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EXPERIMENTAL STUDIES ON THE DEGENERATION OF THE PERIPHERAL NERVE FROM THE HISTO-MORPHOLOGICAL VIEW-POINT

by

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Received for publication Dec. 8, 1959

I. INTRODUCTION

The AUERBACH's plexus in the intestinal wall is ready to degenerate comparing with the ganglia in the sympathetic trunk. It is due to the fact that the intestine has numerous bacilli in the lumen and the infection and other stimulus easily invade the intramural plexus.

As the degenerative figures caused by non-pathological causes the degeneration due to age and the post-mortal change are well known. These non-pathological degenerative changes must be always remembered when one studies the neuropathological change of AUERBACH's plexus in relation to the clinical symptoms of patients.

HERZOG gave us an advice that one must confirm the significance of the degenerative change of nerve plexus with strict experiments when he considers it in connection with the clinical syndrome of patient.

In our clinic many authors reported the neuropathological study on the degenerative change of AUERBACH's plexus in various diseases; INOUE studied on the chronic constipation and caecum mobile, INOUE and LEE on congenital megacolon, YAMANOUCHI on gastric ptosis, YAMADA on the gastric ulcer and cancer, and SEKIYA on cancer of the rectum. However, they did not give definite conclusion on the significance of the degenerative change, because of lack of experimental data.

The present author made, therefore, a plan to get some experimental evidences on significance of degeneration of AUERBACH's plexus occurred with the application of the culture fluid of B. coli, conc. alcohol and Quinapon (a mixed solution of chinin chlorate and strychnin nitrate).

II. MATERIALS AND METHODS

Dogs were used as experimental animals.

The culture fluid of B. coli, 50～96% alcohol or 50～100% Quinapon were injected into the wall of the ileum of dogs respectively. 0.5 cc of each chemical agent was once injected and 3～5 days after the injection the local ileum was removed and

specimen was used for the observation of the acute stimulated state of AUERBACH'S plexus.

Other specimens were removed 30 days after the application of the same chemical agents and observed the chronic change of AUERBACH'S plexus.

For the microscopic observation these specimens were fixed for months, sliced into 30μ thick sections with freezing microtome and impregnated with BIELSHOWSKY'S method.

Quinapon is a mixed solution having following composition; chinin chlorate 0.14g, strychnin nitrate 0.001g and caffcin 0.037g in 2cc aq. dest.

Ⅲ. FINDINGS

Macroscopically there was no abscess of necrosis at the ileum, to which these chemical substances were injected, except the inflammation signs such as hyperaemia, adhesion, edematous swelling or hypertrophy of the local intestine.

A. Microscopical findings of specimen to which the culture fluid of B. coli. was injected

1. The change of AUERBACH'S plexus 3～5days after the injection of the culture fluid

AUEREACH'S plexus remarkably increased the width due to edema of acute inflammation.

The capillary blood vessels which were not observable in normal AUERBACH'S plexus were found numerously and in marked dilatation. Arterioles and venules detoured and some of them were filled with erythrocytes. The leucocytes were infiltrated in the connective tissues around the collagen fibers and accessory cells. There were large or small pools of lymph fluid (Fig. 1).

In the heavy edema, isolated nerve cells or islands of cell groups scattered between the lymph pools. Some parts of nerve cells were not visible covered with leucocyte infiltration, but some others which were surrounded by lymph spaces gave rather clearer contours as those in normal distribution (Fig. 2).

In a island consisting of several nerve cells they formed one or two lines of cell arrangements and each nerve cell lied in so close contact with adjascent ones that the contour of each nerve cell was rather obscure.

The protoplasm of these cells gave diffuse and heavy appearances and neither the capsule nor nuclei were distinguishable. They may be called the nerve cells in the primary stimulation (I. Reizung).

An isolately lying nerve cell in the fluid of edema looked swollen. The lymph invaded the subcapsular slit and formed small pools between the capsule and the cell body (Fig. 2, E_2, E_1). The nerve cell body seems to be shrinked, but the author supposed it to be under compression of edema fluid.

Some nerve process of these cells, swelled and heavily stained, ran through the edema and gave rise of several detoured branches at a point on the outside of the capsule (Fig. 2, P_1).

Some small nerve cells had short and long process radiately running through the edema and they divided the subcapsular space into pseudofenestrae (Fig. 2, G_2).

The degree of the edema was in proportion to the intensity of the stimulus: undiluted culture fluid of B. coli presented a higher degree of edema than 50% solution did.

The degenerative change of the nerve cell seemed to be proportional with the degree of edema. Even in the same specimen, nerve cells lying in more marked edema showed severer change than those in less marked edema.

A remarkable edema resulted in the invasion of fluid into the muscular layers and formed various sized spaces between loosened muscle fibers. In such a place some nerve cells presented only a vague contour with faint protoplasm but the nuclei were rather pyknotic or kept stainability in marginal zone and gave a ring form figure (Fig. 3, G_2, G_4).

The roots of the neurites arising from these nerve cells were thickened markedly and looked like the tad pole (Fig. 3, G_3, G_5). This nerve cell change is called degenerative homogenization of neuroplasm with swelled process.

The G_2 in Fig. 3 was a large nerve cell with faint protoplasm and pyknotic nucleus, but it had no thick neurite. This might be a stimulated figure of the DOGIEL's second type nerve cell.

The figure reminded the author of the nerve cell described by INOUE in the AUERBACH's plexus in dilated intestine, which was obturated and swelled like a tumor with stagnated hard feces. INOUE rightly described it as a stimulated state of a DOGIEL's second type nerve cell.

At a place, where edema was slight, AUERBACH's plexus was stained dark. Nerve cells were in primary stimulated state. Nerve fibers were rather thickened and they kept good argentaffinity.

2. 30 days after the injection of culture fluid of B. coli into the wall of ileum

Edema was still left in places between muscular layers, though it was slight.

Greater part of AUERBACH's plexus was stained dark, but the plexus markedly decreased the width in some parts, while rather increased it in other parts.

Fig. 4 shows a regenerated figure of AUERBACH's plexus with many nerve cells and proliferated accessory cells. A great number of nerve fibers covered the plexus.

The hypertrophy and hyperargentaffinity of nerve fibers seen in the acute stadium already returned normal. They looked somewhat slender and frizzled.

Some of the nerve cells were still hyperchromatic, but the subcapsular edema were not observed; the nerve cell bodies were in close contact with the capsules.

As the degenerative change of the nerve cell, the central liquefaction or spottedly stained protoplasm with pyknotic or marginally pyknotic nuclei were shown. On the other hand, there were lightly stained nerve cells with homogenous neuroplasm giving only cell shadows in severe case. The nerve fiber bundles, forming a large meshworks ran out of AUERBACH's plexus to the muscle layers with small nerve cells scattered their course. They formed the AUERBACH's plexus of the second order in the connective tissue of the muscle layer. Hence many fine nerve fibers or the nervous syncytia ran along the partially destructed muscle fibers as the AUERBACH's plexus of the third order. These fine nerve fibers and nervous syncytia

were well stained, suggesting that they were regenerated nerve elements. Some of the regenerated nerve fibers and nervous syncytia extended toward submucous layer and connected to the MEISSNER's plexus.

In the MEISSNER's plexus remained the cell infiltration and the plexus was still widened.

Passing through the MEISSNER's plexus some fine nerve fibers and nervous syncytia reached the mucous membrane. The spread of these regenerated nerve elements accompanied the proliferated accessory nuclei without exception.

Among these regenerated nerve elements, there were left a few of degenerated nerve fibers with frizzled figures in a loosened nerve bundle.

B. Microscopical findings of specimens to which 50～100% alcohol were injected

1. The change of AUERBACH's plexus 3～5 days after the injection of alcohol

Spotted hemorrhages were found in the muscle and the submucous layers. Edema, the dilatation of capillaries and the infiltration of leucocytes were seen in widened AUERBACH's plexus just as the case of the culture fluid of B. coli.

In highly edematous places, the nerve cell groups were found scattered like islands.

In the slightly edematous places the nerve cells kept a normal arrangement, but the degree of degenerative change of these nerve cells were more remarkable than the case of B. coli filtrate. Besides the nerve cells in primary stimulation, there were some which had markedly lower affinity to silver with swelled homogenous protoplasm and vague contour of nuclei. The nuclei of the nerve cell capsule and accessory cells were very obscure. Some swelled nerve cells had the protoplasm loosened from the capsule in whole circumpherences (Fig. 6, K), of which the author considered as cell figures with subcapsular edema rather than as shrinked cell bodies.

The pathological changes of nerve fiber were broadly divisible into two aspects. One was atrophic thin fibers loosely arranged in a nerve fiber bundle with very poor stainability, the other was heavily stained very thick fibers. The former might be strongly stimulated nerve fibers with alcohol, while the latter lightly stimulated ones. The atrophic nerve fibers often had fine waves and detours on their course (Fig. 9). The swelled nerve fibers with strong argentaffinity arranged loosely in a fiber bundle, each presenting a clear figure sharply out-lined in other tissues.

The endings of the fine nerve fibers constituted a characteristic feature near the nerve cell with globular, spindle or ampule like expansions and rings. They might not be the roots of nerve process but the free endings of the dendrites or neurites, because there was no sign that they had direct continuity with nerve cells near by. According to DE CASTRO and HERZOG these expansions can not exclude the free endings of preganglionic fibers. It was very interesting that ACHUCARRO had described the same figures in the sympathetic ganglion of chronic alcoholism.

The difference between the findings in specimens stimulated with 50% and 100% alcohol was only in the intensity of inflammatory change.

2. The change of AUERBACH's plexus 1 month after the injection of alcohol

The infiltration of the leucocytes had practically been absorbed.

There were many vacant spaces spotted in the nerve plexus, which were supposed to be formed after the disappearance of nerve cells. Indeed, the rest of the nerve cell bodies could be found within the spaces as extremely thin plasma bridges and free ending expansions of nerve fibers (Fig. 11). However, one could find crevices between muscle fibers containing none of the nerve element, which must be residual edema or artifacts. There were found as the degenerative changes of nerve cells, central liquefaction or lowered argentaffinity of protoplasm, chromatolysis of nuclei, deviation or dislocation of nuclei, marginal distribution of the tigroid, homogenized neuroplasm with swelled process, and nerve cells with antler shaped hypertrophic process.

Above all, various shaped expansions of free endings around degenerated nerve cells remarkably appeared in the specimens applied with alcohol.

The regenerative tendency of the gliocyte was relatively poor. There was neither marked proliferation of accessory cells, nor replacement of degenerated protoplasm with satellite cells.

C. Microscopical findings of specimens, to which Quinapon was injected

1. The change of AUERBACH's plexus 3～5 days after the injection of Quinapon

More intense degree of edema, leucocyte infiltration, dilatation of blood vessels and hemorrhage were observed in this case comparing with specimens to which culture fluid of B. coli or alcohol was injected (Fig. 14).

In highly edematous places the nerve cell groups were scattered like islands in extended AUERBACH'S plexus.

The nerve cells, pressed with edema, arranged so closely one another that some of them were deformed between two adjascent cells (Fig. 16).

In such a place, many of nerve cells were in state of primary stimulation (Figs. 15 and 16), but other degenerative figures with central liquefaction of protoplasm and pyknotic nucleus, (Fig. 16, G_1), subcapsular edema (or socalled shrinked protoplasm) (Fig. 16, G_2), or gigantic enlargement were observed, too (Fig. 14, G). The most specific features in this case were the early disappearance of some degenerated cells. Only shadows of nerve cells or vague plasma bridges remained here and there and they presented clear spots around them in the plexus (Fig. 15). The most remarkable change of this kind was represented with Fig. 17. A part of the plexus was entirely demarcated and formed a round cavity, in which several nerve cell shadows and leucocytes were enclosed (Fig. 18). The cavity could be called an intraganglionic abscess or necrosis, but it lacked the reactive infiltration of leucocytes in the wall of the cavity. Therefore, the occurrence of the cavity might be due to the strong toxity of Quinapon on the nerve elements and not to the infection. Accessory cells and satellite cells had only faint nuclei. Most of nerve fibers were atrophic with frizzled figures and with thin and poor argentaffinity, while others were swelled, granulated and had a hyperstainability to silver.

The terminal expansions of free nerve ending around the nerve cell were rarely found in this case.

2. 30 days after the injection of Quinapon into the wall of ileum

In and around AUERBACH's plexus, there were many vacant spaces probably caused by atrophic change of muscle fibers, rest of edema, and destructed nerve cells.

Most of nerve cells fell in atrophic degeneration with shrinked cell bodies or homogenous cell shadows without any sign of satellite cell proliferation.

The accessory cells did not proliferate, too.

A small number of nerve fibers which remained in the plexus were also atrophic with appearances of faintly stained slender lines or worn out threads. Some were severed and others granulated.

The globular or ampule shaped terminal expansions of nerve fibers could be seen around the degenerated nerve cells (Fig. 19, f), but the number of them were far less than the case of alcohol application.

The damage of AUERBACH's plexus were severest in this case with very poor regeneration sign of nerve elements.

Ⅳ. DISCUSSION

The author observed the degenerating and regenerating tendency of nerve elements in AUERBACH's plexus after the local injection of culture fluid of B. coli, 50～100% alcohol or Quinapon. The acute or the stimulated changes of AUERBACH's plexus were observed 3～5 days after the injection of these chemical substances and the chronic or regenerative change 30 days after the injection.

Many authors of our laboratory studied the pathological change of AUERBACH's plexus in various patients, i. e. OTSU in gastric ulcer, MAKINO in duodenal diverticula, INOUE in chronic constipation, YAMANOUCHI in gastric ptosis, YAMADA in cancer of the stomach, SEKIYA in cancer of the rectum.

These pathological changes or socalled degeneration of nerve elements can be classified into 2 groups: one shows the stimulated state and another the atrophic state.

However, these degenerative changes are shown not only in various diseases but in physiological state of the alimentary canal. DE CASTRO, HERZOG, TERPLAN, WOHLWILL, HERMANN and others emphasize that these figures are variously interpreted by authors concerning whether they are significant to explain the clinical symptoms or not. If it is pathological, what does it mean? Is it the cause or the result of the disease? Can these changes be reversible or not? On these points, interpretations have been done only with supposition in the light of clinical symptoms.

HERZOG maintains the importance of full understanding on these figures by means of strict experiments.

The author tried in these experiments to represent the degenerative changes of AUERBACH's plexus which have been shown by many authors in various diseases and given varied interpretations.

He observed the change of AUERBACH's plexus by stimulating it with chemical agents and followed after it for 30 days to know whether it would be reversible or

not. Thus he could affirm these questions to a certain degree.

The culture fluid of B. coli was used as a chemical stimulant for the reason that it could imitate the occurrence of common intestinal inflammation.

Alcohol was used for its stimulating and anesthetic action on the nerve, and Quinapon considering the violent stimulating action of strychnin on the nerve.

The morphological changes after application of these chemical substances include many common figures which must be understood as non-specific changes.

In acute stadium after the injection of these agents, marked edema, dilatation of capillaries, infiltration of leucocytes and some times hemorrhage occurred.

Nerve cells in AUERBACH'S plexus scatter in the edematous tissue isolately or forming small cell groups.

They have diffusely stained dark figures without showing clear out inner structures; they must be called nerve cells in primary stimulation.

Some nerve cells have subcapsular edema with apparent shrinkage of cell bodies; their protoplasm were partially or in whole circumference loosened from the capsule and vacant spaces appear between capsules and the cell bodies.

A nerve cell body, partially loosened from the capsule, retract inward and takes figure of a lunette or a thin protoplasm hanging on the opposite side of the capsule.

The nerve cell, the capsule of which is exfoliated in whole circumference, show a star shaped figure with its process. The subcapsular space is divided into several pseudofenestrae with these process. Most of these star shaped nerve cells reduce the size, though there is a few exceptions to keep a large sized cell body. The latter means a swelling of the nerve cell body with more marked distension of the capsule by the subcapsular edema (Fig. 5, K). As a stimulated figure of the DOGIEL'S 2 type nerve cell, there appears, the spherical giant cell without neurite (Fig. 3, G_2). Its protoplasm is usually very lightly stained and a pyknotic nucleus is seen at the center (Fig. 3, G_2).

The degree of these common degenerative changes in the acute stimulated stage is severer in Quinapon case comparing with other two cases.

Specific changes in the acute stage

50～100% alcohol seems to have specific action on nerve fibers, above all on the ending of them. A nerve fiber adequately stimulated with alcohol gives a thick and well stained figure (Fig. 9). The free endings of nerve fibers around a nerve cell show marked expansion such as globulated, ring shaped, or ampule like swellings (Figs. 7 and 8).

The specific figure of the specimens, to which Quinapon was injected, is the severe destruction of nerve elements. Large or small round spaces appear in AUERBACH'S plexus (Figs. 14, 15 and 17).

Some of small spaces are, without doubt, caused by subcapsular edema with shrinked cell bodies, while a large cavity represents the partial necrosis of AUERBACH'S plexus with early demarkation.

Common degenerative figures of AUERBACH'S plexus one month after the injection of chemical agents

In this stage, most part of edema fluid and infiltrated leucocytes are already absorbed.

Small round spaces are found in AUERBACH'S plexus, in which one can find, not so infrequently, very thin plasma bridges or cell shadows on a part of the spaces (Figs. 11, 12, 13 and 20). They are the rest cavity occurred after disappearance of cells. Satellite cell nuclei are not observed around the rest cavities, nor is there any tendency of proliferation of accessory cells in the neighbourhood.

The specific figure of morphological change 30 days after the injection of chemical agents

The specimens, to which culture fluid of B. coli was injected, show a marked proliferation of the accessory cells and the regenerated nerve fibers. There are only very small number of rest cavities of nerve cells. Therefore, the regeneration of the gliocytes (satellite cells and accessory cells) must be necessary for the reversibility of degenerated nerve cells and nerve fibers.

These findings suggest that the culture fluid of B. coli has relative slight toxity on the nerve elements and once degenerated nerve cells have a fair chance to regenerate.

In the specimens, to which concentrated alcohol was injected, the regenerative tendency is relative poor.

There appear many rest cavities of nerve cells without active proliferation of satellite cells. Well stained free terminal expansions of nerve fibers are more distinct on the nerve cell shadows or in the rest cavities. The hyperargentaffinity of nerve fibers stimulated with alcohol is still kept in the chronic stage of degeneration.

The specific change in the specimen, 30 days after the injection of Quinapon

The rest cavities of nerve cells are found most numerously in this case.

There is no tendency of gliocytes proliferation.

The appearance of the rest cavities of the nerve cells without proliferation of gliocytes suggests that the lack of the proliferation of gliocytes are unfavourable for the regeneration of nerve cells.

Comparing with two other substances, Quinapon (strychnin solution) has most violent toxity on the nerve elements. Throughout his findings, the author can maintain that the gliocytes play an important role in the regeneration of nerve cells and nerve fibers.

A shrinked nerve cell, even once partially loosened from the capsule, may be very difficult to maintain its life.

Other degenerative figures of nerve cells such as deviation of the nucleus or pyknotic nucleus, do not necessarily mean the irreversible change of the nucleus as a lethal figure of nerve cell. HERZOG detected the severe change of the nucleus means the irreversible change of the nerve cell. However, one cannot fully understand what kind of nucleus change does he mean as lethal.

As the sign of regeneration, the author detects the recovery of nerve cell structures i. e. reappearance of tigroids, normal stainability of nucleus, proliferating tendency of gliocytes and nerve fibers. However, it is difficult to predict the destiny

of degenerative cells in the acute stimulated stage, except shrinkage or the complete destruction of nerve cell such as found in a necrotic cavity.

V. SUMMARY AND CONCLUSION

The author morphologically observed the degenerative and regenerative changes of AUERBACH'S plexus in the ileum of the dog after the stimulation with a culture fluid of Bacillus coli, 50～100% alcohol or Quinapon (a strychnin preparation), and has come to following conclusions.

1) The morphological changes of AUERBACH'S plexus after the local injection of these chemical substances include a non-specific change which is caused by an acute or chronic inflammation as a common reaction to stimulus, as well as a specific change due to the specific actions of these substances on the nerve elements.

2) As the acute non-specific changes of AUERBACH'S plexus, there appear edema, infiltration of leucocytes, hemorrhage and dilatation of capillaries in the connective tissues 3～5 days after injection, while as chronic changes atrophic figures of tissues, such as narrowing of AUERBACH'S plexus, crevices between muscle fibers and connective tissues 30 days after injection.

3) Most of nerve cells in AUERBACH'S plexus in acute stadium present the primary stimulation figures with diffuse and heavy stainability, and other small number of nerve cells different degenerative changes, such as deviation of nuclei, pyknotic or marginal pyknotic nuclei, tigrolysis, central liquefaction, poor stainability of protoplasm and degenerative homogenization with swelled process, subcapsular edema, shrinkage of cell body with pseudofenestrae, plasma bridge and etc.

The nerve fibers show stimulated figures with swelling, granules, and loosened arrangement of nerve fibers in a bundle.

4) As the chronic non-specific changes of AUERBACH'S plexus, there appear atrophic change of nerve elements such as nerve cell shadows, rest cavities formed after disappearance of nerve cells, and plasma bridges without any proliferating sign of satellite cells. Nerve fibers are atrophic with thin and frizzled, corkscrew like or granulated figures. In nerve fiber bundles nerve fibers loosely arrange showing fine detours.

5) The regeneration figures appear as proliferation of nerve fibers, satellite cells and accessory cells.

These figures are found in the specimens 1 month after the injection of a culture fluid of B. coli, but poorly with other specimens.

6) The specimens, to which 50～100% alcohol was injected, give hyperstainability of nerve fibers including their terminal free ending around nerve cells such as end globules, end ampules and end rings.

7) The specimens, to which Quinapon was injected, present most severe destructive change in AUERBACH'S plexus, i. e. a large necrotic cavity with many destructed nerve cells within it and small cavities with shrinked nerve cells in acute stage.

8) Among various kind of degenerative figures of nerve cells, one can detect

as irreversible change, the shrinkage of nerve cells without any tendency of satellite cell proliferation.

The same can be said with regard to regeneration of nerve fibers; the proliferation of accessory cells must be necessary for the regeneration of nerve fibers.

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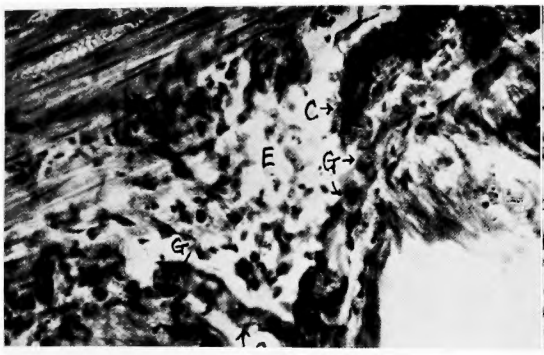

Fig. 1. 3~5 days after injection of culture fluid of B. coli
E : Edema
G : Nerve cell (stimulated)
C : Dilated capillaries

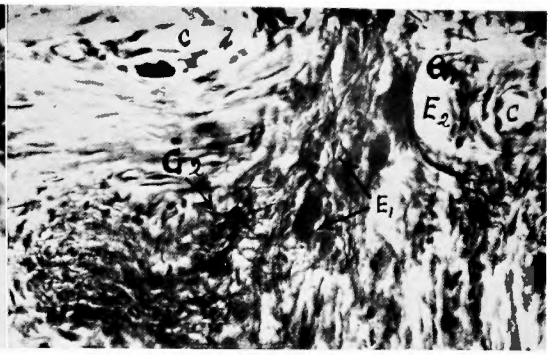

Fig. 2. 3~5 days after injection of culture fluid of B. coli
E_1 : Edema
E_2 : Subcapsular edema
G_1 : Shrinked nerve cell with atrophic process (subcapsular edema)
G_2 : Starlike figures of nerve cells with subcapsular edema
C : Dilated capillaries

Fig. 3. 3~5 days after the injection of culture fluid of B. coli
G_2 : Gigantic swelling of DOGIEL's 2 type nerve cell
G_3, G_4, G_5 : Degenerative homogenization of neuroplasm with swelled process

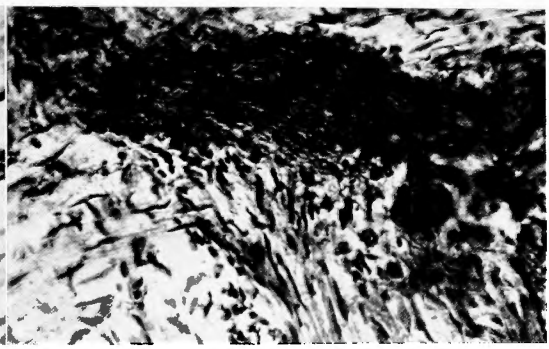

Fig. 4. 1 month after injection of culture fluid of B. coli
Regenerated nerve fibers and proliferated accessory cells are shown.

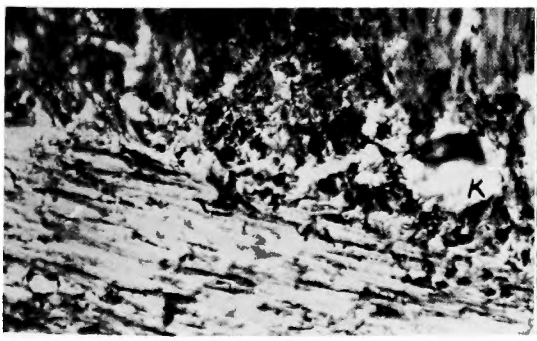

Fig. 5. 3~5 days after alcohol injection
K : A large nerve cell with subcapsular edema
f : granulated nerve fibers

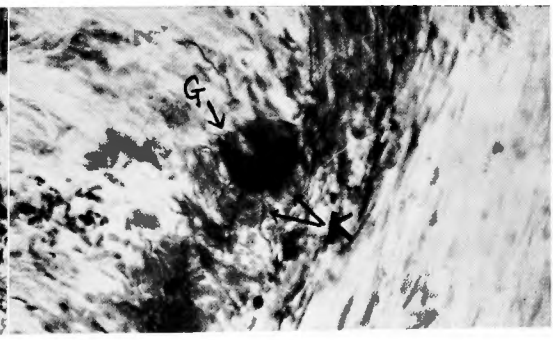

Fig. 6. 3~5 days after alcohol injection
G: A swelled nerve cell with process radiately running
K: Capsule (satellite cells) closely contacted with nerve cell

Fig. 7. 3～5 days after alcohol injection
←show terminal globules of free ending of nerve fibers on a degenerative nerve cell.

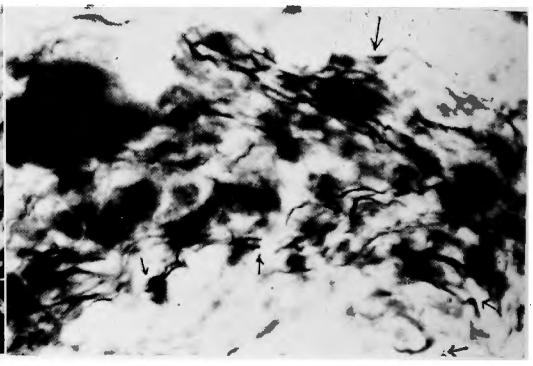

Fig. 8. 3～5 days after alcohol injection
Spindle, ampule and globular shaped terminal expansions of nerve fibers

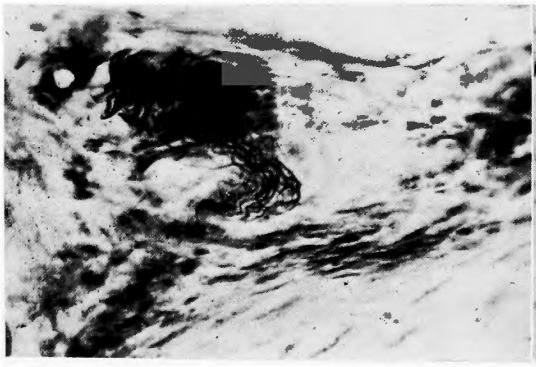

Fig. 9. 3～5 days after alcohol injection
Swelled, heavy stained nerve fibers

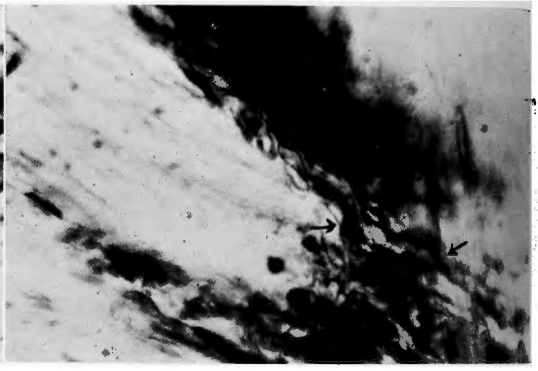

Fig. 10. 3～5 days after alcohol injection
Hypertrophic neurites growing out of degenerative cells

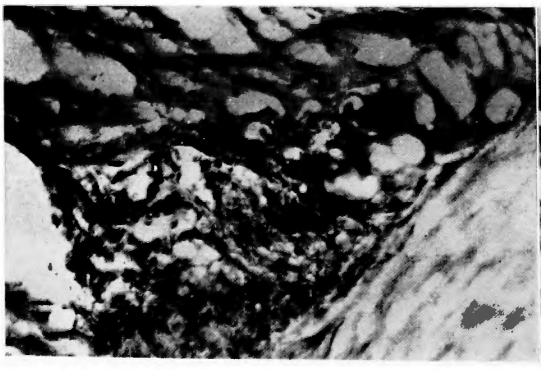

Fig. 11. 1 month after alcohol injection
Rest cavities appeared around the shrinked nerve cells or after the disappeared nerve cells
A few satellite cell nuclei are found in the rest cavity of the nerve cell.

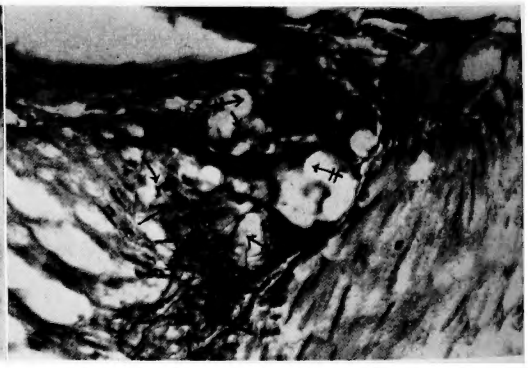

Fig. 12. 1 month after alcohol injection
→ : Shrinked nerve cell
⇸ : Terminal expansions of the nerve fibers ending at the nerve cell.
R. E.: Rest edema in muscle layer
The satellite cell and accessory cell are not observed.

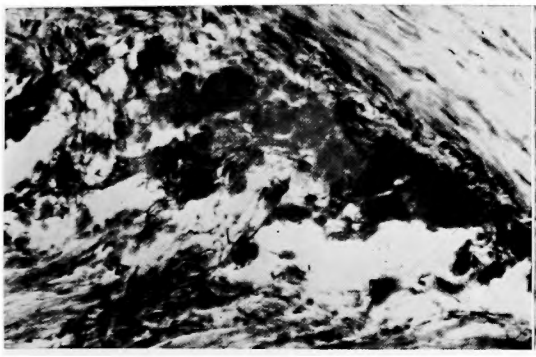

Fig. 13. 1 month after alcohol injection
Fine nerve networks with nodules

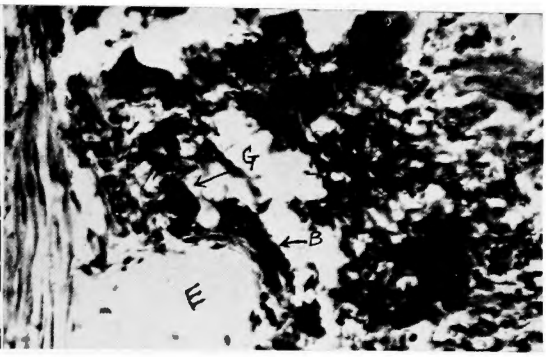

Fig. 14. 3~5 days after Quinapon injection
Intense edema

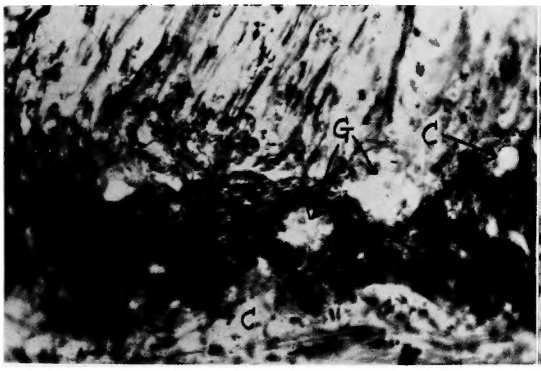

Fig. 15. 3~5 days after Quinapon injection
G : Rest cavities of nerve cells
C : Dilated capillaries

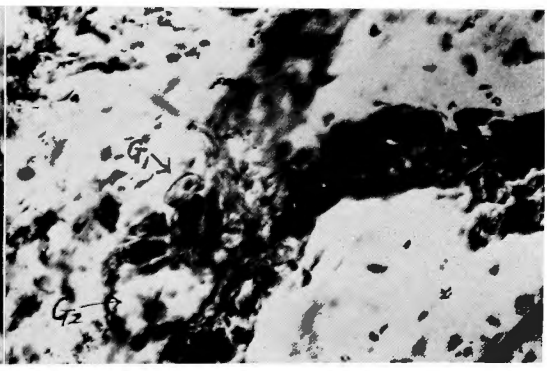

Fig. 16. 3~5 days after Quinapon injection
Nerve cell groups arranging in one or two lines pressed by edema fluid.
G : Central liquefaction with pyknotic nuclei
G_2 : Shrinked nerve cell

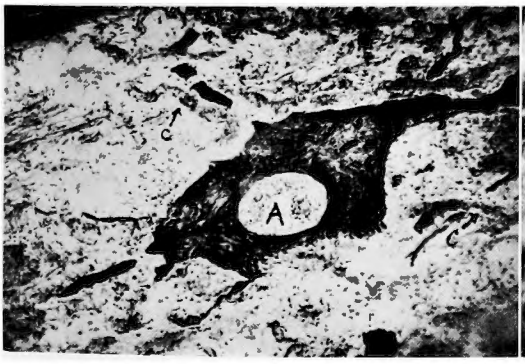

Fig. 17. 3~5 days after Quinapon injection
A : Nectrotic cavity at the center of AUERBACH'S plexus
C : Dilated capillaries
Marked infiltration of leucocytes

Fig. 18. Enlarged fig. of Fig. 17
3~5 days after Quinapon injection
Destructed nerve cells in a necrotic cavity

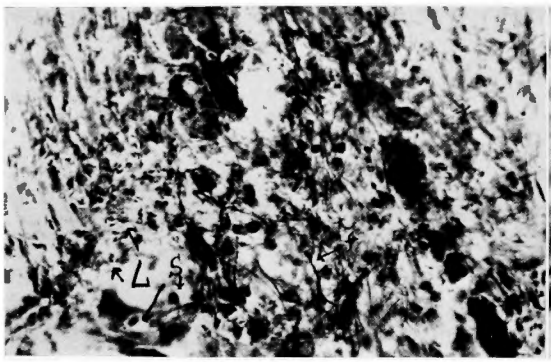

Fig. 19. 3～5 days after Quinapon injection
L : Leucocyte infiltration
S : Swelled satellite cells with pyknotic nuclei
f : Swelled nerve fiber ending
Some satellite cells are swelled, while others atrophic.

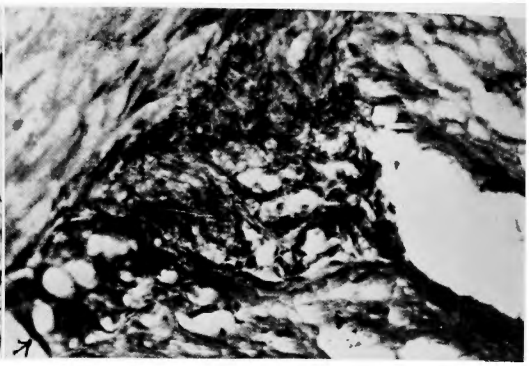

Fig. 20. 1 month after Quinapon injection
Remarkable rest edema and rest cavities of nerve cells, with few satellite cells

和文抄録

大腸菌濾液，濃厚アルコホール，キナポン液の局所注入に依る犬のAUERBACH氏神経叢の変性並びに再生に関する実験的研究

京都大学医学部外科学教室第2講座（指導：青柳安誠教授）

満田久和

大腸菌濾液，50%～96%アルコホール，50%～100%キナポン液を成犬の回腸腸壁に注入し，該部に於けるAuerbach氏神経叢の変性，再生を観察し，次の結果を得た．

1) これら薬剤の局所注入による壁在神経叢の変化は各薬剤の神経に対する特異的作用に基く特異的変化の他に，多くの非特異的変化をもたらすものである．

2) 急性非特異性変化としてはAuerbach神経叢の浮腫，白血球浸潤，出血及び毛細管の拡張等があり，慢性非特異性変化としては該神経叢の狭隘化，筋線維並びに結合織間に出現する空隙等がある．

3) 急性期に於ける神経細胞は一般に所謂初期刺戟形を示し，胞体は濃染し内部構造は不明瞭となるが，他の一部の細胞は核の変位，核の濃染，原形質の融解，中央部の融解，胞体の染色性低下，変性均等化並びに突起腫脹，被膜下浮腫，胞体の萎縮並びに偽窓形成，橋状胞体等がみられる．

4) 慢性期に於けるAuerbach神経叢の非特異性変化としては，神経要素の萎縮状態として神経細胞の淡影化，神経細胞消失後に残る小空洞形成等があり，又神経線維は細く且つちぢれ，神経束内に疎なる排列を示す．

5) 再生像としては著明なものに神経線維の増殖及び被膜細胞と副細胞の増殖がある．斯かる再生像は大腸菌濾液注入標本に著明であるが，アルコホール及びキナポン液注入群に於ては貧弱である．

6) アルコホール注入標本に於ける特異変化像としては神経線維並びにその終末部の著明な嗜銀性の亢進をあげる事が出来，終末部は球状，棍状又は環状体を示すことである．

7) キナポン液注入部の特異性変化は，この薬物によるAuerbach神経叢の甚しい破壊像であって，薬物注入部Auerbach神経叢には大なる空洞を生じ，その中に壊死した神経細胞の残骸を認めたことであり，又個々の神経細胞の死滅により多数の小空洞も形成される．

8) 神経細胞の種々な変化の中で，著者は被膜細胞，副細胞等の増殖を伴わざる神経細胞の萎縮を不可逆的変化として指摘することが出来る．同様に神経線維の再生に於ても先ず副細胞の増殖が起ることが必要欠くべからざる現象と考えられる．