Why The Eye Gets Dry— And What To Do About It

A clinical guide to ocular surface disease.

Some 10 million Americans suffer chronic dry eye symptoms. About two-thirds of these patients elect to treat themselves, often choosing their medications on a random, hit-or-miss basis. The rest turn to eye doctors like yourself for the expert care they deserve.

TEAR PHYSIOLOGY AND FUNCTION

Ocular surface disease is a general term for a group of disorders including dry eye. Proper management of ocular surface disease begins with a thorough understanding of the physiology and function of the lacrimal system.

Dry eye occurs when the quantity or quality of the tear film is not sufficient to maintain a healthy corneal epithelium. The tear film is complex and relatively unstable, and consists of three layers of fluid—lipids, aqueous and mucin. The tear film lubricates the cornea and provides its oxygen supply.

The tear film also plays an immunologic role, as tears contain IgA immunoglobins. Secreted by the lacrimal gland, these antibodies provide a natural defense for the ocular surface. Exactly how this works is not known, but it is thought that IgA may inhibit the adherence of bacteria to the conjunctiva. Patients with decreased tear volume have less IgA and thus a higher incidence of infection.

A group of glands produce the fluids which make up the tear film. The Meibomian glands and the glands of Zeis produce the outermost lipid layer. The aqueous component is generated by the lacrimal gland and accessory glands of Krause and Wolfring. The goblet cells secrete the innermost mucin layer.

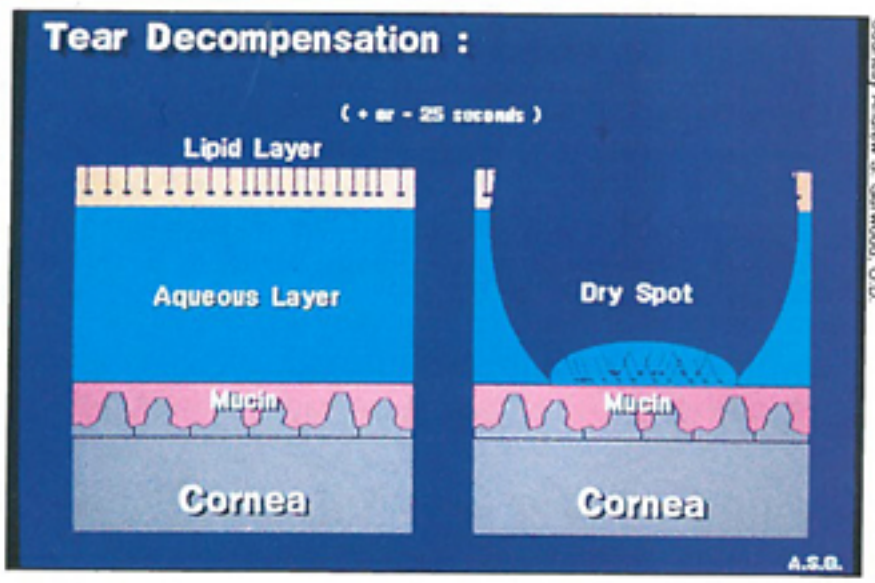
In a normal eye tears are secreted continuously. Many stimuli can increase tear production: sensory stimuli such as irritants (chemicals for example), inflammations or foreign bodies; retinal stimuli such as bright light; and even psychological stimuli such as crying.

Once produced, the tears spread across the eye by the blinking action of the eyelids. A normal tear film is about 7-10 micrometers thick, with a volume of 6 microliters. The aqueous comprises some 90 percent of the overall tear volume.

A sufficient quantity of tears will not ensure adequate lubrication if the quality of tears is lacking or the blinking process is compromised. During blinking a correct balance of lipids, aqueous and mucin forms a continuous film layer. Between blinks, evaporation begins. The tear film thins, allowing the lipids to migrate through the aqueous layer toward the mucin layer. Lipid contamination increases the corneal surface tension.

Eventually a rupture forms in the tear film and a dry spot results. Subsequent blinks remove the lipid contaminants, repair the rupture and restore the normally thick aqueous layer of the tear film.

A normal blink rate is 5-10 seconds, or about 6-



Courtesy Andrew S. Garwood, O.D.

GEORGE E. WHITE, O.D.
PHILADELPHIA

12 times per minute. Age has a significant effect on blink rates. Newborns blink less than once a minute, whereas elderly patients blink as often as every three to six seconds.

Various factors can upset the balance of tear-film components: age, disease, lacrimal gland dysfunction, systemic medications, contact lens wear and preservatives in ophthalmic preparations. When tear-film stability is compromised, rupture may occur more quickly. If the breakup time happens to be shorter than the interval between blinks, dry spots develop on the ocular surface. These dry spots signal the first stage of ocular surface disease.

Closing the eyelids creates negative pressure in the lacrimal sac. This forces tears and debris through one of the two puncta, through the canaliculi and into the lacrimal sac. The tears then drain into the lacrimal duct on their way to the sinuses.

Just as important as the quantity of the tear film is its quality, which is a function of four characteristics:

- *Surface tension.* This influences the ability of a liquid to spread across a surface. For the tear film to spread evenly, its surface tension must be lower than that of the ocular surface. Both the mucin and lipid layers reduce the surface tension of the tear film.
- *PH level.* The pH of the tears varies slightly, but generally ranges from 7.0 to 7.4. Buffers in the tear film resist pH changes and help maintain a normal pH level. Once the pH falls outside the 6-8 range, patients are likely to experience discomfort.

Instructions

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burning and stinging.

• *Tonicity.* This is a measure of the particles dissolved within the tear film. Normal tonicity is around 300m Osm/L. If the tears become hypertonic, water diffuses out of the corneal epithelium and results in dehydration, cellular damage and subsequent irritation. Hypotonicity, on the other hand, causes excessive hydration of ocular tissues. This in turn results in corneal swelling and blurred vision.

• *Viscosity.* Normal tears have a viscosity of 2cp. (Water, by comparison, has a cp value of 1.) The viscosity of tears affects the focusing of light as it enters the eye. Abnormally high viscosity can result in blurred vision.

WHAT CAN GO WRONG

Ocular surface disease may arise from changes to any of the three tear-film layers. In addition, abnormalities of the lid or corneal surface can trigger dry-eye symptoms. Certain neurological lesions can also disrupt tear production.

Lipid abnormalities. The lipid layer, about 0.1 um thick, acts to prevent evaporation of the tears from the surface of the eye. Lipid deficiency can arise from different causes.

One cause is a rare congenital condition called anhydrotic ectodermal dysplasia, in which the Meibomian glands are absent altogether. A more common cause of lipid deficiency is Meibomian gland infection, which may occur in blepharitis. The invading bacteria secrete enzymes called lipases, which break down the oily lipids into free fatty acids. These fatty acids cause surface film breakdown.

Aqueous abnormalities. By far the thickest part of the tear film (7 um), the aqueous contains most of the tear-film components and carries virtually all of the oxygen supply to the cornea. Nine out of 10 cases of ocular surface disease are due to aqueous deficiency.

Also known as keratoconjunctivitis sicca, aqueous deficiency tends to occur more frequently in elderly patients and post-menopausal women. It also affects rheumatoid arthritis patients and hypertensive patients taking thiazide diuretics. In addition, Sjogren's syndrome is a rare condition that includes aqueous deficiency, dry mouth and rheumatoid arthritis.

Disorders that affect the main and accessory lacrimal glands result in aqueous deficiency. These conditions include trauma, inflammation, tumors, drug toxicity, neurologic disease and congenital absence of the glands. Several diseases that affect the tear film cause a reduction in lysozyme, a bacteriolytic enzyme found in the aqueous layer.

Mucin abnormalities. The mucin layer provides a hydrophilic coating to the normally hydrophobic corneal epithelium. Mucin deficiency results from conjunctival disease. Such disorders disrupt the conjunctival goblet cells, which produce most of the mucous.

Lid surface abnormalities. A normal blinking rate is essential to maintain the integrity of the tear film and good corneal health. Anything that interferes with blinking can cause drying on the surface of the eye.

Eyelid problems are one source of disruption. These include lagophthalmos and subsequent exposure keratitis, entropian, ectropian, trichiasis, symblepharon, lid notches, Bell's palsy and incomplete blinking.

An abnormal blink rate can allow lipid contamination of the mucin layer. In a normal eye, a blinking lid acts like a windshield wiper to remove the lipid contaminant, resurfacing the epithelium with a fresh coat of mucin. When the blink rate is less than the tear breakup time, dry spots subsequently form on the epithelium and lead to subsequent tissue damage.

Corneal abnormalities. Corneal problems can also lead to tear-film disruption. Corneal dystrophies, recurrent erosions, dellen, 3-9 staining due to rigid lens wear and post-herpetic corneal anesthesia are examples of such changes.

Microscopic, finger-like projections called microvillae hold the tear film in place on the cornea. Infections and certain chemicals can disrupt this process, creating an unstable tear film and reduced surface tension. Diseases affecting the fifth cranial nerve, which innervates the cornea, can also destabilize the tear film. Post-herpetic neuralgia and corneal anesthesia exemplifies this phenomenon.

Neurologic abnormalities. The parasympathetic pathway governs tear production. The sympathetic pathway provokes vasoconstriction of the lacrimal gland arterioles. Certain neurologic lesions affecting these pathways can decrease tear production.

CASE HISTORY

The history is often the most important tool in

diagnosing ocular surface disease.

Patients often report burning, itching, tired eyes. They may complain of redness, dryness, and a gritty foreign body sensation. Pain and photophobia are also common. Patients may say they have difficulty blinking, and may report mucous strands in the cul-de-sac.

Impaired vision, especially later in the day, is another common symptom. Patients may also report intolerance to wind, smoke, altitude, cold, low humidity, air conditioning, forced-air heating systems or wood-burning stoves. Symptoms may be exacerbated by drafts, reading, fresh paint or bright lights.

Probe all of these areas during the history. Also, inquire about any concurrent diseases that dry eye symptoms may accompany. These include former rashes of lupus, skin lesions of pemphigoid, old scars from Stevens-Johnson syndrome, seborrheic dermatitis, icthyosis and psoriasis.

Certain medications, both ophthalmic and systemic, also contribute to ocular surface disease. Chlorpheniramine and other antihistamines, for example, decrease tear production.

EXAMINATION

While various tests are helpful in gauging tear deficiency, none by itself is a conclusive diagnostic measure.

A careful slit lamp exam is essential. Common findings include corneal filaments, meniscus height, mucous strands, and particles in the tear film. Also seen in conjunctivitis and blepharitis, these particles are actually desquamated corneal epithelial cells.

Rose bengal staining, tear breakup time and the Schirmer tear test all contribute to a diagnosis. Damaged epithelial cells stain with rose bengal, making this the most useful of all diagnostic tests. An interpalpebral staining pattern is pathognomonic of ocular surface disease.

The Schirmer tear test, the oldest method, has become highly controversial because its results are inconsistent. Even so, this test of the wettability of filter paper in the cul-de-sac does offer some clinical data.

The tear breakup time test is also helpful, but, like the Schirmer test, has become controversial because of the many variables that can affect test results. Such factors include slit beam light intensity, humidity and the fluorescein dye solution that is used. If you control these variables you can obtain valuable diagnostic data.

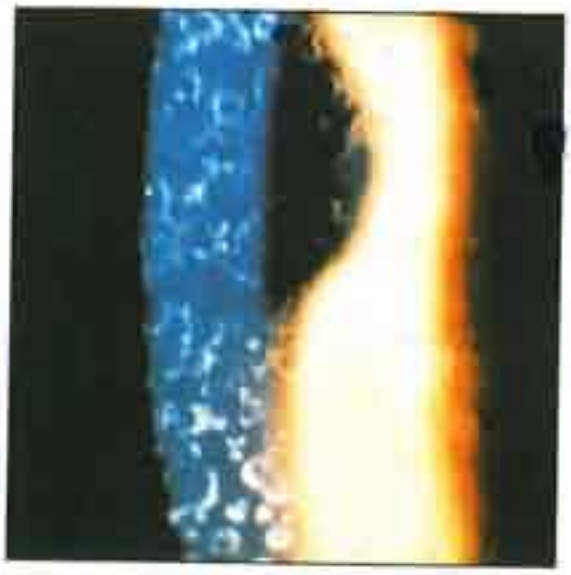
To perform the tear breakup time test, instill fourescein dye in the eye. Have the patient blink a few times and then hold his eye open. Note when a dry spot forms. A dry spot appears as a dark island in the green pool of fluorescein. A normal breakup time is 15 to 20 seconds; a time less than 10 seconds may indicate an abnormal tear film.

Doctors typically use a grading system to indicate the severity of ocular surface disease. Grade 1 denotes a mildy red and irritated eye; grade 2 indicates deterioration of the corneal epithelium; grade 3 suggests keratinization of the epithelium due to severe, chronic dryness.

TREATMENT

Treatment of ocular surface disease entails three goals: supplementing the tears, preserving existing

Filamentary keratitis can result from long-term ocular surface disease.



Map-dot corneal dystrophy can lead to tear film disruption.

Eyelid problems such as lagophthalmos can result in exposure keratitis.

tears and stimulating tear production. Patient education is also crucial.

Tear supplementation. Ocular lubricants or tear substitutes are formulated to mimic the physical and chemical characteristics of tears. Most importantly, an ocular lubricant must promote stability of the tear film. The lipid layer must be preserved, and the damaged epithelium must be allowed to regenerate without interference.

Therapy varies according to the degree of severity. Polyvinyl alcohol-type solutions are usually sufficient to alleviate the symptoms of a mildly dry eye.

Moderate ocular surface disease requires more viscous agents, such as methlycellulose preparations or hypotonic tear substitutes. Patients who require frequent doses of artificial tears are better off using preservative-free preparations. These help patients avoid the risk of toxic reactions to preservatives. Both drops and ointments are available in preservative-free formulations, and most come in convenient, single-dose containers.

A methylcellulose pellet called a Lacrisert is helpful for patients who have difficulty applying drops frequently. The pellet dissolves slowly over 24 hours, providing continuous lubrication to the ocular surface.

While helpful for certain patients, the Lacrisert has some inherent disadvantages. One problem is that those most likely to benefit from its use are elderly, often arthritic patients. These individuals usually do not see very well for a number of age-related reasons; their dexterity may also be compromised. So they may have difficulty placing and retaining the tiny pellet. Also, the existing tears may be insufficient to dissolve the pellet, which may then cause irritation. At $30, the Lacrisert is also an expensive treatment option.

In severe cases of ocular surface disease, frequent application of lubricants may be used in conjunction with ocular ointments. The latter are semi-solid petrolatum or mineral oil-base preparations. These ointments are retained on the ocular surface much longer than drops; the molecules in these preparations are large and do not evaporate or escape through the lacrimal drainage system. Their higher viscosity, however, can be a drawback in that it causes blurred vision. Thus, ointments are typically recommended only for bedtime.

Preserving existing tears. Besides supplementing the tears, your treatment should also aim to preserve existing tears.

A number of methods and devices can help prevent tear loss. These include swimmer's goggles, clear food wrap shields, silicone rubber shields, moist chamber spectacles, lid taping, bandage contact lenses and punctal occlusion. Of these, the latter two are the most useful.

Bandage soft contact lenses are useful in treating filaments or filamentary keratitis resulting from long-term keratoconjunctivitis sicca. The pain relief is almost immediate. What's more, the filaments will not recur under the lens.

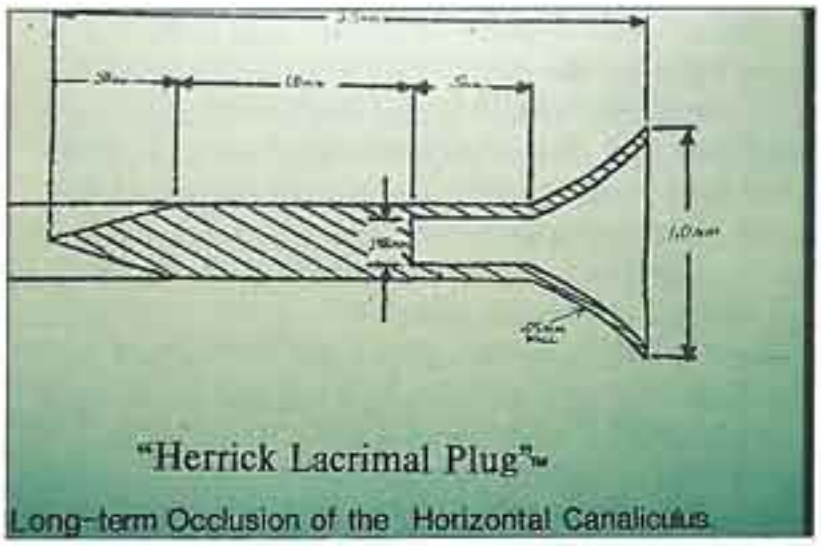
But bandage lenses are not without problems. Normal contact lens dynamics rely on adequate hydration of the eye and lens. With an inadequate aqueous layer, the lens tends to become very dry. Thus, these patients must be monitored closely for lens tightening and subsequent anoxic reactions and secondary infections. Also, dryness frequently results in heavy lens deposits or loss of the lens. Consider starting these patients on prophylactic, broad-spectrum topical antibiotics while using bandage contact lenses.

Punctal occlusion has gained favor in recent years as an effective way to preserve existing tears in cases of ocular surface disease. Punctal occlusion may be achieved with temporary or permanent plugs. If these prove beneficial, permanent surgical methods of punctal occlusion may be considered.

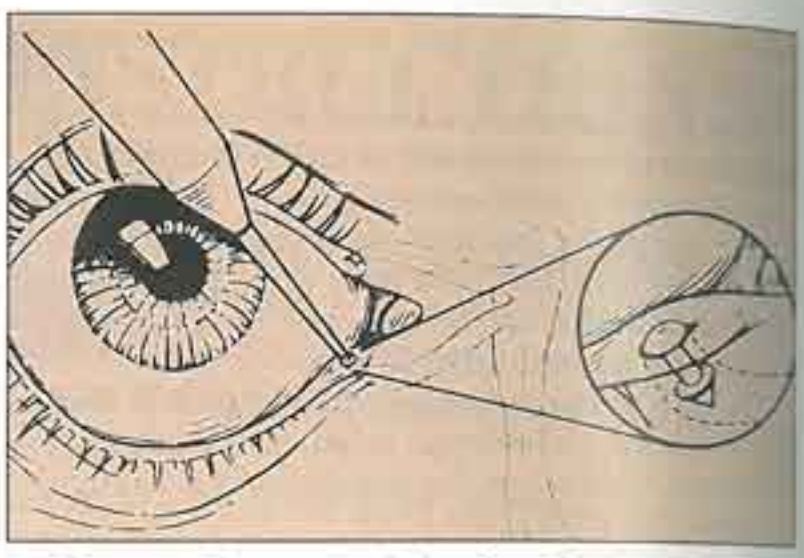
Temporary punctal occlusion with collagen plugs is a simple and painless approach to tear preservation. The collagen rods are available in three sizes, 0.2mm, 0.3mm and 0.4mm.

The procedure requires no anesthetic. Using your jeweler's forceps, simply place the collagen rods in all four punctal openings. If the punctal ring is not completely open or is stenosed, you may need to dilate the puncta. Push the plug down below the punctal opening until it disappears from slit lamp view. Thus positioned, the plug will not become dislodged by normal lid manipulation.

Like a grain of boiling rice, the collagen rod swells to the point where it mechanically blocks the drainage of tears. The blockage lasts about seven to 10 days. Caution the patient about the possibility of epiphora during the first few days. As the collagen starts to dissolve, drainage gradually resumes and the remaining tears subsequently decrease. Because of this, you should see the patient



The Herrick golf-tee silicone punctal plug, from Lacrimedics.



The Freeman silicone punctal plug, from Eagle Vision.

again in two weeks.

If a temporary plug eliminates most of the symptoms, consider going with a permanent silicone punctum plug. Two types of silicone plugs are available: the Freeman plug, available through Eagle Vision, which comes in five sizes (cost: $75 for two); and the Herrick, or golf-tee-shaped, plug, marketed by Lacrimedics ($68 for two).

Placing permanent plugs is different than placing temporary ones. Success with the Freeman plug depends on selecting the proper size. The right selection allows for proper placement and retention.

Place the Freeman plugs only in the inferior puncta because these are the channels through which most of the tears drain. Placement is sometimes difficult, requiring patience on your part. Also, the lids must be stable. You can stabilize the lids with two cotton-tipped applicators, or a modified forceps with two lid-stabilizing teflon tips. Freeman plugs have an advantage in that you can easily see them and monitor their position.

The Lacrimedics golf-tee silicone plug is placed in the cul-de-sac, similar to the collagen plug. Its flanged top impedes lacrimal drainage. The main advantage of the golf-tee plug is its ease of insertion. It is also less expensive than the Freeman plug. It does have one disadvantage: You cannot see it once the plug is pushed below the punctal opening. This makes it difficult to know whether the plug is in place or has been flushed through the lacrimal system.

If these methods prove inadequate, consider referral for permanent punctal occlusion surgery. These techniques, which alter the structure of the lacrimal drainage system, include: cyanoacrylate adhesives, argon laser punctoplasty, cryopexy and electrocautery. All of these procedures may allow spontaneous reopening of the puncta over time. Thus, repeat procedures may be necessary.

Stimulating tear production. A number of pharmaceutical agents are now being tested to spur tear production by direct stimulation of the lacrimal gland. None have gained FDA approval.

Bromhexine hydrochloride, usually used to treat asthma, has shown promising results in treating dry-eye patients. It prolongs tear breakup time and improves Schirmer test results, but has yet to receive FDA approval for ophthalmic use.

Finally, patient education is essential. Explain the underlying disease process. Point out that chronic disease has no cure. Advise patients about the environments that precipitate symptoms and the systemic factors that complicate the disease.

Patients should avoid certain medications that can trigger dry-eye symptoms, such as antihistamines, sympathomimetic agents, diuretics and atropine-like agents. Advise patients to obtain prompt medical attention for ocular infections, since diminished tear production hampers the ability to fight infection. Also instruct them to avoid HVAC vents and arid areas. Recommend that patients install a humidifier in their homes and work areas, and tell them to wear glasses to protect their eyes from wind.

CONCLUSION

No single ocular lubricant is a panacea for all cases of ocular surface disease. Choosing the right agent requires knowledge of both the condition at

hand and the ophthalmic preparations most likely to redress the problem. In addition, punctal occlusion may help preserve existing tears and alleviate dry eye symptoms. ■

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OSC TEST

Read and study the article. Then answer the following questions by circling the *one* correct answer on the Test Form enclosed. Note: Be sure to read all answers before choosing the one best answer. Test fee: $25.

1. Immunoglobin IgA, which helps prevent external infections, is produced by the:
a. glands of Zeis.
b. gland of Moll.
c. lacrimal gland.
d. Meibomian gland .
e. goblet cells.

2. The innermost mucin layer is produced by:
a. glands of Zeis.
b. gland of Moll.
c. lacrimal gland.
d. Meibomian gland.
e. goblet cells.

3. Among the stimuli that can cause tearing are:
a. noxious chemicals.
b. bright lights.
c. foreign bodies.
d. emotions.
e. all of the above.

4. The tear film is:
a. stable and consists of about 90 percent aqueous layer.
b. unstable and consists of about 90 percent aqueous layer.
c. stable and consists of about 50 percent mucin layer.
d. unstable and consists of about 90 percent mucin layer.
e. unstable and consists of equal parts of lipids, aqueous and mucin.

5. Lipid contamination of the tear film results in:
a. increased surface tension.
b. decreased surface tension.
c. reflex tearing.
d. psychological tearing.
e. none of the above.

6. The normal pH of the tears is:
a. 6.2—6.6.
b. 6.6—7.0.
c. 7.0—7.4.
d. 7.4—7.8.
e. 7.8—8.2.

7. The tonicity of the tears is approximately:
a. 250m Osm/L.
b. 300m Osm/L.
c. 350m Osm/L.
d. 400m Osm/L.
e. 500m Osm/L.

8. Hypotonicity of the tears causes:
a. corneal swelling.
b. cataracts.
c. increased IOP.
d. decreased IOP.
e. none of the above.

9. Which of the following conditions may cause lipid deficiency?
a. corneal dystrophy.
b. keratoconjunctivitis sicca.
c. anhydrotic ectodermal dysplasia.
d. goblet cell damage.
e. none of the above.

10. Most of the oxygen stored in the tear film is in which layer?
a. aqueous.
b. mucin.
c. lipid.
d. microvillae.
e. none of the above.

11. Aqueous deficiency tends to occur more frequently in:
a. older patients.
b. post-menopausal women.
c. patients taking thiazide diuretics.
d. patients with Sjogren's syndrome.
e. all of the above.

12. Post-herpetic neuralgia:
a. promotes tear-film stability.
b. destabilizes the tear film.
c. has no effect on the tear film.
d. destroys microvillae.
e. causes increased surface tension.

13. Typical symptoms of ocular surface disease include:
a. burning and itching.
b. foreign body sensation and redness.
c. pain and photophobia.
d. mucous strands and intolerance to wind and smoke.
e. all of the above.

14. The most useful test to diagnose ocular surface disease is:
a. tear breakup time.
b. Schirmer test.
c. Jones test.
d. Rose bengal staining.
e. collagen plugs.

15. If moderate ocular surface disease has been treated with lubcricants without success, you might next consider:
a. cyanoacrylate adhesives.
b. bromhexidine hydrochloride.
c. punctal plugs.
d. polyvinyl alcohol.
e. argon laser punctoplasty.

16. Temporary punctal occlusion is achieved by:
a. polyvinyl alcohol.
b. cyanoacrylate adhesives.
c. mucomimetics.
d. collagen plugs.
e. silicone plugs.

17. Surgical approaches to permanent punctal occlusion include:
a. cyanoacrylate adhesives.
b. argon laser punctoplasty.
c. cryopexy.
d. electrocautery.
e. all of the above.

18. A normal blink rate is:
a. every two to three seconds.
b. every five to 10 seconds.
c. every 15 to 20 seconds.
d. every 20 to 30 seconds.
e. every 30 to 40 seconds.

19. A normal tear breakup time is:
a. five to 10 seconds.
b. 10 to 15 seconds.
c. 15 to 20 seconds.
d. 20 to 25 seconds.
e. 25 to 30 seconds.

20. What are the two most useful methods of preserving existing tears?
a. swimmer's goggles and clear food wrap shields.
b. silicone rubber shields and moist chamber spectacles.
c. lid taping and bandage contact lenses.
d. bandage contact lenses and punctal occlusion
e. swimmer's goggles and lid taping.