NEUROHISTOLOGICAL STUDY ON THE FEMORAL ARTERIAL OCCLUSION OF DOGS

by

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I INTRODUCTION

Recently acute arterial occlusion has been attracting more and more attention as a serious disease which requires urgent therapies. Usually surgical procedures such as embolectomy and sympathectomy have been performed at an early stage, however, it is still open to discussion that how could its prognosis be without treatment.

There have been many investigators studying on the histopathology of experimental acute arterial occlusion, but reports are scarce concerning the experimental works on the influences of such an occlusion upon the nervous system.

In this report the acute occlusion of femoral artery was made in dogs using melted paraffin and histological studies were made upon sympathetic ganglia and spinal ganglia within 9 weeks from the onset of femoral arteral occlusion. Then, the application of sympathectomy as a surgical treatment for acute arterial occlusion was discussed.

II EXPERIMENTAL METHODS

The materials used in this experiment were adult dogs. Under intravenous nembutal anesthesia, a rapid injection of desinfected and melted paraffin (m. p. 42-44°C) was carried out into the femoral artery on one side with a catheter or an injector. In order to avoid the injury of the femoral arterial wall, the author injected 0.5-1.0cc of the melted paraffin through a tree between the deep femoral artery and the anterior femoral artery. After the injection the tree was ligated near its root arising from the femoral arterial trunk. The paraffin coagulated rapidly and produced an extensive occlusion of the femoral arterial trunk. The stoppage of blood flow and the stiffness of blood vessel's wall were observed.

The blood circulation of affected leg was investigated by the arteriographic examination 1 to 4 weeks after the occlusion. Using 10cc of 76% urografin, X-ray picture of the femoral artery was taken in each case.

The animals were divided into two groups, namely sympathectomized and non sympathectomized, and in the former sympathectomy was performed either before or after the occlusion (Table I).

For the microscopic observation the thoraco-lumbar sympathetic ganglia (Th 6 to L.6) were resected 7-30 days after the occlusion, fixed in a 99% alcohol for a week, stained by NISSL's staining and H. E. staining method. The spinal ganglia of the thoraco-lumbar region (Th11 to L5) were removed 13-62 days after the occlusion, fixed in a 20%

Table 1

Non-Sympathectomized group
Group A : Occlusion of left or right femoral artery
Sympathectomized group
Group B : Left lumbar sympathectomy after occlusion of left femoral artery
Group C : Occlusion of left femoral artery after left lumbar sympathectomy
Group D : Occlusion of right femoral artery directly after left lumbar sympathectomy

neutral formalin for more than 3 weeks, sliced 25 microns thick frozen sections and impregnated with SUZUKI's modification of BIELSCHOWSKY's method, and other specimens were fixed in a 99% alcohol, stained by NISSL's staining.

III RESULTS

1. Macroscopic findings of occluded leg

In this experiment the condition of affected leg was independent of sympathectomy. Immediately after the occlusion the coldness of affected leg followed. One day after the occlusion the dog began to crriple, bent the knee and cried at a touch. On the 3 rd day the swelling of leg from knee to toe developed. In the earliest cases, gangrene of the toe occured on the 5 th day. Gangrene developed in the majority of dogs, gradually extended and the gangrenous part finally demarcated.

On the other hand the crripling and the swelling of the leg without gangrene began to disappear about 2 weeks after occlusion, and then the leg returned almost normal. As shown in the group B which had received lumbar sympathectomy 7-10 days after the occlusion, sympathectomy was not effective for the improvement of the gangrene which had already developed (Table 2).

2. Macroscopic findings of occluded artery

The slight adhesions between the occluded artery and its surrounding tissues were found after 7 days. Its color was dark reddish, its wall looked hard and thick. Its lumen was filled with a lot of paraffin-coagulation and clots. Paraffinmass was movable by finger-pressure, but the thrombi adhered firmly. The range of the occlusion in the femoral arterial trunk was 7-10cm in length (Fig. 1).

After 30 days the degree of adhesion increased. The small, winding, swollen collateral vessels were found in the neighborhood of the occluded artery. In the proximal part of the occluded artery many thrombi were found, and in the peripheral part coagulated paraffin distributed. The range of the occlusion was the same as found 7 days after the occlusion.

3. Arteriographic findings of occluded leg

a) Arteriographic picture of normal leg

The external iliac artery which arises from the aorta in front of the 4 th lumbar vertebra passes into the femoral artery. The latter branches into 3 main trees, namely the deep femoral artery, the anterior femoral artery and the saphenous artery until it

Table 2

Group	A							B						C			D		
Dog No.	25	27	28	29	31	33	34	21	22	24	26	30	32	35	36	37	23	41	42
Days from occlusion to onset of gangrene		6	8	10	7	5	10	12	10			6		10		18	10	6	
Range of Gangrene		a	t	t	k	a	t	a	t			t		a		t	t	t	
Development of collateral vessel	+	−	−	−	−	−	−	±	−	+	+	−	−	−	+	−	−	−	+
Daysfrom occl. to affected side	25	18	30	13	21	23	18	10	7	7	30	7	7	b 26	b 20	b 25	0	0	0
Sympathectomy opposite side		18	30		21	23	18	25	23	21	30	7	7	20	30	21	22	20	30
Daysfromoccl. to resection of spinal ganglia	25	18	30	13	21	23	18	25	23	62	37	26	23	20	30	21	22	20	30

a : Ankle. t : Toe. K : Knee. b : before Occlusion

passes into the popliteal artery. After giving off the posterior inferior femoral artery at the knee, the poplieal artery divides into the anterior tibial artery and the posterior tibial artery. The saphenous artery begins at the midway of the femoral artery. Its dorsal branch traverses obliquely the median side of tibia and runs along the dorsal surface of the foot. Its plantar one runs in the posterior part of the leg and divides into two branches of the plantar artery. In the posterior part of the thigh there is distributed the posterior gluteal artery arising from the internal iliac artery (Fig. 2, Fig. 2′).

b) Arteriographic picture of affected leg

In the non-sympathectomized group, the X-ray picture showed no arterial figure in the periphery of the femoral artery including the occluded part until two weeks after the occlusion. After 3-4 weeks, the X-ray picture showed that the femoral artery was interrupted about 5-10 cm corresponding to the occluded part and it anastomosed with the popliteal artery by colleteral vessels. In the thigh the posterior gluteal artery and the deep femoral artery take part in the formation of the collateral vessels. Their branches, anastomosing mutually like a tendril, are connected with the posterior inferior femoral artery. The part where the posterior inferior femoral artery anastomosed with the popliteal artery gave a swollen, dense figure (Fig. 3, Fig. 4). In one case, the posterior inferior femoral artery or the saphenous artery developed well for the formation of collateral vessels, but in 6 cases even 4 weeks after there was no collateral vessel in the leg below the knee (Fig. 5). The definite findings could not be obtained about the collateral circulation in the leg below the knee.

In the sympathectomized group the disappearance of arterial figure continued two weeks after the occlusion. The development of collateral vessel 3 - 4 weeks after the occlusion is similar to the non-sympathectomized group. One case (No. 21 dog) showed a remarkable development of collateral vessel in 4 weeks, but there was no other case, whose collaterals developed better than those of the non-sympathectomized group (Fig. 6).

4. Histological findings of sympathetic ganglia

LERICHE and others described that if there was angitis or thrombosis in an artery, arterial spasm might be evoked by the irritated symathetic nerve fiber. When embolus

once occurs in an artery, a severe circulatory disturbance will cover the whole in the region due to the angiospasm. Here is a question what influence has arterial occlusion upon the sympathetic nervous system for such a long period of time.

Hitherto, among the reports on the degeneration of sympathetic ganglion cell many studies have been done upon the change concerned with intoxication or various diseases. Especially in regard to the chronic arteriopathy such as BUERGER's and RAYNAUD's disease, there are many investigators, some of which observed the degeneration of ganglion cell. It must be noticed that some of these reports supposed the sympathetic nervous system acted a major role in the etiology of these diseases.

On the other hand REILLY proved experimentally that when autonomic nerve suffered from some attack, non-specific change might occur not only in the organ innervated by the nerve but also a remote organ. Then, many investigations were done on experimentally made angitis making use of REILLY phenomenon and tried to examine the pathological picture of peripheral arteriopathy at an early stage of vascular stimulation. However, concerning the problem whether an organic change appears in sympathetic ganglion or not under the stimulus of arterial occlusion, there was scarcely found any experimental investigation. The auther's opinion on this problem is as follows : sympathetic ganglion, kept in continuous excitement by means of widespread femoral arterial occlusion, may possibly cause the pathologic change of the sympathetic ganglion cells.

From the above mentioned viewpoint, sympathetic ganglia were histologically examined in all cases. Generally speaking, the characteristics of change in the sympathetic ganglia were vascular changes, while the degenerative change of ganglioncell was scarcely observed.

a) Vascular change

Concerning the vessels in a sympathetic ganglion of human being, HERZOG and others stated that although capillaries and lymphcanals were proved to be existent, it was difficult to find histologically them in their normal condition. As to the lumbar and thoracic ganglia of normal dogs, only a few capillaries with narrow lumen were found in each ganglion (Fig. 7, Fig. 8).

The following vascular change obtained by the author may be considered to belong to the pathologic change such as congestion and inflammation, because capillary dilatation, edema, increase of leucocyte and extravascular infiltration of leucocytes were observed.

In the case with slight degree of change the dilatation of capillaries and a slight increase of leucocyte appeared in the capillaries (Fig. 9, Fig. 10). In the case with severe degree of change edema and infiltration of leucocytes jointed them. The greater part of ganglion was occupied by numerous dilated capillaries, so that the groups of ganglion cells were scattered like islets among the dilated capillaries (Fig. 11, Fig. 12).

The degree of such a change was various and seemed to relate to the segment of the ganglion and to the duration of time after the occlusion (Table 3, Table 4).

In the first place the group A showed the following interesting data : in the thoracic ganglia (Th 6, Th 8, Th 11) all of 4 cases had no change, bnt in the lumbar (L. 2, L. 3, L. 4) some changes were localized without exception. Similar changes appeared also in the non-affected side regardless the position of the lumbar sympathetic ganglia.

Moreover the degree of such a change observed in the lumbar ganglia varied case to

Table 3 (Group A)

Dog No.	Days affter Occlusion	Removed ganglion: affected side	Removed ganglion: Height of Ganglion	Thoraco-Lumbar Sympathetic Gangiion: Vascular change: Capillary dilatation left	Capillary dilatation right	Edema l	Edema r	Increase of Leucocyte l	Increase of Leucocyte r	Infiltration of Leucocytes l	Infiltration of Leucocytes r	Degeneration of Cell: Change of nucleus l	Change of nucleus r	Hyperchromatic of Cell body l	Hyperchromatic of Cell body r
29	13	l	L 2	−		−		−		−					
			3	+		+		++		+		+			
			4	+		−		+		+					
27	18	l	Th 6	−		−		−		−					
			8	−		−		−		−					
			11	−		−		−		−					
			L 1	−		−		−		−					
			2	+	+	+	+	−	++	−	+		+		+
			3	+	+	−	−	++	−	+	−	+		+	
			4	+	+	+	+	+	+	−	−		+		
			6	−	−	−	−	−	−	−	−				
34	18	r	Th 6		−		−		−		−				
			8		−		−		−		−				
			11		−		−		−		−				
			L 1		+		−		−		−				
			2	+	++	−	+	+	+	−	−			+	+
			3	+	+	−	+	+	+	−	−				
			4	+	−	−	−	−	−	−	−				
			6		+		+		−		−				
31	21	l	L 2	+		+		++		+				+	
			3	+	+	−	−	+	+	+	−				
			4	+	+	−	−	+	+	−	−				
33	23	l	Th 6	−		−		−		−					
			8	−		−		−		−					
			11	−		−		−		−					
			L 1	−	−	−	−	−	−	−	−				
			2	++	+	+	−	++	+	+	+	+		+	
			3	+	+	−	−	+	+	−	−	+	+		
			4	+	−	−	−	++	−	−	−				
			6	−		−		−		−					
25	25	l	L 2	++		++		++		+		+		+	
			3	++		++		++		++		+		+	
			4	−		+		−		−					
28	30	l	Th 6	−		−		−		−					
			8	−		−		−		−					
			11	−		−		−		−					
			L 1	+		−		−		−					
			2	++	++	+	+	++	++	+	+	+		+	+
			3	++	+	−	−	+	+	+	+	+	+	+	+
			4	++	+	+	−	++	++	+	+				
			6	++	+	+	−	++	−	++	−				

Table 4 (Group B. C. D)

Dog No.	Days after Occlusion l	Days after Occlusion r	affected side	Height of Ganglion	Lumbar Sympathetic Ganglion: Vascular change: Capillary dilatation l	Capillary dilatation r	Edema l	Edema r	Increase of Ceucocyte l	Increase of Ceucocyte r	Infiltration of Ceucocytes l	Infiltration of Ceucocytes r	Degeneeation of cell: Change of nuclaus l	Change of nuclaus r	Hyperchromatic of Cell body l	Hyperchromatic of Cell body r
30	7	7	l	L 2	+	+	−	−	+	+	−	−	+			
				3	+	+	+	−	+	−	−	−				
				4	−	−	−	−	−	−	−	−				
32	7	7	l	L 2	+	−	+	−	+	−	−	−				
				3	+	+	−	−	+	+	−	−				
				4	−	+	−	+	−	+	−	−				
22	7	23	l	L 2	+	+	−	+	+	+	−	−				
				3	−	+	−	−	−	+	−	+				
				4	+	+	+	+	+	+	−	−		+		
24	7	21	l	L 2	+	+	−	+	+	+	−	+	+		+	
				3	+	−	+	−	±	+	+	−			+	+
				4	−	−	−	−	−	−	−	−				
21	10	25	l	L 2	+	+	+	+	+	+	−	−				
				3	−	+	−	−	−	−	−	−				
				4	−	−	−	−	−	−	−	−				
26	30	30	l	L 2	+	−	+	+	±	+	+	+				
				3	+	+	+	−	±	+	+	−	+		+	
				4	+	−	+	−	+	−	−	−				
35		20	l	L 2		+		−		−		−				
				3		−		−		−		−				
				4		−		−		−		−				
36		30	l	L 2		−		−		−		−				
				3		+		−		−		−				
				4		−		−		−		−				
37		21	l	L 2		−		−		−		−				
				3		+		−		−		−				
				4		−		−		−		−				
23		22	r	L 2		−		−		−		−				
				3		−		−		−		−				
				4		−		−		−		−				
41		20	r	L 2		−		−		−		−				
				3		+		−		+		−				
				4		−		−		−		−				
42		30	r	L 2		−		−		−		−				
				3		−		−		−		−				
				4		−		−		−		−				

case, for example No. 28 and No. 33 dog showed some changes in every ganglion, but in No. 29 and No. 34 dog some of the ganglia did not show any change. To take the left L. 2 for example, in all of 6 cases some changes were obsered with the exception of No. 29 dog (Fig. 13, Fig. 14).

From these data the ununiformity of change was found in the lumbar ganglia. The same fact was fitted to the bilateral ganglia of the corresponding segment.

In order to investigate more inferior ganglia than L. 4, the author took up L. 6 corresponding to S. 1 of human. Such a difference was observed in some degree in 2 of 4 cases in which all of the bilateral lumbar ganglia (L. 2, L. 3, L. 4 showed intensive changes (Fig. 15).

Concerning the relation between the degree of change and the duration of time after the occlusion, the results obtained were as follows : after one week, in all of 4 cases, slight changes appeared already in some of lumbar ganglia (Fig. 16, Fig. 17). In 20-30 days the degree of change and the number of pathologic ganglia increased (Fig. 18, Fig. 19).

On the other hand, in the group B, of which the left lumbar ganglia (L. 2 to L. 4) were resected 7-10 days after the occlusion and the right ganglia about two weeks later, the right of 3 cases showed slight changes and were similar to the left in regard to the degree of change. Therefore it may be considered that the change of the cases sympathectomized after the occlusion was not so severe as the one of the group A though it could not be reduced by lumbar sympathectomy on one side (Fig. 20).

Moreover in the group C and D, of which the left lumbar ganglia were resected either about 3 weeks or directly before the occlusion, slight changes were observed. Only congestion was found in 4 of 6 cases, but no change in the other two.

b) Degenerative change of sympathetic ganglion cell

The figure of initial stimulation by HERZOG was a common pathological change. The hyperchromatic of the cell body was found in all cases of which the vascular change was remarkable. Other pathologic change was scarcely observed in the majority of cases except the group A. In some cases among the group A, the change of nuclei such as the atrophy and the disappearance of nuclei, especially excentric nuclear location was found. But as other pathologic sign only atrophy of cell body was found at the same time (Fig. 21, Fig. 22).

5. Histological findings of spinal ganglia

In the beginning of the discussion about the degeneration of the spinal ganglion cell, it must be considered that nerve cells show individually various appearances and some are occasionally thought degenerative with NISSL's staining, while with silverimpregnating method as normal spinal ganglia. For example, sometimes NISSL's granules are arranged irregularly, and show the same change as tigrolysis or chromatolysis. Nuclei show especially various appearances, sometimes they vanish and look like a vacuole. Also the size of cell is various. As to nerve process someone is clearly impregnated, but the other is not. Occasionally cells are observed to be standing close or overlapping each other, therefore it is difficult to distinguish whom the processes belong to. Accordingly, in this study the number and the kind of degeneration were in question. In the cases where numerous

cells show a change or more than two kinds of degeneration appear simultaneously, they are diagnosed to be pathologic (Fig. 23).

a) Concerning the degeneration of spinal ganglia (Th11 to L5) following bilateral lumbar sympathectomy (L2 to L4)

Histological study on the spinal ganglia from Th11 to L5 was performed in 12 dogs which had undergone lumbar sympathectomy 3-40 days before, and the following results were obtained.

From 3 to 15 days after the sympathectomy the degeneration of nerve process and nerve fiber was seen in the spinal ganglia, especially in Th12 and L2 it was intensive, while in L5 none. More than 20 days after sympathectomy such a change could not be observed.

As a remarkable change of nerve process at the pericellular plexes, globular bodies appeared and they presented an increased argyroaffinity, about 10 microns in size and mostly round shape (Fig. 24, Fig. 25). In some case there was observed a partial hyperplasy of the body or a conjunctive fiber between the body and the nerve cell (Fig. 26).

HERZOG explained that the globular body would appear as a spherical phenomenon in the ending of nerve process, and they could be considered pathologic.

As other kinds of degeneration hypertrophy and hyperplasy of nerve process were observed. They are well known phenomenon described by STOEHR as disharmonious nerve process, and mostly accompanied with the spherical phenomenon and the fenestration between the cell and the nerve process (Fig. 27, Fig. 28, Fig. 29).

On the other hand, in the author's investigation on the normal spinal ganglia extending from Th11 to Co1, the above mentioned globular body was not found excepting S1 and S3, and even in those two ganglia only a few bodies were seen (Fig. 30).

Accordingly, the result that some pathologic changes appeared in the spinal ganglia (Th12 and L2) suggests the existence of a certain nerve fiber connecting the spinal ganglia to the sympathetic trunk, though it is unsolved whether the appearance of such a change in the spinal ganglia is specific following sympathectomy or not. Possibly they may be the figures of retrograde degeneration of visceral afferents which passed the sympathetic trunk.

b) Concerning the histological findings of spinal ganglia after the femoral arterial occlusion

The author studied on the problem whether the femoral arterial occlusion may have an influence on spinal ganglia to resolve the following two question : (1) if a secondary change of somatic sensory nerve is caused by circulatory disturbance in the leg, does it extend to spinal ganglia ? In other words, does spinal ganglion show a retrograde change ? (2) if the vasomotor nerve is disharmonious or in irritated state, does it have an influence upon spinal ganglia ? It seems certain that vasomotor nerve receives stimulus from the occluded artery. How does it respond to the stimulus ?

In the histological findings with NISSL's staining, pathologic change of the spinal ganglion cells were scarcely observed in all cases. As to the degeneration of nerve process, some pathologic change such as the increase of globular body, the hypertrophy and the hyperplasy of nerve process were found only in the group B and D, but in the group A

and C, such a change was not seen. Then, the degeneration of nerve process in the group B and D was analogous to the change which was found following lumbar sympathectomy and was not influenced by the femoral arterial occlusion (Fig. 31, Fig. 35).

From the data, it may be concluded that spinal ganglia after the arterial occlusion show neither retrograde change nor change due to the dysfunction of vasomotor nerve.

Ⅳ DISCUSSION

It has been believed, up to this time, that sympathetic nerve participates only in the conduction of efferent impulse, but it is not deniable that the nerve fibers with different functions are contained within the sympathetic trunk and they contract or dilate blood vessel by the suitable impulse in each occasion. OKINAKA stated that sympathetic nerve conducts not only efferent impulse but also afferent one. INOUE demonstrated the fact that by the continuous stimulation of lips, some pathologic changes were evoked in the caeliac sympathetic ganglia.

In the literatures concerning the vasomotor nerve innervating the leg, there are the reports proved that the vasodilatation of the leg was evoked by the irritation of dorsal root. KOZUKA affirmed the existence of vasomotor nerve in dorsal root.

The results of the author's investigation on the influence of the femoral arterial occlusion upon the nervous system revealed that the pathologic change, i. e., sympathicoganglionitis was found only in the lumbar sympathetic ganglia, and the change was localized in L2 to L4.

The inflammation of sympathetic ganglion is usually recognized in such diseases as typhus, hydrophobia and shingles among the infectious diseases, while the congestion of sympathetic ganglion is well known to appear secondarily in congestive heart failure among the non-infectious diseases. FISCHER and KAISERLING reported that they produced experimentally intensive inflammation in the prevertebral ganglia by means of serum injection into the lymphcanal of sensitized rabbit's lesser pelvic organ.

The relation between a morphologic change of sympathetic ganglion and a disease or an experiment which may cause this change must be discussed cautiously. Especially the author had to exclude an infectious factor from this experiment for the recognition of experimentally made sympathicoganglionitis. the author would like to present the following fact as the counterevidence. Operative maneuvers were done under sterile conditions to prevent infection. If hematogenous infection occured, inflammation should appear in every sympathetic ganglion, however, no inflammation was found in the thoracic sympathetic ganglia and also in the spinal ganglia. Moreover in the operative wound of affected leg, no inflammatory sign was observed, so that lymphogenous infection would be excluded.

Accordingly, the author would rather think that the same change as sympathicoglionitis appeared in the form of inflammation or congestion as a result of a neurogenic stimulative phenomenon due to some direct stimulus into the sympathetic ganglion than that the change originated from infection. In other words, it may be said that sympathetic nerve would conduct afferent impulse under continuous vascular stimuli, and then evoke a morphologic change in the lumbar ganglia which may receive the impulse by way of the irritated sympathetic nerve.

Next, the degree of change in the opposite side of the sympathetic ganglia in the group C and D, of which the sympathetic ganglia were resected in one side beforehand, was found to be less than the one of the non-sympathectomized group. This finiding suggests that the endogenous factor suppressing the sensitivity of afferent impulse may be prodced in dog by lumbar sympathectomy.

Further, concerning the problem whether special connections exist or not within sympathetic ganglia and spinal ganglia, HIRT stated that tigrolysis appeared in spinal ganglion cells after the resection of the greater splanchnic nerve. The author's result, as mentioned in the preceding chapter, that some degeneration of nerve process appeared in the spinal ganglia (Th12 and L2) within 20 days after lumbar sympathectomy, revealed that a certain nerve fiber passing through lumbar sympathetic ganglia reaches the spinal ganglia by way of the white rami distibuted above L2 of the spinal cord. It has been considered that there is least sympathetic fiber which goes out of the spinal cord below L2, because there is no white ramus below L 2, and there is a difference between the segment of the spinal cord to which the somatic sensory nerve innervating leg enters and the one from which the sympathetic nerve innervating it starts.

Therefore it may be considered that a certain nerve fiber passing through the sympathetic ganglia (L2 to L4) does not contain the somatic sensory nerve fiber, but contains the fiber belonging to the visceral nerve, and after the interruption of this nerve fiber, the degeneration of pericellular nerve fiber appeared in the spinal ganglia as a stimulated sign. From the result that the degeneration of spinal ganglia was remarkable in the upper ganglia such as Th12 and L2, the existence of the ascending fiber which reaches the spinal ganglia through the white rami above L 2 may be infered.

V CONCLUSION

The author investigated the neurohistologcal picture of the sympathetic ganglia and spinal ganglia of the 19 dogs whose femoral artery was occluded experimentally and reached the following conclusion :

1. In the lumbar sympathetic ganglia 7 days after the occlusion, congestion and inflammation were already observed in certain degree. 20-30 days later, the inflammation was in more intensive degree. Especially in L2 to L4, the change was remarkable.

2. The figure of initial stimulation was a common pathologic sign in the sympathetic ganglion cell. The hyperchromatic of the cell body was found in all cases of which the vascular change was remarkable.

3. The degree of inflammation in the opposite lumbar sympathetic ganglia in the cases of which the lumbar ganglia had been resected on one side, was found to be slight.

4. Both in normal dogs and in the dogs whose femoral artery was occluded, within 20 days after lumbar sympathctomy the spinal ganglia (Th12 and L2) showed the same change of nerve process and nerve fiber at the pericellular plexes as spherical phenomenon or disharmonious nerve process.

5. No morphologic change was observed in the spinal ganglia after the experimental femoral arterial occlsion.

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和 文 抄 録

犬の股動脈閉塞に於ける神経組織学的研究

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交感神経線維に求心性の Impulse を伝導する線維があるか，また遠心性線維に antidromic の作用があるかの問題は，末梢循環障碍の病因を議する上に屢々論議される所であるが，私は軟パラフィンを注入して片側股動脈閉塞を起こした犬の腰部並びに胸部の交感神経節及び脊髄神経節を組織学的に検索し次の結論を得た．

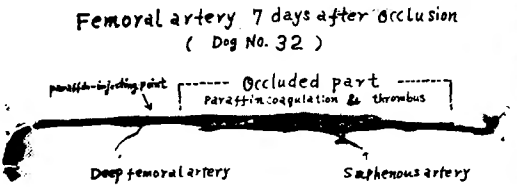
1) 腰部交感神経節には，術後7日に既に充血，軽度の炎症が認められ，20～30日後には強度の炎症像が認められた．特に L.2, L.3, L.4 に変化が著明であつた．併し胸部交感神経節には変化を認めなかつた．この変化は血管性の刺戟によつて二次的に腰部交感神経節に出現したものと考えられるもので，交感神経線維に持続的に刺戟が加わると，求心性衝動の伝導が起り，その結果として所属神経節に刺戟現象が現われ得ることを暗示する．

2) 腰部交感神経節細胞の変化として最も著明なものは，細胞体の濃染で，これは Herzog の言う初期刺戟像のように思われた．而も炎症の強いものに大部分斯かる変化が認められた．

3) 予め一側腰部交感神経節切除を行つておくと，股動脈閉塞に於ける他側腰部交感神経節に出現する変化は軽度であつた．これは求心性の興奮伝導が一部遮断されるのみならず，求心性の衝動に対する生体の反応性が低下したためと解釈される．

4) 脊髄神経節には何らの形態学的変化が認められなかつた．従つて股動脈閉塞は下肢の体性神経には影響をあたえないものと考えたい．

5) 正常犬及び股動脈閉塞犬の両側腰部交感神経節を切除すると，脊髄神経節 (T_{12}, L_2) の神経突起には突起失調 (増殖，肥厚)，球現象が認められた．この変化は恐らく腰部交感神経幹を通る内臓神経の求心性線維に於ける逆行性変化であろう．



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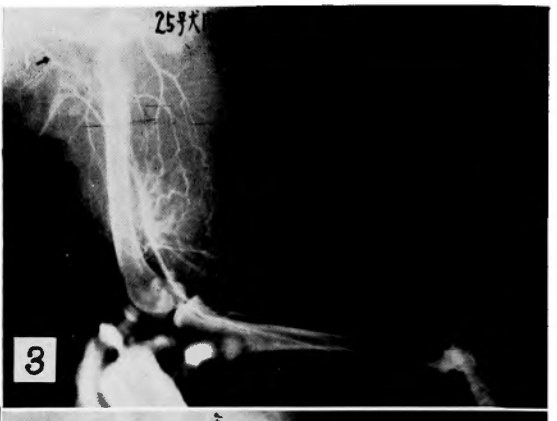

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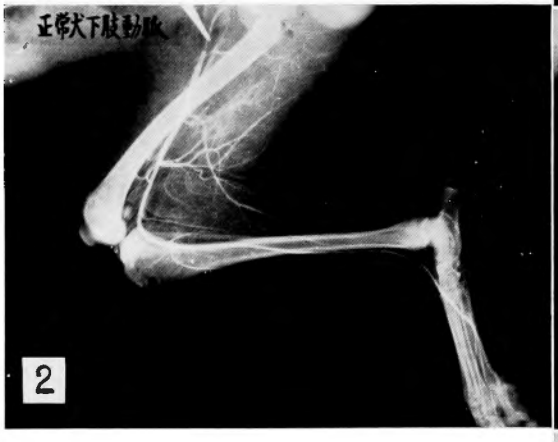

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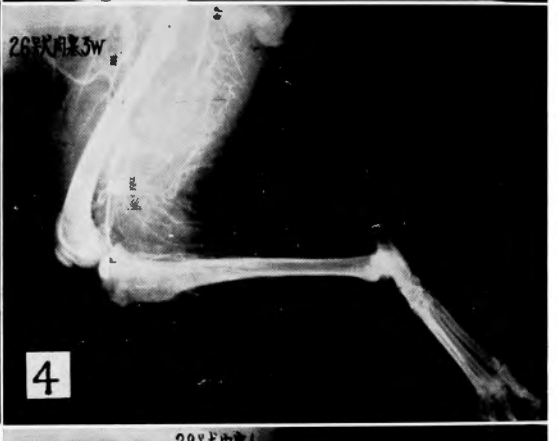

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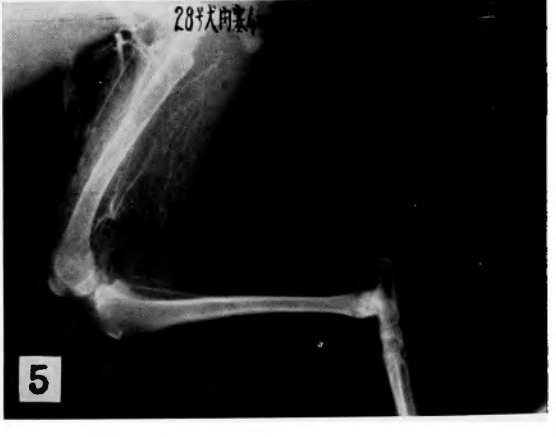

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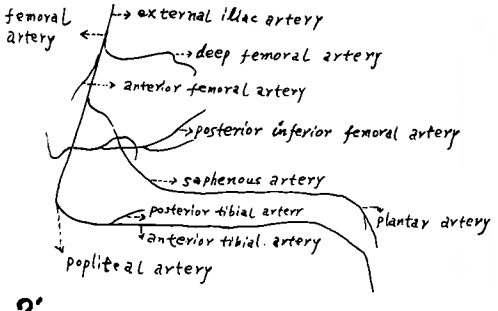

2′ Fig. 2′

Fig. 3 Well-developed collateral vessels in the thigh.

Fig. 4 Posterior gluteal artery anastomosing with posterior inferior femoral artery.

Fig. 5 Interrupted femoral arterial trunk.

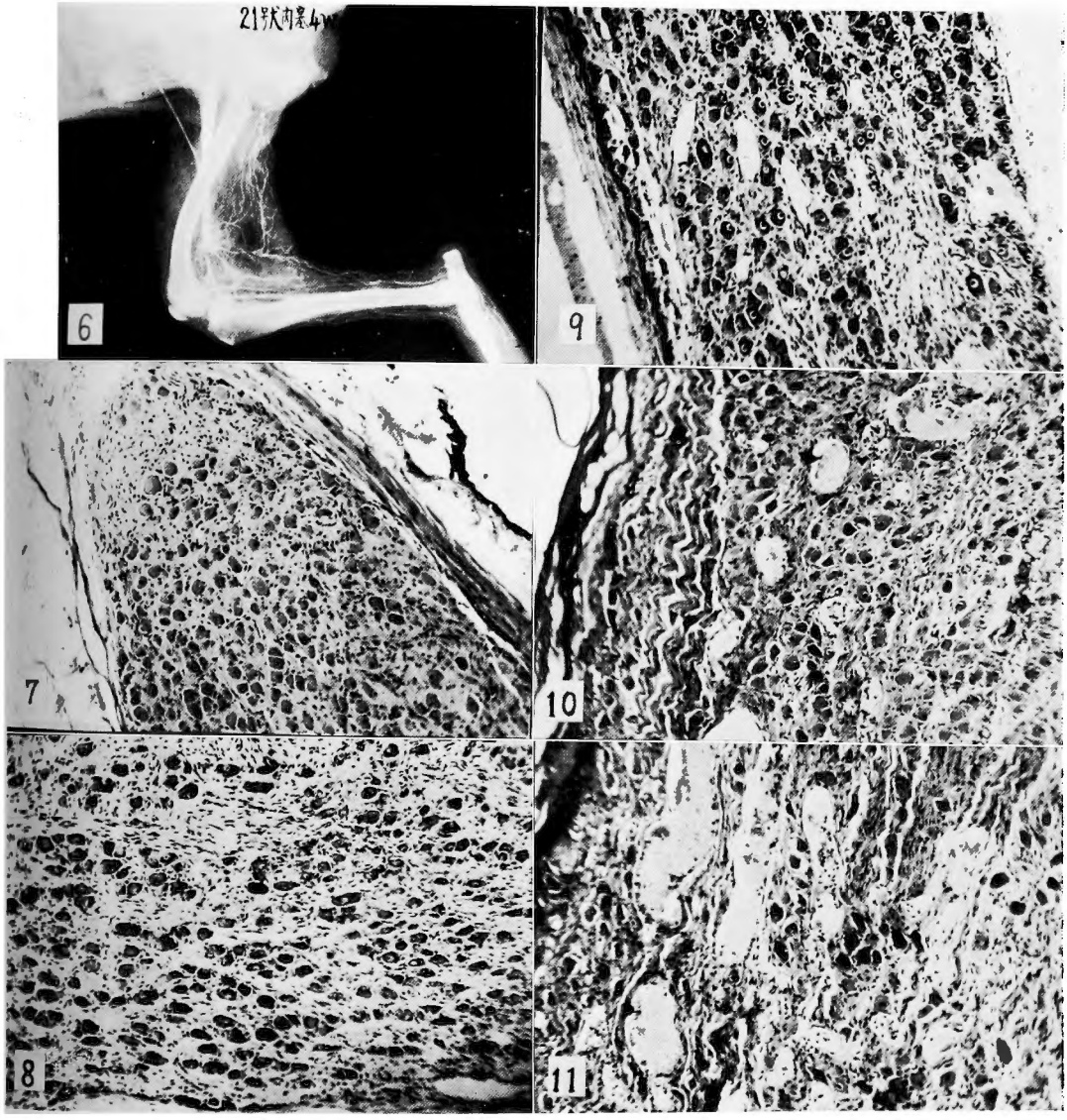

Fig. 6 Remarkable development of collaterals in one case of sympathectomized group.
Fig. 7 Normal sympathetic ganglion (Th 8). H. E. ×150.
Fig. 8 Normal sympathetic ganglion (L 2). NISSL ×150.
Fig. 9 7 days after occlusion capillary dilatation in L 2 (No. 30 dog). NISSL ×150.
Fig. 10 13 days after occl. edema, capillary dilatation and increase of leucocyte are shown in L 4 (No. 29). H. E. ×150.
Fig. 11 25 days after occl. intensive inflammation, hyperchromatic cells in L. 2 (No. 25). H. E. ×150.

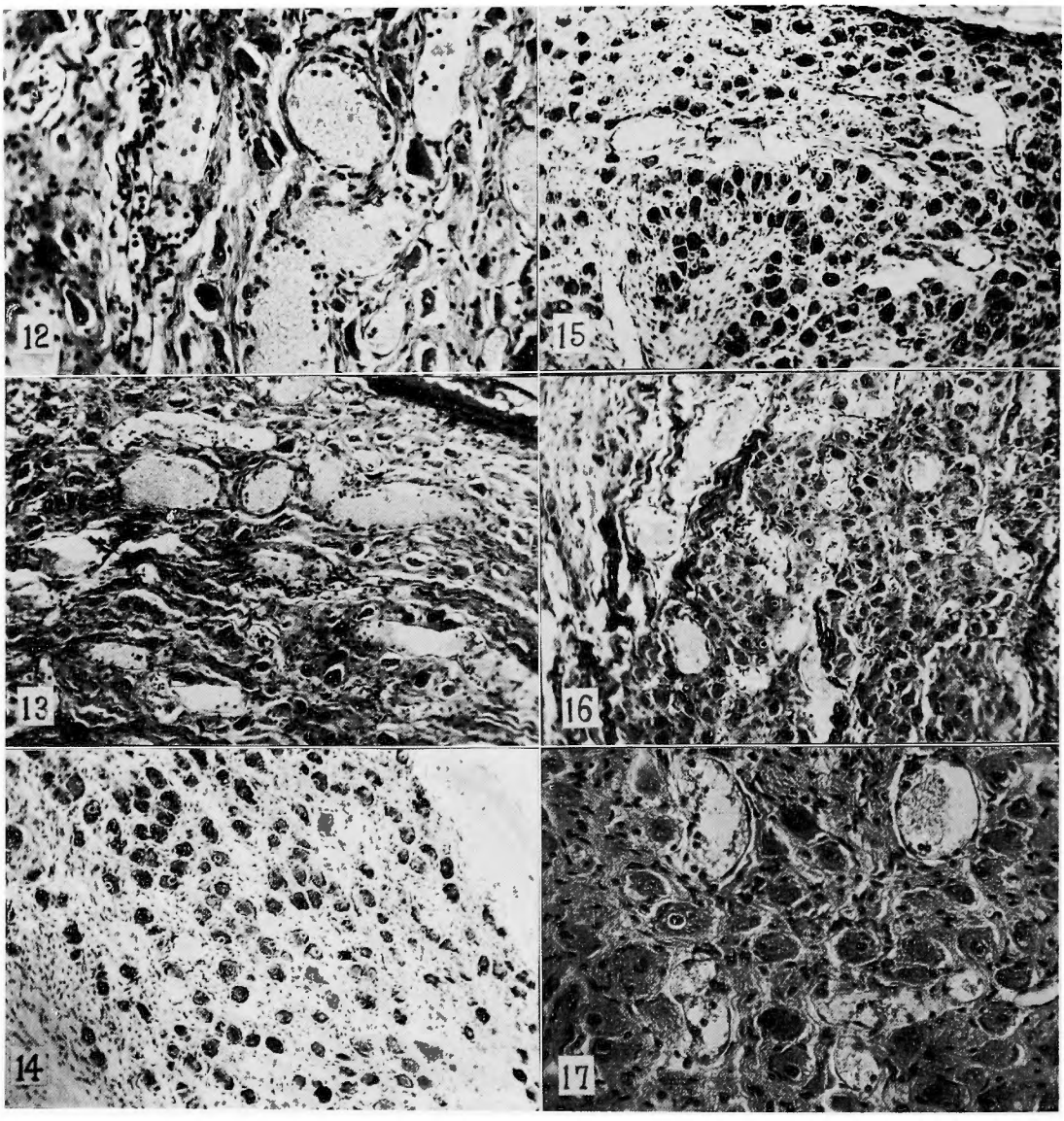

Fig. 12 30 days after occl. leucocyte infiltration. hyperchromatic nerve cells in L 3 (No. 28). H. E. ×300.

Fig. 13 18 days after occl. remarkable inflammation in L 2 (No. 27). H. E. ×150.

Fig. 14 13 days after occl. no inflammatory change in L2 (No. 29). NISSL ×150.

Fig. 15 30 days after occl. capillary dilatation, edema in L6 (No. 28). NISSL ×150.

Fig. 16 7 days after occl. capillary dilatation, leucococyte infiltration in L 3 (No. 24). H. E. ×150.

Fig. 17 7 days after occl. capillary dilatation in L 3 (No. 32). H. E. ×300.

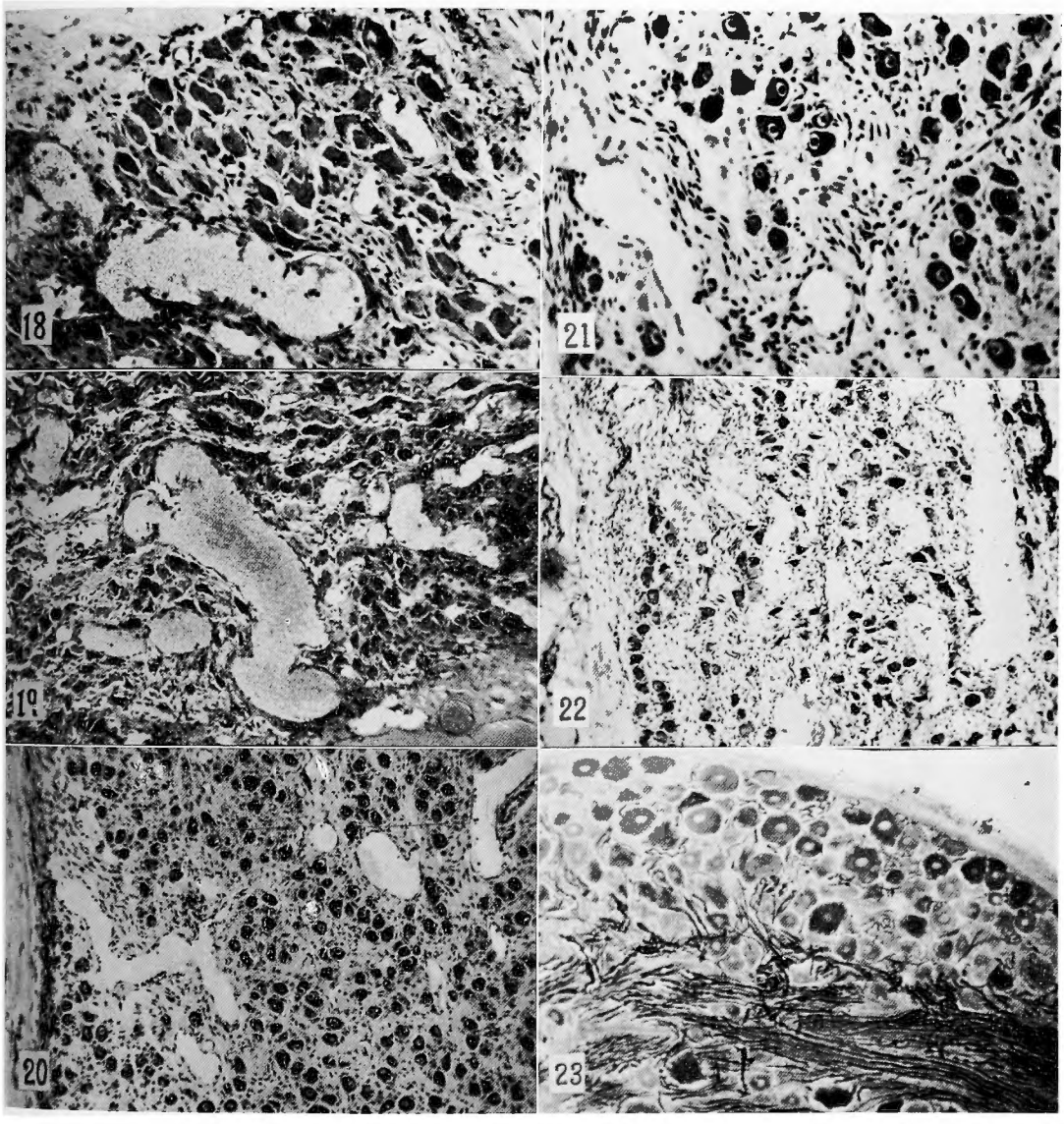

Fig. 18 21 days after occl. remarkable congestion, hyperchromatic nerve cells, leucocyte infiltration in L 2 (No. 31). H. E. ×300.

Fig. 19 23 days after occl. intensive congestion in L 2 (No. 33). H. E. ×150.

Fig. 20 23 days after occl. congestion in right L 4 (No. 22). NISSL ×150.

Fig. 21 23 days after occl. atrophy and excentric location of nuclei in L 3 (No. 33). NISSL ×300.

Fig. 22 25 days after occl. shrinked nerve cells in L 3 (No. 25). NISSL ×150.

Fig. 23 Normal spinal ganglion (Th12). B-S. ×150.

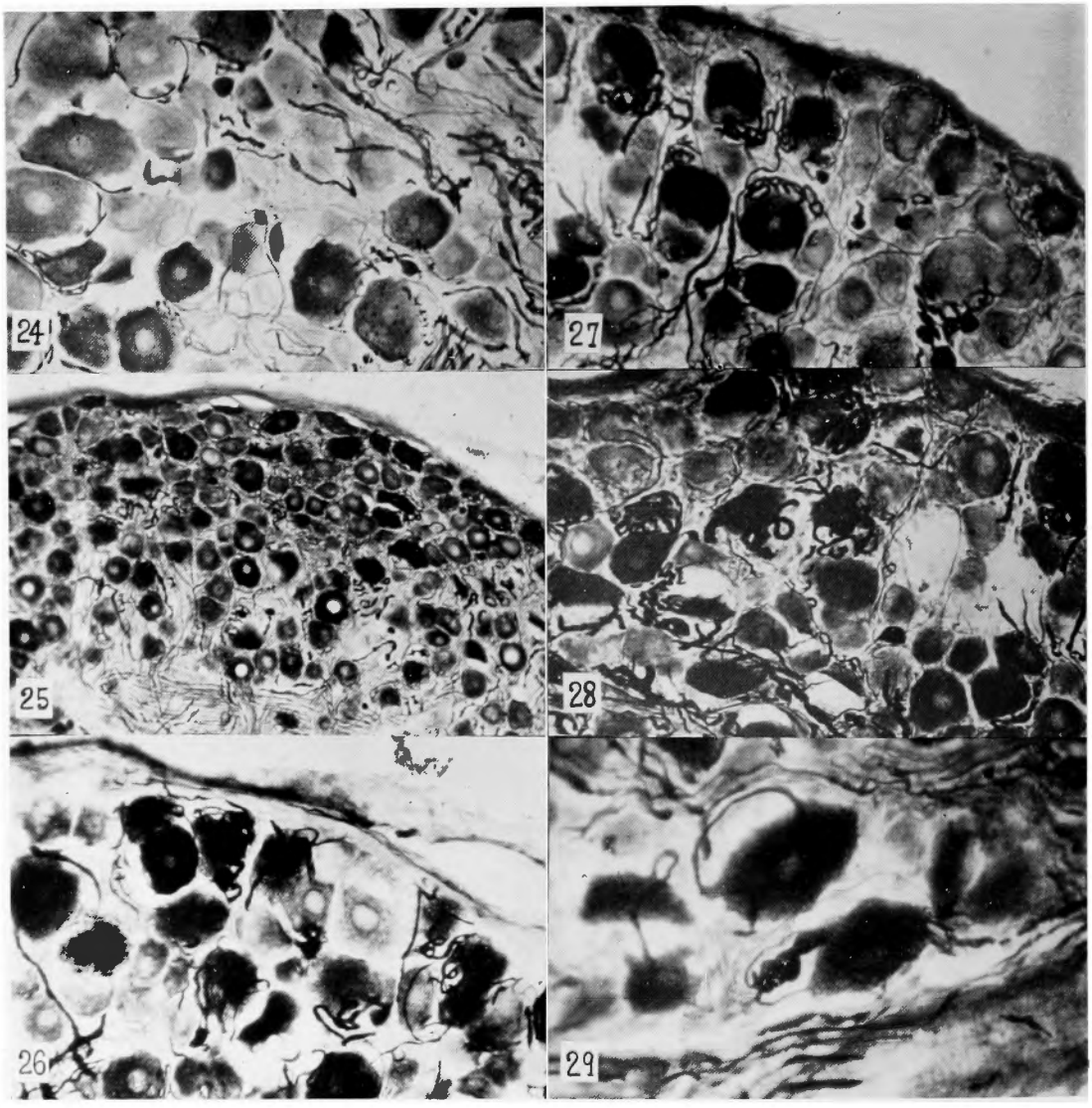

Fig. 24 3 days after lumbar sympathectomy, globular bodies appeared in spinal ganglion Th12. B-S. ×300.

Fig. 25 7 days after lumbar sympathectomy, many globular bodies in L 2. B-S. ×150.

Fig. 26 15 days after lumbar sympathectomy, globular bodies connected with nerve cells in L 2. B-S. ×300.

Fig. 27 7 days after lumbar sympathectomy, hyperplasy and hypertrophy of nerve process and fenestration are shown in Th12. B-S. ×300.

Fig. 28 10 days after lumbar sympathectomy, hypertrophic nerve process in L 2. B-S. ×300.

Fig. 29 7 days after lumbar sympathectomy, degeneration of nerve process in Th12. B-S. ×300.

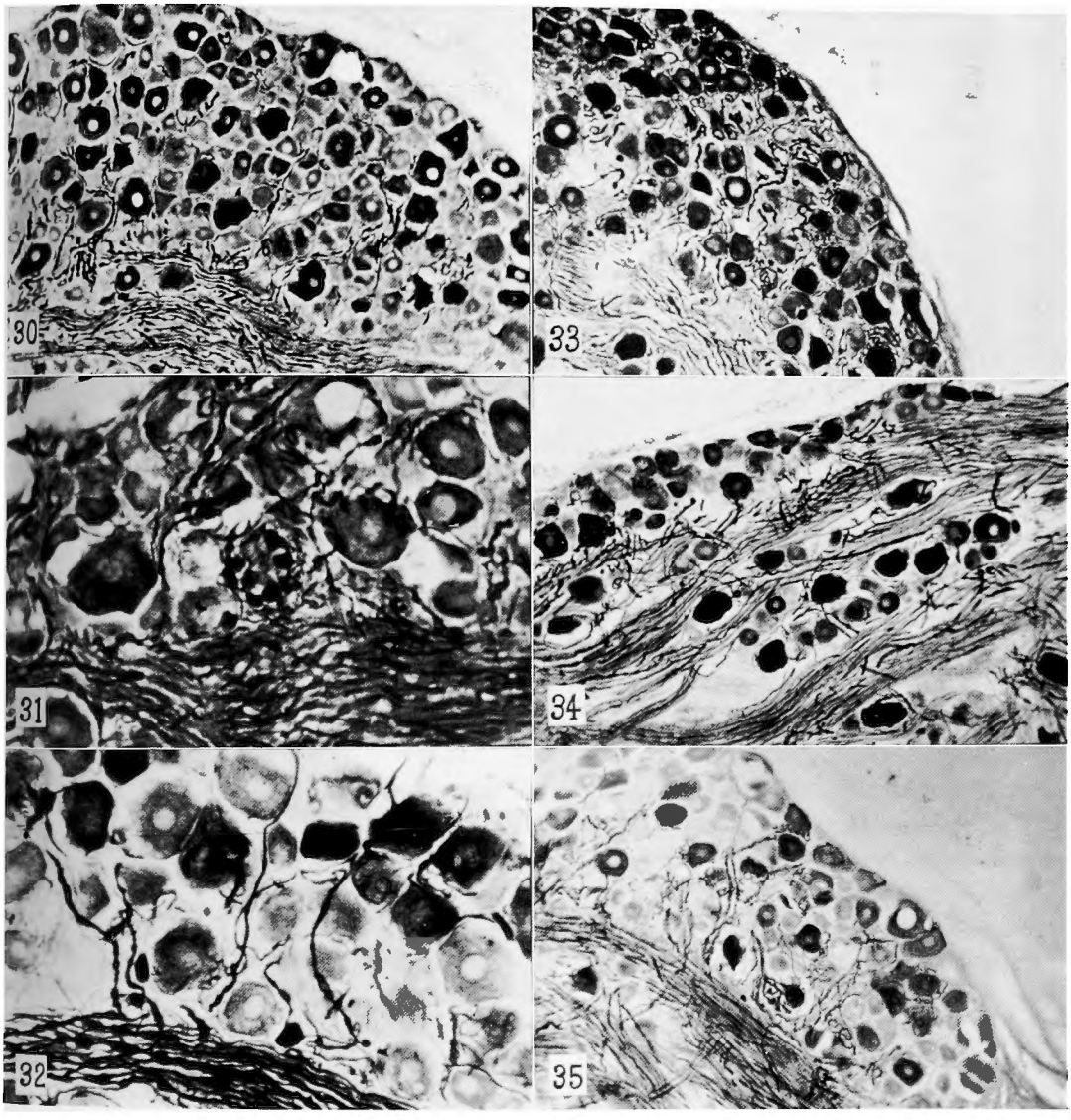

Fig. 30 Only a few globular bodies in S 1 of normal dog. B-S. ×300.

Fig. 31 15 days after lumbar sympathectomy, a group of globular body in L 2 (No. 21). B-S. ×300.

Fig. 32 A few globular bodies in Th12 (No. 21). B-S. ×300.

Fig. 33 16 days after lumbar sympathectomy, many pericellular globular bodies in Th12 (No. 22). B-S. ×150.

Fig. 34 7 days after lumbar sympathectomy, a few globular bodies in L 2 (No. 26). B-S. ×150.

Fig. 35 13 days after occlusion, no change of nerve process in L 2 (No. 29). B-S. ×150.