

## **STRONGYLES**

The Strongyle group of equine parasites includes both large and small strongyles. They are the most important internal parasites of horses because they are the most common and because of the type of damage the immature stages do to the horse. The worms occur in horses of all ages and are commonly referred to as bloodworms.

### **Life Cycle**

Large strongyles produce one generation per year. Small strongyles complete their cycle every eight weeks. Sexually mature females in the large intestine pass unembryonated eggs in the feces. Under favorable environmental conditions, the infective or third-stage larvae develop from these eggs within a week. They are surrounded by sheaths that protect them from the outside environment.

This allows some larvae to survive over winter and endure drought. The third larval stage become quite active and migrate up and down blades of pasture grasses. This infective stage is swallowed while the horse grazes, eats off the ground or drinks water from a contaminated source. Since millions of eggs are laid, the likelihood for exposure to the infective larvae is greatly increased.

### **Pathology**

Ingested larvae migrate through the intestinal wall into the arteries, ending up mainly in the mesenteric artery as it branches from the aorta. The worm's irritation makes the arterial wall thicken, cutting the amount of blood flowing to the small intestine, colon and cecum. As the artery becomes more restricted, the blood pressure causes the wall to balloon and form a "verminous aneurysm." Thrombi or vegetative blood clots in the vessel are released and lodge within the arteries of the intestines, heart, kidney, liver and legs. Later these same larvae develop into adults. Adult large strongyles attach to the mucosa of the cecum and large colon and suck blood, causing inflammation and small bleeding ulcers in the intestinal lining.

The large strongyle, *S. vulgaris*, causes severe damage to the anterior mesenteric artery and its branches. This results in aneurysms, emboli and thrombi to body organs as described above.

The larvae of another large strongyle, *S. edentatus*, localize in the cecal or portal veins and cause perivascular thickening. The liver becomes swollen, looks bluish-red and has the white larvae embedded under its capsule. Prolonged infestation causes chronic fibrosis and may also lead to inflammation of the abdominal lining (peritonitis) and adhesions.

The third large strongyle, *S. equinus*, produces submucosal cysts in the liver, pancreas and intestine.

Small strongyles (cyathostomes) create less harmful reactions. Inflammation (catarrhal, hemorrhagic or fibrinous) of the ventral and dorsal colon may cause intestinal ulcers and possible perforation of the intestinal wall. A major difference between large and small strongyles, however, is the encysting or burrowing of the small strongyle larvae into the large intestine. This encysted stage remains for long periods of time and produces nodules in the deeper mucosal layers. Few species of small strongyles attach to the lining and suck blood.

### **Clinical Signs**

The clinical signs of a large strongyle (primarily *S. vulgaris*) infection are directly associated with the larval migration in the walls of the mesenteric arteries. The inflammation constricts the arteries and reduced blood supply (ischemia) to the intestinal wall kills tissue (infarction). "Verminous aneurysm" or "worm aneurysm" is classic of this parasite.

Acutely affected horses have fever, poor appetite, rapid weight loss, depression, lethargy, abdominal distress (colic) and death. The frequency of colics is directly related to the parasite control program. More colic in untreated young horses will mean more in the adults. Horses will have episodes of diarrhea and intermittent bouts of constipation. They may also have persistent diarrhea with hepatitis and phlebitis. Lameness from emboli in the legs may appear

with exercise and disappear with rest. The horse's general body condition is exemplified by unthriftiness, a dull or roughened hair coat and/or impaired performance even with adequate nutrition.

It will usually dramatically improve after proper anthelmintic treatment. When the mucosal ulcerations of encysted small strongyle larvae rupture, anemia may result.

The number of cases involving large strongyles has decreased with the use of effective dewormers. In contrast, there have been more small strongyle problems due to increased drug resistance. Larval cyathostomiasis (or small strongyle infection) is a distinct clinical syndrome that occurs with the mass emergence of encysted larvae from the intestinal wall. This release happens more in winter and spring. Small strongyles are less damaging than large strongyles since the larval migration is limited to the intestinal lining. However, the damage impairs normal gut motility, leading to signs of colic. It is unusual for a horse, particularly a younger animal, to be absolutely free of parasites. It is not uncommon for horses to have large numbers of small strongyles.

## **Diagnosis**

Bloodworms are diagnosed from fresh or refrigerated feces with a qualitative (what type) flotation method. A quantitative (how many) procedure is the Stoll or McMaster technique, which uses a counting chamber to total the eggs per gram of manure but doesn't differentiate the large bloodworm eggs from the small strongyle eggs. Take samples before giving any product and again two weeks after a treatment to monitor its effectiveness. With the six- to 11-month prepatent period (egg to mature adult) for large strongyles, infections will not be evident until late weanling or yearling age. Fecal exams do not detect the number of migrating large strongyles in body tissues. While requiring sophisticated and expensive equipment, ultrasound scanning and radiographic arteriography are newer diagnostic tools that complement rectal palpation in examining mesenteric arteries and medical symptoms.

## **Treatment**

Many anthelmintics are active against large and small strongyles. Several species of small strongyles have developed resistance to the benzimidazole group of chemicals. The combination of a benzimidazole (*e.g.*, fenbendazole) and piperazine overcome this resistance. Some deworming treatments are less effective because the encysted larvae remain protected within a barrier wall for up to two and a half years. Migrating larvae can be treated effectively with thiabendazole or fenbendazole daily for five days, two doses of oxfendazole on alternate days or ivermectin. Controlling the strongyle population is similar to that for other equine parasites. This includes sound pasture management, a regularly scheduled deworming program and routine fecal exams. The goal for any parasite control plan is to keep the number of adult, egg-bearing strongyles at low subclinical levels through the strategic use of selected, approved anthelmintics and programs that limit the horse's exposure to infected manure.

## **ROUNDWORMS**

### **Life Cycle**

The large roundworm of horses is *Parascaris equorum*. Its direct life cycle requires no intermediate host and takes 10 weeks to develop from egg to egg-producing adult. A foal ingests the sticky infective egg from a contaminated pasture, nursing surface of the udder, bedding or waterer. The larvae penetrate the intestinal wall and migrate to the liver through the portal vein. After one week in the liver, the bloodstream carries the larvae to the lungs where they move into the alveoli or air spaces. The larvae are coughed up and re-swallowed and mature in the small intestine. Eggs from the adults pass into the feces and, in a favorable environment, develop into the infective second stage within two weeks.

Roundworm eggs are resistant to their surroundings because of a thick, tough outer egg shell. This protective shell permits the eggs to remain in the soil as a source of reinfection for an extended time. The adult female parasite is prolific. An infected foal may pass an estimated 50 million eggs per day.

### **Clinical Signs**

Clinical signs from *Parascaris* are most common in young horses (sucklings and weanlings). Few adult horses have problems because they acquire immunity to the parasite after six months of age. Intestinal infections may show no clinical sign. However, accumulations of large numbers of immature and mature roundworms can decrease gut motility. This slower bowel can trigger an intussusception (the intestine telescoping onto itself) with any increased gut motility. Intestinal impactions from clustered roundworms may cause rupture of the intestine at the mesenteric attachment and produce inflammation of the abdomen (peritonitis). Typical symptoms include: unthriftiness, depression, a pot-bellied appearance, rough hair coat, loss of appetite, a "summer cold" with a dry cough and nasal discharge, enteritis, colic and even death.

### **Pathology**

Roundworm larvae migrate through the liver causing local hemorrhages that result in white tracts of scar tissue. The small intestine may be inflamed. Large numbers of adult roundworms can obstruct the intestine and possibly rupture the intestinal wall.

### **Diagnosis**

A positive diagnosis is made by a microscopic fecal exam, noting the thickened, brown-colored, roughened sticky-shelled eggs. These are first found at three months of age because of the 10- to 12-week development time. Adult roundworms are the size of a pencil (8-15 inches long). The immature adult round-worm can be confused with the pinworm. It has a blunt, tapered tail instead of the sharp, pointed one of the pinworm.

### **Treatment**

Roundworms are susceptible to every class of anthelmintic except phenothiazine. Some veterinarians recommend dosing mares two weeks before foaling, sucklings six to eight weeks old and weanlings every two months for the first year of age. Such treatment does not remove the immature migrating stages in the lungs and liver. Heavily parasitized young horses should not be treated with quick-acting drugs that cause massive deterioration of worms that may block the intestines. Toxemia may also occur. A reduced dose of piperazine or benzimidazole product is suggested with full retreatment in 7-14 days.

### **Control**

Sanitation and good management offer the best control of roundworms. Disinfection of the environment, particularly waterers and feeders, decreases the horse's exposure to any infective larvae. Washing the udders of mares that are exposed to infected surroundings may be valuable. Foals receive their parasite load from a previous year's contamination, so continued use of the same barn for foaling and housing a variety of horses in the same barn promote parasite problems. Egg survival in the outside environment and prolific females increase a horse's exposure to roundworms in a pasture. Coprophagia (*i.e.*, eating manure) should not transmit an ascarid infection unless the soil has been infested previously and the mare is a roundworm carrier. The rate of egg development correlates directly with the temperature. The highest number of embryonated eggs will be present during warmer weather, which usually also coincides with the highest number of horses on pasture.

Fecals done two to three times annually or 14 days after treatment will provide a good evaluation of deworming product(s). Roundworms have not become resistant to anthelmintics.

### **BOTS**

There are two adult bot flies: *Gasterophilus intestinalis*, or the common bot, and *Gasterophilus nasalis*, or the throat bot. The larval stage of the bot or nit fly is found in the horse's stomach. The eggs are easily seen attached to the hairs of the face, neck and forelegs.

## **Life Cycle**

The parasite completes one generation each year. The life cycle is indirect, so an intermediate step is required before reaching maturity. After the adult flies copulate, the pregnant female deposits her eggs on the hairs of the forelegs and chest area (*G. intestinalis*) and between the lower jaws (*G. nasalis*). This fly activity coincides with warm months (May to October). Bot fly activity peaks in early spring and late fall. The eggs hatch within a week. It is thought that the larvae need the external stimulation of the warm, moist lips of the horse to emerge. However, *G. nasalis* eggs spontaneously hatch in a week after being deposited.

Larvae may remain viable in their egg cases for a long time in the fall and winter months after fly activity has ceased. The hatched larvae enter the mouth when the horse chews, bites or licks its legs, or by migration from the jaw area. They spend about a month in a growth and development stage (instars), burrowing into the tongue and gums. They emerge, are swallowed and migrate to the stomach where they accumulate and attach themselves to the stomach lining. The bot larvae grow to 16-20 mm (3/4 inch) long during the nine to 10 months of overwintering. The bots detach themselves and pass out in fecal material. They may also attach to the rectum a few days before exit. This spontaneous discharge of larvae begins in late spring and continues into the summer months, thus lengthening the period for fly activity. The bots burrow into loose soil or dry manure to pupate and emerge as adults in three weeks.

## **Pathology**

Bot larvae are the stage that cause damage in the horse. These cigar-shaped bots make pits in the stomach wall that can perforate at their point of attachment. Complete penetration results in peritonitis. The pits begin to heal after removal by a boticidal deworming. Rupture of the stomach is possible with heavy infections. The larvae also irritate the gum and tongue by burrowing in the oral cavity.

## **Clinical Signs**

The major clinical sign is the horse's annoyance and nervous frustration from the adult fly. The female depositing her eggs may frighten some timid horses to the point of bodily injury. The bot fly, however, is incapable of biting or stinging. The tell-tale yellowish egg cases are easily seen attached to hair of the chest, legs, neck, throat and around the mouth in the summer and fall. The burrowing first- and second-stage larvae (*G. intestinalis*) produce mouth and tongue ulcers during the summer and fall. These sores may impair feeding and lead to poor mastication, unthriftiness and/or retarded growth. Digestive upsets (*i.e.*, colic or obstruction) are due to the continued irritation of the stomach wall and bowel by the attached bots. The larvae impair normal gastric function.

## **Diagnosis**

Bots are diagnosed primarily by the appearance of the characteristic egg cases on the hair shaft. The larvae are usually not seen in a fecal examination.

## **Treatment**

Treatment involves different classes of anthelmintics than used for other groups of parasites. Organophosphates (trichlorfon and dichlorvos) and ivermectin are effective for both the oral and gastric stages.

## **Control**

Control of this parasite comes from proper grooming techniques, such as clipping or scraping egg cases from the hair. Warm water rinses can be sponged on the eggs to stimulate an early, artificial hatching of the eggs. Use caution during bathing to avoid possible human infection from the emerging larvae. Use a boticide dewormer a month after the first killing frost in the fall and at mid- summer for instars.

## **PINWORMS**

There are two equine pinworms, *Oxyuris equi* (common pinworm) and *Probstmayria vivipara* (the minute pinworm). Immature pinworms occur in horses of all ages but are most common in younger stock (*i.e.*, weanling, yearling, adolescent).

### **Clinical Signs**

The anus of the horse becomes inflamed from the egg deposits and their gelatinous substances which produce the intense itching (or pruritis). The animal is restless and loses body condition because of the constant irritation, and the hair loss on the tailhead detracts from their normal appearance and behavior. The horses stand and rub their rear quarters and tail.

### **Life Cycle**

The direct life cycle begins with the horse ingesting the infective eggs found in feed or water contaminated with manure. Horses may also ingest the eggs while licking or chewing on infested fences and walls. The eggs hatch in the horse's intestines, where the larvae mature into adults. The adult pinworms develop in the large colon over five months but do not penetrate or migrate into the intestinal tissue. The female pinworms, loaded with eggs, move to the rectum and anus. There, they rupture and deposit their sticky eggs on the perianal region. These eggs become the infective-stage larvae within three to five days.

### **Pathology**

Pathological lesions are produced by the pinworm's contact and irritation to the tissue. The worms feed on the mucosa of the colon, causing surface erosions. The eggs near the rectum prompt an intense itch and subsequent loss of tail hair.

### **Diagnosis**

The best method to diagnose pinworms is the direct microscopic exam, since eggs are not commonly found in a routine fecal flotation. An impression of the perianal area is made with transparent, adhesive tape. Close inspection of the anal region may reveal the cream-colored gelatinous masses of attached eggs. Female worms are voided spontaneously and may be seen in fresh feces. They are grey-white with a sharp-pointed tail. The female *P. vivipara* is comma-shaped with a pointed tail as seen with the microscope. Infections of these minute pinworms are ordinarily diagnosed in postmortem (autopsy) examination.

### **Treatment**

Routine deworming programs used for roundworms and bloodworms also control pinworms. Strategic deworming schedules or monthly therapy during the times of heavy exposure lower the parasite count in the manure. A lower number of infective larvae means less tail rubbing on fences and stall walls. The perianal area can also be bathed to help remove any attached eggs.

## **TAPEWORMS**

Tapeworms of the horse include *Anoplocephala perfoliata* and *Anoplocephala magna* and *Para-anoplocephala mamillana*. They occur in horses of all ages.

### **Life Cycle**

The life cycle requires free-living pasture insects known as oribatid mites. In the horse, segments near the end of the tapeworm's body detach and are passed out with the feces. These segments dry and burst open to release the eggs. The oribatid mites ingest the eggs and serve as hosts for the developing tapeworm until cysticercoids mature within two to four months. The horse eats the infected mites while grazing the pasture or hay. The parasite reaches maturity only

after months inside the horse. Therefore, tapeworms don't finish growing until the infected foal becomes a weanling, yearling or much older adult. The mite population lives in low-lying or wet pastures. They are more common in areas that have been grazed for many years. Wet weather and overgrazing increase the potential for transmission.

### **Pathology**

The pathology for tapeworms is primarily confined to the cecum and adjacent colon. The tapeworms form clusters that attach around the ileocecal valve or produce a catarrhal enteritis of the distal portion of the small intestine.

### **Clinical Signs**

In general, affected horses have a record of poor growth and unthriftiness. Digestive disturbances are often obscure because tapeworms cause only local ulceration of the cecal mucosa. This erosion can perforate, though, and lead to peritonitis in severe cases. With persistent infestation, the intestinal wall of the ileum thickens to produce a tough, membrane-like obstruction. Several consequences may occur such as occlusion of the ileocecal valve, rupture of the cecum and intussusception of the ileum and cecum. Most horses, however, show no clinical signs of a tapeworm infection.

### **Diagnosis**

Tapeworms can be diagnosed by fecal flotation or sedimentation exam. The egg is angular-shaped with a hyaline-thickened wall. The embryo has six hooklets. Since the release rate into manure is inconsistent, take fecal samples over several days. The proglottids, or portions of the tapeworm, are rarely seen in a fecal examination because the worm's integrity is lost in passage through the intestinal tract.

### **Treatment**

No specific product is labeled for equine tapeworms. Some effective drugs include niclosamide, praziquantel, fenbendazole, mebendazole or a double dosage of pyrantel pamoate. Ivermectin does not control tapeworms, so caution should be advised for programs that use only this product. Infestation is controlled by deworming before the heavy, spring grazing season and again in late fall.

## **THREADWORMS**

*Strongyloides westeri* is a hair-like parasite 8-9 mm (1/3 inch) long. Most common among foals, the threadworm is the first nematode that can appear as eggs in the feces, as early as two weeks of age.

### **Life Cycle**

Threadworms have a unique method of transmission. These larvae persist in the body tissue of the mare for years and are thus called somatic larvae. Larvae pass into the foal through the mare's milk between four and 44 days postpartum. Therefore, the mare becomes a persistent, perennial source for contamination to each foal crop. The mare does not, however, carry an open infection that actively sheds eggs. Ingested larvae stimulate an immune response in the suckling foal. This self-cure begins by six months of age.

Eggs shed by the foal become infective-stage larvae under favorable environmental conditions. The larvae can also penetrate the skin or be ingested during grazing. They migrate to the lungs, up the trachea and are swallowed. Once reaching the small intestine, they continue to mature until the females begin laying eggs. The parasitic form of *Strongyloides* is the female adult. Free-living male and female threadworms can sexually reproduce outside the host. The infective-stage larvae do not survive outside the horse over the winter.

## Pathology

The lesions of pathology result from erosions or sloughing of the intestinal mucosa. Any migrating larvae may also cause hemorrhage in the lungs.

## Clinical Signs

The primary symptom is diarrhea of young foals with subsequent weight loss, poor appetite and anemia. The loose stool may occur simultaneously with foal heat scours, and, if so, the outbreak is strictly coincidental. (Note: fecal egg counts can be high or low during the diarrhea). Any respiratory distress results from larval damage. Skin irritation from the penetrating infective-stage larvae produces a dermatitis.

## Diagnosis

Threadworms are diagnosed by finding eggs on fecal flotation. The typical oval-shaped, thin-walled, embryonated egg is smaller and lighter in color than the strongyle egg. Within the egg, the larvae may have a crease line and be weakly motile. Fresh or refrigerated fecal samples must be submitted since eggs hatch within a few hours. The larvae are not easily floated, so a direct fecal smear is suggested to see the eggs. If strongyle eggs are found in the young foal, they come from the foal's eating its mare's feces (coprophagia) and are just passing through the foal's intestines. When strongyle and threadworm eggs are found in a two- to three-month-old suckling, do not rule out a concurrent infection. Other causes for the clinical diarrhea include viral, bacterial, nutritional or idiopathic reasons. *S. westeri* produces a non-fetid, normal, green-color stool with no febrile response. Appropriate medication for parasitism gives a prompt remission.

## Treatment

Treatment with anthelmintics includes a proper rotational deworming program that lowers contamination. Thiabendazole, oxbendazole and ivermectin are effective, but other benzimidazoles require higher than normal dosages. A single dose of ivermectin to the mare has been suggested on the day she gives birth or within 12 hours of delivery. For threadworms and roundworms, combine oxbendazole or thiabendazole with piperazine. For threadworms and resistant strongyles, select oxbendazole or thiabendazole plus piperazine or just ivermectin. If the foal's diarrhea is caused by *S. westeri*, begin treatments at two weeks of age and continue every two weeks in more serious cases. Foals with a prior history of threadworms are routinely given a prophylactic treatment. It's hard to eradicate the mare's somatic larvae -- no exact regimen has been studied.

## Control

Threadworms are controlled with basic sanitation and management. Proper removal of manure and a good mare/foal deworming program will minimize annual infestation.

## ONCHOCERCA

Cutaneous onchocerciasis is prevalent in several geographic regions and has a worldwide distribution in horses. *Onchocerca cervicalis* (neck threadworm) is found in the ligamentum nuchae of the horse. *Onchocerca reticulata* (ligament worm) is in the ligamentum nuchae and the flexor tendons and suspensory ligaments of the lower front legs. It's hard to differentiate between the two species.

## Life Cycle

The life cycle of *Onchocerca* requires as an intermediate host, the biting midge (*Culicoides spp.*). This insect has such intense feeding habits that just its biting causes an allergic dermatitis. The adult *Onchocerca* female in the ligamentum nuchae produce microfilariae. These offspring also migrate through the superficial layers of the skin. The number of microfilariae and their distribution in the skin vary with the season of the year. The microfilariae are ingested as the *Culicoides* feed on the horse. Within two to three weeks the infective larvae reach the insect's

mouthparts and can be transmitted to the horse during the midge's feeding. The larvae are deposited primarily in the ventral abdomen near the umbilicus. They are also found in the pectoral (girth) area, eyelids, withers and the skin inside the upper thigh.

## **Clinical Signs**

Clinical signs are usually seen in horses older than five years. Symptoms appear during both the fly season (summer) and winter. The disease can be debilitating and restrict a horse's freedom of movement. Skin lesions appear on the head, neck, withers and ventral midline of the abdomen. The microfilariae stimulate a mild diffuse dermatitis that produces alopecia or thinning hair, small patches of dry scaly skin, small papules and pruritis. Itching intensifies and then becomes chronic. Large abrasions, excoriation (skin loss) and ulceration result in depigmentation and scarring. Hair regrowth is scanty and either darker or lighter than normal color.

Microfilariae may infiltrate the eye and irritate the cornea and iris, causing inflammation of the cornea, eyelids and eyeball and possible blindness. White nodules (less than 1 mm) are seen in the pigmented conjunctiva near the corner of the eye.

The ligamentum nuchae becomes swollen and painful from the infiltration of the worm. However, the individual adult onchocerca is hard to isolate. Lesions gradually develop that thicken the ligament with nodules. These cavities enlarge with caseous debris and necrotic worms. The dead worms calcify and form focal areas of mineralization. Hyaline degeneration discolors the nuchal ligament.

Swelling of the flexor tendons and suspensory ligaments occurs in the front fetlocks. This edema produces lameness. The legs may remain enlarged with nodules of palpable parasites. The symptoms subside within three to four weeks.

## **Pathology**

The pathological lesion is inflammation of both perivascular tissue and the subcutaneous layer. The hypersensitivity to the microfilariae may be acute or chronic. There is no correlation between the number of parasites in the horse and the outward signs of the disease. Some horses harbor *Onchocerca spp.* but show no illness. Anthelmintic treatment may temporarily increase the severity of a systemic reaction to the killed microfilariae.

## **Diagnosis**

The diagnosis for cutaneous onchocerciasis is the demonstration of a microfilarial dermatitis. Strict isolation of the organism is difficult. A positive conclusion of onchocerciasis is implied with a matching history and symptoms that regress each fall and return the next summer. It can be confirmed with a deep skin biopsy or skin scraping that contains the microfilariae. Failure to find the parasite suggests other possibilities. The biopsy punch or snip of skin (1-2cm) is mixed with saline and allowed to incubate at room temperature for five to 24 hours before examination with the microscope. Any skin scraping should include tissue fluid.

Both *Onchocerca* parasites are long and coiled so identification is based on microscopic length. *O. cervicalis* is 207-240 microns (the male 6-7 cm and the female up to 30 cm). *O. reticulata* is 330-370 microns, and its microfilariae are 0.25 mm long. In most cases, the final diagnosis of onchocerciasis is based upon the horse's response to therapy.

Other disease problems that show similar skin conditions include: (1) dhobie itch, (2) Queensland itch, (3) sweet itch, (4) summer itch, (5) kasen or (6) a simultaneous *Culicoides* sensitivity. The biting midge produces a more intense itching over the rump and tailhead, causing a "rat-tail" appearance.

## **Treatment**

Three drugs are available to treat *Onchocerca spp.* Since the parasite's life span may be five years, therapy must be continued for adequate control. Ivermectin at its regular dosage results in spontaneous resolution within days. Microfilariae disappear and allow clinical improvement of the dermatitis. Initially, however, routine doses can cause

adverse reactions, primarily edema to the lower abdomen or pruritis from the killed filariae. There may also be limb and eyelid edema and a fever. These side effects can be reduced with steroid administration. A relapse in the disease is possible two to nine months after treatment because of reinfestation with migrating microfilariae. Little therapeutic activity has been reported against the adult *Onchocerca spp.* found in the ligamentum nuchae.

Diethylcarbamazine (DEC) is a safe drug for onchocerciasis in horses. Give a daily dose of 1 mg/kg for 21 days. Again, dying microfilariae produce a pruritis that requires corticosteroid treatment. If eye lesions are present, use steroids during the first few days of DEC to help reduce any irritation. This medication has been used for the entire summer in heavy infested areas. It is not 100 percent effective, and relapses do occur.

Levamisole may be placed as a top-dressing on grain when combined with more palatable syrups or peanut butter. At 10 mg/kg for seven days, the anthelmintic can produce some side effects, so a steroid supplement is advisable.

To best control the parasite, give a regular ivermectin treatment two to four times annually. Since biting midges and face flies can complicate and delay recovery, keep stables insect-free in the late afternoon during warmer weather. Screens, insecticides and residual sprays are recommended. Surgical removal of infected skin nodules is an alternative.

## **STOMACH WORMS**

Stomach worms are classified as two kinds of nematodes. The first category, Spiruroids, are *Draschia megastoma*, *Habronema muscae* and *Habronema majus*. They occur in horses of all ages and have a seasonal prevalence directly related to house and stable flies. These insects act as the intermediate hosts in the stomach worm life cycle. The second category is *Trichostrongylus axei*, or the minute stomach worm. This parasite is more common among sheep and cattle but can also infect horses. Its occurrence varies widely, depending on the close association of horses and ruminants. If the ruminant population is heavily infested, there is a chance for the same infection rate in horses.

### **Life Cycle**

Stomach worm larvae are ingested by the house or stable fly maggots that thrive in manure. This immature stage develops inside the maggots, which become adult flies in just three weeks, posing a problem since it could lead to quick reinfection. The flies deposit the infectious larvae in unusual places such as skin lesions or sites with natural body fluids. If licked or swallowed by the horse, the larvae become adults in the stomach. External larvae irritate the skin enough to produce the familiar granulomatous lesion or "summer sore."

### **Pathology**

The pathology of stomach worms is limited to the inflammation and thickening of the stomach wall and skin ulcerations. Any stomach reaction is usually found only on postmortem examination. Skin wounds, if left untreated, become infected and do not heal.

### **Clinical Signs**

Clinical signs mimic their name by causing inflammation of the gastric mucosa (gastritis) and digestive disturbances. Irritation to the stomach lining prompts colic and diarrhea. In particular, *Draschia* forms a tumor-like lesion along the margo plicatus in the glandular portion of the stomach. This round nodule has two small central openings and is filled with a mass of worms living in necrotic debris. The horse may display no illness unless the gastritis perforates, leading to peritonitis. Sometimes, fistulous tracts develop from the stomach to the spleen.

*H. muscae* live in the mucus secretions of the glandular stomach. Its irritation increases gastric secretions that may or may not include hemorrhage and ulceration. Externally, "summer sores" from *Habronema* are the reaction of fly strikes on the skin. The house or stable flies attack skin lacerations or abrasions, depositing larvae on the lower limbs, ventral abdomen and shoulders. These larvae then sensitize the skin and produce pruritis. If the wounds do not heal, they become chronic, proliferative, granulomatous masses that bleed easily. The horse's eyes, lips, nose and male

external genitalia are targets for flies because of the fly's attraction to moisture. Males have increased frequency and difficulty with urination when lesions occur on the penis. Excessive tearing, conjunctivitis and inflamed eyelids or nictitating membranes are common.

*T. axei* infections often go unnoticed unless their rapid life cycle produces quick, heavy accumulations of parasites. An acute, catarrhal inflammation of the gastric mucosa sometimes extends into the duodenum. With time in chronic cases, the stomach lining thickens and looks uneven, bumpy and mulberry-like. Diarrhea results. Historically, the typical sign is sudden weight loss or poor condition in the horse that is grazing with ruminants.

### **Diagnosis**

Typically, infection by spiruid parasites go undetected in fecal flotation because the eggs are thin-walled and collapse during dilution. Minute stomach worms, however, can be found on fecal flotation since their eggs look like strongyle eggs. A biopsy and scraping of skin lesions help identify larvae.

### **Treatment**

Stomach worms are not isolated for treatment by a specific anthelmintic. Routine deworming products such as the organophosphates, benzimidazoles or ivermectin eliminate these and other parasites. Local treatments for "summer sores" include surgery, electrocautery or cryosurgery, caustics or astringents, and topical trichlorfon or fenthion. Repellents control the flies. Composting manure and other pasture management techniques decrease exposure. Rotational grazing with ruminants may need to be stopped if *T. axei* infection becomes a problem.

### **EYE WORMS**

*Thelazia lacrymalis*, another spiruid nematode, is related to the stomach worm. The eyeworm is found primarily in young horses less than eight years old, particularly in yearlings to three-year-olds.

### **Life Cycle**

Diagnosed predominantly during postmortem, the eyeworm uses the face fly (*Musca autumnalis*) as its intermediate host. The eyeworm larvae are picked up by the fly and continue to mature for two to four weeks. The parasite migrates to the fly's mouthparts and then into the horse's eye as the face fly feeds. The eyeworm may go undetected until later that year since the life cycle takes three months to complete. The viviparous female sheds larvae (not eggs) into the tear secretions to start another cycle.

### **Pathology**

Any pathological lesions are confined to the reaction to these larvae. Cellular infiltration of the lacrimal gland and fibroplasia of the tear ducts are the main lesions.

### **Clinical Signs**

Clinical signs are typically not seen. Besides the common annoyance of the face fly, a conjunctivitis results from the constant irritation to the eyes. This inflammation may be compounded by a secondary bacterial infection that causes corneal vascularization or opacity (blindness). Bleeding into the anterior chamber of the eye also occurs.

### **Diagnosis**

No clinical test is available for a diagnosis. Observing the worm in the eye or identifying the larvae in the tears is the best method. Eyeworms are not routinely seen on clinical exam because the parasite invades the ducts of the lacrimal gland. A local anesthetic facilitates their release. Differential diagnosis includes the stomach worm (*Habronema*) or

*Onchocerca* larvae. The eyeworm may even penetrate the eyeball structures, causing small, raised white nodules in the pigmented and nonpigmented conjunctiva near the outside corner.

## Treatment

One method of treatment is mechanical removal with a local anesthetic. Also an ophthalmic solution of 50-75 cc of 0.5% iodine and 0.75% potassium iodide can be irrigated in the eye. Ointments such as 0.03% echothiophate (Phospholine<sup>R</sup>, Ayerst) or 0.02% isofluorophate (Floropryl<sup>R</sup>, Merck) are used. Larvacidal doses of fenbendazole (10 mg/kg for five days) or oxifendazole (10 mg/kg, twice at a two-day interval) have been suggested.

## Control

Control of the face fly through improved sanitation helps decrease the breeding grounds for flies. Regular manure removal, composting away from the barn and insecticidal sprays help. Horses may also need stall or face screening and insecticide application to protect them from the continual annoyance of the fly.

## Pasture Management

Few horses escape exposure to internal parasites. Favorable temperatures and moisture help the eggs in manure hatch and develop into infective-stage larvae. As a horse grazes, it eats these immature adults that attach to the blades of grass. The horse's own feces maintains the supply of worms for itself and other horses in the same pasture. Several techniques are used on many horse farms to lower the amount of fecal contamination and reduce the likelihood of exposure to the horse.

1. Rotate the confined area of grazing often. Move horses kept in small areas from one pasture to another field every month to maintain adequate grass cover. Electric fencing can subdivide pastures into smaller acreages. Thus the horse eats grass that does not contain excess larvae. Since some eggs mature in six weeks, their life cycle is broken when the host is removed from the contaminated premise.
2. Graze younger animals separately from older horses. Certain parasites survive primarily in horses less than two years old. The increased volume and variety of parasites associated with young horses simply expose adults to more worms. Weanlings and yearlings develop their own immunity against *Strongyloides* and *Parascaris*.
3. Don't overgraze and overstock pastures, which forces horses to eat any remaining weeds, dirt, parasites and wood. The manure piles provide incubators for hatching eggs and storage for potential migrating larvae. The parasites attach to water droplets on weeds or wooden posts. Overgrazed pastures mean that the few living plants left in the field are heavily contaminated with larvae.
4. Groom pastures to grow a better blend of grasses. If composting is impossible, spread or thin manure on ungrazed land to provide fertilizer and expose parasite eggs to sunlight and other elements which kill them. A chain drag or harrow pulled through the pasture breaks open fecal piles and removes thatch buildup. This process also aerates the soil and levels rough areas. Some soil types allow rolling to smooth loose ground.
5. Mow often to control weeds and mechanically disperse manure mounds. Regular cutting keeps the young grass thriving and older plants at a shorter height that is more palatable for grazing. This also reduces fescue toxicity and growth of noxious weeds. Grass maintained at four to six inches keeps seed heads from growing. Mature fescue contains the fungus that produces reproductive problems in mares. Lush pastures are more likely to maintain adequate nutrition for horses if groomed periodically.
6. Use tractor attachments that vacuum manure from a pasture or paddock. The implement picks up feces and eggs by "sweeping" over the ground. This equipment is more practical for intensive housing on limited grazing areas. It also helps on farms in warmer climates by assisting in fly control.
7. Cattle can clean pastures, too. They have different eating and digestive patterns. Cattle eat the taller grasses while horses like the young, shorter growth. The cattle tongue acts as a prehensile tool to grab and pull. Horses nibble closer to the ground with their front incisor teeth. This preference for different types of grass helps remove those tufts typically seen in horse pastures. Most internal parasites (except *T. axei*) of horses cannot survive in cattle's digestive systems. The larvae die and, therefore, reduce the parasite load the horse

gets from the same field. Cattle and sheep follow the horses during pasture rotation or are left with the horses. Besides the benefit for parasite control, it makes the most economical use of available pasture.

8. Soil test to find the present conditions and the fertilizers needed for good pasture. The best time to fertilize is the rest period between horse rotations. Herbicides can be applied at the same time.
9. Don't overlook stall sanitation. A horse's surroundings, inside or outside, are potential sites for ingesting parasites. If a horse is confined to a stall, its manure is just as alive with hatching eggs as those found in a field. Daily picking and weekly stripping of bedding is mandatory to decrease contamination. Spray disinfectants on walls and floors, scrub buckets and spread lime over the ground to "sweeten" the ammonia odor and adjust the soil pH. The stall flooring should be layered and adequate ventilation provided to handle urine and excess moisture.
10. Compost for 2-4 weeks the solid waste cleaned from stalls. The heat generated by the compost kills parasite eggs and some larvae. Dump the bedding several yards away from the barn to discourage flies. Weeks later, spread the material on ungrazed pastures or use as fertilizer for bare ground. Avoid the tendency not to allow enough time for proper decay. Spreading the contents immediately after stripping the bedding simply moves the manure and parasites from the stall to the pasture.
11. Don't feed horses on the ground. The primary source for internal parasites is the ground. The less a horse is exposed to contaminated areas, the less likely it is to consume worms. Keep hay, grain or salt blocks in feed bunks, racks or buckets.
12. Filth attracts flies. If compost piles are close to the barn, the stalled horses are easily bitten. Stalls left dirty for a long time lure insects, and controlling insects is also important for disease transmission (EIA, Sleeping Sickness), skin conditions and annoyance. Use sprays, wipes and biological insecticides to decrease or kill their population.
13. Keep clean water available at all times and remove any standing water. Scrub water buckets daily. Empty water tanks and brush the algae from the sides. Horses that defecate in their waterers require extra attention. Insects use stagnant water as a breeding ground.

### Summary

The proper selection of a product for treatment is based on:

1. Previous history of a product's effectiveness.
2. Fecal examinations.
3. How much the horse is exposed to parasite larvae in the pasture.
4. External or clinical signs of parasitism: *e.g.*, pot-bellied, poor hair coat.
5. Consultation with the local veterinarian.

The decision to deworm should be based on research that has proven which anthelmintics are most effective and during which months they are indicated. Geographical locations is also important. *Table 1* lists, by name and company, available products and the internal parasite for which each is recommended. The chart indicates the different chemical classes of drugs, which assists in understanding the idea behind rotational deworming programs. Once the correct diagnosis has been made, the best product and management can be selected to treat the problem.