A Summary of Blindness in Domestic Animals
Part II

Kenneth Fertig
HORSE
PERIODIC OPHTHALMIA
(MOON-BLINDNESS)

Periodic ophthalmia is a disease of the eye affecting horses, mules and asses only. Etiology of this disease is not known, although existing evidence points to a virus as the possible cause. It is characterized by a recurring inflammation which may effect one eye alone, or both eyes either simultaneously or alternately, or one eye until blindness occurs and then the other eye.

The onset of the attack is sudden, usually being noticed first in the morning, the animal having appeared normal the night before. The eyelids are swollen and tender and the eyes are kept closed. A watery discharge runs from the eyes. There is some rise of body temperature, accompanied by other evidences of a generalized disturbance. After a week or ten days the inflammation subsides and the eye, or eyes, either may be little affected or may be chalk white and completely blind. The interval between attacks may vary from a few weeks to several months and the number of attacks may vary from one to twenty, occurring over a period of years.

Blindness following early attacks is usually due to adhesions of the iris to the lens. If these adhesions break down before they are attached too firmly, the lens will usually clear. The cornea, or outside surface of the eye, is usually cloudy during an attack but almost always clears in a few days and usually remains clear even after the animal is totally blind. The fluid in the eye also presents a cloudy appearance during an attack. Following an attack, this fluid will sometimes slowly clear, often presenting a hazy appearance for weeks following. Frequently an agglutination or clumping of cells within the eye fluid takes place, which looks like sediment or strings floating around whenever disturbed by movement of the head.

EXTERNAL & INTERNAL OPHTHALMIA

The next possibility, after periodic ophthalmia, is either external ophthalmia (conjunctivitis) or internal ophthalmia (iritis, choroiditis, or retinitis). The etiology is similar for both of these conditions. The causes of internal ophthalmia are largely those of the external form only acting with greater intensity or on a more susceptible eye. Blows with whips, clubs and twigs; the presence of foreign bodies (hayseed, chaff, dust, sand, etc.); road dust often contains infective micro-organisms which enter via scratches in the conjunctiva; bright sun-
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[1] 100,000 Pen. 100 mg. Strep. 10%-merazine 10%-thiazole 5mg. Cobalt

[2] 300,000 Pen. 300 mg. Strep. 10%-merazine 10%-thiazole 5mg. Cobalt

[3] 500,000 Pen. 500 mg. Strep. 10%-merazine 10%-thiazole 5mg. Cobalt

The Following in Standard 7½Gm. Tubes

[5] 100,000 Pen. 100mg. Strep. [8] 300,00 Pen. 300mg. Strep. 10%-merazine 10%-thiazole
[6] 100,000 Pen. 100mg. Strep. 10%-merazine 10%-thiazole [9] 100,000 Pen. 100mg. Strep. 5000 Bacitracin
[10] And 25Gm. Tube with — 1,000,000 Pen., 1Gm. Strep., 500mg. Merazine, 500mg. Thiazol, 5mg. Cobalt

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light on snow or water; exposure to cold, rain, wet quarters and ammonia from their own urine are all possible causes of disease. Ophthalmia is a constant result of inflammations of the mucous membrane of the nose and throat, such as nasal catarrh, sore throat, influenza, strangles and nasal glanders.

Pathogenesis of external ophthalmia:
If the affection has resulted from a wound of the cornea or the localization of the disease organisms mentioned, the area becomes white and opaque. Often the blood vessels begin to extend from the sclera to the white spot and that portion of the cornea is rendered permanently opaque. If the wound is severe, ulceration may occur, leaving a break in the tissue which is filled in by opaque fibrous tissue. Pus may form and effectively paste the lids shut, preventing sight.

Pathogenesis of internal ophthalmia:
If the cause is from superficial injury, the pathogenesis is similar to what is given above. When the attack occurs from general causes or as the result of some distinct disease, the affection will be largely confined to the deeper structures. The aqueous humor is usually turbid and has numerous yellowish-white flakes floating in its substance or deposited in the lower part of the chamber. The iris is a brownish color. Both of these facts suggest that the vision of the horse at this stage would be very dim. The animal in this state is very highly photophobic and keeps its eyes closed in light. Occasionally, if the inflammation is severe, exudates overfill the globe of the eye and put excessive pressure on the retina, paralyzing it. Also direct exudation into the retinal cells leads to paralysis. When the retina is thus paralyzed, vision is heavily clouded or entirely lost. Pus may also form in the choroid or iris, escape into the cavity of the aqueous humor, and show as yellow-white stratum. In nearly all cases there is resulting degeneration of the lens and its capsule, constituting a cataract. Black cataracts are formed by an adhesion of the pigment. These diseases may or may not cause permanent blindness, depending on the severity of the infection and the natural resistance of the host and structures involved.

**EQUINE INFLUENZA (PINK EYE)**
This is an acute, highly contagious, general infectious disease of equines. The cause is a filtrable virus with which secondary infection is often associated. The period of incubation is from five to ten days. The virus is present in the blood and other body fluids of the sick. The virus is spread readily by the sick and objects with which they come in contact.

Pink eye is generally considered to be a form of influenza marked by edema of the subcutis of the limbs, ventral parts of the body and often the eyelids. The virus can get to the eye by the bloodstream or it may get into the conjunctival sac by direct contact. When in the eye, the lids may be closed by an edematous swelling. There may also be conjunctivitis, turbidity of the cornea and even lesions within the eyeball such as iritis and cataract.

**PURPURA HEMORRHAGICA**
Purpura is a noncontagious malady of equines occurring secondary to infections such as strangles, influenza, pneumonia and wound infection diseases. The essential cause is unknown, but it is believed that the unknown causative agent enters the body after the above conditions and thus gets into the blood stream. After it enters the blood stream it is believed to destroy the endothelial cells of the capillaries. The eyes may be the seat of severe edema and hemorrhage. The lids close entirely, while the membrana nictitans and conjunctival sclera are edematous and hemorrhagic. When the lids are forced open, reddish fluid or blood escapes.

**FOWL**

**FOWLPOX**
Fowl pox is an acute, virus disease of birds caused by Borrelia avium and is
characterized by wartlike nodules on the external surface of the head and by the formation of raised ulcers in the mouth.

The virus is contained within the nodules occurring on the comb, wattles, ear lobes and face of the affected birds. It is transmitted to healthy birds through breaks in the continuity of the skin of the head. At certain seasons and in certain localities this occurs by means of mosquitoes. At the site of infection the virus causes a hyperplasia of the epithelium; this is followed by degeneration and necrosis. Cells of the infected area become swollen and exhibit vacuoles causing the formation of small pustules which soon dry and become transformed into warty epithelial crusts. When the mucous membranes of the eye and infraorbital sinus are involved, blindness may result because of: (1) the proliferating epithelium mechanically obstructs vision, (2) the purulent exudate in the conjunctival sac may glue the eyelids together, (3) pressure on the eye from the lesions may cause it to rotate, or (4) the eye may become distorted in the orbit.

The disease runs its course in three to four weeks, after which time the pox heal, usually without scar formation.

**AVIAN LEUKOSIS (FOWL LEUKEMIA, RANGE PARALYSIS, LYMPHOMATOSIS)**

This is a complex viral disease occurring in several forms. Some of these forms affect the eye, others don't. Only those affecting the eye will be discussed. The etiologic agent is Trifur gallinarum.

The avian leukosis complex is divided into two general forms, lymphomatosis and leukosis, and each of these is further divided into several different forms.

Ocular lymphomatosis is the form that affects the eye. In this form the iris becomes discolored and the pupil distorted, resulting in blindness. The pathogenesis of the avian leukosis complex lacks much of having been completely determined. The manner in which the transmissible agent enters the body under natural conditions is not known. It is apparent that lymphomatosis spreads by contact and there is strong circumstantial evidence that it is transmitted through the egg. After a variable period of incubation, depending upon the stock and its genetic constitution, the lymphomatous form makes its appearance by symptoms which are traceable to abnormal, extravascular accumulations of lymphoid cells. These infiltrations can occur in any part of the body. When this happens in the eye, the optic nerve and the iris are frequently infiltrated with lymphoid cells. When the iris is involved, it becomes grayish white and the pupil small. The eyeball may bulge. This brings about partial or total blindness.

**GENERAL VITAMIN A DEFICIENCY**

This condition can affect any of the species of animals that become deficient in vitamin A but it is more apt to occur in the animals that normally receive food containing low amounts of vitamin A. Avitaminosis A is most apparent in poultry that is fed on a ration which is deficient in yellow corn and green feed for a period of two to three months, in young turkeys kept under dry range conditions, in hogs kept under dry range conditions, in hogs deprived of pasture and fed on grain alone and in range cattle pasturing on dry forage in the semiarid regions of the Southwest.

The vitamin does not occur as such in plant products but rather as its precursor, carotene. This compound is commonly spoken of as provitamin A because the body can transform it into the active vitamin. This is the way in which vitamin A needs of farm animals are met, for the most part, because their ration consists mainly or entirely of foods of plant origin.

Vitamin A is combined with a protein in visual purple and is essential to its production. This compound breaks down in the physiological process of sight as a result of a photo-chemical reaction. A deficiency of the vitamin results in night blindness, which is a symptom in all animals. The deficiency first manifests itself as a slowness of eye to adapt itself to darkness. There are various other eye
symptoms that vary markedly among species, some of which represent secondary infections. Xerophthalmia is characterized by a dry condition of the cornea and conjunctiva, cloudiness and ulceration. Copious lacrimation is a more prominent eye symptom in cows. In the case of chickens, the secretions of the tear glands dry up and an infection may then occur resulting in a discharge that causes the lids to stick together. Another condition that occurs, is the replacement of the normal epithelium by a stratified keratinizing epithelium, in various parts of the body. This also occurs in the eye.

Another way by which blindness may result is as follows: Vitamin A is concerned in the normal development of bone. A failure of the spinal and some other bones to develop normally results in pressure on the nerves and their degeneration. For example, a blindness in calves results from a constriction of the optic nerve caused by narrowing of the bone canal through which it passes.

LISTEROSIS

Listeriosis is a highly fatal, specific, infectious disease of sheep, goats, cattle and to a lesser degree of swine. It is caused by Listeria monocytogenes. The mode of entrance of the organism is not known; however, the respiratory and alimentary tracts are probable routes. The sheep nose bot, Oestrus ovis, has been incriminated as a possible vector of the disease.

While blindness is not seen in many of the cases of the disease, it can occur. It is due to the localization of the organism in either the optic nerve or some of the tracts leading to the center of sight, or to active lesions in the center of sight in the brain. The lesions are characterized by monocytic infiltration with localization. Sight is lost due to blockage of nerve impulses coming from the retinal rods and cones. When the lesion has been present for a period of time, the actual nerve tracts degenerate, the optic nerve degenerates and sight is irretrievably lost.

The involved animals circle and may run into fences if blind. The eyes appear dull when affected.

EYEWORMS

Eyeworms, while not common, are occasionally found in the eyes of domestic animals. Thelazia californiensis is the only species found in North America. This occurs in dog, cat, sheep, deer and man. The worms are whitish and about one-half inch long. They live underneath the eyelids and in the tear ducts. This worm is related to Manson’s eye worm found beneath the nictitating membrane and in tear sacs in poultry.

The mode of transmission is unknown. The worms move about actively over the surface of the eyeball, causing considerable inflammation and a profuse flow of tears. Scarification and ulceration of the eyeball may result from the activity of the worms and opacity of the cornea and blindness may be the final outcome. These symptoms are probably due to the mechanical irritation involved when the animal blinks or turns his eyes, or possibly it is due to the liberation of a mild type of toxin by the parasites.

RINDERPEST

Rinderpest is a highly contagious, general disease of cattle characterized by a rapidly fatal, febrile course, with inflammation and necrosis on the mucous membranes, especially those of the digestive tract. The cause is a filtrable virus, Tortor bovis, that apparently is in some way adherent either to the erythrocytes or the leucocytes.

The methods of infection are chiefly by contact with diseased animals or their products, such as flesh and hides. Infection occurs through the digestive tract. The young are most susceptible and from cattle it may be transmitted to sheep, goats, swine and wild ruminants, though these animals are less susceptible than cattle.

The virus enters the body through the digestive tract, passes to the blood in the usual way, propagates rapidly and possesses a selectivity for the mucous membranes in which the toxins injure the capillary endothelium. This causes an inflammatory process accompanied by early necrosis and liquefaction of the epithelial
cells which results in erosions and ulcers. Blindness results in the following manner: The virus is carried to the mucous membrane of the eye where it causes severe reddening. This is followed by a sero-mucous discharge from the eyes. This mucous may cause the eyelids to stick together and thus the animal can’t see.

**ARSENICAL & LEAD POISONING**

This can occur in any species of animal if they ingest the chemical. These chemicals are absorbed into the blood stream and they are carried to the nervous system. They affect the eye by causing the pupils to dilate, if these animals so affected get into bright light the retinal cells will become paralyzed and blindness will result.

The use of these chemicals in weed sprays, rodent poisons, insecticides and paints has increased the incidence of this condition in animals.

**MISCELLANEOUS CAUSES**

There are a number of things that may affect the eye directly and cause blindness. These are foreign bodies such as pieces of weeds, thorns, seeds and sticks; chemical irritants such as acids; scratches such as those from a barbed wire fence or from bites as occurs in dogs. These conditions may also allow secondary infections to enter the eye and produce blindness.

Blindness may also result from congenital defects such as absence of one or both eyes, failure of development of optic foramina as a result of vitamin A deficiency and improper development of any part of the eye of the fetus.

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   - Rinderpest ................ pp. 256-284.
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   - Diabetes Mellitus .... pp. 244-250.
   - Leukosis .............. pp. 118-126.
   - Pregnancy Disease .... pp. 664-665.
   - Rinderpest ........ pp. 637-638.
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   - Bovine Infectious Keratitis pp. 736-738.

*Antibiotic (continued from page 76) known to have tuberculosis before the revalent drug was in general use, and it is presumed that their original bacilli were drug-sensitive and they were superinfected with resistant bacilli. To them the future is drugless.

Finally, an important economic problem is presented in the dairy industry. It is known (31) that there may be serious interference with lactic acid fermentations if minute amounts of antibiotics are present in the milk. Since

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