Pathology of Tenosynovitis with Rupture of the Gastrocnemius Tendon in Young and Adult Broiler Chickens

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Abstract. A pathological investigation was carried out on tenosynovitis with rupture of the gastrocnemius tendon occurring in young and adult broiler chickens. Tenosynovitis has been seen mainly in the gastrocnemius tendon sheath and tended to be chronic in character, showing a proliferation of juvenile fibrous tissue in the subsynovial layer. Complete rupture of the gastrocnemius tendon occurred in eight of 12 young chickens (50–71 days of age). Nodular fibrous thickening in the tendon-muscle junction was observed in fourteen of 16 adult chickens (181 or 235 days of age) and one young chicken. It was associated with partial rupture of the tendon in four of these chickens. The thickening was regarded as a healing process of the partial rupture. Tenosynovitis was regarded as a precursor of rupture of the tendon. Accordingly, tenosynovitis and rupture of the gastrocnemius tendon may be in the same entity of disease. Secondary lesions were observed in the muscle and nerve fibers in the leg.

Since the first report on rupture of the gastrocnemius tendon in chickens in the United States of America in 1944 [1], similar disorders have been reported from England [2, 9, 15], the United States of America [5] and Canada [3] up to 1963. The etiology and pathogenesis of the disease remain unknown. The same condition as seen in the chicken has occurred in turkeys [1, 8]. Johnson and van der Heide [13] suggested in 1971 that tenosynovitis might be the precursor of the rupture of the gastrocnemius tendon. Jones et al. [14] isolated a reovirus from the lesion in 1975. This condition occurs in both broiler and layer chickens most of which are at, or near, maturity, although sometimes young birds are involved. In Japan two outbreaks of the disorder were found recently in adult [10] and young [19] broiler chickens.

The term “tenosynovitis” in chickens

was first used in 1967 in order to distinguish it from infectious synovitis [4]. The incidence seems to have been increasing in several countries, including Japan. Recently, it became evident that tenosynovitis is caused by a reovirus [20]. Spontaneous tenosynovitis has been reported to occur in young broiler chickens ranging from about 3 weeks of age to marketing age [4, 13, 21, 25]. It appears that with the lapse of time the lesion becomes chronic in character, showing thickening, hardening and fibrosis of the tendons and their sheaths in the metatarsus and the posterior area above the hock joint [4, 7, 21, 23, 25]. In addition, some cases are associated with rupture of the gastrocnemius tendon [13, 25]. A virus has frequently been isolated from these cases [13, 21, 23, 25]. Lesions induced by the isolate were identical to those of the natural cases [12, 17, 21, 25].

From the reports cited above it is postulated that pathogenetically there may be a relationship between the rupture of the gastrocnemius tendon and tenosynovitis. Such a relationship has not been investigated, although it has been suggested by Johnson and van der Heide [13].

The present authors encountered outbreaks of tenosynovitis associated with rupture of the gastrocnemius tendon in young and adult broiler chickens. This paper describes the pathological findings and discusses the pathogenesis of the ruptured tendon.

Materials and Methods

Materials used for this study were 28 broiler chickens (Table 1). They were collected from two outbreaks occurring on unrelated farms and named groups A and B. The case history is as follows.

Group A (Nos. 1–12): On this farm a flock of 6,000–6,500 chickens to be reared as broilers was placed at weekly intervals with intention for the birds to be processed at about 70 days of age. During the summer and autumn of 1976, some chickens over 50 days of age in the flock showed leg weakness or difficulty in standing. Rupture of the gastrocnemius tendons with marked hemorrhages was noticed at the processing plant. The average incidence of this condition was about 5% in each age group, with males being affected in an overwhelming majority. The owner reported that no abnormality had been observed in any of the chickens before about 50 days of age. The materials used for this investigation were obtained from 12 birds ranging from 50 to 71 days of age. They were collected from six affected flocks. Of the 12 birds, ten were male, one was female, and one of unknown sex.

Group B (Nos. 13–28): On this farm a flock consisting of about 450 male and 3,000 female broiler breeder chickens was being raised in the spring of 1975, when this disorder occurred. The disorder began to be noticed at 117 days of age. The affected birds were in good bodily condition, but exhibited difficulty in walking and frequently displayed a unique symptom of standing on tiptoe (Fig. 1). The morbidity was 2.2% at 181 days of age and 3.3% at 235 days of age. The same condition as described above had occurred with about 10% of morbidity in the spring of 1974. The materials for this study were obtained from 16 chickens collected at 181 and

235 days of age. Of the 16 birds, eleven were female and five male.

Following postmortem examination, tissues were fixed in 10% formalin and embedded in paraffin. Sections were stained with hematoxylin and eosin. Selected sections were subjected to Azan, elastica van Gieson, periodic acid-Schiff (PAS), Luxol fast blue-eosin, and Bodian stainings. Frozen sections were prepared from some muscles and peripheral nerves and stained with Sudan III. As a rule, the following tissues from both right and left legs of each bird were examined in addition to all the visceral organs: gastrocnemius tendon and its sheath, tendons and their sheaths around the hock, metatarsophalangeal and interphalangeal joints and around the metatarsus, such muscles as *Musculus* (M.) *gastrocnemius*, *M. peroneus longus*, *M. biceps femoris*, and *M. tensor fasciae latae*, and the ischiatic nerve. In addition, synovial membranes and the bones composing all the joints of the legs were examined in some birds. They had been treated by electric decalcification, celloidin embedding, and hematoxylin and eosin staining.

Results

1. Gross pathology

Lesions characteristic of this condition were seen in the gastrocnemius tendon and its surrounding tissues. There were some differences in the lesion between groups A and B.

Group A: The gastrocnemius tendon and its surrounding tissue were slightly swollen and edematous. Complete rupture of the tendon (Fig. 2) occurred in eight of 12 birds, bilateral in five and unilateral in three (Table 1). It occurred about 2 cm above the hock joint (in the middle area of the tendon) in 7 birds and in the gastrocnemius tendon-muscle junction in 1 bird. The free ends of the ruptured tendon showed necrosis with marked hemorrhage. Hemorrhage with edema spread to the surrounding tissue and resulted in detachment of other flexor tendons from the tibial bone. The hock joint of 2 birds (Nos. 3 and 7) was slightly swollen with excess turbid synovial fluid. Tibial dyschondroplasia coexisted in 3 birds (Nos. 8, 11 and 12).

Table 1. Phase of lesion of gastrocnemius tendon sheath and incidence of rupture of the tendon

Bird No.	Group	Age (days)	Sex	Phase of lesion of tendon sheath and incidence of rupture of tendon*							
				Left				Right			
				Acute	Subacute	Chronic	Rupture of tendon	Acute	Subacute	Chronic	Rupture of tendon
1	A	50	?	—	●	—	—	—	—	—	—
2	A	50	Male	—	●	—	—	—	●	○	×
3	A	52	Female	—	●	○	—	—	●	○	—
4	A	57	Male	○	●	—	—	●	—	—	—
5	A	69	Male	●	—	—	—	●	—	—	—
6	A	69	Male	○	●	—	×	○	●	—	×
7	A	70	Male	○	●	○	×	○	●	—	×
8	A	70	Male	○	●	—	×	○	●	—	×
9	A	70	Male	—	—	—	×	○	●	—	×
10	A	71	Male	○	●	—	×	○	●	○	▲
11	A	71	Male	—	—	—	—	○	●	—	×
12	A	71	Male	●	○	—	×	○	●	—	×
13	B	181	Female	—	○	●	△	—	—	—	—
14	B	181	Female	—	○	●	—	—	—	—	—
15	B	181	Female	—	—	●	—	—	—	—	—
16	B	181	Female	○	○	●	△	—	—	—	—
17	B	181	Male	—	○	●	△	—	—	—	—
18	B	235	Female	—	○	●	△	—	—	●	△
19	B	235	Female	—	—	—	—	—	—	●	△
20	B	235	Female	○	○	●	△	○	○	●	▲
21	B	235	Female	—	●	○	△	—	—	●	△
22	B	235	Female	—	○	●	△	—	●	○	△
23	B	235	Female	—	—	—	—	—	—	●	△
24	B	235	Female	○	●	○	▲	○	●	○	▲
25	B	235	Male	○	○	●	△	○	●	○	△
26	B	235	Male	○	○	●	▲	—	○	●	△
27	B	235	Male	—	○	●	△	—	—	●	—
28	B	235	Male	—	—	●	—	○	●	○	△

Remarks.

* ○: Observed, ●: Main lesion in each bird, ×: Complete rupture, ▲: Partial rupture, △: Nodular fibrous thickening, —: Not observed, No symbol: Not examined.

Group B: Marked thickening and hardening of the region of the gastrocnemius tendon were felt by palpation. After skinning the whole area of the tendon was seen to be involved, frequently showing a nodular thickening in the tendon-muscle junction (Fig. 3). The lesion was grayish-white in color. The nodular thickening in some cases reached 1.5 cm in diameter, within which small hemorrhagic and necrotic foci were occasionally observed. The affected tendon and its surrounding flexor tendons

were adherent, losing their function. Such lesions were bilateral in fourteen of 16 birds and unilateral in two. A small-nodular thickening was seen in the tendon-muscle junction of the M. peroneus longus of 2 birds (Nos. 19 and 22). Excessive clear synovial fluid was frequent in the hock joint.

Pneumonia, hydropericardium and splenomegaly were present in a few birds of group A. In group B, a degenerative change of the pectoral muscle was seen in 3 birds

and egg-rupture peritonitis in 5 others.

2. Histopathology

The essential histological lesions in both groups were the same in character and are described as an entity.

a. Tendon sheath and tendon

Acute phase: There was a fibrinous exudation on the synovial membrane of the tendon sheath with proliferation of fibroblasts and formation of new blood vessels in the subsynovial layer (Fig. 4). Swelling and slight hyperplasia of synovial cells were seen in the remaining synovial membrane, especially in the membrane of areolar type (Fig. 6).

Subacute phase: The subsynovial layer of the tendon sheath was comparatively thickened due to proliferation of fibroblasts and mesenchymal cells, frequently with formation of new blood vessels in deeper areas (Fig. 5). The cellular proliferation spread to the tendon and adjacent connective and adipose tissues. A sparse heterophilic infiltration was present in the lesions. Fairly marked hyperplasia of synovial cells was seen in the remaining synovial membrane of areolar type (Fig. 7).

Chronic phase: The subsynovial layer of the tendon sheath was markedly thickened with proliferation of dense juvenile fibrous tissue (Figs. 8, 9). Juvenile fibrocytes in some areas were irregularly intermingled in a fascicular arrangement resembling fibroma. There were a few scattered hemosiderin-laden cells, lymphocytes and heterophils in the lesions. Fibrous adhesion of the tendon sheaths frequently occurred. The juvenile fibrous tissue also spread to the surrounding tissue. Within the tendon around the tendon sheath involved there were proliferation of juvenile fibrocytes, swelling and increase of nuclei of tendon cells, infiltration of a few lymphocytes and plasma cells, appearance of a few hemo-

siderin-laden cells and heterophils, and atrophy of tendon cells (Fig. 10). Sometimes, the tendon was replaced by juvenile fibrous tissue and fairly thickened. The nodular thickening observed grossly consisted of dense juvenile fibrous tissue containing many blood vessels (Figs. 12, 13). This fibrous tissue invaded the muscular interstitial connective tissue (see below).

In 6 birds (Nos. 2, 7, 8, 10, 12 and 16) endarteritis obliterans was scattered in medium-size arteries in the juvenile fibrous tissue of the tendon sheath and its surrounding tissue (Fig. 15). The alteration consisted of proliferation of juvenile fibrocytes, sometimes with lymphocytic infiltration, in the tunica intima. Lymphocytic aggregations around blood vessels were dotted in some birds. Near the distal end of the gastrocnemius tendon in a few birds of group B, there was an enlargement of hyaline cartilage consisting of proliferation of cartilage cells (Fig. 11), rarely with bone formation resulting from endochondral ossification.

There were hemorrhage, fibrin exudation, and edema with proliferation of granulation tissue at the complete rupture of the tendon which was observed grossly. Microscopical partial rupture occurred to the gastrocnemius tendon-muscle junction showing nodular fibrous thickening (Fig. 14). Its histological pictures were identical to those of the complete rupture.

The gastrocnemius and its surrounding flexor tendon sheath were involved in most of the birds examined. The lesions were bilateral in most cases (Table 1). The birds of group A had mostly subacute lesions and sometimes acute and chronic ones. Conversely, the chickens of group B had mainly chronic lesions and occasionally acute and subacute ones. Nodular fibrous thickening of the gastrocnemius tendon-muscle junc-

tion was noticed in 1 bird of group A and in 14 birds of Group B, four of which had the partial rupture, unilateral in three and bilateral in one.

No significant lesions were seen in the tendon sheath in any other region, although there were swelling and slight hyperplasia of some synovial cells. No changes were observed in the capsule, bursa or articular cartilage of the hock or any other joint, except purulent inflammation in 2 cases (Nos. 3 and 7).

b. Leg muscle

Muscle lesions tended to occur in the muscles belonging to the tendons and their sheaths involved, such as the M. gastrocnemius and M. peroneus longus. They were prominent in the junction. Their severity was dependent upon that of the lesion of the tendon sheath. In acute and subacute lesions there were swelling, myolysis, fragmentation, and vacuolar and hyaline degenerations of muscle fibers (Figs. 16, 17). An increase in number and central displacement of muscle nuclei were occasionally observed in the swollen muscle fibers (Fig. 16). PAS and Sudan III positive substances were not seen in the vacuoles. In the interstitial connective tissue there were a proliferation of immature or mature fat cells and edema, some with slight fibroblastic proliferation (Fig. 16).

In the chronic lesion of the tendon sheath, a markedly proliferating juvenile fibrous tissue was seen replacing muscle fibers in the tendon-muscle junction, especially around the nodular fibrous thickening of the tendon and its sheath. Muscle fibers in these lesions showed vacuolar and hyaline degenerations, and rarely regeneration. In other areas there were dotted swelling, and hyaline and vacuolar degenerations of muscle fibers. In the interstitial connective tissue a proliferation of fat cells

was very remarkable and quite extensive, frequently associated with atrophy of muscle fibers (Fig. 18). Lymphocytic aggregations were dotted around blood vessels.

c. Other changes

In the ischiatic nerves of many birds there were scattered focal swelling and fragmentation of the myelin sheath (Fig. 19), and swelling, irregular atrophy and fragmentation of the axis cylinder (Fig. 20). Focal loss of nerve fibers with the appearance of scavenger cells (Fig. 21) was occasionally observed in some nerve fiber bundles in the legs of the birds of group B.

There were no significant lesions related to this condition in any visceral organ.

Discussion

The present examination indicates that the primary and essential lesion of tenosynovitis in chickens is non-purulent in character. The lesion mainly involves the gastrocnemius tendon sheath and is characterized by a proliferation of juvenile fibrous tissue, suggesting a chronic condition. These changes are quite similar to those of naturally occurring tenosynovitis [4, 7, 13, 21, 23, 25] and of experimentally induced one by inoculating a virus which is believed at present to be a reovirus [12, 14, 17, 20, 21, 25]. Moreover, some workers [4, 17, 21, 23, 25] observed the involvement of the tendon sheaths of the metatarsus in addition to those in the posterior part of the hock joint. From the histological findings and the epizootic outbreaks studied in the present investigation, it is likely that the present cases might also be due to a viral infection, although no virus isolation has been made. Rossi et al. [23] stated that the virus in viral tenosynovitis attacked the tibometatarsal extensor and digital flexor tendons. The morphological changes of the synovial membrane in the present cases

were different from those induced by the infection of *Mycoplasma synoviae* [18], *Mycoplasma gallisepticum* [16, 24] and *Staphylococcus aureus* [11, 18].

The histological pictures observed in this study suggest that tenosynovitis may be a precursor of rupture of the gastrocnemius tendon, as described by Johnson and van der Heide [13]. A relationship between the rupture of the tendon and tenosynovitis has also been implied by other workers [12, 14]. The juvenile fibrous tissue having proliferated in the subsynovial layer of the tendon sheath invaded the tendon itself actively in young chickens of group A. Consequently, the tensile strength of the tendon was weakened, which might result in a complete rupture in the middle area of the affected tendon.

In adult chickens of group B, on the other hand, fibrous thickening and adhesion of the tendon sheaths were predominant, but the proliferative invasion of the juvenile fibrous tissue into the tendon was not so marked. As a result, the fluctuation of the tendon was restricted and a partial rupture might have occurred to the tendon-muscle junction. Moreover, the nodular fibrous thickening observed in the junction was regarded as a healing process of the partial rupture.

The difference in the invasion of the lesion into the tendon between groups A and B might be influenced by the degree of maturity of the tendon. It is interesting to note that the tensile strength of the third digital flexor tendon is the highest in laying breed hens, the second highest in broiler breed hens, and the lowest in broiler breed cocks [26]. The male chickens of group A were affected with tenosynovitis as frequently as with rupture of the tendon. This suggests that there may be a sex disposition in the incidence.

As described in the Introduction, only the rupture of the gastrocnemius tendon has frequently been observed in young and adult broiler and layer chickens since 1944 [1-3, 5, 9, 10, 13-15, 19]. Most of the pathological changes of this disorder were similar to those manifested by the birds of group B. Above all, marked nodular thickening and fibrosis of the tendon were frequently noticed [1, 3, 5, 13-15]. It is not generally believed that tendon cells contribute very much to the repairing process of severed tendons. Most of the fibroblasts that repair the tendon come from the inner tendon sheath. If there is no such sheath, they come from the loose connective tissue at the periphery of the tendon [6]. A recent report characterized a reovirus isolated from ruptured gastrocnemius tendon [14]. It was revealed that a reovirus producing tenosynovitis had already been reported in 1957 [20] and 1954 [22]. Accordingly, tenosynovitis and rupture of the gastrocnemius tendon may be categorized into the same disease entity.

The alterations of muscles and peripheral nerves in the present cases are considered secondary changes of tenosynovitis and the complete or partial rupture of the tendon. Muscle lesions almost identical to those seen in this study have been described in broiler chickens affected with rupture of the gastrocnemius tendon [3, 5, 10, 19]. The endarteritis obliterans observed here is thought to be a histological manifestation occurring in the chronic inflammation and not to be pathognomonic. Similar arterial lesions have been found in the chronic type of experimental viral tenosynovitis [17] and in birds affected with rupture of the gastrocnemius tendon [19]. The enlargement of the hyaline cartilage near the distal end of the gastrocnemius tendon seems to be characteristic of a more chronic phase. This

disorder is characterized by a proliferation of mesenchymal tissue in the tendon and its sheath. It is generally known that the tendon cells near the insertion of the tendon exhibit certain properties of the cells that produce cartilage or bone [6].

References

- [1] Bullis, K. L., and van Roekel, H. (1944). Uncommon pathological conditions in chickens and turkeys. *Cornell Vet.*, **34**, 312-319.
- [2] Carnaghan, R. B. A., and Hanson, B. S. (1958). Rupture of the gastrocnemius tendon in fowls. *Brit. vet. J.*, **114**, 360-366.
- [3] Chute, H. L. (1950). Rupture of the gastrocnemius tendon in chickens. *Canad. J. comp. Med.*, **14**, 218-221.
- [4] Dalton, P. J., and Henry R. (1967). Tenosynovitis in poultry. *Vet. Rec.*, **80**, 638.
- [5] Devos, A. (1963). Rupture of the gastrocnemius tendon in chickens. *Avian Dis.*, **7**, 451-456.
- [6] Ham, A. W. (1965). In *Histology*, 5th Ed., J.B. Lippincott Co., Philadelphia and Montreal, 251-254.
- [7] Hanson, B. S., and Roberts, D. H. (1967). Tenosynovitis in broilers. *Vet. Rec.*, **81**, 329.
- [8] Harris, A. H. (1963). Rupture of the gastrocnemius tendon in the turkey (*Meleagris gallopavo*). *Vet. Rec.*, **75**, 969-970.
- [9] Harris, S. T. (1947). Rupture of the tendon achilles in chickens. *Vet. J.*, **103**, 356-357.
- [10] Ishiguro, H. (1974). Rupture of the gastrocnemius tendon in breeding flocks of meat type chickens: Pathogenesis. The 73th Meet. Jap. Soc. Vet. Sci.
- [11] Itakura, C., Kurisu, K., and Goto, M. (1976). Histopathology of purulent arthritis of chickens. *Jap. J. vet. Sci.*, **38**, 451-459.
- [12] Johnson, D. C. (1972). Diagnosis, pathology, and etiology of tenosynovitis in broilers and broiler breeders. *Avian Dis.*, **16**, 1067-1072.
- [13] Johnson, D. C., and van der Heide, L. (1971). Incidence of tenosynovitis in Maine broilers. *Avian Dis.*, **15**, 829-834.
- [14] Jones, R. C., Jordan, F. T. W., and Lioupis, S. (1975). Characteristics of reovirus isolated from ruptured gastrocnemius tendons of chickens. *Vet. Rec.*, **96**, 153-154.
- [15] Jordan, F. T. W. (1955). Rupture of the gastrocnemius tendon in the fowl. *Vet. Rec.*, **67**, 548-551.
- [16] Kerr, K. M., and Olson, N. O. (1967). Pathology in chickens experimentally inoculated or contact-infected with *Mycoplasma gallisepticum*. *Avian Dis.*, **11**, 559-578.
- [17] Kerr, K. M., and Olson, N. O. (1969). Pathology of chickens experimentally inoculated or contact-infected with an arthritis-producing virus. *Avian Dis.*, **13**, 729-745.
- [18] Moorhead, P. D., Cross, R. F., and Henderson, W. (1967). Pathological manifestations of experimental infectious synovitis and staphylococcosis in chickens. *Avian Dis.*, **11**, 354-365.
- [19] Nishimura, H., Fujie, Y., and Tsuchiya, T. (1976). Rupture of gastrocnemius tendon in broiler chickens. *J. Jap. Soc. Poult. Dis.*, **12**, 176-181 (in Japanese).
- [20] Olson, N. O. (1972). Viral arthritis. In *Diseases of Poultry*, 6th Ed., Hofstad, M. S., editor, Iowa State University Press, Ames, 756-760.
- [21] Olson, N. O., and Solomon, D. P. (1968). A natural outbreak of synovitis caused by the viral arthritis agent. *Avian Dis.*, **12**, 311-316.
- [22] Olson, N. O., and Weiss, R. (1972). Similarity between arthritis virus and Fahey-Crawley virus. *Avian Dis.*, **16**, 535-540.
- [23] Rossi, C., Babini, A., Beccaria, E., and Guarda, F. (1969). Isolation of an avian teno-synovitis viral agent. Virological and histological researches. *Atti. Soc. ital. Sci. vet.*, **23**, 1049-1052 (in Italian with English summary).
- [24] Sato, S., Shoya, S., Horiuchi, T., and Nonomura, I. (1972). An outbreak of synovitis caused by *Mycoplasma gallisepticum* in chickens. *Nat. Inst. anim. Hlth Quart.*, **12**, 54-62.
- [25] van der Heide, L., Geissler, J., and Bryant, E. S. (1974). Infectious tenosynovitis: Serologic and histopathologic response after experimental infection with a Connecticut isolate. *Avian Dis.*, **18**, 289-296.
- [26] van Walsum, J. (1975). Contribution to the aetiology of synovitis in chickens, with special reference to non-infective factors. II. *T. Diergeneesk.*, **100**, 76-83.

Explanation of Figures

- Fig. 1. Bird exhibiting a unique clinical symptom of standing on tiptoe. 181 days old (No. 13).
- Fig. 2. Complete rupture of gastrocnemius tendon with hemorrhage and edema. 70 days old (No. 7).
- Fig. 3. Marked thickening and fibrous adhesion of gastrocnemius tendon and its surrounding tissue with nodular thickening in the tendon-muscle junction (arrow). 235 days old (No. 26).
- Fig. 4. Acute lesion of gastrocnemius tendon sheath. Fibrinous exudation on the synovial membrane, with fibroblastic proliferation and formation of new blood vessels in the subsynovial layer. 235 days old (No. 28). Hematoxylin and eosin staining (H-E). $\times 236$.
- Fig. 5. Subacute lesion of gastrocnemius tendon sheath. Marked proliferation of fibroblasts and mesenchymal cells in the subsynovial layer. Tendon is seen in the lower area. 70 days old (No. 9). H-E. $\times 118$.
- Fig. 6. Acute lesion of gastrocnemius tendon sheath. Swelling of synovial cells with slight proliferation of fibroblasts in the subsynovial layer. 235 days old (No. 28). H-E. $\times 465$.
- Fig. 7. Subacute lesion of gastrocnemius tendon sheath. Hyperplasia of synovial cells and fibroblastic proliferation with formation of new blood vessels in the subsynovial layer. Tendon is seen in the lower area. 235 days old (No. 25). H-E. $\times 284$.
- Fig. 8. Chronic lesion of gastrocnemius tendon sheath. Marked proliferation of juvenile fibrocytes with newly formed blood vessels and edema in the subsynovial layer. 235 days old (No. 20). H-E. $\times 236$.
- Fig. 9. Chronic lesion of gastrocnemius tendon sheath. Marked proliferation of juvenile dense fibrous tissue in the subsynovial layer. Tendon is seen in the lower area. 181 days old (No. 16). H-E. $\times 66$.
- Fig. 10. Fibroblastic proliferation with slight infiltration of plasma cells and lymphocytes in gastrocnemius tendon. 70 days old (No. 9). H-E. $\times 236$.
- Fig. 11. Enlargement of hyaline cartilage at distal end of gastrocnemius tendon. 181 days old (No. 13). H-E. $\times 46$.
- Fig. 12. Nodular fibrous thickening in gastrocnemius tendon-muscle junction (see Fig. 13). 235 days old (No. 19). H-E. $\times 7$.
- Fig. 13. High-power magnification of Fig. 12 showing juvenile fibrocytes. 235 days old (No. 19). H-E. $\times 284$.
- Fig. 14. Nodular fibrous thickening with partial rupture in gastrocnemius tendon-muscle junction. There are fibrinous exudation, hemorrhage and proliferation of granulation tissue. 235 days old (No. 24). H-E. $\times 7$.
- Fig. 15. Endarteritis obliterans in a medium-size artery in the subsynovial layer of gastrocnemius tendon sheath. 70 days old (No. 7). H-E. $\times 236$.
- Fig. 16. Proliferation of fibroblasts and immature fat cells with formation of new blood vessels in the interstitial connective tissue of gastrocnemius muscle adjacent to the tendon. Swelling and hyaline degeneration of muscle fibers with increase of nuclei (arrow). 71 days old (No. 12). H-E. $\times 236$.
- Fig. 17. Swelling and vacuolar degeneration of muscle fibers, and proliferation of interstitial connective tissue in gastrocnemius tendon-muscle junction. Tendon is seen in the lower area. 235 days old (No. 26). H-E. $\times 142$.
- Fig. 18. Atrophy of muscle fibers with marked proliferation of fat cells in the interstitial connective tissue of M. biceps femoris. 181 days old (No. 13). H-E. $\times 284$.
- Fig. 19. Focal swelling and fragmentation of myelin sheaths (arrows) in ischiatic nerve. 235 days old (No. 19). H-E. $\times 515$.
- Fig. 20. Swelling, fragmentation and irregular atrophy of axis cylinder (arrow) in ischiatic nerve. 235 days old (No. 19). Bodian staining. $\times 515$.
- Fig. 21. Focal loss of nerve fibers with the appearance of scavenger cells in nerve fiber bundle around gastrocnemius tendon. 235 days old (No. 21). H-E. $\times 284$.