Nagoya J. med. Sci. 31: 155-170, 1968.

ELECTROPHYSIOLOGICAL STUDIES OF SPINAL ACTIVITY BY OCCLUSION OF AORTA

YOSHIMASA TANAKA

1st Department of Surgery, Nagoya University School of Medicine
(Director: Prof. Yoshio Hashimoto)

ABSTRACTS

The primary interest of the study is to investigate the spinal cord disturbance by aortic occlusion by electrophysiological means. Cats were chosen for the study and spinal reflexes and spontaneous discharge of motoneuron were electrophysiologically recorded on oscilloscope with complete occlusion of arch of aorta through the thoracotomy.

The monosynaptic reflex of spinal cord showed 3 phasic or 5 phasic changes in amplitude before disappearance, while multisynaptic reflexes acted in reversed phases.

Monosynaptic reflex decreased its amplitude when aortic occlusion was released during its excited stage, while it increased when it was done before the excited stage.

Spontaneous discharge of motoneuron was also investigated, which showed direct change in efferent fibers but not in afferent fibers.

In spinal cats, spontaneous discharges of motoneuron showed no relationship to amplitude of monosynaptic reflex but parallel relationship to the multisynaptic reflex.

Negative and positive potentials of anterior horn of spinal cord reduced their amplitude by aortic occlusion, but no spike of intramedullary evoked potential were changed.

Spinal reflex and spontaneous discharge of motoneuron recover following the release of aortic occlusion, and the latter anticipates in recovery.

INTRODUCTION

Recent advance in the cardio-vascular surgery has widen the indications of surgical treatment of congenital heart diseases, vascular anomalies, aortic aneurysms or other cardio-vascular disorders which have been considered surgically untreatable. Therefore, the necessity to occlude the aorta temporarily during the surgical procedures have been frequently encountered clinically which might cause spinal cord dysfunction due to circulatory disturbances. Many fundamental studies remain to be solved as to the pathogenesis of this surgical complication.

田 中 良 正
Received for publication October 25, 1967.

It was thought there were no actual complications by resection of abdominal aortic aneurysm as to spinal cord function, although Mehrez, *et al.*[1] reported a case of paraplegia which occurred following the removal of aneurysm from the infrarenal aorta. Further, Edward, *et al.*[2] observed the motor weakness of the left leg following the teflon graft in a case of ruptured abdominal aneurysm, and at autopsy it was revealed there were necrosis of gray matter of spinal cord between T_{12} and S_1 segments. Adams, *et al.*[3] reported the development of spinal cord disturbances in the case of resection of thoracic aorta aneurysm with multiple ligation of intercostal arteries of wide area. DeBakey[4] stated that there were 3 to 4% occurrence of spinal cord disturbances following the resection of thoracic aortic aneurysm even when hypothermic procedures were used.

Although clinical manifestations of spinal cord disturbances are important, the primary purpose of the study remains on the fundamental basis to analyse by the physiological condition in acute stage of anoxic condition of spinal cord, when circulatory disturbance of spinal cord by short period of aortic occlusion is given, such as the electrophysiological properties of the spinal cord. Häggqvist[5] investigated the early histological changes of large nerve cells of anterior horn of the spinal cord by temporal occlusion of thoracic aorta. It was found that more damages were seen in the nerve cells after longer occlusion time. Rexed[6] stated the permissive time for occlusion of thoracic aorta was within 15 minutes in their experiments with rabbits. Tureen[7] stated it was within 15 minutes in cats, Cooley[8] stated it was 20–30 min in human being. Iyomasa, *et al.*[9] claimed 18 minutes in dogs even though hypothermic procedure was used. Duncan, *et al.*[10] classified the aortae of dogs into 4 divisions, and they observed that occlusion at the level of infrarenal aorta caused no paralysis of hindlimbs, while occlusion of all lumbar arteries and intercostal arteries caused paraplegia. Similar results have been observed by Spencer[11].

There are many reports concerning early changes of spinal activity by blood flow disturbances. Montgomery and Luckhardt[12] studied the change of knee jerk during oxygen lack in dogs, following intraspinal pressure and occlusion of abdominal aorta. Electrophysiological studdies of the subject were carried out by Van Harrveld[13] with spinal cord compression, by Kirstein[14] and Oka[15] with occlusion of abdominal aorta, and by Gelfan[16] and Murayama[17] with occlusion of thoracic aorta, and by Kimura[18] with aortic occlusion by means of balloon-catheter.

They studied mainly the spinal reflexes or changes of evoked potential of spinal cord. However, systematic study of the simple motoneuron or activities of simple ganglion cells is a relatively recent discipline, and few investigations have been reported along this line.

In this experiment, activity of single motoneuron was studied in its anoxic condition and spinal reflexes, intraspinal evoked potential. The activity of

motoneuron was also investigated in the early stage of disturbance of blood circulation of spinal cord.

MATERIAL AND METHOD

Thirty-six adult cats weighing 2.5 kg to 3.5 kg were used in the study. They were anesthetized generally through intratracheal tube with Fluothan inhalation, and laminectomy was carried out from L_3-S_1 levels. Exposed lumbosacral spinal cord was soaked with liquid paraffin. Flaxadil was given to immobilize the animals after their arousal and then animals were fixed in the frame. Artificial respiration was carried out throughout the experiment. Anterior as well as posterior nerve roots of L_6–S_1 were divided as peripherally as posible from spinal cord. Bipolarplatinum electrodes were hooked onto the anterior and posterior roots of L_7–S_1 level and stimuli was given onto posterior roots with supramaximal single shots with 1/1 second intervals. Monosynaptic reflex (M.S.R.) and polysynaptic reflex (P.S.R.) were recorded on oscilloscope from anterior roots through the bipolar electrodes.

In another experiment, anterior roots were divided and separated under the microscope. Fibers of anterior roots were then hooked by the platinum electrode, through which motoneuron spontaneous discharge was recorded. Intraspinal evoked potential of anterior horn was recorded on oscilloscope with single shot stimulation of the ipsilateral sciatic nerve, through the metalic microelectrode of 10 μ in diameter inserted into the anterior horn from dorsal surface of spinal cord of L_6 level.

Thoracotomy was carried out with cat being fixed in the frame, so that the arch of aorta was made free from the posterior wall. Aorta was clamped and occluded at the angle of III intercostal artery origin. Then, microscopic observation of dorsal surface of spinal cord reveals immediate slowness of blood circulation, and the appearance of spinal cord becomes very pale, and slight movement of blood corpuscle in the small vessels can be visualized. Distal portion of aorta and femoral arteries are not palpated. Spinal cats were made by aspiration of cervical spinal cord through the C_5–C_7 laminectomy.

RESULTS

(*1*) *Changes of spinal reflexes by occlusion of aorta* (Fig. 1)

a) Spinal reflex before occlusion.

b) 15 seconds after clamping of aorta:

M.S.R. decreases its amplitude 50% of normal, while P.S.R. increases.

c) 60 seconds after clamping of aorta:

M.S.R. increases its amplitude up to 250% of normal and this is the maximum amplitude while P.S.R. decreases reversely.

e) 120 seconds after clamping of aorta:

M.S.R. decreases its amplitude at this time, while P.S.R. shows tendency

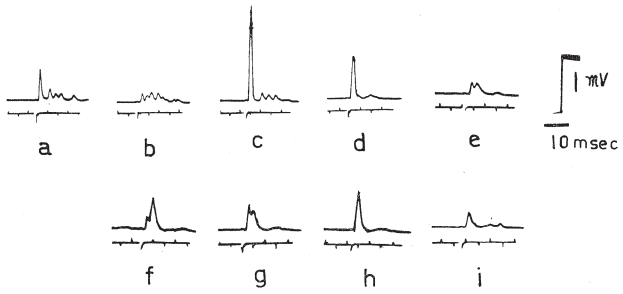

FIG. 1. Changes of spinal reflexes by occlusion of arch of arch of aorta (spinal cat).
a) Spinal reflex before occlusion.
b)—e) Spinal reflex during occlusion.
f)—i) Spinal reflex following opening of aorta clamp.
Stimuli: Single shot of 0.5 msec. 0.5 V on L_6 posterior root.
Recording from L_6 anterior root.

to increase to some extent.

f) 10 seconds following opening of aorta clamp:
Amplitude of M.S.R. decreases while amplitude of P.S.R. increases.

g) 20 seconds following the opening of aorta clamp:
Amplitude of M.S.R. increases again and amplitude of P.S.R. decreased.

h) Amplitude of M.S.R. increases further.

i) 60 seconds following the opening of aorta clamp:
Amplitude of both M.S.R. and P.S.R. decreased to minimum, then they gradually turn back to normal.

(*2*) *Change of spinal reflexes by occlusion of aorta* (Fig. 2)

When the aorta is occluded for more than 90 seconds and less than 180 seconds, changes of amplitude of M.S.R. showed 5 stages.

When aorta is occluded for either less than 90 seconds or more than 180 seconds, there were 3 stages.

Amplitude of the M.S.R. decreases immediately following the occlusion of aorta and shows its minimal amplitude—about half of normal at around 15 to 20 seconds (the first decreased stage). Then, amplitude of M.S.R. increases and reaches its maximum up to 150% to 250% at around 40 to 80 seconds (the first increased stage). Subsequently, amplitude decreases rapidly and disappears if occlusion of aorta continues usually after 2 min. 30 sec. to 3 min. 30 sec. So, in this case, 3 stage-curve is observed.

If occlusion of aorta is discontinued before M.S.R. activity disappears, the amplitude increases temporarily (the second increased stage) and this makes the 5 stage-curve.

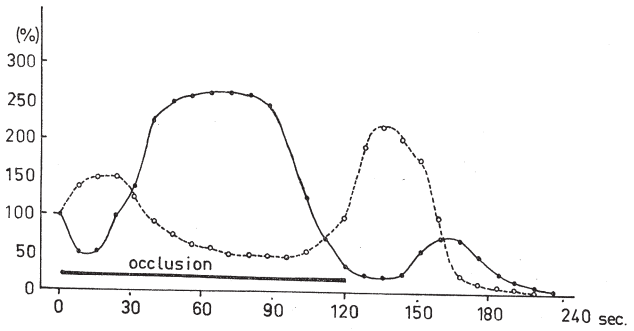

FIG. 2. Changes of spinal reflex by occlusion of aorta of a spinal cat (continuous observation).
—o— M.S.R. ---o--- P.S.R. ——— Occlusion time

Change of amplitude of P.S.R. seems to have reversed relationship to M.S.R., and the amplitude decreases and shows minimum at around 40 to 80 seconds when amplitude of M.S.R. reveals its maximum.

Curves of P.S.R. amplitude, therefore, are 4 stages.

(3) *Changes of monosynaptic reflex by occlusion of aorta of spinal cats* (Fig. 3 a)

When aorta is occluded within 30 sec., M.S.R. increases its amplitude temporarily following the release of aortic occlusion, and recovers its normal shape within a short period.

When aorta is occluded in the range of 60–80 seconds, M.S.R. reaches its maximum of the first increasing phase. When it is released, its M.S.R. am-

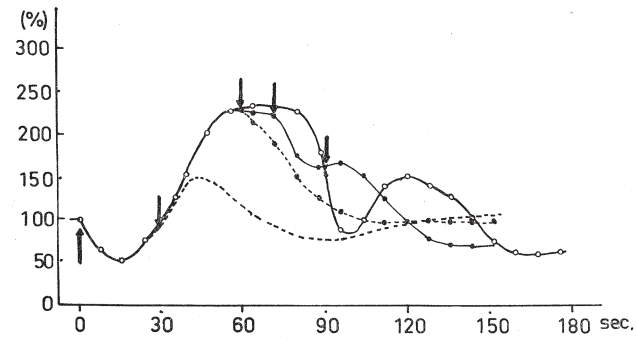

FIG. 3 a. Changes of amplitude of monosynaptic reflex by occlusion of aorta (spinal cat).
Occlusion time 30″ ------ 60″ ---●--- 70″ —●— 90″ —o—
↑: occlusion began ↓; occlusion released
This experiment was performed with 2 cats.

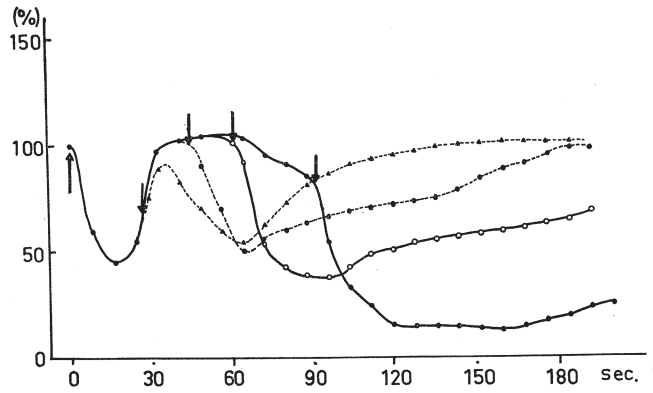

FIG. 3 b. Changes of amplitude of monosynaptic reflex by occlusion of aorta (normal cats).
Occlusion time 30″ ---▲--- 45″ ---●--- 60″ —○— 90″ —●—

plitude becomes small quickly and then it recovers gradually to normal.

When aorta is occluded in the range of 90 to 120 seconds, M.S.R. shows the second decreasing phase. When it is released, its amplitude increases temporarily and again decreases. When occlusion is continued, the second decreasing phase becomes as it is and M.S.R. disappears.

When occlusion was released in the first and the second decreasing phases, amplitude of M.S.R. becomes large temporarily.

(4) *Changes of M.S.R. by occlusion of aorta of normal and spinal cats*

When the aorta of spinal cats are occluded, its amplitude of M.S.R. decreases 50% in 15 seconds, the amplitude increases gradually to about 200% in

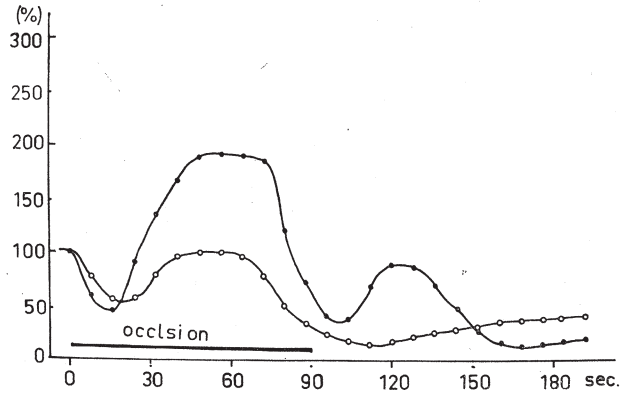

FIG. 4. Comparison of M.S.R. of normal cats and spinal cats with aortic occlusion.
Normal cats —○— Spinal cats —●—

50 to 70 seconds, then it decreases gradually, and when occlusion is released, the amplitude increases temporarily. Contrary, when the aorta of normal cats are occluded, the amplitude decreases 50% in 15 seconds as seen in spinal cats but amplitude of the first increasing phase reveals 100% at maximum and no increase in amplitude is seen following the release of occlusion. It gradually recovers to normal amplitude. In the case of aortic occlusion, amplitude of M.S.R. of spinal cats shows larger maximum than those of normal cats, and spinal cats reveal temporary increase in amplitude following the release of aortic occlusion and then recover.

(5) *Change of spontaneous discharge of motoneuron by aortic occlusion (normal cats)*

Spontaneous discharge of motoneuron of L_6 anterior root decreases slightly for 5 seconds immediately after occlusion of aorta. Then, it increases temporarily up to 200% to 250% for 10 to 25 seconds, then it disappears in 30 seconds. Furthermore, different discharges of various amplitudes were observed, which were not seen before occlusion. In other word, they reveal the recruitment of motoneuron. These motoneuron discharges disappear 30 seconds later.

Fig. 5a illustrates the changes of spontaneous discharge of many motoneurons by aortic occlusion of 30 seconds, and they show immediate decrease in short period but increase 150% to 250% in 10 to 25 seconds. Around 30 seconds later, discharge disappears suddenly.

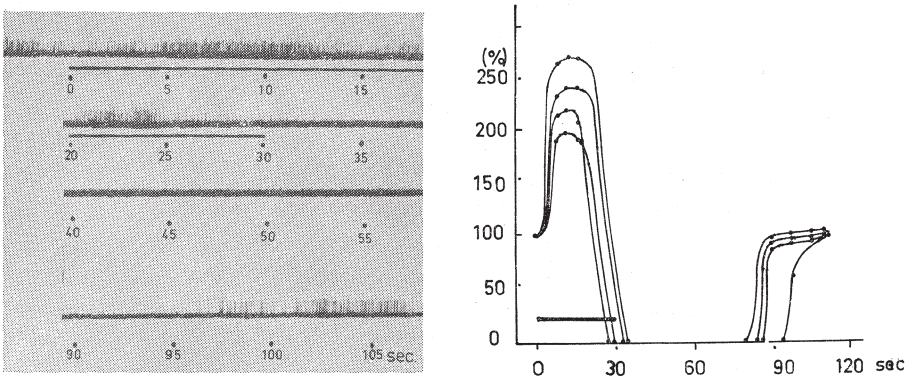

FIG. 5a. Changes of spontaneous discharge frequency of L_6 motoneuron by aorticocclusion of 30" (normal cats)
—— aortic occlusion.

When occlusion was released after 30 seconds, no spontaneous discharge was seen even following release, and silent phase was observed. Spontaneous discharge comes back in 50 to 60 seconds following release of occlusion of aorta.

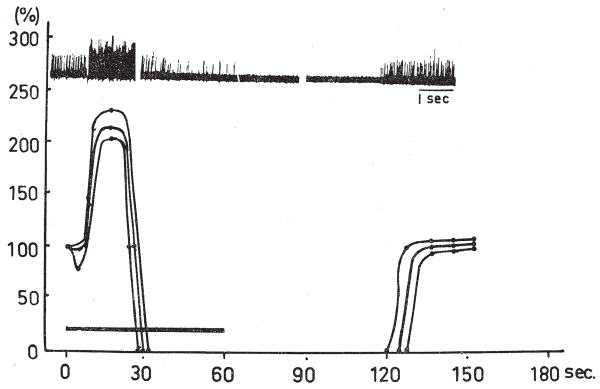

FIG. 5 b. Changes of spontaneous discharge frequency of L_6 motoneuron by aortic occlusion of 60″ (normal cats).
Vertical axis: changes of discharge frequency
Horizontal axis: time (seconds)
Photo: Spontaneons discharge.

Fig. 5 b shows the changes of spontaneous discharge of motoneuron by aortic occlusion of 60 seconds, and it shows similar change to that of occlusion of 30 seconds, but spontaneous discharge recovers promptly in 70 to 80 seconds following release.

It shows the longer occlusion time, the longer silent phase.

(6) *Changes of spontaneous discharge of afferent fibers of L_6 posterior root and efferent fibers of L_6 anterior root by occlusion of aorta (normal cats)*

Spontaneous discharge of efferent fibers of normal cats shows temporary

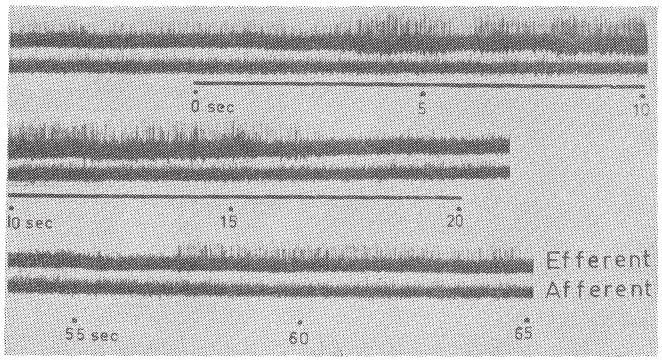

FIG. 6. Changes of spontaneous discharge of afferent fibers of L_6 posterior root and efferent fibers of L_6 anterior root by aortic occlusion of 20 seconds.
—— occlusion of aorta.

decrease in small degree immediately after the occlusion of aorta and 10 to 30 seconds later, and various discharge of recruitment of motoneuron appears with sudden increase of spontaneous discharge. Spontaneous discharge of efferent fibers decreases suddenly and disappears in 25 to 30 seconds. When occlusion is released, discharge recovers following silent phase. Contrary, spontaneous discharge of afferent fibers shows no change after the aortic occlusion as if posterior roots were separated and afferent fibers were divided.

(7) *Changes of M.S.R. and spontaneous discharge of motoneuron of normal and spinal cats* (Fig. 7 a and 7 b)

In the case of aortic occlusion, frequency of spontaneous discharge of motoneuron of L_6 anterior root increases at the first decrease stage of amplitude of M.S.R. Then, it stops at the first increase stage of M.S.R. Spontaneous

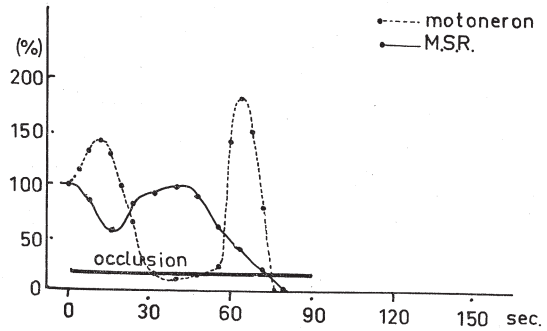

FIG. 7 a. Changes of amplitude of M.S.R. and frequency of spontaneous discharge of motoneuron of normal cats by occlusion of aorta of 90 seconds.

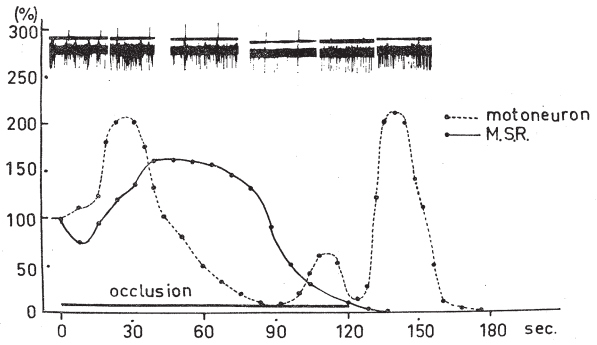

FIG. 7 b. Changes of frequency of spontaneous discharge of motoneuron and amplitude of M.S.R. of spinal cats by occlusion of aorta of 120 seconds.
—— Occlusion time
Photo: Upper trace shows amplitude of M.S.R.
Lower trace shows spontaneous discharge.

discharge shows its recruitment temporarily at the second decrease stage of M.S.R.

Amplitude of M.S.R. and spontaneous discharge of motoneuron change in the reversed relationship in the case of aortic occusion. As illustrated in Figure 2, P.S.R. and M.S.R. change in the reversed relationship, also. Therefore, spontaneous discharge changes seemingly in the parallel relationship to amplitude of polysynaptic reflex.

Following the sectioning the cervical spinal cord of a cat, its spontaneous discharge of motoneuron decreases to compare with normal cats, but amplitude of M.S.R., in the case of aortic occlusion, changes in the reversed relationship to spontaneous discharge as it was seen in a normal cat.

No difference was seen in this respect between spinal and normal cats.

(8) Evoked potential of spinal anterior horn by occlusion of aorta

A microelectrode was inserted into anterior horn of spinal cord (Fig. 8 a and 8 b) through posterior root of L_6 and evoked potential was recovered by stimulation of the ipsilateral sciatic nerve. This shows initial spike, negative potential of 7 or 8 spikes of about 5 msec. duration and positive potential of 7 or 8 spikes of fairly long duration.

Evoked potential (b) following aortic occlusion shows no change to compare with pre-occlusive potential (a) and potential after aortic occlusion of 90 sec. (c) shows small negative and positive potentials although initial spikes show no change. Negative potential reduced to one half in amplitude and 150 seconds later (e), negative and positive potentials disappear although no change was seen of initial spikes. If aortic occlusion was released after 150 seconds, evoked potentials of 8 minutes following recovers (f).

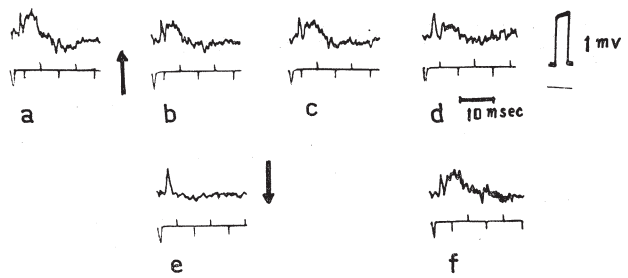

FIG. 8 a. Evoked potential of L_6 anterior horn of spinal cord.
Stimulation: 0.5 msec. 0.5 V once per min. ipsilateral sciatic nerve.
↑: aortic occlusion. ↓: released.
a) preocclusive evoked potential.
b)~e) during occlusion.
f) 8 min. following relese of occlusion.

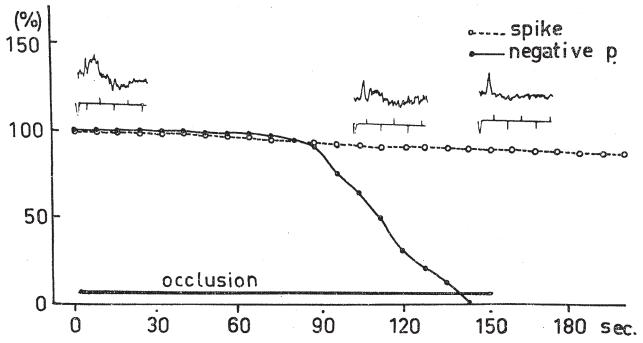

FIG. 8 b. Amplitude of intramedullary evoked potential by occlusion of aorta.

Change of the initial spike and negative potential by occlusion of aorta.

Initial spikes showed slight change by occlusion of aorta, but gradual reduction of amplitude was observed. Contrary, negative potential decreases its amplitude from 90 seconds of occlusion and disappears by occlusion of 150 seconds.

Time length for disappearance of negative potential is same as the time for disappearance of spinal reflex.

Amplitude of the spinal reflex changes in reverse relationship by occlusion of aorta, but amplitude of negative potential decreases suddenly around 90 seconds of occlusion and then disappears.

(*9*) *Recovery of M.S.R. and spontaneous discharge of motoneuron* (Fig. 9)

By occlusion of aorta, amplitude of M.S.R. decreases with previously described course and finally disappears.

By release of occlusion of aorta, amplitude of M.S.R. gradually increases its size to recover completely to normal.

In the case of aortic occlusion within 2 min. 30 seconds, the fashion of M.S.R. recovery seems in proportionally related course to the time of occlusion.

Spontaneous discharge of motoneuron, however, increases immediately following the occlusion and stops at about 30 seconds later and shows silent phase. When occlnsion was released, it appears abruptly after some period of silent phase and recovers to normal frequency. If occlusion time is within 1 min. recovery course shows fairly smooth curve, but if it is longer than 1 min., curve shows quick elevation. Recovery of motoneuron anticipates to the recovery of M.SR.

DISCUSSION

Kirstein[14] stated that when the abdominal aorta of the unanesthetized

spinal cat was occluded, M.S.R. changed its amplitude in 3 phases of initial, depress and augmentation and P.S.R. did its amplitude in 2 phases.

In the present experiment, it was observed the accelerated M.S.R. before disappearance of spinal reflex and amplitude of M.S.R. changed in 3 or 5 phasic mode, depending upon the time of oc clusion of aorta. Similar results were noted also by Gelfan *et al.* in 1955.

M.S.R. disappears in 2 minutes 30 seconds to 3 minutes 30 seconds by aortic occlusion.

Van Harreveld *et al.*[13] stated spinal reflex disappears in 2 min. 25 sec. to 4 min. 30 sec. by occlusion of thoracic aorta. Brooks *et al.*[19] reported the initial suppressin of M.S.R. was secondary change due to anoxic change of interneuron. Van Harreveld[20] stated the increased amplitude of M.S.R. by occlusion of aorta was due to the excitement of anterior horn cells themselves and not due to the release of suppression from interneuron. On the contrary, Gelfan and Tarlov[22] revealed this was due to the release phenomenon of anterior horn cells.

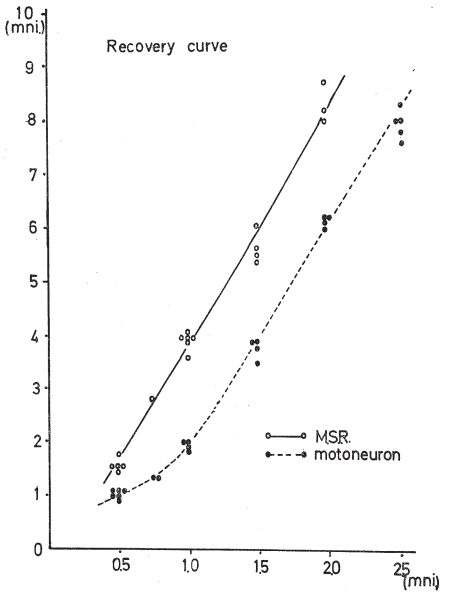

FIG. 9. Relationship between M.S.R. and spontaneous discharge of motoneuron.
Vertical axis: Occlusion of aorta.
Horizontal axis: Time needed for recovery.

In the present experiment, spinal reflex changes its M.S.R. and P.S.R. in the reversed relationship and the activity of the motoneuron depressed when M.S.R. showed the maximal amplitude. It seems likely that anoxic condition of interneuron was responsible for the change in the spinal reflex. In other word, the anoxic change of spinal cord by aortic occlusion influenced first on the synapsises and this makes the blockage of impulse conduction of spinal cord. These changes of spinal reflex are not by the result in central neural mechanism but the activity of spinal cord itself.

When the aortic occlusien was released, the M.S.R. of the spinal cat shows immediate and remarkable changes in its amplitude. For example, when it was released in the excitement phase of 40 sec. to 60 sec,, the amplitude of M.S.R. reduced remarkably, while it increased if the occlusion was released after 80 seconds.

Brooks *et al.*[23] pointed out the similar facts that the amplitude of M.S.R. decreased promptly by inhalation of oxygen in the state of excited phase and increased before and after the excited phase. Amplitude of spinal reflex

changes not only the anoxic condition of spinal cord but the additional inhalation of oxygen.

While the amplitude of M.S.R. of intact cats shows its increase around 100 to 150 per cent at maximum. The fact may be suggested the M.S.R. gets the influence of central neural mechanism.

Suda[21] pointed out that the stimulation of reticular formation made the suppression or accelerating effect upon the spinal reflex.

Occlusion of the aorta makes the circulatory disturbance of the spinal cord and increase of amplitude of M.S.R. in the spinal cats.

In intact cats, the spontaneous discharge of motoneuron of spinal cord increased up to 250% in 10 to 25 seconds following the occlusion of aorta with various other recruitment phenomena of motoneuron. The spontaneous discharge disappeared suddenly 30 seconds after the occlusion of aorta. These changes of spontaneous discharges of motoneuron seem due to either the anoxic change of spinal cord or the influence of central or peripheral neural mechanism.

The recording of spontaneous discharge from the corresponding posterior nerve root showed no change by occlusion of aorta but the spontaneous discharge of motoneuron showed remarkable change. Furthermore, the same change is observed in the experiment with spinal cats, so it was revealed that the spontaneous discharge of motoneuron was not due to central or peripheral neural mechanism but due to anoxic change of spinal cord itself. Gelfan *et al.*[22] and Van Harreveld could demonstrate the depolarization of motor cells of spinal cord by anoxic change. Brook[23] also demonstrated the depolarization and hyperactivity of motoneuron by anoxic change of spinal cord.

In the present experiments, anoxic change of spinal cord by occlusion of aorta made the increase of spontaneous discharge for 10 to 25 seconds following occlusion of aorta, as well as the development of recruitment of motoneuron of spinal cord.

In spinal cats, changes of amplitude of M.S.R. and changes of spontaneous discharge of motoneuron by occlusion of aorta show no correlation but spontaneous discharge of motoneuron increases when amplitude of M.S.R. are small.

Fujimori *et al.*[24] also stated that M.S.R. did not mean the excitement of motoneuron of spinal cord.

As Van Harreveld[20] reported, the changes of M.S.R. by aortic occlusion had no parallel relationship to the activity of motor cells of spinal cord. It is, therefore, presumed the excitement of M.S.R. by anoxic change of spinal cord never indicates the excitement of motoneuron of spinal cord. Similarly, silent stage of motoneuron activity does not necessarily indicate the arrest of spinal cord activity.

The evoked potentials of spinal cord surfaces have been investigate by Gelfan[16] and Kimura[18]. They stated that negative and positive potentials

were affected by occlusion of aorta. As described above, it was revealed from the present study that negative potentials showed the reduction of amplitude starting at 90 second following the occlusion and disappeared in 2 min. 30 seconds to 3 min., while spike activities showed no damage within 3 min. following the aortic occlusion.

Gasser[25] states that the spike activity is the result of orthodromic stimulation of G_{1a} while negative potential complex is of interneuronal activities.

Changes of negative potential complex seem for the interneurons to respond to anoxic condition of spinal cord.

The stage that negative phase disappeared by aortic occlusion corresponds almost to the stage of disappearance of spinal reflex. Clinically, disappearance of knee jerk at this stage may be the outcome of spinal activity to anoxic change of spinal cord.

Amplitude of M.S.R. changes by aortic occlusion and this returns to normal after occlusion is released.

Spontaneous discharge of motoneuron also shows abrupt change by occlusion of aorta and stops at around 30 seconds following occlusion, but this returns gradually following its release.

In aortic occlusion within 5 minutes, recovery of spontaneous discharge of motoneuron anticipates the recovery of amplitude of M.S.R.

The longer the occlusion is made, the more difference is noted between these two activities electrophysiologically.

SUMMARY

1) In spinal cats, the change of amplitude of M.S.R. by aortic occlusion is seen in 3 or 5 phasic mode, and change of amplitude of P.S.R. is reveresed phasic to the change of amplitude of M.S.R.

2) In spinal cats, when the aortic occlusion is released during the excitement stage of M.S.R., the amplitude reduces abruptly. When it is released before or after the excitement stage, the amplitude increases temporarily.

3) M.S.R. of spinal cats shows bigger amplitude in excitement stage than that of intact cats.

4) In intact cats, spontaneous discharge of motoneuron increases immediately by occlusion of aorta, together with various other motoneuron recruitment. Spontaneous discharge arrests 30 seconds after the occlusion of aorta and shows silent period, but afferent input from the posterior roots shows no change.

In spinal cats, changes of M.S.R and spontaneous discharge of motoneuron by aortic occlusion don't correspond and excitement of spinal reflex doesn't indicate the excitement of spinal refiex.

5) Negative potential which is evoked from anterior horn of spinal cord reduces the amplitude at around 90 seconds and disappears at around 2 min

30 seconds to 3 min. 30 seconds following aortic occlusion.

Time of disappearance of negative potential corresponds to the time of disappearance of spinal reflex in close relationship.

6) Recovery of spontaneous discharge of motoneuron by release of aortic occlusion anticipates the recovery of M.S.R. and the longer occlusion gives the more dissociation of two activities in recovery process.

ACKNOWLEDGEMENT

The author wishes to express cordial acknowledgement to Professor Dr. Yoshio Hashimoto, Professor Dr. Keiichi Murata and other members of the 1st Surgical Department, School of Medicine, Nagoya University for their guidance and encouragement.

REFERENCES

1) Mehrez, I. O., Nabseth, D. G., Hogan, E. L. and Deterbig, R. A. Jr., Paraplegia Following Resection of Abdominal Aortic Aneurysm, *Ann. Surg.*, **156**, 890, 1962.
2) Hogan, E. L. and Romanul, F. C. A., Spinal Cord Infarction Occurring During Insertion of Aortic Graft, *Neurology*, **16**, 67, 1966.
3) Adams, H. D. and van Geertruyder, H. H., Neurologic Complications of Aortic Surgery, *Ann. Surg.*, **144**, 574, 1956.
4) De Bekey, M. E., Changing Concepts in Thoracic Vascular Surgery, *J. Thoracic Cardiovas. Surg.*, **38**, 145, 1959.
5) Häggqvist, G., Die Tonische Innervation der Skeltmuskeln,
6) Rexed, B., Some Observations on the Effect of Compression of Short Duration of the Abdominal Aorta in the Rabit, *Acta Psychiat. Neural. Scond.*, **15**, 365, 1940.
7) Tureen, L. L., Effect of Experimental Temporary Vascular Occlusiou of the Spinal Cord, *Arch. Neurol. Psychiat.*, **35**, 789, 1936.
8) Cooley, D. and Debekey, M. D., Surgical Consideration of Intrathoracic Aneurysm of Aorta Great Vessel, *Ann. Surg.*, **135**, 160, 1950.
9) Iyomasa, Y., Takagi, K. Ogiwara, T., Kamashima, Y., Amari, S., Inoue, K. and Hirose, N., Experimental Study of Surgery of Pulmonary Stenosis and Aortic Stenosis II., *Jap. J. Thorac. Surg.*, **5**, 376, 1952 (in Japanese).
10) Killen, D. A., Edwards, R. H., Adkins, B. Jr. and Boenton, F. H., Spinal Cord Ischemia Following Mobilization of Canine Aorta from Posterior Parties, *Ann. Surg.*, **162**, 1063, 1965.
11) Spencer, F. G. and Zimmerman, T. M., The Influence of Ligation of Inteacostal Arteries on Paraplegia in Dogs, *Surg. Forum*, **9**, 340, 1958.
12) Montgomery, M. F. and Luckhardt, A. B., The Effect of Intracranial and Intraspinal Respons on Knee Jerk, *Amer. J. Physiol.*, **91**, 210, 1930.
13) Van Harreveld, A., The Resistance of Central Synaptic Conduction to Asphyxiation, *Amer. J. Physiol.*, **133**, 572, 1941.
14) Kirstein, L., Early Effects of Oxygen Lack and Carbondioxide Execess on Spinal Reflex, *Acta Physiol. Scand.*, **23**, Suppl., 80, 1958.
15) Oka, Y., Electrophysiological Study of Spinal Reflex of Spinal Cord Injury and Circulatory Disturbance, *J. Yonago Med. Ass.*, **16**, 1965.
16) Gelfan, S. and Tarlov, I. M., Differential Vulnerability of Spinal Cord Structures to Anoxia, *J. Neurophysiol.*, **18**, 170, 1955 (in Japanese).
17) Murayama, M. and Smith, M., Rigidity of Hind Limbs of Cats Produced by Occlusion

of Spinal Cord Blood Supply, *Neurology*, **15**, 565, 1965.
18) Kimura, C., Tsunekawa, K., Nimura, Y. and Ikeda, M., Vascular Dynamic of Spinal Cord and Its Disturbances, *Saigai Igaku*, **8**, 261, 1965 (in Japanese).
19) Brooks, C. McC. and Fuortes, M. G. F., Potential Changes in Spinal Cord Following Administration of Strychine, *J. Neurophysiol.*, **15**, 257, 1952.
20) Biersteker, P. A. and Van Harreveld, A., The Nature of the Rigidity Caused by Spinal Cord Asphyxiation. *J. Physiol.* (*London*), **166**, 382, 1963.
21) Suda, I., Koizumi, K. and Brooks, C. McC., Reticular Formation Influences on Neurons of Spinal Reflex Pathway, *J. Neurophysiol.*, **21**, 113, 1958.
22) Gelfan, S. and Tarlov, I. M., Interneurones and Rigidity of Spinal Origin, *J. Physiol.* (*London*), **146**, 594, 1959.
23) Brooks, C. McC. and Ecoles, J. C., A Study of the Effects of Anesthesia and Asphyxin on the Monosynaptic Pathway, *J. Neurophysiol.*, **10**, 349, 1947.
24) Fujimori, B., Tokizane, T. and Eldred, E., Effect upon Monosynaptic Reflexes of Decamethonium and Succinylcholine. 1) Peripheral Mechanism. 2) Central Mechanism, *J. Neurophysiol*, **22**, 165, 1958.
25) Gasser, H. S. and Graham, H. T., Potentials Produced in the Spinal Cord by Stimulation of Dorsal Roots, *Amer. J. Physiol.*, **103**, 303, 1933.