

## Experimental Study on Repair of Lacerated Tendons<sup>\*)</sup>

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### ABSTRACT

The distribution of intratendinous vessels of the flexor tendon of chickens was observed by microangiography using the ink injection method, and it was confirmed that the distribution was similar to that in man. Semi-tranverse excision of the tendon was performed at a site with an abundance of vessels and to healing process was observed in four groups classified as follows: Those in which the tendon sheath was intact, those in which it was excised, those in which circulation was intact and those in which it was obstructed. In the sheath and circulation intact groups, the endotenon was found to primarily fill the gap at the site of laceration, there was no thickening of the tendon and the tendon was restored to normal earlier. However, in the sheath excised and circulation obstructed groups, there was excessive proliferation of the epitenon, generalized thickening of the tendon and although there was filling of the gap with epitenons, maturation of granulation tissue was markedly delayed. Thus, it was learned that intratendinous circulation is highly important for healing of the tendon. Therefore, a study was undertaken to observe microangiographically the effects on intratendinous vessels of such tendon suture procedures as the Bunnell Method, the Modified Kessler Method and Tsuge Method. Results revealed that the Tsuge Method did not injure the intratendinous vessels, and thus was considered to be one of the outstanding tendon suture procedures.

### INTRODUCTION

Generally, the objective of treatment of injured tendons is to prevent adhesion between the sutured site of the tendon and surrounding tissue so that normal gliding of the sutured tendon can be obtained. However, it is well known that the degree of adhesion is dependent upon such things as the type of injury, treatment administered to the sheath, tendon suture procedure and length of postoperative immobilization. According to Matthews<sup>17)</sup>, adhesion can be prevented or limited to the minimum by one or a combination of two of the following: excision of the synovial sheath, tendon suture (Bunnell Method) and immobilization, but when all three are combined, strong adhesion develops. However, these three procedures are indispensable in the treatment of injured tendons, and thus at present, hope is being

placed upon the development of improvement in the techniques<sup>8)</sup>.

On the other hand, various experimental studies are being made in the area of tendon tissue repair. Poteza (1962)<sup>22)</sup> claims that injury of the tendon is repaired by fibroblasts derived from the surrounding tissue cannot be avoided. However, according to Lindsay (1960)<sup>9)</sup>, the tendon tissue has an intrinsic potential for repair, and that cells of the epitenon and endotenon layers play this role. Subsequently, there have been many reports supporting the latter theory<sup>5, 10, 16, 20, 24, 27)</sup>. At present, it has been confirmed that the tendon tissue definitely possesses intrinsic potential for repair so long as it has an adequate supply of nourishment. Further, the intra-tendinous vessels and diffusion of synovial fluid through the sheath have been found to be two such nutrient supply routes<sup>10, 12, 18)</sup>.

<sup>\*)</sup> 堀 司郎 : 損傷腱治療に関する実験的研究

Thus, the authors studied the effects of presence or absence of intratendinous circulation and synovial sheath on intrinsic potential tendon repair, particularly the effects upon the epitenon and endotenon, and reviewed the effects of the currently used clinical procedure, i.e., the Bunnell Method, Modified Kessler Method<sup>7)</sup> and Tsuge Method<sup>25, 26)</sup>, on tendon circulation.

## MATERIALS AND METHODS

### 1) Materials

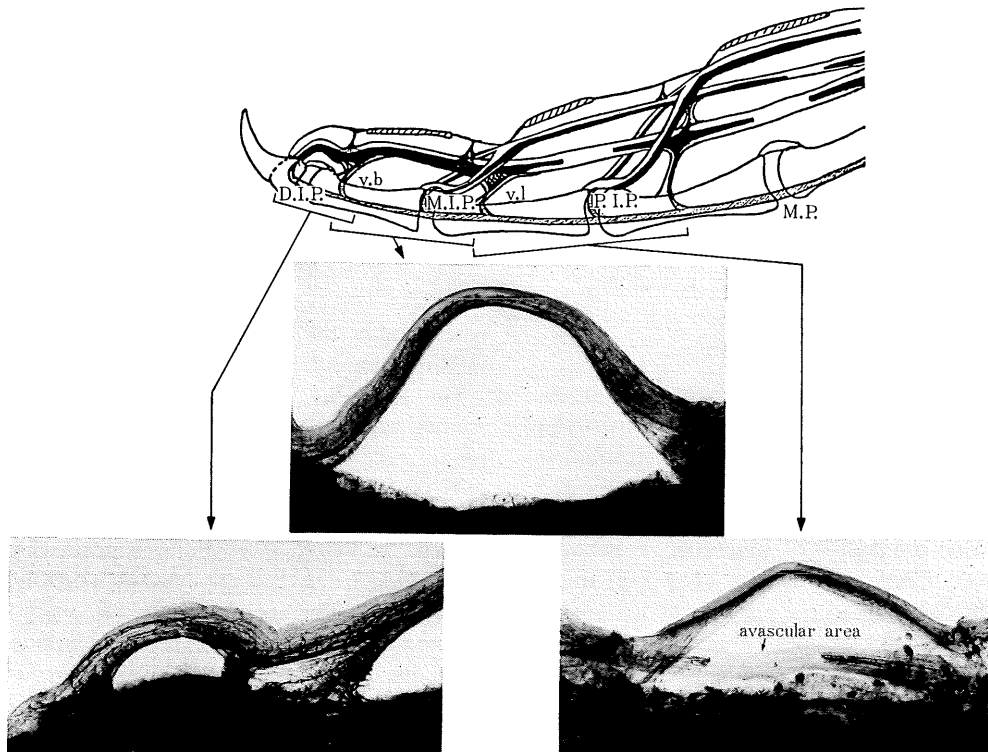
Used as experimental animals were 80 chickens, 2-3 months old, weighing 2-3 kg. There is a close anatomical resemblance between the chicken toe and the human finger<sup>1, 4)</sup>. That is, as the middle toe of the chicken has 4 phalanges as shown in Fig. 1, it has two tendons which correspond to the sublimis tendon in man. The profundus tendon used in the experiment is

covered with a synovial sheath from the osteotendinous junction to the head of the metatarsal bone, has vincula at the distal portion of the 2nd and 3rd phalanges and also a mesotenon which divides the tendon at the upper portion of the neck of the proximal phalanx. These vincula and mesotenon are distributed to the sublimis tendon and are attached to the synovial sheath by the mesotenon structure. That is, these vincula and mesotenon arise from the gliding floor, become connected to epitenon of the superficial layer of the tendon and then to the parietal layer of the synovial sheath forming a bendback structure<sup>1, 6, 24)</sup>.

### 2) Methods

1. Observation of vascular distribution in the normal tendon (20 chickens)

First, the chicken was anesthetized with intramuscular injection of 0.1 mg of atropine



**Fig. 1.** Structure of middle toe of chicken and microangiography of tendon

Intratendinous vessels of the flexor digitorum profundus tendon are well developed between the insertion at the distal phalanx and vincula breva, and between the vincula breva and vinculum longum excluding 1/3-1/4 of the tendon on the palmar side. However, an avascular area is found in approximately the central part of the vascular distribution between the vinculum longum and the mesotenon of the upper portion of the neck of the proximal phalanx,

sulfate and 50 mg/kg of pentobarbitalum, after which 3 ml of heparin was injected intravenously. Next, catheters were introduced into the bilateral femoral arteries. To 1,500 ml of physiological saline were added 4% gelatin and 10% indian ink, which was placed in an incubator bath heated to 45°C. This was perfused into the body of the chicken at a constant pressure of 120–150 mmHg via the catheter. After Confirming that the toes were well stained, the metatarsal region was ligated, and then amputated at an immediately proximal site, and immersed in 10% formalin solution kept in a refrigerator. After the toe has been fixed in formalin, the flexor tendon of the 3rd toe was removed under an operating microscope, and following deaquation and dealcoholization, it was immersed in 1:1 solution of methyl-salicylate and benzylbenzoate to prepare a transparent specimen.

2. Observation of tendon repair process over time (40 chickens)

Operation was performed under an operating microscope. The chickens used in this experiment were divided into four groups consisting of combinations of sheath intact and excised, and tendon circulation intact and obstructed (Fig. 2). First, a zig-zag incision according to Bruner's technique<sup>3)</sup> was made along the midline of the 3rd phalanx of the middle toe and the sheath was exposed. In the sheath intact

group, it was severed transversely slightly on the proximal side of the DIP joint which is located between the 3rd and 4th phalanges, the profundus tendon was drawn out exercising care so as not to injure the vincula breva, and the tendon was excised halfway on the plantar side at about the midsection between the vincula breva and vinculum longum. In the intratendinous circulation obstructed group, the whole circumference of the tendon was ligated twice with 6-0 nylon suture at the insertion of the vincula breva, next the sheath was excised transversely slightly on the proximal side of the MIP joint located between the 2nd and 3rd phalanges, and the tendon was similarly ligated at the insertion of the vinculum longum. In the sheath excised groups, it was excised to an extent whereby the vincula breva and vinculum longum were adequately exposed, the tendon was excised transversely half-way and circulation was obstructed similar to that in the sheath intact group. Postoperative immobilization was not administered. The postoperative repair process was observed over a period of 3 days to 12 weeks, during which histological studies were also made. Staining was performed using the H-E method and a modified Masson-Schaefer method.

3. Effects of various tendon suture methods on intratendinous vessels (20 chickens)

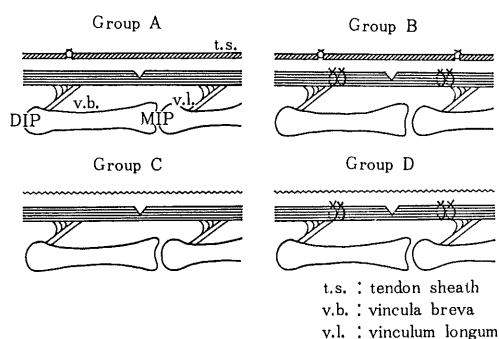


Fig. 2. Group A: Synovial sheath and circulation both intact, Group B: Sheath intact and circulation obstructed, Group C: Sheath excised and circulation intact, Group D: Sheath excised and circulation obstructed.

The obstruction of circulation in Groups B and D was performed by ligating the vincula breva and vinculum longum twice each at the respective tendon insertions.

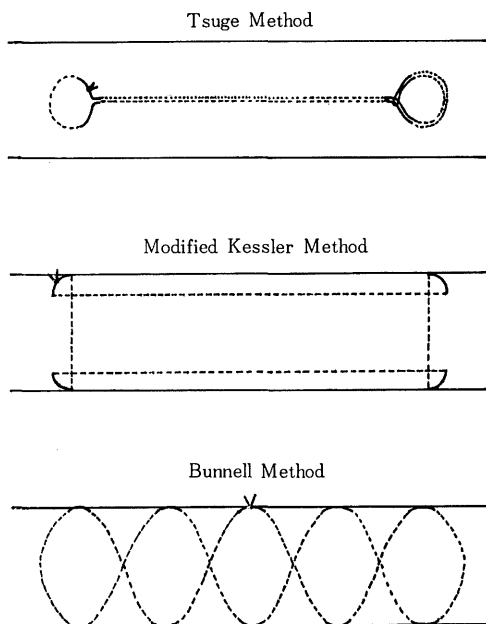


Fig. 3. Various tendon suture methods

The profundus tendon of chickens were exposed similar to that of the tendon intact group in Experiment 2, and without excising it, it was sutured at about the middle between the vincula breva and longum by the Tsuge, Modified Kessler and Bunell Method (Fig. 3). On the 2nd day after surgery, 3 ml of heparin was injected intravenously and both legs were amputated generalized anesthesia. Then, physiological saline with indian ink was perfused from the leg artery, and transparent specimens of the suture site were prepared as in the case of Experiment I.

## RESULTS

### 1. Observation of vascular distribution in the normal tendon (Fig. 1)

The flexor tendon of the middle toe is linked with vessels via the distal phalanx insertion, vincula breva, vinculum longum, vincula-like mesotenon in the upper portion of the neck of the proximal phalanx and paratenon on the outside of the tendon sheath. In addition, it is supplied also with some vessels from the mesotenon which forms a membrane between the sheath at the abovementioned insertion and vincula.

The profundus tendon has vessels which run longitudinally within the tendon between the insertion of the distal phalanx and vincula breva, and vincula breva and vinculum longum, and these give off horizontal branches. Particularly, in the area about 2/3 from the dorsal aspect of the tendon there is a fine well-balanced network between vessels which run longitudinally and those which travel transversely to link with them, but in the area 1/3 to 1/4 from the palmar side there are hardly any vessels.

Near the mid-part of the vinculum longum and mesotenon of the neck of proximal phalanx, the vessels from the proximal side and those from the distal side converge in the middle of the tendon and form loop-like terminals, thus the area between the two groups of vessels are avascular. Review of the serial histological transverse specimens of the tendon at this area failed to demonstrated the presence of vessels.

### 2. Observation of the tendon healing process over time

#### 1) Macroscopic findings

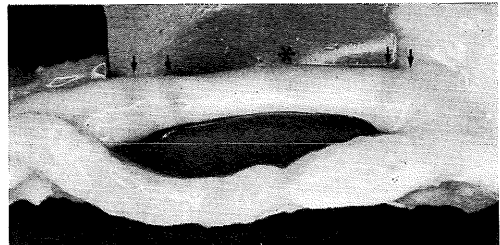
In Group A (Sheath-circulation intact), the excised area became filled in with yellow soft

granulation tissue on the 5th day, and subsequently this granulation tissue became localized and raised, but subsided on the 3rd week and the color became close to white. At 4-5 weeks, the surface became smooth and it was difficult to distinguish the area from the surrounding tendon tissue.

In Group C (Sheath excised-circulation intact), the excised site presented findings similar to those of Group A, but slight thickening persisted along the total length of the tendon in the area where the sheath was excised.

In Group B (Sheath intact-circulation obstructed), the excised site became filled in with granulation tissue, but the nylon suture used to obstruct circulation became embedded in proliferating superficial cells, and on the 11th day new vessels could be seen in these superficial cells and the cell layer became thickened. Thus, the whole tendon between the two sites of ligation became thickened, and the excised site became indistinguishable from the surrounding tissue earlier than Groups A and C. However, as the sheath was intact, there was a limit to the degree of tendon thickening, and on the 6th week, although there was thickening of the tendon, elasticity was not lost.

On the other hand, in Group D (Sheath excised), proliferation of the superficial cells of the tendon became marked from the 1st week thickly covering the whole tendon causing the surface to become irregular. After the elapse of four weeks, the proliferation gradually



**Fig. 4.** Group D—Macroscopic findings 4 weeks after operation

Adhesion between tendon and surrounding tissue is not seen. The ligating suture (↓) is embedded in proliferated cells of superficial layer of tendon, the whole tendon including the vincula is thickened, lacks elasticity and is firm. Distinction between the site of excision (\*) and surrounding tendon tissue is not appear.

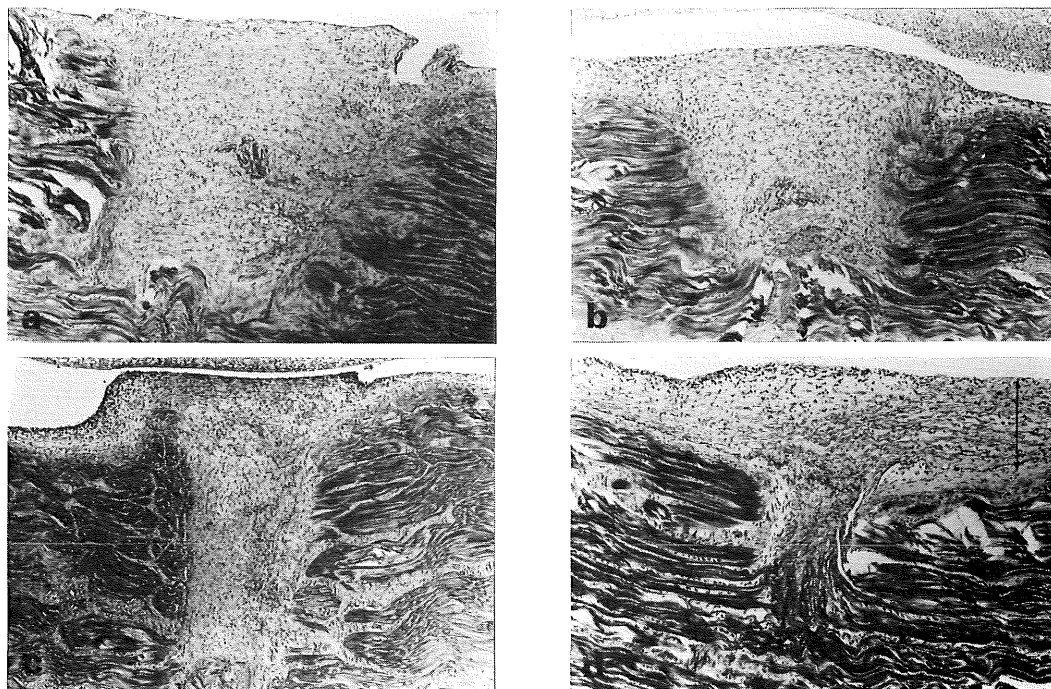
subsided, but different from the normal elastic tendon, it was firm (Fig. 4), and this finding persisted even after 12 weeks.

## 2) Histological findings

### Group A Sheath and circulation both intact

The deep area of the gap at the excised site was filled in with coagulated blood and phagocytes on the 3rd day after surgery. Although no proliferative changes in the epitenon were observed, the endotenon layer between the tendon bundle became enlarged and fibroblast proliferation as well as cell di-

vision were seen. At five days after surgery, there was marked proliferation of the fibroblasts of the endotenon which filled the gap at the excised site and the capillaries of the endotenon became large and clearly visible, but there was only slight cell proliferation in the epitenon. On the 7th day, the excised site was completely filled with granulation tissue. The endotenon was filled with proliferated fibroblasts and the new vessels were larger and became more numerous, and granulation tissue was filling in the gap at the excised site.



**Fig. 5.** Histological findings 1 week after operation (50 $\times$ )

#### a) Group A

Cells of the endotenon located in the tendon bundle proliferate as though pushing apart the tendon bundle at the site of excision and filling the gap. Proliferation of cells of the epitenon can also be seen, but it is localized.

#### b) Group B

Cells of the endotenon located in the tendon bundle did not proliferate and intratendinous vessels have disappeared. There is marked proliferation of the cells of the epitenon which are seen infiltrating into granulation tissue at the site of excision. Cells in the deep layer at the site of excision are sparse.

#### c) Group C

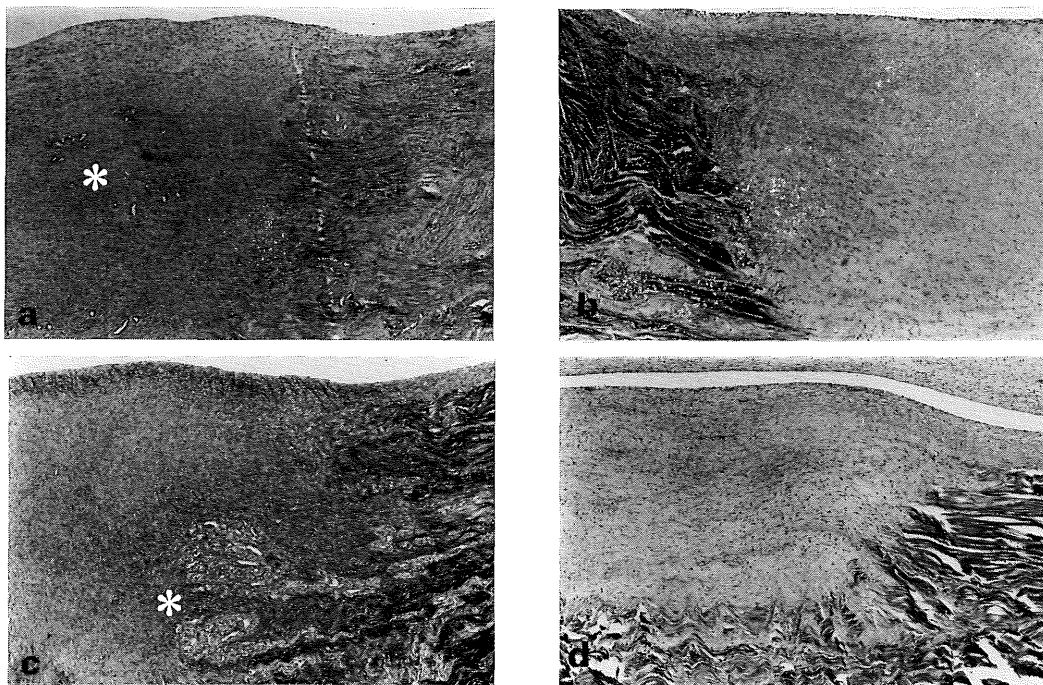
The cells of the endotenon in the tendon bundle proliferate together with the formation of new vessels, and become granulation tissue at the site of excision. Proliferation of the cells of the epitenon is also marked, and both cells take part in filling the gap.

#### d) Group D

Cells of the epitenon cover the whole tendon and continue to proliferate creating several ten layers in thickness ( $\longleftrightarrow$ ), which fill the gap. Cells of the endotenon become degenerated and decrease in number.

Although cell proliferation of the epitenon was slight, it combined with the cell proliferation of the endotenon and formed granulation tissue in the excised area (Fig. 5-a). Subsequently, numerous new capillaries were observed in the granulation tissue, and the cells of the granulation tissue of the excised site gradually started

to align in the direction of the long axis of the tendon. Production of collagen fiber was noted. At three and four weeks, the cell constituents of the excised site decreased, and although the newly produced collagen fibers were wavy, they were aligned almost parallel with the long axis of the tendon. Newly



**Fig. 6.** Histological findings 4 weeks after operation (40 $\times$ )

a) Group A

The cell component of the granulation tissue at the site of excision becomes decreased, and although wave-like in formation, the new collagen fibers run parallel with the long axis of the tendon. New vessels can be seen grouped together (\*) in the deep layer. Clear distinction cannot be made between normal tendon bundle and granulation tissue.

b) Group B

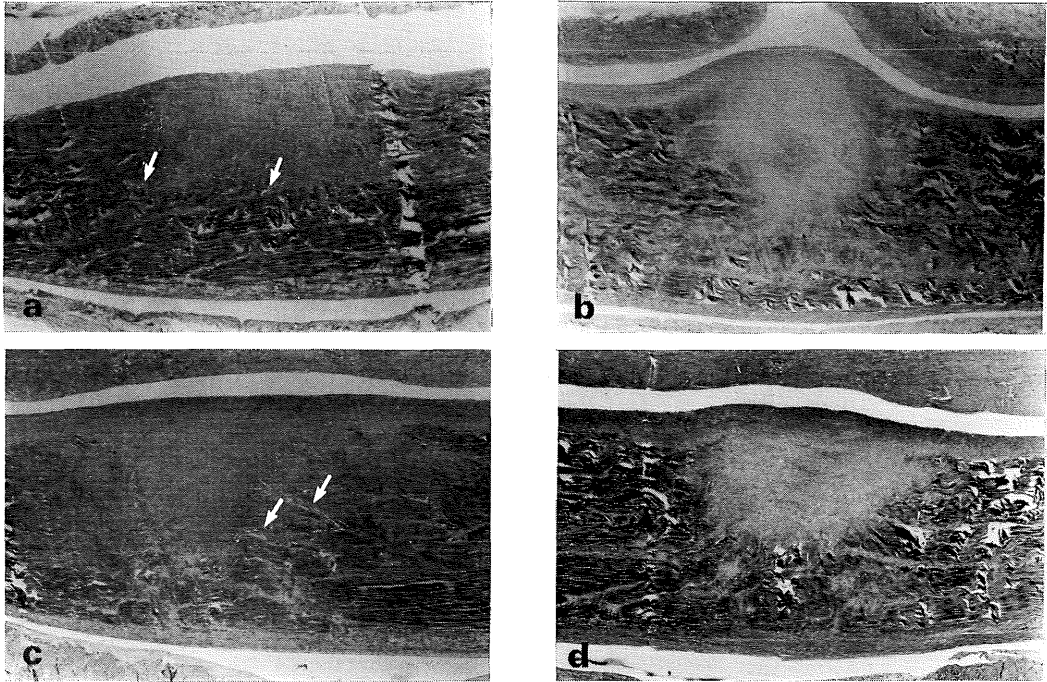
The fibroblasts and collagen fibers in the superficial layer at the site of excision are aligned parallel with the long axis of the tendon, but in the deep layer, the direction of the cells is irregular and the number are few, also the production of collagen fibers is poor. No intratendinous vessels can be seen.

c) Group C

The granulation tissue at the site of excision has an abundance of collagen fibers which link with the tendon bundle rendering it impossible to make distinction from the border between the normal tendon bundle. New vessels (\*) are formed in the deep layer. There is also marked proliferation of the epitenon.

d) Group D

The fibroblasts of the superficial layer in the granulation tissue of the site of excision align parallel with the long axis of the tendon, but in the deep layer the alignment is irregular and the cells are sparse. The border between normal tendon bundle and the site of excision is distinct. No intratendinous vessels can be seen.



**Fig. 7.** Histological findings 6 weeks after operation (20 $\times$ )

a) Group A b) Group B c) Group C d) Group D

In Group A and C, the new collagen fibers are large, firmly linked with normal surrounding tendon bundle, and there is a marked decrease in cell component. There is an abundance of new intratendinous vessels ( $\downarrow$ ), and the state is considered healed.

In Group B and D, new collagen fibers of the epitenon of the superficial layer at the site of excision bridge the site. However, in the deep layer, the direction of the cells are irregular and are yet immature, and the production of collagen fibers are poor. Clear distinction can yet be made with the surrounding tendon bundle. No intratendinous vessels can be seen.

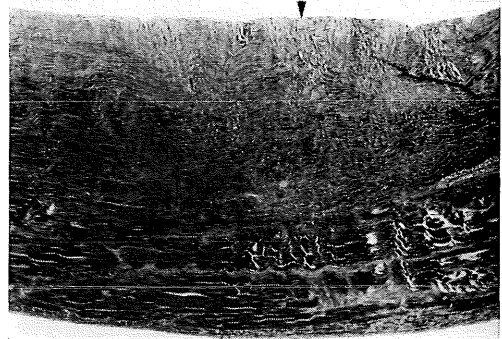
produced vessels were found in the deep layer of the excised site, and maturation of the granulation tissue began at the deep layer (Fig. 6-a).

After 5-6 weeks, the new collagen fibers increased in size and bound firmly together with the neighboring tendon bundle, the cell constituent decreased markedly, while blood vessels increased, and fusion of the tendon was almost complete (Fig. 7-a). Subsequently, the collagen fibers grew into normal tendon bundles, and the fibroblasts in between developed into new tenocytes. (Fig. 8)

Group B Sheath intact-circulation obstructed

There were no findings in the excised site gap on the 3rd day, but on the 5th day, it was filled with fibroblasts. Proliferation of the cells of the epitenon was not marked, but the cells were connected with the superficial cells

of the excised site. However, the cells of the endotenon were decreased, and their distribution



**Fig. 8.** Histological findings in Group A 11 weeks after operation (40 $\times$ )

The collagen fibers at the site of excision had become almost normal tendon bundles and fibroblasts had become tendon cells. Arrow indicates site of excision.

in the deep layer of the excised site was sparse. On the 7th day, the proliferating cells of the epitenon spread beyond the vincula and along the area ligated by sutures to obstruct circulation on to the excised site and covered the whole tendon. The gap at the excised site was filled in by proliferating cells from the epitenon. The cells in the superficial layer were densely aligned, but those in the deep layer were sparse (Fig. 5-b).

On the 11 day, new capillaries extending towards the excised site were clearly visible in the cells of the epitenon which had spread beyond the ligatures. At three and four weeks, some of these superficial vessels in the epitenon began to extend towards the inner side of the tendon, but the number was yet small. The fibroblasts and new collagen fibers in the superficial layer of the excised site were running parallel with the long axis of the tendon, but the direction of the cells in the deep layer was inconsistent, and the cells were few in number, also the production of collagen fibers was low (Fig. 6-b).

Subsequently, the number of superficial cells decreased, and in turn the new collagen fibers became larger in size and began to bridge the gap. Even at six weeks, the direction of the cells in the deep layer was inconsistent and there were few new collagen fibers (Fig. 7-b). However, here after individual difference developed, but generally by extension of reconstructed intratendinous vessels into the deep layer of the severed site, the cells in this layer matured and production of new collagen fibers improved, resulting in the repair of the tendon.

#### Group C Sheath excised-circulation intact

On the 3rd day, only coagulated blood and phlogocytes were observed in the excised site. As in the case of Group A, the cells of the endotenon in the tendon bundle, showed vessels within them, which were enlarged. On the 5th day, the excised site was still pitted, but in the deep area fibroblasts of the endotenon were present together with large vessels and formation of granulation tissue was seen. On the 7th day, there was excessive proliferation of cells of the epitenon covering the whole tendon and the excised site showed an abnormal elevation, which together with the cells of the epitenon filled the gap at the excised site (Fig. 5-c). This proliferation of the cells of the

epitenon continued, and new vessels were noted in the cells which began extending towards the excised site.

On the 3rd and 4th week, there was an abundance of vessels in the excised site, the fibroblasts gradually decreased and new collagen fibers began linking between tendon bundles running parallel with the long axis of the tendon (Fig. 6-c). Subsequently, the collagen fibers became thick, the fibroblasts became mature and transformed into tendon cells. At six weeks, the fibroblasts were still numerous, but it was considered that repair had been concluded (Fig. 7-c).

#### Group D Sheath excised-circulation obstructed

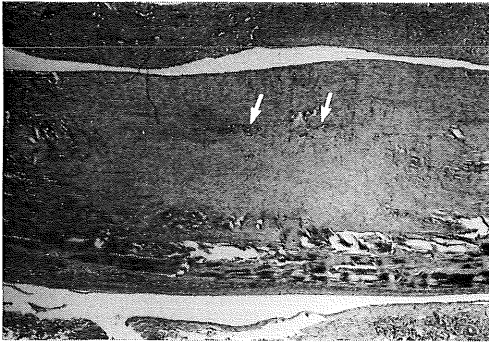
On the 3rd day, no change was noted in the excised tendon site. A small number of fibroblasts were observed mixed with the coagulated blood and inflammatory cells covering the cut end on the 5th day, but they were not abundant. On the 7th day, the cut end was covered with an abundance of cells of the epitenon which had proliferated markedly. These cells got around the ligation which obstructed circulation, proliferated to several ten layers and covered the whole tendon. The cells of the endotenon, on the other hand, became degenerated and decreased in number (Fig. 5-d). Proliferation of the cells of the epitenon, however, continued creating marked thickening. Some becoming twice the thickness of the normal tendon.

At the 3rd and 4th week, the superficial layer of fibroblasts on the cut end was seen running parallel to the long axis of the tendon, but the production of new collagen fibers was inadequate. At the deep layer, the size of the cells were irregular, the direction also was irregular and the number sparse (Fig. 6-d).

At the 6th week, the collagen fibers on the superficial layer were large and ran parallel to the long axis of the tendon, bridging the excised site. However, the cells in the deep layer were immature, and the production of collagen fibers was poor and the direction irregular (Fig. 7-d).

On the 12th week, restructured intratendinous vasculature began to appear at the excised ends, and fibroblasts in the deep layer became more mature and started to be replaced with new collagen fibers (Fig. 9).





**Fig. 9.** Histological findings in Group D 12 weeks after operation (20×)

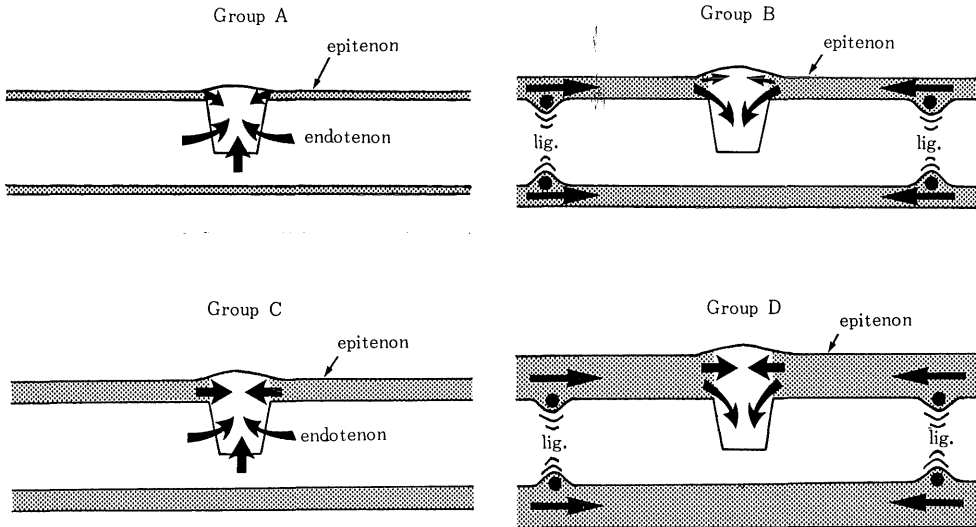
New vessels can be seen (↓) at site of excision, but production of collagen fibers in the deep layer is poor and cell components are voluminous. The distinction between normal tendon bundles is clear.

3) Summarization

From the above, it can be seen that there is a definite difference in the repair process at the excised site among the various groups. That is, the order of quickness in blast tissue maturation which infiltrated the excised site was A, C, B, D, and it was found that the

presence or absence of intratendinous circulation was greatly involved. In Group A and C in which circulation was intact, both the endotenon and epitenon played roles towards complete repair of the tendon, the former playing the primary and the latter the secondary roles. However, in Group B and D in which the tendon circulation had been impaired, the epitenon showed abnormal proliferation, and assumed the primary role in repair. The cells of the endotenon become degenerated due to impairment of nutrition and decreased in number. New vasculature was regenerated within the proliferated epitenon, which continued to develop into intratendinous vasculature, and maturation of cells at the excised site came to be noted (Fig. 10).

The difference between an intact and excised sheath as observed by comparison in the findings of Groups B and D in which circulation was obstructed showed that in Group B the excised site was already filled with cells on the 5th day after surgery due to proliferation of the epitenon, but in Group D only a small number of cells were seen sporadically at the excised site. However, after the 7th day, ex-



**Fig. 10.** Schematic diagrams of location and amount of cells which fill in granulation tissue at site of excision

In Groups A and C, proliferation of cells of the epitenon and endotenon fill in the granulation tissue at the site of excision.

In Groups B and D, the granulation tissue is filled in only with cells of the epitenon.

In Groups C and D in which the sheath had been excised, there is marked proliferation of the superficial cells of the tendon which constitute the epitenon.

cessive proliferation of the epitenon layer was observed (Fig. 5-d), and as the sheath which surrounds the tendon like a wall had been removed, proliferation of the epitenon was even further promoted. Further, since circulation was also obstructed, the tendon as a whole became thickened and firm, and the tendon lost elasticity (Fig. 4).

### 3. Effects of various tendon suture methods on intratendinous circulation

#### 1) Tendon suture methods

The various tendon suture methods were performed with 7-0 nylon suture without excising the tendon as shown in Fig. 3, after which it was replaced into the sheath. Although the tendon was not excised, it was sutured firmly enough by the respective methods to be able to withstand the tension created immediately after excision.

#### 2) Findings of indian ink injected specimens

From external appearance, the Tsuge Method did not have any ill effects on the tendon per se nor on the vessels of the superficial layer. The suture grasps only a portion of the tendon bundle on the palmar side, while the other vessels including those on the dorsal aspect

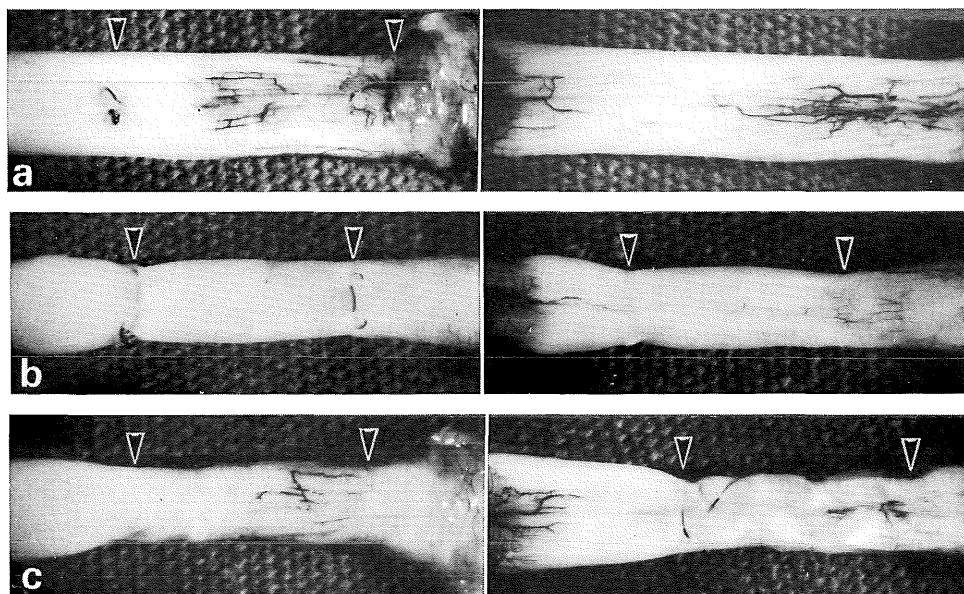
remain the same as normal (Fig. 11-a).

In the Modified Kessler Method, there were slight depressions at the grasp of the tendon bundle as indicated by 4 arrow points, but there was no effect upon the vessels in the superficial layer of the tendon (Fig. 11-b).

However, in Bunnell's Method, the width of the whole tendon was reduced and the superficial vessels were obstructed and strangulated. Blockage of the superficial vessels affects the dorsal vessels, causing compression of not only the suture site but also the tendon as a whole (Fig. 11-c).

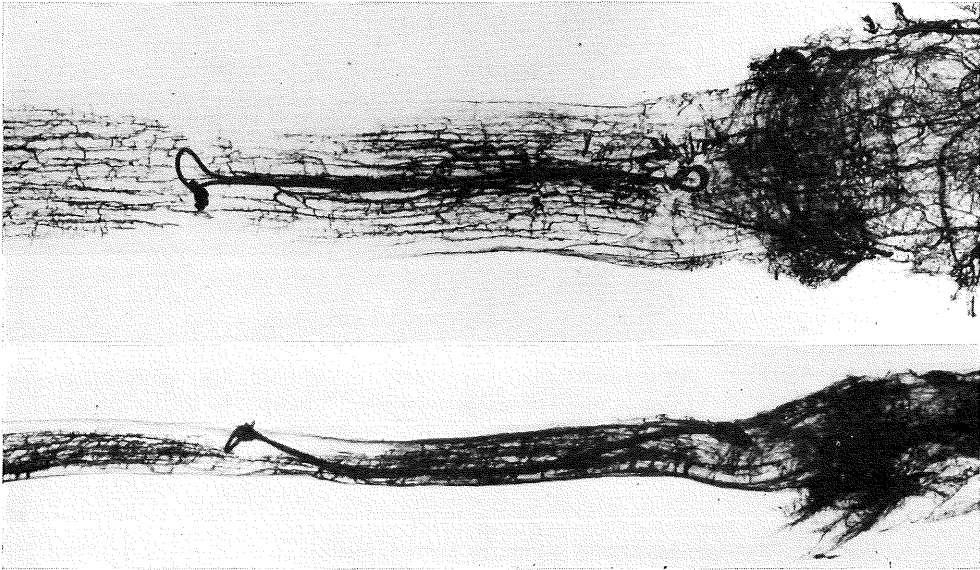
Findings of the transparent specimens showed that in the Tsuge Method, the intratendinous vessels distributed from the vincula breva and vinculum longum were not affected and presented a distribution pattern the same as normals, and there was an abundance of vessels at the suture site (Fig. 12).

In the Modified Kessler Method, slight strangulation of the tendon width at the four grasp points of the tendon bundle could be observed. There were effects on some of the sutured that transversed at right angle with the long axis of the tendon, or route of the vessels, but there



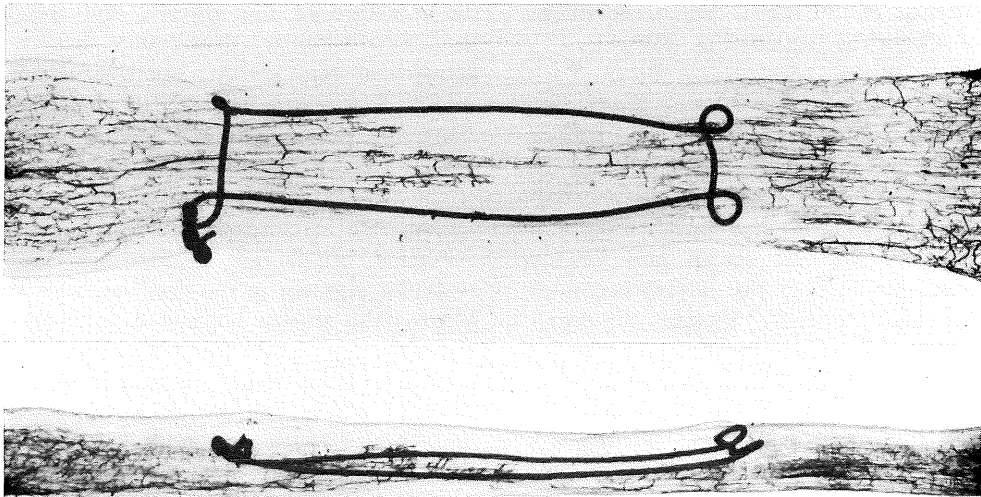
**Fig. 11.** External view of the tendon after suture by a) Tsuge Method, b) Modified Kessler Method and c) Bunnell Method. (Arrow shows suture site)

The superficial vessels of the tendon were not affected by the Tsuge Method nor Modified Kessler Method, but strangulation of the tendon occurred with the Bunnell Method, obstructing the vessels. (left: palmar aspect, right: dorsal aspect)



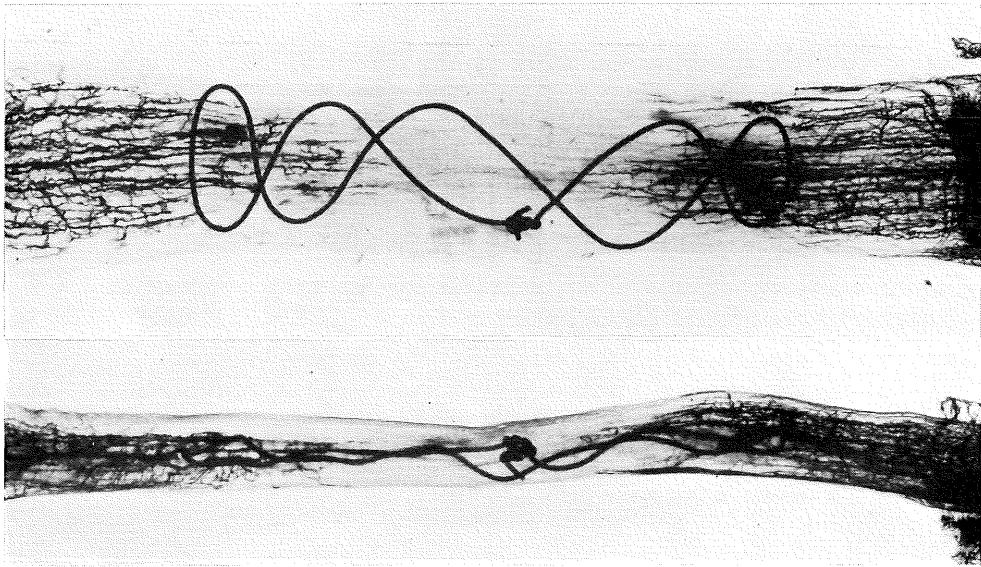
**Fig. 12.** Microangiogram after suture by Tsuge Method

The intratendinous longitudinal vessels are not obstructed and present a distribution which does not differ from normal. There is an abundance of vessels in the suture site, (upper: A-p view, lower: lateral view)



**Fig. 13.** Microangiogram after suture by Modified Kessler Method

Strangulation of intratendinous vessels is seen in the 4 grasp sites of the tendon bundle, but there is no affect on the vascular distribution, (upper: A-p view, lower: lateral view)



**Fig. 14.** Microangiogram after suture by Bunnell Method

The intratendinous vessels are obstructed by squeezing of the suture site, with bleeding occurring in some places, causing the site to become ischemic. (upper: A-p view, lower: lateral view)

were no effects on the vascular distribution of the suture site as a whole (Fig. 13).

In the Bunnell Method, there was compression of the tendon as whole because of ligation with the suture, and interruption or rupture resulting in hemorrhage in the vessels which run longitudinally within the tendon (Fig. 14).

## DISCUSSION

### 1. Distribuion of intratendinous vasculature

The profundus tendon of the middle toe of chickens is frequently used in experimental studies of the tendon because the relation ship of the tendon, synovial sheath and vincula closely resemble that in the flexor tendon of the human hand<sup>4,9,15,27,28</sup>. Further, the distribution of the intratendinous vasculature is also similar to that in man<sup>6,24</sup>. Therefore, the author observed in detail the distribution of the intratendinous vasculature in the chicken toe using the indian ink injection method. The intratendinous vasculature in the profundus flexor tendon runs from its osteo-tendinous junction at the distal phalanx to the vasculature of the vinculum longum, and consists of vessels which travel longitudinal to the long axis of the tendon and traverse vessels which link with them. These vessels are located on the dorsal side of the tendon, and thus the 1/3-

1/4 area on the palmar side is an avascular layer. These resemble the vascular distribution in man. According to Lundborg<sup>11</sup> and Matsui et al.<sup>14</sup>, blood circulation in the tendon within the sheath of the finger is supplied through the structure of the vincula, and the intratendinous vascular distribution has a fine network of anastomosing branches which pass among the vessels which run longitudinally along the dorsal aspect. These longitudinal vessels give off vascular loops in the direction of the palmar aspect, but they claim that the palmar side is composed of avascular layers.

Further, Lundborg<sup>11</sup> reports that there is an avascular segment in the distribution of vessels between the vincula breva and proximal synovial reflexion in the flexor digitorum sublimis tendon, and that there are poorly vascularized segments among the distribution of vessels in the flexor digitorum profundus tendon. Further, nourishment of this portion of the tendon and the avascular layer on the volar aspect of the tendon is supplied by diffusion of synovial fluid. In the flexor digitorum profundus tendon of a chicken, there are definite avascular areas intermediate between the vinculum longum and mesotenon at the upper portion of the neck of the proximal phalanx, and also in the intermediate area of the vascular distribution from

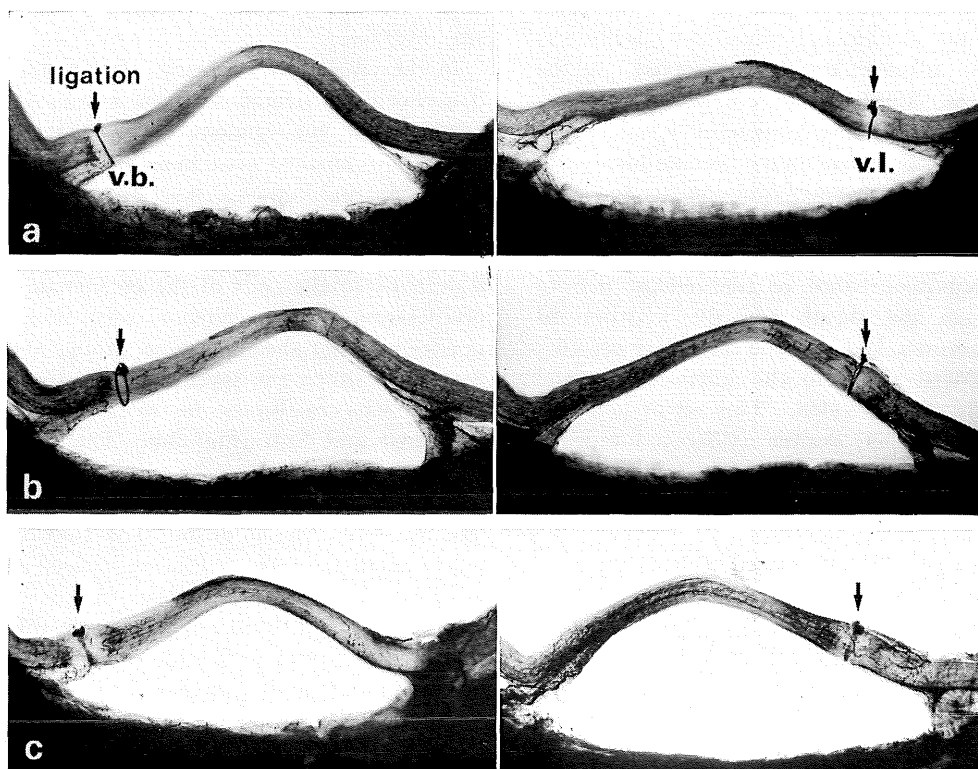
this mesotenon and the paratenon structure.

The presence of these avascular areas should be given special consideration when using chicken tendons for experiments in tendon repair. The author observed the distribution of intratendinous vessels, and used the vascularized area between the vincula breva and vinculum longum for this experiment.

With the purpose of ascertaining the intratendinous hemodynamics of this area, the vincula breva or vinculum longum of different chickens were ligated at their respective insertions with 6-0 nylon suture, and microangiography using the indian ink injection method was performed. The results are as shown in Fig. 15. On the 3rd day after ligation, the respective vessels from the vincula on the ligated side were obstructed, but a decrease in only a small area could be seen without complete disappearance of the vessels. On the 7th day, the vessels from the vincula on the non ligated side developed into

the poorly vascularized segment at the ligated side. By the 11th day, no difference between normal vascular distribution could be observed, and anastomosis between vessels of the epitenon layer which had passed over the surface of the ligating suture and intratendinous vessels was seen.

According to the experimental results of Smith<sup>23</sup>, Peacock<sup>21</sup> and Young<sup>30</sup>, blood flow from only one of the two is not sufficient enough for perfusion of the total length of the tendon in the sheath, and they advocate the need for segmental blood supply by the vincula. However, the experiments performed by Matsui<sup>14</sup> indicate that there are variation in the vincula of the human flexor tendons, and also there is a difference in vascular distribution by age. It was found that when the tendon is injured, the vincula breva proved to be most important, possessing a wealth of blood supplying pathways.



**Fig. 15.** Microangiogram after tendon ligation

On the 3rd day (a) after operation, the vessels from the vincula on the ligation side are obstructed and the number of vessels governed by it decreased, but on the 7th day (b), compensatory circulation from the vincula on the other side developed, and on the 11th day (c), the distribution of vessels no longer differed from that of normal tendons. (left: right toe, right: left toe)

The author's experiment was based on chickens, but the fact that there is an avascular area, suggests the possibility of segmental blood supply, but it is felt that between the vincula breva and vinculum longum where there is a wealth of vasculature, if the vessels on one-side should become injured, those on the other side would assume compensatory control.

## 2. Repair of lacerated tendons

In 1960, Lindsay<sup>9)</sup> reported on his detailed observation of the repair process of the flexor tendon of chickens, and emphasized that all cells composing the tendon were capable of producing immature cells. However, subsequently his theory was suppressed and neglected for a long time because of the theory advocated by Potenza<sup>22)</sup> that there can be no repair of the tendon without the occurrence first of adhesion to the surrounding tissue.

In 1974, Tokita<sup>24)</sup> demonstrated by a partial transverse resection of the tendon of a chicken that the epitenon alone is involved in the repair of the injured site, and when the sheath was intact, no adhesion could be observed. In the same year, Matthews<sup>16)</sup> performed a similar experiment using the tendon of a rabbit toe and stated the site of injury became filled with tenoblasts without any adhesion to the surrounding tissue. He also reported that the experimental results of Potenza were caused by nutritional disturbance of the tendon due to damage to the sheath and the insertion of tendon sutures, and that the secondary reaction thus induced shrouded the intrinsic potential for repair of the tendon. This repair potential was reported by Furlow (1976)<sup>9)</sup>, Lundborg (1976)<sup>10)</sup>, McDowell (1970)<sup>20)</sup> and Umeda (1978)<sup>27)</sup>, and thus this theory is now generally accepted.

Further Matthews<sup>18)</sup> placed a free fragment of tendon in the sheath and made observation over time. He noted that the superficial layer of the tendon was nourished by immersion in synovial fluid while the central portion received nutrition from circulation within the tendon. Lundborg<sup>10,12,13)</sup> returned a resutured tendon into the knee joint cavity and observed the repair process, through which he was able to demonstrate that the tendon can be repaired with synovial fluid alone. He claims that the fluid plays a role as nutrient of the superficial layer of the tendon not only in the normal state,

but also during the repair process as well. However, he noticed that there was nutritional disorder in the central portion of the tendon, and realized there was a limitation to the permeability of synovial fluid. That is, nutritional deficiency occurs with synovial fluid alone, and participation of the intratendinous vessels is also necessary.

Thus, in their study of tendon repair, the author observed the changes in the epitenon and endotenon which are the intrinsic cells of the tendon, in relation to the presence or absence of intratendinous circulation and the tendon sheath. Lindsay<sup>9)</sup> reports that at time of tendon repair, proliferation of the epitenons is the first to be observed, but in the circulation-sheath intact group, proliferation of the cells of the endotenon occurred earlier and in large number, and within the tendon was marked appearance of new vasculature. The endotenon assumed the primary role and the epitenon the secondary role in filling the gap of the lacerated site (Fig. 5a).

In the circulation obstructed group, the tendon which was ligated at 2 sites did not develop necrosis, and the epitenon layer on the outer side of the blocked area immediately began to proliferate and together with the new vasculature extended over the ligated site and filled the gap, while there was no proliferation, but a decrease in the cells of the endotenon. The proliferation of the epitenon was more pronounced in the sheath excised group, and the tendon in which the layer had proliferated to several tens of layers in thickness became thickened and firm (Fig. 5d). The speed in maturation of the granulation tissue that fills the lacerated site depends on how quickly the intratendinous vessels grow. The slowest was the circulation obstructed-sheath excised group, in which immature cells were still present in the deep layer 12 weeks after surgery (Fig. 9).

The sheath serves as the so-called tendon pulley and contains synovial fluid to ensure smooth gliding of the tendon. Also, as stated earlier, the role of synovial fluid as nutrient to the superficial layer of the tendon cannot be overlooked. Also the sheath serves as a wall to prevent the tendon from developing adhesion to the surrounding tissue during the repair process<sup>24)</sup>. In the author's experiment, there was a delay in epitenon proliferation up to the

5th day after surgery in the sheath excised group, but thereafter there was an excessive proliferation of the epitenon layer which completely covered the tendon. This phenomenon is considered due to temporary imbalance of supply of nutrition to the superficial layer of tendon caused by loss of synovial fluid, but thereafter since there is an excessive proliferation of the epitenon, it becomes possible to cope with the change.

From the above, it has been found that if the lacerated tendon is under the ideal conditions of having both circulation and synovial sheath intact, there will be no thickening of the lacerated site due to proliferation of epitenon and the gap will be filled primarily of endotenon cells which mature rapidly into a normal tendon. This indicates that in the treatment of injured tendons, it is most important to keep the supply of nutrition to the tendon intact. In order to accomplish this, a tendon suture method which does not impair tendon circulation is required and it is important to keep the sheath intact. Lundborg<sup>12)</sup> also draws similar conclusions from his experimental results on nutritional supply of the tendon.

Also Wray et al.<sup>28)</sup> carried out studies using the tendon of chickens in which the tendon was partially excised transversely and comparison was made of a group without immobilizing the site and those in which tendon suture and immobilization were performed. In the latter group, close adhesion with the sheath developed whereas in the former only slight adhesion occurred. They have also reported very good clinical results whereby they had left partially injured tendons untreated, and subjected the site to movement after several days of immobilization. It is felt that adhesion did not develop in the author's experiment because neither tendon suture nor immobilization procedures were performed.

### 3. State of intratendinous vessels after tendon suture

Matthews (1977)<sup>19)</sup> observed the changes in the cut ends of tendons after simple tenotomy and those in the intratendinous vessels after suture by Bunnell's method. The results showed that in tendon cut ends in which vessels could be observed, there was no adhesion to the surrounding tissue, whereas in those in which vessels were absent, there was adhesion.

In Bunnell's method, the suture causes the vessels in the suture site to disappear causing formation of very firm adhesion to the surrounding tissue. From these findings, he became aware of the strong relationship between the ischemic state of tissue and adhesion, and pointed out the need to devise a suture method which could withstand the proximal muscle pull and at the same time not impair circulation within the stumps.

Bergljung<sup>2)</sup> made comparison of the state of intratendinous vessel changes following tendon suture by the Bunnell and Mason-Allen Methods, and demonstrated that the Bunnell Method showed an ischemic state due to strangulation of the tendon.

In 1975, Tsuge<sup>25,26)</sup> introduced the intratendinous tendon suture as a method whereby the intratendinous vessels are not impaired. The procedure is simple, causes little damage to the cut ends and can be performed leaving the sheath intact<sup>20)</sup>.

Wray et al.<sup>29)</sup> made comparison of the state of tendon suture 4 weeks after operation by the Tsuge, Kessler, Kleinert and Bunnell Methods, and reported that although there was no difference in tendon tension, the Tsuge Method was slightly superior than the others with regards to tendon gliding.

In the author's experiment, the tendon was not severed and the synovial sheath was left intact, and only intratendinous suture was performed. Thus, the conditions of suture were different from those of the usual tendon rupture, but it was possible to observe the effects of tendon suture upon the intratendinous vessels. That is, in the Bunnell Method the suture is inserted into the center of the tendon and although no direct injury is inflicted upon the longitudinally routed vessels that run along the dorsal aspect, because it is a suture tying of the tendon, the intratendinous vessels are obstructed. On the other hand, the Modified Kessler Method has sutures which transverse the tendon, which when tightly ligated have effects upon the longitudinal vessels which cannot be neglected. However, in the Tsuge Method, as there are only two sutures which run along part of the tendon bundle on the palmar side of the tendon, no matter how strongly ligated there is no strangulation of the tendon. Thus, the blood vessels which are

primarily located on the dorsal aspect and run longitudinally between the tendon bundle receive hardly any effects from the suture nor the ligation. Therefore, it is considered that this method meets the points outlined by Matthews<sup>19)</sup>, and on the basis of the experiments of Wray et al.<sup>29)</sup> and those of the author, it is felt it is one of the ideal suture methods.

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