STRONGYLUS VULGARIS IN THE HORSE: A REVIEW

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Introduction

Strongylus vulgaris is the most notorious parasite of the horse and is certainly one of the most pathogenic. The adult worm is a bloodsucker and resides in the cecum and right ventral colon where it is strongly attached to a plug of mucosa drawn into its deep buccal capsule. In Ontario, adult S. *vulgaris* have been found in more than 85% of horses (53). In Britain, Poynter (50) recorded the highest incidence of infection in year old animals (nearly 90%) and the lowest (46.6%) in foals. Found throughout the world, S. *vulgaris* is feared most for the arteritis induced by the larval forms during their migration. The occurrence of larvae in the cranial mesenteric artery was apparently known to the ancient Romans (54). Verminous arteritis in the cranial mesenteric artery (and its branches) has been reported as the cause of death in 10 to 33% of abdominal crises in the horse (39, 51).

A long history of controversy has surrounded the development of S. *vulgaris* in the equine host. A major issue has been whether migrating larvae move with the circulation, or against it, or even follow some direct path to bring about lesions in arteries. Olt (43) believed the normal route was similar to that of *Ascaris* and that larvae found in the cranial mesenteric artery were aberrant, reaching this site by migration within the mesentery. In his earlier view, Ershov (27) evidently considered migration within the mesentery to be normal, and both Ershov (27) and Olt (43) concluded that larvae reached the lumen of the cranial mesenteric artery by penetrating the vessel wall. Ottaway and Bingham (44) agreed with Olt (43) insofar as it seemed unlikely that the development of larvae in arterial lesions occurred normally. From necropsies, Farrelly (29) found no evidence of penetration of vessels from the outside by S. *vulgaris* larvae, and proposed a venous route to the heart ("by way of the posterior vena cava and/or the vena azygos") with passage through the lungs and return to the left heart, thus attaining the cranial mesenteric artery via the aorta. Migration of larvae from the heart along the aorta and cranial mesenteric artery (29, 45) became known as the "intra-arterial hypothesis" (49). Vague on how the left heart is attained, this concept encountered resistance because it required that larvae be found in the lungs, and several workers failed to record such a finding (14, 21, 26, 37).

In general, inferences drawn from the necropsy of naturally infected horses did not reveal the unusual migration of S. *vulgaris*. Wetzel and Enigk (56) were the first to infect foals experimentally with S. *vulgaris*, and Enigk (24, 25) continued this study to establish the direct arterial route whereby larvae reached the cranial mesenteric artery by penetration of the arterioles in the intestinal wall. Remarkably, while observing recent lesions in naturally infected foals, Kikuchi (33) in 1928 suggested the route later confirmed by Enigk in 1950 and 1951 (24, 25). Moreover, as Georgi (30) recently pointed out, even Kikuchi (33) was anticipated nearly 60 years before by Bollinger (4) who believed that in its migration S. *vulgaris* was confined to the cranial mesenteric artery.

Development of S. *vulgaris*

The experimental evidence is clear that S. *vulgaris* develops in the following way. From the egg on the ground or on pasture there emerges a first stage larva which grows and molts to a second stage larva and then again to the third stage. The third stage larva is the infective stage and under optimal summer conditions probably requires about ten days to two weeks to develop from the time the egg is passed (41). It is surrounded by the loosened cuticle of the second stage but after ingestion by the horse, this cuticle is cast off (termed exsheathment) during passage through the alimentary canal. The exsheathed third larvae rapidly penetrate the mucosa and submucosa of the ileum, cecum and ventral colon. This is accomplished at least as early as two days postinfection (PI) and is accompanied in the submucosa by edema and marked dilation of small arteries, veins and capillaries (18, 21, 24). In the submucosa the third larva molts to the fourth stage on about day 4 or 5 PI (26). After this molt migration evidently begins. These larvae penetrate arterioles of the

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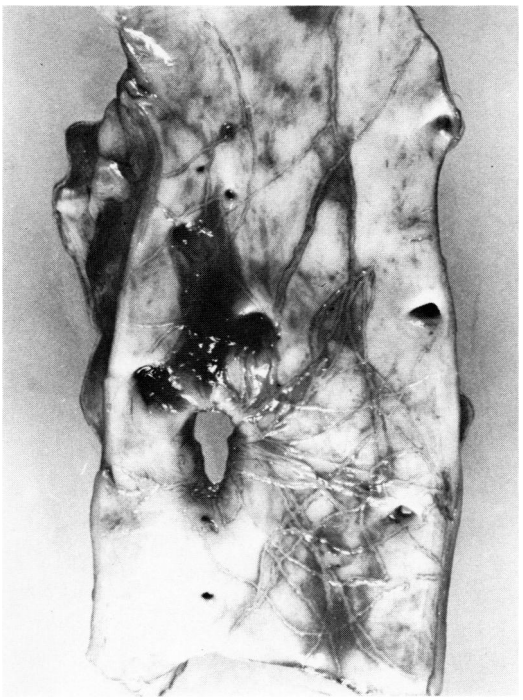

FIGURE 1. Fibrin tracks of migrating *Strongylus vulgaris* on the intima of the aorta near the origin of the cranial mesenteric artery. From a six week old pony foal inoculated with 5000 ± 6% *S. vulgaris* larvae and examined 17 days PI.

submucosa (26) and are found close to the intima or in the lumina of small arteries on day 7 PI (21). Severe submucosal arteritis may be apparent by this time.

Working against the flow of blood, the fourth stage larvae gradually move up the arterial system of the intestine. By eight days PI larvae have reached the cecal and ventral colic arteries (24, 26). When these larger arteries are attained, the route of migration is marked by a meandering thread of fibrin on the intima (24, 26) (Figure 1) and by day 14 larvae may be found in mural thrombi (21). The ileo-ceco-colic and cranial mesenteric arteries are reached between 11 and 14 days PI (21, 26, 28). The migratory advance is said to attain its height by the 19th day at which time larvae may be found in almost any part of the arterial system (26) but are always most numerous in arteries close to the origin of the cranial mesenteric artery.

The recent work of Duncan and Pirie (21) has added numerous refinements to the later development of *S. vulgaris*. These authors found that on the 25th day PI marked thrombosis was evident in the ileo-ceco-colic and cranial mesenteric arteries from which many fourth stage larvae 1 to 2 mm long were recovered or seen in sections. The molt to the fifth stage (preadults) occurs as early as 90 days PI (25, 26), and by 120 days most larvae are preadults measuring up to 18 mm long (21). *S. vulgaris* larvae tend to remain in the arterial site until they molt to the fifth stage (21), although many fourth stage larvae are apparently swept away before the final molt occurs (25). No doubt their size and the thrust from the flow of blood are important factors in the separation of larvae from arterial lesions. Eventually the preadult *S. vulgaris* reach the small arteries on the serosal surface of the large intestine and terminal small intestine. Unable to migrate further in arteries, the young *S. vulgaris* become encased in pea-sized nodules (21, 25). These nodules are numerous four months after infection; after their escape from nodules into the lumen of the intestine, *S. vulgaris* require another six to eight weeks before reaching sexual maturity (20, 21). The prepatent period is about six and one-half months (25, 55). There is no evidence of prenatal infection (14, 25).

PATHOGENESIS OF *S. vulgaris* INFECTION

Within two days of infection Duncan and Pirie (22) observed hemorrhagic foci on the mucosa of the ileum, cecum and colon. By one week PI these authors noted a severe inflammatory reaction in the submucosa, with widespread arteritis, accompanied by thrombosis and infiltration by neutrophils. These changes were correlated with the molt of larvae to the fourth stage and soon extended to the muscularis and serosa. Infarction of the ileum, cecum and colon is observed two to three weeks PI along with thrombosis and thickening of the cranial mesenteric artery and its branches (14, 22). At about this time tortuous fibrin tracts, often thread-like and so characteristic of early *S. vulgaris* infection, are observed on the intima of arteries and these may extend into the abdominal aorta (Figure 1). The tracks are overgrown with endothelium. Between one and four months PI the predominant lesions are in the cranial mesenteric and ileo-ceco-colic arteries, where the principal changes are thrombus formation and marked infiltration and fibrosis of the tunica media (22). Fourth stage, and later fifth stage larvae are associated with thrombi. Larvae returning to the intestine become encapsulated in nodules (21). Approximately nine months after infection with 750 *S. vulgaris* larvae a considerable reduction of lesions in the cranial mesenteric artery was found by Duncan and Pirie (21), suggesting

that repair to earlier damage caused by larvae may occur.

Lesions brought about by *S. vulgaris* are most common in the cranial mesenteric and ileo-ceco-colic arteries (45, 49). Poynter (49) found the incidence to be 86% in the cranial mesenteric artery followed by 62.5% in the cecal and colic arteries. Colic has long been considered related to thrombosis or embolism in these vessels. Aneurysms, especially of the cranial mesenteric artery, are often mentioned in the literature but a true aneurysm associated with *S. vulgaris* infection is rare if it occurs at all. While the lumen of an affected artery is often wider than normal the wall of the vessel is generally thicker (Figure 2) and the lumen of extensive sections of an artery may be several times its normal diameter. In older horses the cranial mesenteric and ileo-ceco-colic arteries are often encased in a large nodular mass. Within five or six weeks following infection, the intimal surface of the cecal and ventral colic arteries is often rough and may have a corrugated appearance (Figure 3).

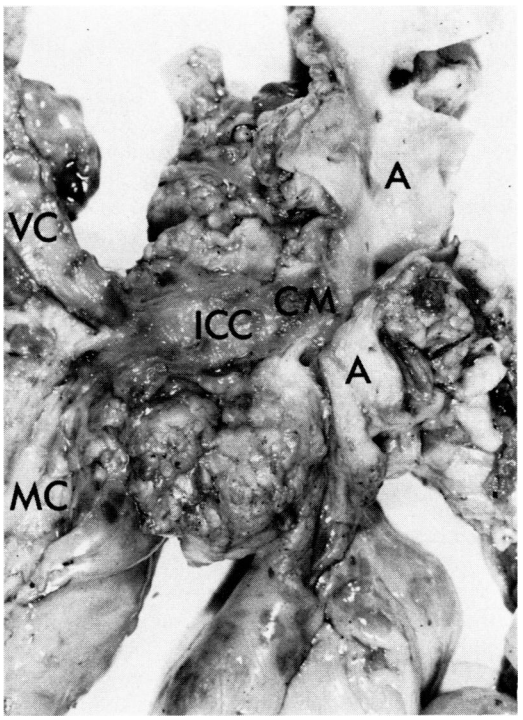

FIGURE 2. Large nodular mass surrounding the cranial mesenteric and ileo-ceco-colic arteries. The lumina of these arteries were wider than normal. From a four month old pony foal inoculated with 1000 *Strongylus vulgaris* larvae and examined 35 days PI. (A—aorta; CM—cranial mesenteric artery; ICC—ileo-ceco-colic artery; MC—medial cecal artery; VC—ventral colic artery).

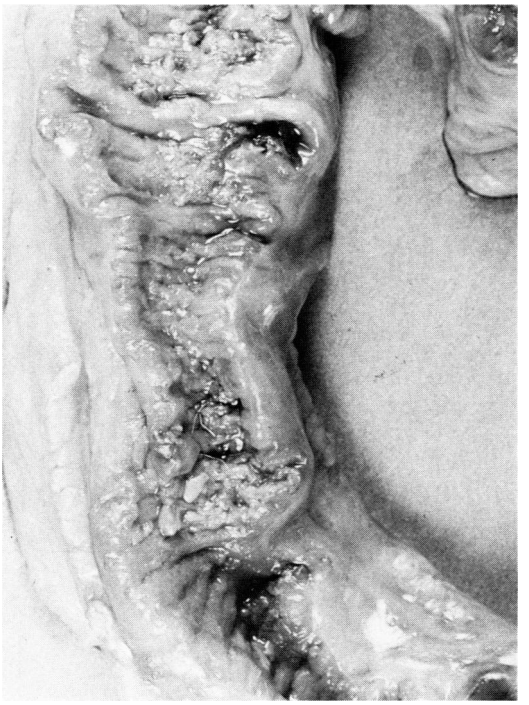

FIGURE 3. Rough and corrugated intimal surface of the ventral colic artery. From a seven month old pony foal inoculated with 2000 *Strongylus vulgaris* larvae and examined 36 days PI.

Lesions in remote or unusual sites have been associated with *S. vulgaris* larvae. Cronin and Leader (9) recorded a case of occlusion of the right coronary artery which they considered due to *S. vulgaris* larvae. *S. vulgaris* in the kidney is recorded (37). Intermittent lameness has been attributed to thrombosis or embolism of the external iliac artery, brought about by verminous arteritis (57). In a study of several cases of equids with neurological signs, *S. vulgaris* was suspected by Little (34) as an important cause of cerebrospinal nematodiasis. Little *et al* (35) later induced signs and lesions of acute verminous encephalitis by inoculation of fourth and fifth stage *S. vulgaris* into the internal carotid artery of ponies.

CLINICAL SIGNS

Naturally infected horses generally carry a mixed burden of large and small strongyles in the intestine. In heavily infected animals especially, it is difficult to distinguish the signs due to large strongyles inhabiting the intestine from those of the small strongyles (often called trichonemes). However, diarrhea is said to be a more frequent sign of infection with small strongyles than with the large (23).

Adult *S. vulgaris* are most commonly found in the cecum and feed by drawing in a plug of mucosa into the buccal capsule. At necropsy, the crater-like ulcers caused by large strongyles are often more numerous than the worms, suggesting that they move periodically to new sites of attachment. Anemia, emaciation, poor coat and poor performance are frequently attributed to large strongyles in the intestine. However, the number of adult *S. vulgaris* in the intestine required to induce clinical signs is unknown.

The acute signs associated with *S. vulgaris* are due to migrating larvae and are seen during the first few weeks after infection. The severity of these is related to the number of larvae ingested and undoubtedly to the age and previous experience of the host. A well-defined syndrome results from the experimental inoculation of infective larvae into worm-free foals (14, 24). Drudge *et al* (14) found that following infection of two to nine month old pony foals with large numbers (2500–5000) of *S. vulgaris* larvae, an acute reaction developed. This was characterized by a marked increase in temperature, loss of appetite, rapid loss of body weight, depression and recumbency, abdominal distress, constipation, or occasionally, intermittent diarrhea. Most animals died 14 to 22 days after infection. Sudden onset of fever (as early as the second day after infection) with readings of 40.0 to 41.1°C (104 to 106°F) were recorded during the first ten days of infection. Loss of appetite coincided with fever, and loss of weight ranged up to 26% during a two to three week period. Typically, fecal output was reduced and consistency was hard.

Drudge *et al* (14) also induced a chronic infection with repeated doses of 250 larvae over a 16 week period. Again, fever was a consistent feature, frequently between 38.9 and 39.4°C (102 and 103°F) along with poor weight gain as well as intermittent periods of abdominal distress. Recently, Duncan and Pirie (22) made similar observations following doses of 750 larvae given to nine worm-free pony foals, but noted a wide variation in temperature during the first two or three weeks of infection (from slightly above 37.2 to over 40.6°C) (99 to over 105°F). From experiental studies, our experience has been that one to two month old pony foals given 750 *S. vulgaris* larvae in a single dose generally survive, while those given 1000 larvae frequently die within one to two weeks. Amborski *et al* (1) found that clinical responses of previously uninfected ponies to *S. vulgaris* were more severe than those of reinfected ponies. Older horses are often observed to have arterial lesions without a history of specific signs, although signs detected in field cases can be correlated with findings at necropsy (10, 31, 47).

Under experimental conditions, during the first three weeks of acute or subacute infection it has been found that hemoglobin gm% (Hb gm%), red blood cells per mm^3 (RBC/mm^3) and packed cell volume (PCV%) may decline slightly indicating a moderate anemia (14). The most consistent change in early *S. vulgaris* infection would appear to be a rapid increment in total white cell (WBC) counts. These values have been observed to rise sharply during the first three weeks to levels of 17,000 to 22,700/mm^3 (14, 18, 22). Eosinophil values increase after the second week but may show little change in acute infection (14). A similar low eosinophil response of calves with acute *Dictyocaulus viviparus* and *Ascaris suum* infection has also been reported (11, 32). Increments in serum total protein and globulin fractions occur as early as the first week following infection (14); however, with moderate doses of larvae (700–750) these parameters, especially beta globulins, remain relatively unchanged for six or seven weeks (18, 22, 52). Amborski *et al* (1) also found less pronounced changes in Hb gm%, PCV, and serum protein values in reinfected ponies. Fever in *S. vulgaris* infection is attributed to tissue damage or a toxic substance elaborated by larvae (14, 17).

Diagnosis

Although infection with adult *S. vulgaris* may be diagnosed by allowing eggs to hatch and the larvae to develop to the (infective) third stage when they can be identified, it is impossible to make a diagnosis from the recently passed egg alone. It is also difficult to diagnose early *S. vulgaris* infection (one to three weeks PI) as well as the extensive arteritis that is present when the wall of the cranial mesenteric and ileo-ceco-colic arteries may be greatly thickened. Adhesions may be present in *Strongylus* infections (38), tending to complicate rectal examination for nodular enlargements. Moreover, in mixed *Strongylus* infections (*S. vulgaris* and *S. edentatus*) we have observed at necropsy that lymph nodes in the region of the cranial mesenteric artery are sometimes enlarged and hard, thus making the clinical determination of an enlarged mass due to *S. vulgaris* alone difficult. Experimental studies suggest that in addition to general condition (especially in foals), an increase in temperature as well as in total white cell counts

should be taken into consideration in diagnosis. Beta globulins are also known to increase after infection (52). However, as Drudge *et al* (14) pointed out, the change in the blood picture associated with *S. vulgaris* is not unlike that seen in bacterial infections. There is a great need for precise criteria in the diagnosis of verminous arteritis in horses.

Epidemiology

Strongyle egg output is known to vary seasonally. In Britain, Poynter (48) found that minimal egg production occurred in winter, rising during the spring with maximal production in August or September. In Ontario, Slocombe and McCraw (53) also found that strongyle egg counts were high in August but during the period May to July counts were lower for thoroughbred, standardbred and show horses than for pleasure or commercial animals. Management and the time of administration of anthelmintics probably accounted for these differences. Following Poynter's (48) study, Ogbourne (40) showed that the percent of *S. vulgaris* (and *S. edentatus*) viable eggs was lowest in winter but reached a peak in May and remained high during the summer. Thus, seasonal variations in adult populations of *S. vulgaris* (and *S. edentatus*) could be accounted for by seasonal differences in infection rate and the lengths of the prepatent periods.

The survival of eggs and larvae is a complex and perennial question. Eggs and larvae are affected by the same environmental factors (temperature, moisture, etc.) but in varying degrees. Parnell (46) stated that winter conditions in Canada do not kill unembryonated strongyle eggs. Ogbourne (41) found that eggs incubated at 6.5°C developed but did not hatch. Preliminary studies by Slocombe and McCraw (unpublished data, 1975) indicated that *Strongylus* eggs, freshly passed by worms in saline, do not survive more than one half hour at freezing temperatures. There appears to be no development of larvae to the infective stage on British pastures during winter (41) but infective larvae already present can survive the winter to infect grazing horses the following spring (42). Evidently the rate of drying affects both the number of larvae surviving as well as the degree of development (41). In Britain (where rainfall is generally greater than in Ontario), there is a high mortality rate of larvae on pasture during the summer (42).

Ingenious experiments by Duncan (19) revealed that a primary source of infection of foals was from infected mares. He found that if mares were treated with an anthelmintic prior to foaling and again every two weeks while grazing with their foals (which remained untreated) fecal egg counts and cultures as well as pasture larval counts remained negative from May to mid-September. In a second group in which the mares (as well as the foals) remained untreated, fecal counts of mares and the number of pasture larvae increased rapidly during the summer. The foals from the two groups were examined at necropsy when approximately one year of age, and those which had been with treated mares during the previous summer had a mean mixed strongyle burden of only 3532 worms whereas those running with their untreated mares had a mean burden of over 57,000 worms (19). In a similar experiment where mares were dosed while with the foals only when a positive egg count was recorded (about every three weeks), he found that at necropsy foals again had much lower worm burdens compared to controls.

Control and Treatment

Prevention of the damaging effects of larvae in arteries is the most important aspect in the management of *S. vulgaris* infection. Routine treatment for the removal of adult worms is essential as the large egg output of strongyles can bring about contamination of a confined area very quickly. It is evident that relatively small numbers of infective larvae may have serious effects on foals, and thus mares in foal or nursing should be routinely examined for strongyle eggs and treated with an appropriate anthelmintic. Thiabendazole has had wide use and recently several other anthelmintics have been developed or approved for use in adult horses, including benzimidazole compounds (2, 5, 15, 16), tetrahydropyrimidines (8, 36) and organic phosphorus compounds (7, 13).

Drudge *et al* (17) and Drudge and Lyons (12) have shown that high levels of thiabendazole subdue the pathogenic effects of early *S. vulgaris* infection in pony foals. Following infection with 5000 *S. vulgaris* larvae (sufficient to induce acute disease) two doses of thiabendazole at the 440 mg/kg level were administered on successive days (12). Three of four foals treated on days 7 and 8 PI were examined approximately three weeks PI and gross lesions were found to be in an advanced state of resolution. Treatment of one foal on days 11 and 12 PI and another on days 17 and 18 PI resulted in prompt remission of body temperature and general clinical improvement.

Recently, Coffman and Carlson (6) reported on the treatment of two quarter horse mares in which a nodular mass was found in each by rectal examination. Both were in a deteriorated condition with recurrent or occasional fever. Verminous arteritis was confirmed in one horse by an exploratory laparotomy, carried out because neoplasia was tentatively diagnosed. Thiabendazole at the rate of 250 mg/kg body weight was given via stomach tube on two consecutive days. Coffman and Carlson (6) observed that both mares were improved two to three weeks after treatment and had begun to gain weight. In one mare, the nodular mass was found by rectal examination to have decreased to approximately 16 by 16 cm, about one third the original size.

Summary

Early concepts and recent experimental findings on the development of *Strongylus vulgaris* are reviewed. Infective larvae penetrate the intestinal wall, molt and migrate directly in the arterial system reaching the cranial mesenteric artery about 14 days postinfection. Early migration of fourth stage larvae is marked by tortuous tracks of fibrin on the intima of arteries. Fifth stage larvae become separated from lesions in arteries and are carried back to the wall of the intestine where they become encapsulated in nodules. After escaping into the lumen of the intestine the preadults mature in six to eight weeks. The prepatent period is about six and one-half months and there is no evidence of prenatal infection.

By one week postinfection, arteritis, thrombosis and infiltration of neutrophils are noted in the intestine. Infarction of the intestine is observed two to three weeks postinfection along with thrombosis and thickening of the cranial mesenteric artery and its branches. Later, thrombus formation, infiltration and fibrosis of the tunica media of arteries are the main lesions.

Clinical signs due to adult S. *vulgaris* are ill-defined but acute signs develop following inoculation with large numbers of infective larvae. These consist of an increase in temperature, loss of appetite and weight, abdominal distress, constipation or diarrhea. An early change is a rapid increment in total white cell counts.

A primary source of infection of foals is evidently from infected mares. Prevention of the damaging effects of larvae in arteries is the most important aspect in the management of S. *vulgaris* infection.

Résumé

Les auteurs passent en revue les premières données et les observations expérimentales récentes, relatives à *Strongylus vulgaris*. Les larves infestantes pénètrent la paroi intestinale, muent, émigrent directement dans le système artériel et atteignent l'artère mésentérique antérieure, environ 14 jours après l'infestation. Le début de la migration des larves du quatrième stade provoque la formation de sillons de fibrine tortueux, dans l'intima des artères. Les larves du cinquième stade quittent les lésions artérielles et retournent dans la paroi de l'intestin où elles s'enkystent. Après s'être libérés dans la lumière intestinale, les jeunes adultes atteignent la maturité en six à huit semaines. La période de prépatence dure environ six mois et demi et il n'existe aucune évidence relative à une infestation pré-natale.

Une semaine après l'infestation, on note la présence d'artérite, de thrombose et d'une infiltration de l'intestin par des neutrophiles. L'infarction de l'intestin se produit environ deux à trois semaines après l'infestation, en même temps que la thrombose et l'épaississement de l'artère mésentérique antérieure et de ses branches. Plus tard, la formation d'un thrombus, l'infiltration et la fibroplasie de la média des artères constituent les principales lésions.

Les signes cliniques attribuables aux S. *vulgaris* adultes sont mal définis; des signes aigus apparaissent toutefois à la suite de l'ingestion d'un grand nombre de larves infestantes. Ils se caractérisent par de l'hyperthermie, une perte d'appétit et de poids, de la douleur abdominale et de la constipation ou de la diarrhée. Une augmentation rapide du nombre total des leucocytes représente un changement précoce.

Les juments parasitées représentent évidemment une source importante de contamination pour les poulains. La prévention des lésions artérielles constitue le facteur le plus important de la lutte contre l'infestation par S. *vulgaris*.

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ANALYSE DE VOLUME

Outlines of Avian Anatomy. A. S. King et J. McLelland. Publié par Baillière Tindall, Londres. 1975. 154 pages. Prix $6.00.

Ce petit livre, riche en diagrammes bien choisis, est destiné avant tout aux étudiants en médecine vétérinaire et à ceux que l'anatomie comparée des oiseaux intéresse.

Tous les grands systèmes y sont traités avec un souci de concision et de clarté pour ne conserver que les éléments nécessaires à la compréhension de l'anatomie aviaire.

Le professeur King enseigne l'anatomie à Liverpool et le professeur McLelland dirige cette chaire à Edinbourg. L'ouvrage de ces auteurs traduit des préoccupations qui débordent les laboratoires d'anatomie. Les pathologistes y réfèreront avec avantage; les vétérinaires engagés dans l'inspection de la volaille y trouveront aussi d'excellents rappels.

Détail que je me permets d'ajouter et que j'apprécie, à la fin du volume, les auteurs donnent une excellente bibliographie à consulter pour approfondir le sujet de chaque chapitre. *O. Garon.*