moult. The parasites then migrate into the lumen of the bronchi to be coughed up and swallowed. They pass into the intestinal tract and become adults in 8 to 9 weeks.

Not all of the larvae coming into the lungs may be stopped in the pulmonary capillaries. A few may slip through the lungs into the general circulation and be carried to such organs as the spleen, kidney, or the pregnant uterus. Upon reaching the uterus, the larvae can pass from the capillaries of the maternal placenta to the capillaries of the fetal placenta, and be carried to the embryonic lungs. The larvae will then migrate as in the adult finally lodging in the intestinal tract of the fetus, there to mature and lay eggs.

In an extended survey conducted by Dr. M. T. Sloss at the Stange Memorial Clinic, some interesting figures have been compiled. Of the 67 parasitisms in pups under 4 months of age seen in the course of 1 year (1938), 51 were infected with either *Toxocara canis* or *Toxascaris leonina*. The distribution of these 51 infections is shown in the following table.

| **Toxocara canis** | | **Toxascaris leonina** | |
|---|---|---|---|
| Age | cases | Age | cases |
| 4 weeks | 1 | 4 weeks | 1 |
| 5 weeks | 14 | 8 weeks | 4 |
| 6 weeks | 1 | 12 weeks | 14 |
| 7 weeks | 4 | 16 weeks | 9 |
| 9 weeks | 1 | | — |
| 10 weeks | 2 | | 28 |
| | — | | |
| | 23 | | |

Prenatal infection has definitely been proved in the case of *T. leonina*, but has not been so shown for *T. canis*. However, it is theorized that the 8 or 9 weeks required for the ascarid larvae to mature in the adult intestine are also needed for them to develop in puppies. Assuming this to be true, when an ascarid infection is found in any pup under 2 months of age, it should be assumed that the animal was infected prenatally.

The only way to prevent this prenatal infection is to worm the bitch before or soon after mating, and keep her freed of parasites throughout the gestation period.

—*J. M. Gooch, '44*

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**5** **Clostridium Welchii in Mastitis.** In the practice of Dr. B. B. Palmer, I.S.C. '11, of Wayzata, Minn., a few sporadic cases of an unusual mastitis occurred which had not previously been amenable to treatment. Death occurred soon after the onset of symptoms. On Nov. 7, 1943, another of these cases was brought to the attention of Dr. Palmer.

The patient, a 7-year-old purebred Guernsey cow, had been fresh 6 weeks. The evening before the veterinarian was called, she gave 17 lbs. of milk and appeared quite normal. The next morning one quarter of the udder was hard and swollen; the remaining quarters gave 6 lbs. of milk. The animal was seen by the veterinarian at 8 a.m. at which time the temperature was 103° F., the appetite small and no fluid could be obtained from the affected quarter. A diagnosis of mastitis was made. Treatment at this time consisted of 900 gr. of sulfanilamide per orum. At 6 p.m. the cow was again observed. An extensive swelling had begun to form on the underline just anterior to the udder. A fulminating mastitis was present; the prognosis was unfavorable. A sample for bacteriological examination was obtained from the affected quarter and sent to a laboratory.

The next morning, crepitation of the skin over the entire abdomen revealed the presence of gas in the subcutis. The milk flow had diminished to 4 lbs. per milking from the 3 quarters remaining functional. The bacteriology laboratory reported finding a pure culture of *Clostridium welchii*. Treatment was then changed to sulfathiazole sodium sesquihydrate, 25 Gm., administered intravenously in 500 cc. of sterile water. Sulfathiazole, 300 gr., was given per orum that morning and repeated in the afternoon.

The morning following the revised treat-

ment, the cow was much improved. Its temperature had returned to normal, the swelling no longer increased, and the appetite was returning. The sulfathiazole sodium sesquihydrate treatment was repeated. Sulfathiazole, 300 gr., was given per orum and repeated daily for 2 more days. The swelling slowly receded. Milk production in the non-infected quarters rose from the amount obtained by stripping to 10 lbs. per day.

The success of the sulfathiazole sodium sesquihydrate treatment was again demonstrated a few days later in a similar case in which the disease was farther advanced before the veterinarian was called. The cow did not succumb, but the quarter most affected sloughed away. The udder had been damaged to such an extent that milk production was unprofitable so the animal was fattened for beef.

*—R. E. Norton, '44*

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**6** **Severed Tendons in Equine.** A rather common farm accident with a horse-drawn mower, brought a young roan Belgian mare to the Stange Memorial Clinic on July 20, 1943, with severed superficial and deep flexor tendons of the left hind leg. The gaping wound was inflicted between the middle and distal third of the metatarsus. The horse placed considerable weight on the leg, allowing the fetlock to touch the ground at times.

It is usually not difficult to diagnose this condition in the equine. In the majority of cases a definite history which involves an accident with farm implements is known. The severity and the location of the cut tendons primarily determine the prognosis.

A very marked degree of dorsal flexion of the fetlock joint indicates a rupture or division of the flexor perforans (deep digital flexor). The dorsal flexion is such as to allow the fetlock to reach the ground, causing the toe to be turned up so as to expose the sole.

When only the suspensory ligament is divided the fetlock drops just slightly and the solar surface of the foot remains flat on the ground. When the flexor perforatus (superficial digital flexor) is ruptured there is a marked dropping of the fetlock sufficient to cause the toe to turn slightly upward. The fact remains, however, that a rupture of either ligament is followed by approximately the same symptoms and the two conditions may be confused.

### Symptoms

The grave condition resulting from a rupture of all three tendinous structures posterior to the metatarsal bone is marked by the dangling position taken by the phalanges. The animal appears to stand on the distal end of the metatarsal bone while the phalanges extend inertly to the anterior.

A division of the digital extensor tendons anterior to the cannon bone is principally manifested by dragging of the toe and various degrees of inability to properly extend the phalanges while in motion, especially in placing the foot forward prior to the supporting phase of the stride. No abnormal position of the phalanges is noted while the limb supports weight as in quiet standing.

### Anatomy

The mechanism of the hock and stifle joints is such that they are unable to move or flex independently. The peroneus tertius or the tendinous portion of the flexor metatarsi is an inextensible cord which unites the stifle and the hock joint in such a way that movement of one joint produces movement of the other. The tendon arises in the extensor fossa of the femur in common with the long extensor muscle and is inserted by three slips into the hock and metatarsus. A rupture of this tendon produces a marked flexion of the stifle joint and excessive extension of the hock. This produces a lack of harmony in the function of both joints and the uncertain movements of the limb may give the impression of a broken bone. The absence

*(Continued on page 158)*