hyphemas must rest and avoid strenuous activity to allow the blood to reabsorb.

http://www.stlukeseye.com/Conditions/Hyphema.asp

HYPOPYON

An accumulation of pus in the front of the eye. This condition is most often seen following a penetrating trauma to the eye and should be examined by an eye doctor as soon as possible. In this image, the pus is seen as a pool of whitish fluid between the iris and cornea.

http://www.stlukeseye.com/conditions/Hypopyon.asp

IRITIS

Iritis is a form of anterior uveitis and refers to the inflammation of the iris of the eye.

There are two main types of iritis: acute and chronic. Acute iritis is a type of iritis that can heal independently within a few weeks. If treatment is provided, acute iritis improves quickly. Chronic iritis can exist for months or years before recovery occurs. Chronic iritis does not respond to treatment as well as acute iritis does. Chronic iritis is also accompanied by a higher risk of serious visual impairment.

Signs and symptoms

- Ocular and periorbital pain
- Photophobia
- Consensual photophobia (pain in affected eye when light is shone in unaffected eye)
- Blurred or cloudy vision
- Reddened eye, especially adjacent to the iris
- White blood cells (leukocytes) (resulting in a grey or near-white haze) and protein (resulting in tiny white dots) in the anterior chamber, often called "cells and flare."
- Synechia (adhesion of iris to lens or cornea)

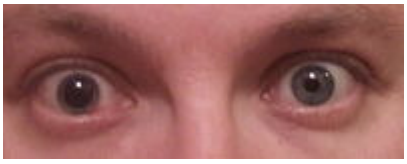
Causes

People with ankylosing spondylitis and other HLA-B27 related disorders are prone to iritis, iridocyclitis, and other forms of uveal tract inflammation. Iritis is also found in those with rheumatoid arthritis, Behcet's disease, Crohn's disease, lupus, Reiter's disease, chronic psoriasis, psoriatic arthritis, sarcoidosis, scleroderma, and ulcerative colitis. Iritis is usually secondary to some other systemic condition, but can be the only apparent somatic symptom.

Complications

Complications of iritis may include the following: Cataract, glaucoma, corneal calcification, posterior uveitis, blindness, band keratopathy, and cystoid macular oedema.

Treatment



Eye treated with dilating eye drops (Atropine).

- Steroid anti-inflammatory eye drops (such as prednisolone acetate)
- Dilating eye drops (to help prevent synechia and reduce photophobia)
- Pressure-reducing eye drops (such as brimonidine tartrate)
- Oral steroids (such as prednisone)
- Subconjunctival steroid injections
- Steroid-sparing agents such as methotrexate (for prolonged, chronic iritis)

http://en.wikipedia.org/wiki/Iritis

KERATOCONUS

Keratoconus (from Greek: *kerato-* horn, cornea; and *konos* cone), is a degenerative non-inflammatory disorder of the eye in which structural changes within the cornea cause it to thin and change to a more conical shape than its normal gradual curve. Keratoconus can cause substantial distortion of vision, with multiple images, streaking and sensitivity to light all often reported by the patient. Keratoconus is the most common dystrophy of the cornea, affecting around one person in a thousand, and it seems to occur in populations throughout the world, although some ethnic groups experience a greater prevalence than others. It is typically diagnosed in the patient's adolescent years and attains its most severe state in the twenties and thirties.

Keratoconus is a little-understood disease with an uncertain cause, and its progression following diagnosis is unpredictable. If afflicting both eyes, the deterioration in vision can affect the patient's ability to drive a car or read normal print. In most cases, corrective lenses

are effective enough to allow the patient to continue to drive legally and likewise function normally. Further progression of the disease may require surgery including transplantation of the cornea. Despite its uncertainties, keratoconus can be successfully managed with a variety of clinical and surgical techniques, and often with little or no impairment to the patient's quality of life.

Symptoms

A simulation of the multiple images seen by a person with keratoconus.
"... a candle, when looked at, appears like a number of lights, confusedly running into one another" — Nottingham

People with early keratoconus typically notice a minor blurring of their vision and come to their clinician seeking corrective lenses for reading or driving. At early stages, the symptoms of keratoconus may be no different from those of any other refractive defect of the eye. As the disease progresses, vision deteriorates, sometimes rapidly. Visual acuity becomes impaired at all distances, and night vision is often quite poor. Some individuals have vision in one eye that is markedly worse than that in the other eye. Some develop photophobia (sensitivity to bright light), eye strain from squinting in order to read, or itching in the eye. There is however normally little or no sensation of pain.

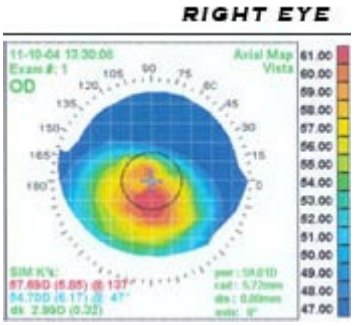
The classic symptom of keratoconus is the perception of multiple 'ghost' images, known as monocular polyopia. This effect is most clearly seen with a high contrast field, such as a point of light on a dark background. Instead of seeing just one point, a person with keratoconus sees many images of the point, spread out in a chaotic pattern. This pattern does not typically change from day to day, but over time it often takes on new forms. Patients also commonly notice streaking and flaring distortion around light sources. Some even notice the images moving relative to one another in time with their heart beat.

Signs and diagnosis

Prior to any physical examination, the diagnosis of keratoconus frequently begins with an ophthalmologist's or optometrist's assessment of the patient's medical history, particularly the chief complaint and other visual symptoms, the presence of any history of ocular disease or injury which might affect vision, and the presence of any family history of ocular disease. An eye chart, such as a standard Snellen chart of progressively smaller letters, is then used to determine the patient's visual acuity. The eye examination may proceed to measurement of the localised curvature of the cornea with a manual keratometer, with detection of irregular astigmatism suggesting a possibility of keratoconus. Severe cases can exceed the instrument's measuring ability. A further indication can be provided by retinoscopy, in which a light beam is focused on the patient's retina and the reflection, or *reflex*, observed as the examiner tilts the light source back and forth. Keratoconus is amongst the ophthalmic conditions that exhibit a

scissor reflex action of two bands moving toward and away from each other like the blades of a pair of scissors.

If keratoconus is suspected, the ophthalmologist or optometrist will search for other characteristic findings of the disease by means of slit lamp examination of the cornea.[10] An advanced case is usually readily apparent to the examiner, and can provide for an unambiguous diagnosis prior to more specialised testing. Under close examination, a ring of yellow-brown to olive-green pigmentation known as a Fleischer ring can be observed in around half of keratoconic eyes. The Fleischer ring, caused by deposition of the iron oxide hemosiderin within the corneal epithelium, is subtle and may not be readily detectable in all cases, but becomes more evident when viewed under a cobalt blue filter. Similarly, around 50% of subjects exhibit Vogt's striae, fine stress lines within the cornea caused by stretching and thinning. The striae temporarily disappear while slight pressure is applied to the eyeball. A highly pronounced cone can create a V-shaped indentation in the lower eyelid when the patient's gaze is directed downwards, known as Munson's sign. Other clinical signs of keratoconus will normally have presented themselves long before Munson's sign becomes apparent, and so this finding, though a classic sign of the disease, tends not to be of primary diagnostic importance.

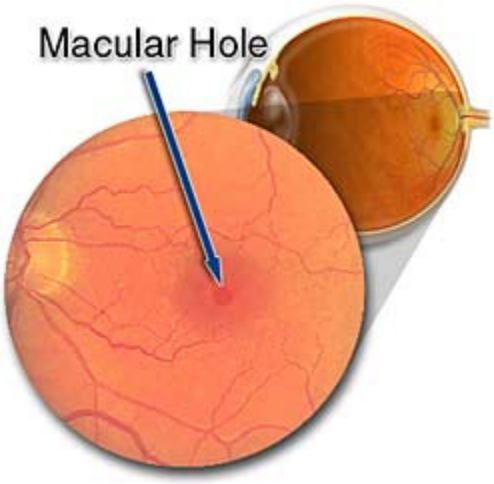


Corneal topogram of a keratoconic eye

A handheld keratoscope, sometimes known as *Placido's disk*, can provide a simple non-invasive visualization of the surface of the cornea by projecting a series of concentric rings of light onto the cornea. A more definitive diagnosis can be obtained using corneal topography, in which an automated instrument projects the illuminated pattern onto the cornea and determines its topology from analysis of the digital image. The topographical map indicates any distortions or scarring in the cornea, with keratoconus revealed by a characteristic steepening of curvature which is usually below the centreline of the eye. The technique can record a snapshot of the degree and extent of the deformation as a benchmark for assessing its rate of progression. It is of particular value in detecting the disorder in its early stages when other signs have not yet presented.

Once keratoconus has been diagnosed, its degree may be classified by several metrics:

- The steepness of greatest curvature from *mild* (< 45 D), *advanced* (up to 52 D) or *severe* (> 52 D);
- The morphology of the cone: *nipple* (small: 5 mm and near-central), *oval* (larger, below-center and often sagging), or *globus* (more than 75% of cornea affected);
- The corneal thickness from mild (> 506 μm) to advanced (< 446 μm).



http://en.wikipedia.org/wiki/Keratoconus

MACULAR HOLE

Macular hole is a problem that affects the very central portion of the retina. It happens for a variety of reasons such as: eye injuries, certain diseases, and inflammation inside the eye. However, the most common cause is related to the normal aging process.

The vitreous gel inside the eye is firmly attached to the macula. With age, the vitreous becomes thinner and separates from the retina. Sometimes this creates traction on the macula, causing a hole to form.

Macular holes often begin gradually and affect central vision depending on the severity and extent of the problem. Partial holes only affect part of the macular layers, causing wavy, distorted, blurred vision. Patients with full-thickness macular holes experience a complete loss of central vision.

Signs and Symptoms

The severity of the symptoms is dependent on whether the hole is partial or full-thickness.

- Blurred central vision
- Distorted, "wavy" vision
- Difficulty reading or performing tasks that require seeing detail
- Gray area in central vision
- Central blind spot

Detection and Diagnosis

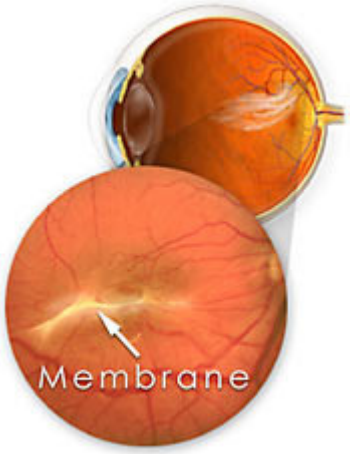
Visual acuity testing, Amsler grid, and ophthalmoscopy are all performed to evaluate the macula's health and function. The retina doctor may also order photographs of the macula prior to performing surgery to repair the hole.

Treatment

Some macular holes seal spontaneously and require no treatment. In many cases, surgery is necessary to close the hole and restore useful vision.

Macular holes are repaired with surgery. During the operation, the surgeon first gently removes the vitreous gel with a procedure called vitrectomy. This eliminates any traction on the macula. A gas bubble is injected in the eye to place gentle pressure on the macula and help the hole to seal. In many cases, patients enjoy functional vision after the bubble has dissipated and the eye has healed.

http://www.stlukeseye.com/Conditions/MacularHole.asp



MACULAR PUCKER

Epi-retinal membrane (ERM) or **macular pucker** is a cellophane-like membrane that forms over the macula. It is typically a slow-progressing problem that affects the central vision by causing blur and distortion. As it progresses, the traction of the membrane on the macula may cause swelling.

ERM is seen most often in people over 75 years of age. It usually occurs for unknown reasons, but may be associated with certain eye problems such as: diabetic retinopathy, posterior vitreous detachment, retinal detachment, trauma, and many others.

Signs and Symptoms

- Blurred vision
- Double vision that is noticeable even with one eye covered
- Distorted vision (straight lines may appear bent or wavy)

Detection and Diagnosis

The doctor is able to detect ERM with ophthalmoscopy during an examination of the retina. It has a glistening, cellophane-like appearance. The affect of ERM on the patient's central vision is assessed with a visual acuity test and the Amsler Grid. If the doctor suspects macular swelling, he may order fluorescein angiography.

Treatment

A procedure called a membrane peel is performed when vision has deteriorated to the point that it is impairing the patient's lifestyle. Most vitreo-retinal surgeons recommend waiting for treatment until vision has decreased to the point that the risk of the procedure justifies the improvement.

The membrane peel is performed under a local anesthesia in an operating room. After making tiny incisions The membrane peel is often done in conjunction with a procedure called a vitrectomy.

http://www.stlukeseye.com/Conditions/EpiRetinalMembrane.asp

NIGHT BLINDNESS

Nyctalopia is a condition making it difficult or impossible to see in relatively low light. It is a symptom of several eye diseases. Night blindness may exist from birth, or be caused by injury or malnutrition (for example, a lack of vitamin A).

The most common cause of nyctalopia is retinitis pigmentosa, a disorder in which the rod cells in the retina gradually lose their ability to respond to the light. Patients suffering from this genetic condition have progressive nyctalopia and eventually their daytime vision may also be affected. In X-linked congenital stationary night blindness, from birth the rods either do not work at all, or work very little, but the condition doesn't get worse.

Another cause of night blindness is a deficiency of retinol, or vitamin A, found in fish oils, liver and dairy products. In the Second World War misinformation was spread by the British to cover up the reason for their pilots' successful night time missions. Their success was, in the misinformation, attributed to improved night vision and pilots flying night missions were encouraged to eat plenty of carrots, which contain carotenoids and can be converted into retinol. The actual reason for their success was their use of advanced radar technologies.

The opposite problem, known as *hemeralopia*, is much rarer.

The outer area of the retina is made up of more rods than cones. The rod cells are the cells that enable us to see in poor illumination. This is the reason why loss of side vision often results in night blindness. Individuals suffering from night blindness not only see poorly at night, but also require some time for their eyes to adjust from brightly lit areas to dim ones. Contrast vision may also be greatly reduced.

Causes

- vitamin a deficiency
- retinitis pigmentosa
- congenital night blindness
- pathological myopia
- peripheral cortical cataract
- oguchi's disease

http://en.wikipedia.org/wiki/Nyctalopia

PHOTOPHOBIA

Photophobia is a symptom of excessive sensitivity to light and the aversion to sunlight or well-lit places. In medical terms it is not fear, but an experience of discomfort or pain to the eyes due to light exposure.

Light sensitivity is usually due to too much light entering the eye, which causes over stimulation of the photoreceptors in the retina and subsequent excessive electric impulses to the optic nerve. This leads to a reflex aversion to light, and discomfort or pain. Too much

light can enter the eye if it is damaged, such as with corneal abrasion and retinal damage, or if a pupil(s) is unable to normally constrict (seen with damage to the oculomotor nerve).

Patients with photophobia will avert their eyes from direct light (sunlight and room lights), or may seek the shelter of a dark room or wear sunglasses.

Photophobia is also a behavior demonstrated by insects or other animals which seek to stay out of the light

Causes

Patients may develop photophobia as a result of several different medical conditions, related to the eye or the nervous system.

- Meningitis
- Eye disease, injury, or infection such as chalazion, episcleritis, glaucoma, keratoconus
- Subarachnoid haemorrhage
- Albinism
- Burns to the eye
- Migraines
- Encephalitis
- Conjunctivitis
- Aphakia
- Iritis
- Corneal abrasion
- Corneal ulcer
- Cataracts
- Retinal detachment
- Chiari malformation
- Cystinosis
- Anticholinergic drugs may cause photophobia by paralyzing the iris sphincter muscle.
- Hangover
- Chikungunya

http://en.wikipedia.org/wiki/Photophobia

REFRACTIVE ERROR

A **refractive error**, or **refraction error**, is an error in the focusing of light by the eye and a frequent reason for reduced visual acuity.

An eye that has no refractive error when viewing a distant object is said to have *emmetropia* or be *emmetropic*. An eye that has a refractive error when viewing a distant object is said to have *ametropia* or be *ametropic*.

Refractive errors are frequently categorized as spherical errors and cylindrical errors:

- Spherical errors occur when the optical power of the eye is either too large or too small to focus light on the retina. People with refraction error frequently have blurry vision.
    - When the optics are too powerful for the length of the eyeball (this can arise from a cornea with too much curvature or an eyeball that is too long), one has myopia.
    - When the optics are too weak for the length of the eyeball (this can arise from a cornea with not enough curvature or an eyeball that is too short), one has hyperopia.

- Cylindrical errors occur when the optical power of the eye is too powerful or too weak across one meridian of the optics. It is as if the overall lens tends towards a cylindrical shape along that meridian. People with this refraction error see contours of a particular orientation as blurred, but see contours with orientations at right angles as clear. When one has a cylindrical error, one has astigmatism.

Causes

Refractive errors are thought to occur due to a combination of genetic and environmental factors. Trauma or ocular disorders such as keratoconus may induce refractive errors.

Diagnosis

Blurry vision may result from any number of conditions not necessarily related to refractive errors. The diagnosis of a refractive error is usually confirmed by an eye care professional during an eye examination using an instrument called a *phoropter* which contains a large number of lenses of varying optical power. In combination with a retinoscope (a procedure entitled *retinoscopy*), the doctor instructs the patient to view an eye chart while he or she changes the lenses within the phoropter to objectively estimate the amount of refractive error the patient may possess. Once the doctor arrives at an estimate, he or she typically shows the patient lenses of progressively higher or weaker powers in a process known as *refraction* or *refractometry*. Cycloplegic agents are frequently used to more accurately determine the amount of refractive error, particularly in children.

An automated refractor is an instrument that is sometimes used in place of retinoscopy to objectively estimate a person's refractive error.

Treatment and management

How refractive errors are treated or managed depends upon the amount and severity of the condition. Those who possess mild amounts of refractive error may elect to leave the condition uncorrected, particular if the patient is asymptomatic. For those who are symptomatic, glasses, contact lenses, refractive surgery, or a combination of the three are typically used.

It is worth noting, however, that in the case of myopia, such treatments may also have the long-term effect of exacerbating that refractive error -- i.e., making the patient even more nearsighted. This would be due to the very same prescription that is tailored for use at a 12-to-20-foot distance also commonly being used for close-up work as well, thus artificially

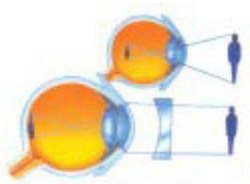
amplifying the focusing stress that would normally be presented to the accommodation mechanisms of the eye at that distance.

http://en.wikipedia.org/wiki/Refractive_error

SHORT SIGHT (MYOPIA) AND LONG SIGHT (HYPERMETROPIA)

Short sight (myopia) and long sight (hypermetropia) are common conditions, both caused by the cornea and lens not focusing properly on the retina.

Short sight is where the eyeball is elongated or the lens is too thick, causing the image to



Nearsightedness (Myopia)

Nearsightedness or myopia, occurs when light entering the eye focuses in front of the retina instead of directly on it. This is caused by a cornea that is steeper, or an eye that is longer, than a normal eye. Nearsighted people typically see well up close, but have difficulty seeing far away. This problem is often discovered in school-age children who report having trouble seeing the chalkboard. Near-sightedness usually becomes progressively worse through adolescence and stabilizes in early adulthood. It is an inherited problem.
Signs and Symptoms

- Blurry distance vision
- Vision seems clearer when squinting

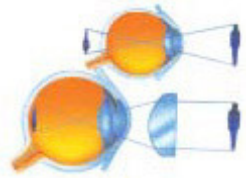
Detection and Diagnosis

Nearsightedness is detected with a vision test and refraction.

The treatment for nearsightedness depends on several factors such as the patient's age, activities, and occupation. Vision can corrected with glasses, contacts, or surgery. Refractive procedures such as LASIK can be considered for adults when the prescription has remained stable for at least one year.

http://www.stlukeseye.com/conditions/Myopia.asp

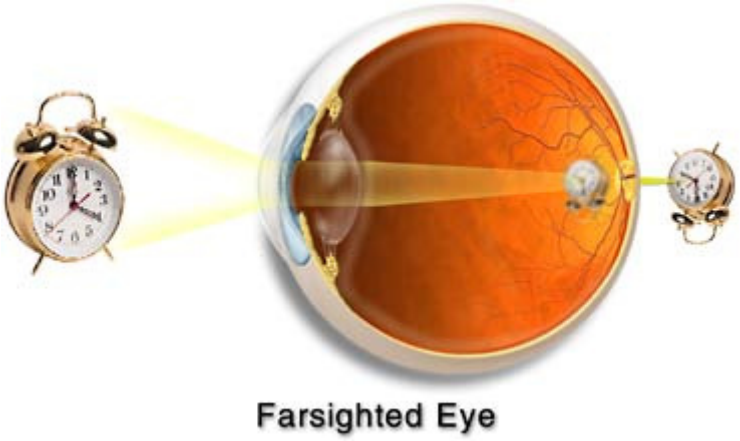
Long sight is where the eyeball is too short or the lens too thin, causing the image to focus behind the retina.



Both long and short sightedness can be overcome by wearing glasses.

FARSIGHTEDNESS (HYPEROPIA)

Farsightedness or hyperopia, occurs when light entering the eye focuses behind the retina, instead of directly on it. This is caused by a cornea that is flatter, or an eye that is shorter, than a normal eye. Farsighted people usually have trouble seeing up close, but may also have difficulty seeing far away as well.



Young people with mild to moderate hyperopia are often able to see clearly because their natural lens can adjust, or accommodate to increase the eye's focusing ability. However, as the eye gradually loses the ability to accommodate (beginning at about 40 years of age), blurred vision from hyperopia often becomes more apparent.

Signs and Symptoms

- Difficulty seeing up close
- Blurred distance vision (occurs with higher amounts of hyperopia)
- Eye fatigue when reading
- Eye strain (headaches, pulling sensation, burning)
- Crossed eyes in children

Detection and Diagnosis

Hyperopia is detected with a vision test called a refraction. Young patients' eyes are dilated for this test so they are unable to mask their farsightedness with accommodation. This is called a wet refraction.

Treatment

The treatment for hyperopia depends on several factors such as the patient's age, activities, and occupation. Young patients may or may not require glasses or contact lenses, depending on their ability to compensate for their farsightedness with accommodation. Glasses or contact lenses are required for older patients.

Refractive surgery is an option for adults who wish to see clearly without glasses. LASIK, Clear Lens Extraction And Replacement, LTK and intraocular contact lenses are all procedures that can be performed to correct hyperopia.

http://www.stlukeseye.com/conditions/Hyperopia.asp

PRESBYOPIA

Presbyopia describes the condition where the eye exhibits a progressively diminished ability to focus on near objects with age. Presbyopia's exact mechanisms are not known with certainty, however, the research evidence most strongly supports a loss of elasticity of the crystalline lens, although changes in the lens's curvature from continual growth and loss of power of the ciliary muscles (the muscles that bend and straighten the lens) have also been postulated as its cause.

Similar to grey hair and wrinkles, presbyopia is a symptom caused by the natural course of aging. The first symptoms (described below) are usually first noticed between the ages of 40-50. The ability to focus on near objects declines throughout life, from an accommodation of about 20 dioptres (ability to focus at 50 mm away) in a child to 10 dioptres at 25 (100 mm) and leveling off at 0.5 to 1 dioptre at age 60 (ability to focus down to 1-2 meters only).

Symptoms

The first symptoms most people notice are, difficulty reading fine print, particularly in low light conditions, eyestrain when reading for long periods, blur at near or momentarily blurred vision when transitioning between viewing distances. Many advanced presbyopes complain that their arms have become "too short" to hold reading material at a comfortable distance.

Presbyopia, like other focus defects, becomes much less noticeable in bright sunlight. This is not the result of any mysterious 'healing effect' but just the consequence of the iris closing to a pinhole, so that depth of focus, regardless of actual ability to focus, is greatly enhanced, as in a pinhole camera which produces images without any lens at all. Another way of putting this is to say that the circle of confusion, or blurredness of image, is reduced, without improving focusing.

A delayed onset of seeking correction for presbyopia has been found among those with certain professions and those with miotic pupils. In particular, farmers and housewives seek correction later, whereas service workers and construction workers seek eyesight correction earlier.

Focusing mechanism of the eye

In optics, the closest point at which an object can be brought into focus by the eye is called the eye's **near point**. A standard near point distance of 25 cm is typically assumed in the design of optical instruments, and in characterizing optical devices such as magnifying glasses.

There is some confusion in articles and even textbooks over how the focusing mechanism of the eye actually works. In the classic book, 'Eye and Brain' by Gregory, for example, the lens is said to be suspended by a membrane, the 'zonula', which holds it under tension. The tension is released, by contraction of the ciliary muscle, to allow the lens to fatten, for close vision. This would seem to imply that the ciliary muscle, which is outside the zonula must be circumferential, contracting like a sphincter, to slacken the tension of the zonula pulling outwards on the lens. This is consistent with the fact that our eyes seem to be in the 'relaxed' state when focusing at infinity, and also explains why no amount of effort seems to enable a myopic person to see further away. Many texts, though, describe the 'ciliary muscles' (which seem more likely to be just elastic ligaments and not under any form of nervous control) as pulling the lens taut in order to focus at close range. This has the counterintuitive effect of steepening the lens centrally (increasing its power) and flattening peripherally.

Presbyopia and the 'payoff' for the nearsighted

Many people with myopia are able to read comfortably without eyeglasses or contact lenses even after age 40. However, their myopia does not disappear and the long-distance visual challenges will remain. Myopes with astigmatism will find near vision better though not perfect without glasses or contact lenses once presbyopia sets in, but the greater the amount of astigmatism the poorer their uncorrected near vision. Myopes considering refractive surgery are advised that surgically correcting their nearsightedness may actually be a disadvantage after the age of 40 when the eyes become presbyopic and lose their ability to accommodate or change focus because they will then need to use glasses for reading. A surgical technique offered is to create a "reading eye" and a "distance vision eye", a technique commmonly used in contact lens practice, known as monovision.

Treatment

Presbyopia is not routinely curable - though tentative steps toward a possible cure suggest that this may be possible - but the loss of focusing ability can be compensated for by corrective lenses including eyeglasses or contact lenses. In subjects with other refractory problems, Convex lenses are used. In some cases, the addition of bifocals to an existing lens prescription is sufficient. As the ability to change focus worsens, the prescription needs to be changed accordingly.

Around the age of 65, the eyes have usually lost most of the elasticity. However, it will still be possible to read with the help of the appropriate prescription. Some may find it necessary to hold reading materials farther away, or require larger print and more light to read by. People who do not need glasses for distance vision may only need half glasses or reading glasses.

While bifocals and multifocals offer a working solution to everyday problems, they are hated by many, especially engineers, camera operators, and those used to having a good, sharp, distortion-free image in their work. Varifocals cause straight lines to look bent, and can leave

some feeling dizzy after extended use. Trufocals allows clear, crisp vision wherever the viewer is looking. This is the newest technological improvement to multifocals and bifocals. Trufocals work like the natural eye, when the viewer moves the slider the images are focussed clearly. The power of simple, multiple prescriptions should not be underestimated. Reading glasses hastily prescribed may be fine for reading, but not good for shopping and generally walking around in. This is because of spatial disortion. Poor quality lenses have aberrations and cause visual discomfort. A slightly weaker prescription however, just powerful enough for reading using the full remaining accommodation of the eye, may feel much more comfortable for more general use too. Careful calculation of working ranges, together with a certain amount of trial and error, can restore undistorted vision for critical tasks for many people who do not find multifocals to their liking. In order to reduce the need for bifocals or reading glasses, some people choose contact lenses to correct one eye for near and one eye for far with a method called "monovision". Monovision sometimes interferes with depth perception. There are also newer bifocal or multifocal contact lenses that attempt to correct both near and far vision with the same lens.

Nutrition

At least one scientific study reported that taking lutein supplements or otherwise increasing the amount of lutein in the diet resulted in an improvement in visual acuity, while another study suggested that lutein supplementation might slow aging of the lens. Lutein is found naturally in both the lens of the eye and the macula, the central area of the retina.

Surgery

New surgical procedures may also provide solutions for those who do not want to wear glasses or contacts, including the implantation of accommodative intraocular lenses (IOLs). Scleral expansion bands, which increase the space between the ciliary body and lens, have not been found to provide predictable or consistent results in the treatment of presbyopia.

http://en.wikipedia.org/wiki/Presbyopia

PTOSIS

Ptosis (pronounced toe' sis), or drooping of the upper eyelid, may occur for several reasons such as: disease, injury, birth defect, previous eye surgery and age. In most cases, it is caused by either a weakness of the levator muscle (muscle that raises the lid), or a problem with the nerve that sends messages to the muscle.

Children born with ptosis may require surgical correction of the lid if it covers the pupil. In some cases, it may be associated with a crossed or misaligned eye (strabismus). Left untreated, ptosis may prevent vision from developing properly, resulting in amblyopia, or lazy eye.

Patients with ptosis often have difficult blinking, which may lead to irritation, infection and eyestrain. If a sudden and obvious lid droop is developed, an ophthalmologist should be consulted immediately.

Signs and Symptoms

The causes of ptosis are quite diverse. The symptoms are dependent on the underlying problem and may include:

- Drooping lid (may affect one or both eyes)
- Irritation
- Difficulty closing the eye completely
- Eye fatigue from straining to keep eye(s) open
- Children may tilt head backward in order to lift the lid
- Crossed or misaligned eye
- Double vision

Detection and Diagnosis

When examining a patient with a droopy lid, one of the first concerns is to determine the underlying cause. The doctor will measure the height of the eyelid, strength of the eyelid muscles, and evaluate eye movements and alignment. Children may require additional vision testing for amblyopia.

Treatment

Ptosis does not usually improve with time, and nearly always requires corrective surgery by an ophthalmologist specializing in plastic and reconstructive surgery. In most cases, surgery is performed to strengthen or tighten the levator muscle and lift the eyelid. If the levator muscle is especially weak, the lid and eyebrow may be lifted. Ptosis can usually be performed with local anesthesia except with young children.

http://www.stlukeseye.com/Conditions/Ptosis.asp

SCLERITIS

Scleritis is an inflammatory disease that affects the conjunctiva, sclera, and episclera (the connective tissue between the conjunctiva and sclera). It is associated with underlying systemic diseases in about half of the cases. The diagnosis of scleritis may lead to the detection of underlying systemic disease. Rarely, scleritis is associated with an infectious problem.

The affected area of the sclera may be confined to small nodules, or it may cause generalized inflammation. Necrotizing scleritis, a more rare, serious type, causes thinning of the sclera. Severe cases of scleritis may also involve inflammation of other ocular tissues.

Scleritis affects women more frequently then men. It most frequently occurs in those who are in their 40's and 50's. The problem is usually confined to one eye, but may affect both.

Signs and Symptoms

- Severe, boring pain that can awaken the patient
- Local or general redness of the sclera and conjunctiva
- Extreme tenderness
- Light sensitivity and tearing (in some cases)
- Decreased vision (if other ocular tissues are involved)

Detection and Diagnosis

Along with visual acuity testing, measurement of intraocular pressure, slit lamp examination, and ophthalmoscopy, the doctor may order blood tests to rule out diseases affecting the body. If involvement of the back of the eye is suspected, the doctor may order imaging tests such as CT Scan, MRI, or ultrasonography of the eye.

Treatment

Scleritis is treated with oral steroid and non-steroidal anti-inflammatory medication to reduce inflammation. Eye drops alone do not provide adequate treatment. In very severe cases of necrotizing scleritis, surgery may be required to graft scleral or corneal tissue over the area of thinned sclera.

http://www.stlukeseye.com/conditions/Scleritis.asp

SNOW BLINDNESS

Snow blindness (Niphablepsia) is a painful condition, typically a keratitis, caused by exposure of unprotected eyes to the ultraviolet (UV) rays in bright sunlight reflected from snow or ice. This is especially a problem in polar regions and at high altitudes, as with every thousand feet (approximately 305 meters) increase in elevation, the intensity of UV rays goes up five percent.

The problem is also related to the condition arc eye sometimes experienced by welders.

Snow blindness is akin to a sunburn of the cornea and conjunctiva, and may not be noticed for several hours from exposure. Symptoms can run the gamut from eyes being bloodshot and teary to increased pain, feeling gritty and swelling shut. In very severe cases, snow blindness can cause permanent vision loss.

When trekking, mountaineering or skiing, sunglasses that offer the following are frequently recommended:

- 99-100% UV absorption
- Polycarbonate or CR-39 lens
- 5-10% visible light transmittance
- Large lenses that fit close to the face and cover the whole eye
- Wraparound, side-shielded, or dark-lensed 'glacier' glasses to prevent incidental light exposure
- Wear even when the sky is overcast, as UV rays can still filter through clouds

- In the event of lost or damaged sunglasses, make emergency goggles by cutting slits in dark fabric or tape folded back onto itself

www.vikipedia.com

STRABISMUS
(CROSSED OR TURNED EYE)

Strabismus is a problem caused by one or more improperly functioning eye muscles, resulting in a misalignment of the eyes. Normally, each eye focuses on the same spot but sends a slightly different message to the brain. The brain superimposes the two images, giving vision depth and dimension. Here's an easy way to see how the eyes work together: hold your finger at arm's length. While looking at your finger, close one eye, then the other. Notice how your finger changes position. Even though the images are slightly different, the brain interprets them as one.

Each eye has six muscles that work in unison to control movements. The brain controls the eye muscles, which keep the eyes properly aligned. It is critical that the muscles function together for the brain to interpret the image from each eye as a single one.

Strabismus must be detected early in children because they are so adaptable. If a child sees double, his or her brain quickly learns to suppress or block out one of the images to maintain single vision. In a very short time, the brain permanently suppresses vision from the turned eye, causing a weak or amblyopic eye. Children may also develop a head tilt or turn to compensate for the problem and eliminate the double image. Unlike children, adults with a newly acquired strabismus problem typically see double.

There are many causes of strabismus. It can be inherited, or it may be caused by trauma, certain diseases, and sometimes eye surgery.

Signs and Symptoms

Adults are much more likely to be bothered by symptoms from strabismus than young children. It is unusual for a child to complain of double vision. Children should undergo vision screening exams to detect problems early. The younger the child is when strabismus is detected and treated, the better the chance of normal vision. The following are common signs and symptoms:

- Turned or crossed eye
- Head tilt or turn
- Squinting
- Double vision (in some cases)

Detection and Diagnosis

Strabismus is detected with a comprehensive eye exam and special tests used to evaluate the alignment of the eyes such as: the Krimsky test and prism testing.

Treatment

The appropriate treatment for strabismus is dependent on several factors including the patient's age, the cause of the problem, and the type and degree of the eye turn. Treatment may include patching, corrective glasses, prisms, or surgery.

With patching, the better eye is covered, forcing the child to use the weaker eye. Over time, the brain adjusts to using the weaker eye and vision gradually improves. For this treatment to be effective, it must be done at a young age before the child can develop amblyopia.

Surgery is sometimes performed for both adults and children to straighten a crossed eye. The procedure may be done with local or general anesthesia. There are several different surgical techniques used to correct strabismus. The appropriate one is dependent on the muscle involved and the degree of the eye turn.

http://www.stlukeseye.com/Conditions/Strabismus.asp

STYE OR **HORDEOLUM**

Stye or **hordeolum** is an infection of the sebaceous glands at the base of the eyelashes. While they produce no lasting damage, styes can be quite painful

Styes are generally caused by a Staphylococcus aureus bacteria infection. Although they are particularly common in infants, styes are experienced by people of all ages. Styes can be triggered by stress or poor nutrition. Using the same razor to shave hair near both the eyes and a mustache can also spread staphylococcus bacteria, potentially leading to styes or other eye infections.

A stye can be secondary, caused by blepharitis. A blocked oil gland near the eye, a chalazion, is often mistaken for a stye.

Signs and symptoms

The first signs of a stye are tenderness, pain and redness in the affected area. Later symptoms include itching, swelling, watering of the eye, sensitivity to light and discomfort when blinking. A yellowish bump develops in the affected area.

Treatment

While most styes will drain on their own, this process can be accelerated by the application of a hot or warm compress or by pulling out the eyelash. There is also a specialized Polysporin topical ointment for styes. With treatment, styes typically resolve within one week. While a stye is technically a pimple and can be popped, doing so is not recommended without technical expertise due to its proximity to the eye. Styes may also cause a bruised feeling around the eye, which can be treated through the application of a warm cloth.

Medical professionals will sometimes lance a particularly persistent or irritating stye with a needle in order to accelerate its draining. A stye's expansion can also be fought with an erythromycin ophthalmic ointment like Neosporin, a special version of which is available for styes. Medical professionals may also prescribe Amoxicillin over a period of a week.

If a stye bursts care must be taken to cleanse the wound to prevent reinfection.

Contact lenses should never be worn during treatment for a stye. Eye make-up is not recommended, and it is also important to refrain from touching the stye.

Alternative remedies

There are various folk remedies for styes, including rubbing a gold ring or the hair of a cat's tail on the affected area. Other folk remedies suggest applying substances to the stye. Some suggested topical applications are the first urine of the day, a black teabag and lipstick. Also suggested is rubbing the index finger on the palm until warm before applying it to the affected area.

According to Ayurveda, applying saliva to the infected area immediately after waking in the morning will cure the infection.

It is important to note that these techniques have not been empirically proven, and are not recommended as treatments by medical professionals or other experts.

http://en.wikipedia.org/wiki/Hordeolum

TOXOCARIASIS

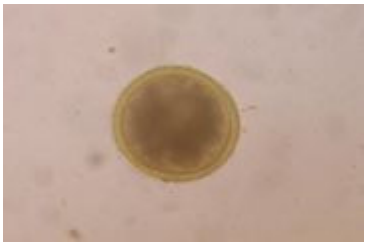
Toxocariasis (Toxocarosis) is a helminth infection of humans caused by the dog or cat roundworm, *Toxocara canis* or *Toxocara cati*, respectively. Humans can become infected by ingestion of embryonated eggs (containing fully developed larva L3) from contaminated sources. There are two main syndromes; visceral larva migrans (VLM), which encompasses diseases associated with major organs, and ocular larva migrans (OLM), in which toxocariasis pathological effects on the host are restricted to the eye and the optic nerve.

Toxocariasis occurs around the world. Epidemiologic surveys show a 2-5% positive rate in healthy adults from urban Western countries and 14.2-37% in rural areas. In tropical countries, surveys show a positive rate of 63.2% in Bali, 86% in Saint Lucia, and 92.8% in Réunion. Toxocariasis is most commonly a disease of children, typically children aged 2-7 years.

Risk factors

- Exposure to contaminated soil.
- Presence of unwormed pups, unhygenic conditions.
- Geophagia (pica)- 2-10% of children aged 1-3 indulge in habitual geophagia.

Pathophysiology



Toxocara canis egg

Adult worms of the *Toxocara* family often live in the small intestine of dogs and cats. They range from 4-12 cm in length. Almost all puppies are infected at or soon after birth. During the summer, *Toxocara* infective eggs are shed. They survive for years in the environment, and humans typically ingest the eggs orally by eating with contaminated hands (Most commonly from handling infected feces with bare hands). Once introduced into the human intestine, the eggs develop into larvae. The larval form is less than 0.5 mm in length and 0.02 mm wide. The larvae penetrate the bowel wall and migrate through blood vessels to reach the liver, muscles, and lungs. Sometimes the parasite penetrates into the eye and brain.

Disease severity is affected by the number of eggs ingested, duration of infection, tissue location of larvae, and the immune response to the infection.

Features

- Weakness
- Pruritus (Itching)
- Rash
- Difficulty breathing
- Abdominal pain / Hepatosplenomegaly
- Hyper-eosinophilia
- Increased total serum Immunoglobulin E (IgE) level
- Elevated antibody titers to *T. canis*

Diagnosis

In suspected cases, diagnosis is confirmed by an increase in the anti-*Toxocara* excretory-secretory antigen IgE level History of exposure to dogs and cats High Sustained eosinophilia + hyperglobulinemia + hepatomegaly Liver Biopsy shows degenerated larvae at the centre of an eosinophilic granuloma

Treatment

Anthelmintic treatment, e.g. mebendazole, thiabendazole and diethylcarbamazine. Albendazole is the more modern form of treatment. Management with anti-inflammatory steroids is also an option.

Prognosis

Toxocariasis is always a benign, asymptomatic, and self-limiting disease, although brain involvement can cause brain damage, meningitis, encephalitis, or epilepsy. Ocular involvement, also known as 'ocular larvae migrans,' may cause loss of visual acuity or

unilateral blindness. Pulmonary and hepatic forms can cause protracted symptoms if the patient does not receive treatment.

Prevention

The eggs of *Toxocara* species are widespread in parks, playgrounds, yards, and in homes and apartments where the occupants have dogs or cats. Elimination of eggs from the environment is not possible; therefore, prevention depends on proper hygiene, including handwashing after contact with pets. Public policies that have attempted to eradicate *Toxocara* infection in dogs and cats have had limited success.

http://en.wikipedia.org/wiki/Toxocariasis

TOXOPLASMOSIS

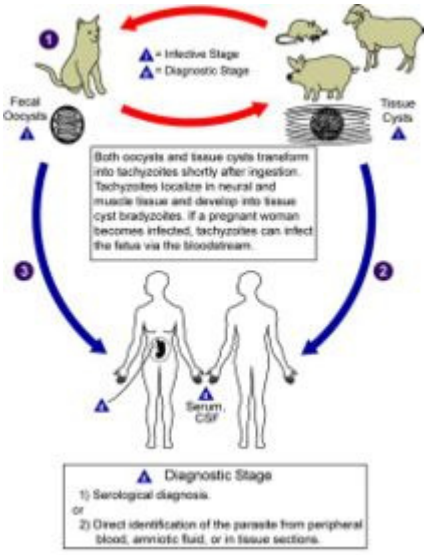
Toxoplasmosis is a parasitic disease caused by the protozoan Toxoplasma gondii. The parasite infects most warm-blooded animals, including humans, but the primary host is the felid (cat) family. Animals are infected by eating infected meat, by ingestion of faeces of a cat that has itself recently been infected, or by transmission from mother to fetus. Cats have been shown as a major reservoir of this infection.

During the first few weeks, the infection typically causes a mild flu-like illness or no illness. After the first few weeks of infection have passed, the parasite rarely causes any symptoms in otherwise healthy adults. However, people with a weakened immune system, such as those infected with HIV, and fetuses, may become seriously ill, and it can occasionally be fatal. The parasite can cause encephalitis (inflammation of the brain) and neurologic diseases and can affect the heart, liver, and eyes (chorioretinitis).

History

The protozoan was first discovered by Nicolle & Manceaux, who in 1908 isolated it from the African rodent *Ctenodactylus gundi*, then in 1909 differentiated the disease from *Leishmania* and named it *Toxoplasmosis gondii*. The first recorded congenital case was not until 1923, and the first adult case not until 1940. In 1948, a serological dye test was created by Sabin & Feldman, which is now the standard basis for diagnostic tests.

Transmission



Life cycle of *Toxoplasma gondii*.

Transmission may occur through:

- Ingestion of raw or partly cooked meat, especially pork, lamb, or venison containing Toxoplasma cysts. Infection prevalence in countries where undercooked meat is traditionally eaten, such as France, has been related to this transmission method. Oocysts may also be ingested during hand-to-mouth contact after handling undercooked meat, or from using knives, utensils, or cutting boards contaminated by raw meat.

- Ingestion of contaminated cat faeces. This can occur through hand-to-mouth contact following gardening, cleaning a cat's litter box, contact with children's sandpits, or touching anything that has come into contact with cat faeces.
- Drinking water contaminated with Toxoplasma.
- Transplacental infection in utero.
- Receiving an infected organ transplant or blood transfusion, although this is extremely rare.

The cyst form of the parasite is extremely hardy, capable of surviving exposure to freezing down to −12 degrees Celsius (10 degrees Fahrenheit), moderate temperatures and chemical disinfectants such as bleach, and can survive in the environment for over a year. It is, however, susceptible to high temperatures—above 66 degrees Celsius (150 degrees Fahrenheit), and is thus killed by thorough cooking, and would be killed by 24 hours in a typical domestic freezer.

Cats excrete the pathogen in their faeces for a number of weeks after contracting the disease, generally by eating an infected rodent. Even then, cat faeces are not generally contagious for the first day or two after excretion, after which the cyst 'ripens' and becomes potentially pathogenic. Studies have shown that only about 2% of cats are shedding oocysts at any one time, and that oocyst shedding does not recur even after repeated exposure to the parasite. Although the pathogen has been detected on the fur of cats, it has not been found in an infectious form, and direct infection from handling cats is generally believed to be very rare.

Pregnancy precautions

Congenital toxoplasmosis is a special form in which an unborn child is infected via the placenta. A positive antibody titer indicates previous exposure and immunity and largely ensures the unborn baby's safety. A simple blood draw at the first pre-natal doctor visit can determine whether or not the woman has had previous exposure and therefore whether or not she is at risk. If a woman receives her first exposure to toxoplasmosis while pregnant, the baby is at particular risk. A woman with no previous exposure should avoid handling raw meat, exposure to cat feces, and gardening (cat feces are common in garden soil). Most cats are not actively shedding oocysts and so are not a danger, but the risk may be reduced further by having the litterbox emptied daily (oocysts require longer than a single day to become infective), and by having someone else empty the litterbox. However, while risks can be minimized, they cannot be eliminated. For pregnant women with negative antibody titer, indicating no previous exposure to T. gondii, as frequent as monthly serology testing is advisable as treatment during pregnancy for those women exposed to T. gondii for the first time decreases dramatically the risk of passing the parasite to the fetus.

Despite these risks, pregnant women are not routinely screened for toxoplasmosis in most countries (France, Austria and Italy being the exceptions) for reasons of cost-effectiveness and the high number of false positives generated as the disease is so rare (an example of Bayesian statistics). As invasive prenatal testing incurs some risk to the fetus (18.5 pregnancy losses per toxoplasmosis case prevented), postnatal or neonatal screening is preferred. The exceptions are cases where foetal abnormalities are noted, and thus screening can be targeted.

Some regional screening programmes operate in Germany, Switzerland and Belgium.

Treatment is very important for recently infected pregnant women, to prevent infection of the fetus. Since a baby's immune system does not develop fully for the first year of life, and the resilient cysts that form throughout the body are very difficult to eradicate with anti-protozoans, an infection can be very serious in the young.

Transplacental transmission:(a) infection in 1st trimester - incidence of transplacental infection is low (15%) but disease in neonate is most severe. (b) infection in 3rd trimester - incidence of transplacental infection is high (65%) but infant is usually asymptomatic at birth.

Clinical manifestations

Infection has two stages:

Acute toxoplasmosis

During acute toxoplasmosis, symptoms are often influenza-like: swollen lymph nodes, or muscle aches and pains that last for a month or more. Rarely, a patient with a fully functioning immune system may develop eye damage from toxoplasmosis. Young children and immunocompromised patients, such as those with HIV/AIDS, those taking certain types of chemotherapy, or those who have recently received an organ transplant, may develop severe toxoplasmosis. This can cause damage to the brain or the eyes. Only a small percentage of infected newborn babies have serious eye or brain damage at birth.

Latent toxoplasmosis

Most patients who become infected with *Toxoplasma gondii* and develop toxoplasmosis do not know it. In most immunocompetent patients, the infection enters a latent phase, during which only bradyzoites are present, forming cysts in nervous and muscle tissue. Most infants who are infected while in the womb have no symptoms at birth but may develop symptoms later in life.

Treatment

Treatment is often only recommended for people with serious health problems, because the disease is most serious when one's immune system is weak.

Medications that are prescribed for acute Toxoplasmosis are:

- Pyrimethamine — an antimalarial medication.
- Sulfadiazine — an antibiotic used in combination with pyrimethamine to treat toxoplasmosis.
- clindamycin — an antibiotic. This is used most often for people with HIV/AIDS.
- spiramycin — another antibiotic. This is used most often for pregnant women to prevent the infection of their child.

(Other antibiotics such as minocycline have seen some use as a salvage therapy).

In people with latent toxoplasmosis, the cysts are immune to these treatments, as the antibiotics do not reach the bradyzoites in sufficient concentration.

Medications that are prescribed for latent Toxoplasmosis are:

- atovaquone — an antibiotic that has been used to kill Toxoplasma cysts in situ in AIDS patients.
- clindamycin — an antibiotic which, in combination with atovaquone, seemed to optimally kill cysts in mice.

However, in latent infections successful treatment is not guaranteed, and some subspecies exhibit resistance.

Biological modifications of the host

The parasite itself can cause various effects on the host body, some of which are not fully understood.

Reproductive changes

A recent study has indicated Toxoplasmosis correlates strongly with an increase in boy births in humans. According to the researchers, *depending on the antibody concentration, the probability of the birth of a boy can increase up to a value of 0.72 ... which means that for every 260 boys born, 100 girls are born.* The study also notes a mean rate of 0.60 to 0.65 (as opposed to the normal 0.51) for Toxoplasma positive mothers.

Behavioral changes

It has been found that the parasite has the ability to change the behavior of its host: infected rats and mice are less fearful of cats — in fact, some of the infected rats seek out cat-urine-marked areas. This effect is advantageous to the parasite, which will be able to sexually reproduce if its host is eaten by a cat. The mechanism for this change is not completely understood, but there is evidence that toxoplasmosis infection raises dopamine levels and concentrates in the amygdala in infected mice.

The findings of behavioral alteration in rats and mice have led some scientists to speculate that toxoplasma may have similar effects in humans, even in the latent phase that had previously been considered asymptomatic. Toxoplasma is one of a number of parasites that may alter their host's behaviour as a part of their life cycle. The behaviors observed, if caused by the parasite, are likely due to infection and low-grade encephalitis, which is marked by the presence of cysts in the brain, which may produce or induce production of a neurotransmitter, possibly dopamine, therefore acting similarly to dopamine reuptake inhibitor type antidepressants and stimulants.

"In populations where this parasite is very common, mass personality modification could result in cultural change. [Variations in the prevalence of *Toxoplasma gondii*] may explain a substantial proportion of human population differences we see in cultural aspects that relate to ego, money, material possessions, work and rules." — Kevin Lafferty

Correlations have been found between latent *Toxoplasma* infections and various characteristics:

- Decreased novelty-seeking behaviour
- Slower reactions
- Lower rule-consciousness and jealousy (in men)
- More warmth and conscientiousness (in women)

The evidence for behavioral effects on humans is relatively weak. There have been no randomized clinical trials studying the effects of toxoplasma on human behavior. Although some researchers have found potentially important associations with toxoplasma, it is possible that these associations merely reflect factors that predispose certain types of people to infection (e.g., people who exhibit risk-taking behaviors may be more likely to take the risk of eating undercooked meat).

Toxoplasma's role in schizophrenia

The possibility that toxoplasmosis is one cause of schizophrenia has been studied by scientists since at least 1953. These studies had attracted little attention from U.S. researchers until they were publicized through the work of prominent psychiatrist and advocate E. Fuller Torrey. In 2003, Torrey published a review of this literature, reporting that almost all the studies had found that schizophrenics have elevated rates of toxoplasma infection.[A 2006 paper has even suggested that prevalence of toxoplasmosis has large-scale effects on national culture. These types of studies are suggestive but cannot confirm a causal relationship (because of the possibility, for example, that schizophrenia increases the likelihood of toxoplasma infection rather than the other way around).

- Acute *Toxoplasma* infection sometimes leads to psychotic symptoms not unlike schizophrenia.
- Some anti-psychotic medications that are used to treat schizophrenia, such as Haloperidol, also stop the growth of *Toxoplasma* in cell cultures.
- Several studies have found significantly higher levels of *Toxoplasma* antibodies in schizophrenia patients compared to the general population.
- *Toxoplasma* infection causes damage to astrocytes in the brain, and such damage is also seen in schizophrenia

Two risk factors for contracting toxoplasmosis are:

- Infants born to mothers who became infected with *Toxoplasma* for the first time during or just before pregnancy.
- Persons with severely weakened immune systems, such as those with AIDS. Illness may result from an acute Toxoplasma infection or reactivation of an infection that occurred earlier in life.

http://en.wikipedia.org/wiki/Toxoplasmosis

TRICHIASIS

Trichiasis is a medical term for ingrown eyelashes. This can be caused by infection, inflammation, autoimmune conditions, and trauma such as burns or eyelid injury.

Standard treatment involves destruction of the affected eyelashes with electrology, specialized laser, or surgery.

Trichiasis in dogs is hair from the eyelid growing in the wrong direction and rubbing on the eye, causing irritation. It usually occurs at the lateral upper eyelid, especially in the English Cocker Spaniel. Trichiasis also refers to hair from a nasal fold rubbing on the eye. This type of trichiasis can be flattened by rubbing petroleum jelly onto it, but surgery is sometimes necessary for permanent correction.

http://en.wikipedia.org/wiki/Trichiasis

UVEITIS

Uveitis specifically refers to inflammation of the middle layer of the eye, termed the "uvea" but in common usage may refer to any inflammatory process involving the interior of the eye.

Uveitis is estimated to be responsible for approximately 10% of the blindness in the United States. Uveitis requires an urgent referral and thorough examination by an ophthalmologist, along with urgent treatment to control the inflammation.

Types

Uveitis is usually categorized anatomically into *anterior*, *intermediate*, *posterior* and *panuveitic* forms.

- Anywhere from two-thirds to 90% of uveitis cases are anterior in location (anterior uveitis), frequently termed *iritis* - or inflammation of the iris and anterior chamber. This condition can occur as a single episode and subside with proper treatment or may take on a recurrent or chronic nature. Symptoms include red eye, injected conjunctiva, pain and decreased vision. Signs include dilated ciliary vessels, presence of cells and flare in the anterior chamber, and keratic precipitates ("KP") on the posterior surface of the cornea.
- Intermediate uveitis consists of vitritis - inflammatory cells in the vitreous cavity, sometimes with *snowbanking*, or deposition of inflammatory material on the pars plana.
- Posterior uveitis is the inflammation of the retina and choroid.
- Pan-uveitis is the inflammation of all the layers of the uvea.

Causes

Myriad conditions can lead to the development of uveitis, including systemic diseases as well as syndromes confined to the eye. In anterior uveitis, no specific diagnosis is made in approximately one-half of cases. However, anterior uveitis is often one of the syndromes associated with HLA-B27.

Systemic disorders causing uveitis

Systemic disorders that can cause uveitis include:

- Acute posterior multifocal placoid pigment epitheliopathy
- Ankylosing spondylitis
- Behçet's disease
- Birdshot retinochoroidopathy
- Brucellosis
- Herpes simplex
- Herpes zoster
- Inflammatory bowel disease
- Juvenile rheumatoid arthritis
- Kawasaki's disease
- Leptospirosis
- Lyme disease
- Multiple sclerosis
- Presumed ocular histoplasmosis syndrome
- Psoriatic arthritis
- Reiter's syndrome
- Sarcoidosis
- Syphilis
- Systemic lupus erythematosus
- Toxocariasis
- Toxoplasmosis
- Tuberculosis
- Vogt-Koyanagi-Harada syndrome

- Whipple disease
- Polyarteritis nodosa

Masquerade syndromes

Masquerade syndromes are ophthalmic disorders that clinically present as either an anterior or posterior uveitis, but are not primarily inflammatory. The following are some of the most common:

- Anterior segment
    - Intraocular foreign body
    - Juvenile xanthogranuloma
    - Leukemia
    - Malignant melanoma
    - Retinoblastoma
    - Retinal detachment
- Posterior segment
    - Lymphoma
    - Malignant melanoma
    - Multiple sclerosis
    - Reticulum cell sarcoma
    - Retinitis pigmentosa
    - Retinoblastoma

Symptoms

- Redness of the eye
- Blurred vision
- Sensitivity to light (photophobia)
- Dark, floating spots along the visual field
- Eye pain

Treatment

The prognosis is generally good for those who receive prompt diagnosis and treatment, but serious complication (including cataracts, glaucoma, band keratopathy, retinal edema and permanent vision loss) may result if left untreated. The type of uveitis, as well as its severity, duration, and responsiveness to treatment or any associated illnesses, all factor in to the outlook.

Uveitis is typically treated with glucocorticoid steroids, either as topical eye drops (such as betamethasone, dexamethasone or prednisolone) or oral therapy with prednisolone tablets. In addition topical cycloplegics, such as atropine or homatropine, may be used. In some cases an injection of PSTTA can also be given to reduce the swelling of the eye.

Antimetabolite medications, such as methotrexate are often used for recalcitrant or more aggressive cases of uveitis. Experimental treatment with Infliximab infusions may prove helpful.

http://en.wikipedia.org/wiki/Uveitis