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Effects of Increased Tissue Pressure upon Nerve Conduction Velocity*

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= Abstract =Studying changes of nerve conduction velocity may be useful in the diagnosis, treatment and prognosis of the compartmental syndrome. An experimental model was made to keep the hind leg of the rabbit at the constant pressure of 80 mm Hg, 120 mm Hg and 160 mm Hg during 6 hours and 12 hours using volume expander.

Experiments were performed in 6 groups (Group 1; 80 mm Hg−6 hours, Group 2; 120 mm Hg−6 hours, Group 3; 160 mm Hg−6 hours, Group 4; 80 mm Hg−12 hours, Group 5; 120 mm Hg−12 hours, Group 6; 160 mm Hg−12 hours) to study the early decrease of nerve conduction velocity and the late recovery for 16 weeks, and following results were obtained.

1. Nerve coduction velocity fell 95.8 per cent at 80 mm Hg in 4 hours, 93.4 per cent at 120 mm Hg in 2 hours and 95.5 per cent at 60 mm Hg in 1 hour.
2. Almost complete recovery was observed in Group 1 with the level of 95 per cent statistical significance.
3. Time and pressure were both important factors to the recovery of the nerve conduction velocity and the longer the pressurized time and the higher the applied pressure the less recovery was seen.
4. Reduction of nerve conduction velocity was observed early in the compartmental syndrome and this was considered significant in the diagnosis of compartmental syndrome.

Key Words: ***Tissue pressure, Nerve conduction velocity, Compartmental syndrome***

INTRODUCTION

Effects of increased tissue pressure on extremities or compartments have been known from the ancient and the results might be disastrous. Recently there was increasing tendency of industrial injuries and traffic accidents and many compartmental syndromes occurred.

In 1881 Volkmann reported his treatise on ischemic muscle paralysis and contracture, pointing out the consequences of excessive pressure on the nerves and the muscles of the extremities. In 1926 Jepson proved that paralysis and contracture could be prevented by prompt decompression.

Many articles were published discussing specific etiologies of compartmental syndromes; burn (Matsen 1975), carbon monoxide poisoning (Suk *et al.* 1971), prolonged limb compression (Mubarak and Owen 1976), prolonged use of tourniquet, Hauser's procedure (Wiggins 1975), vascular surgery (Reneman 1975) and intensive use of muscle (Whitesides *et al.* 1975), *etc.*

Whitesides *et al.* (1975) used needle and manometer for the measurement of the intracompartmental pressure. Mubarak and Owen (1976) used wick catheter and Rorabeck and MacNab (1975) used slit catheter. For the criteria of the fasciotomy, Whitesides *et al.* (1975) took 45-60 mm Hg (normal; 9-15 mm Hg) using needle manometer method and Mubarak *et al.* (1978) took 30 mm Hg (normal; 0-8 mm Hg) using wick catheter method.

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Rorabeck and Clarke (1978) and Hargens *et al.* (1979) studied the effects of intracompartmental pressure upon nerve function but they described the effects in short term and the results of early decompression, and there were little comments on the long term results and recovery.

It is postulated that studying the changes of the nerve conduction velocity might be helpful in the diagnosis, treatment and prognosis of the compartmental syndrome. An experimental model was designed to keep the hind leg of rabbits at the constant pressure of 80, 120 and 160 mm Hg during 6 and 12 hours using volume expander. Serial changes of the nerve conduction velocity were checked in 6 groups to study the early decrease of nerve conduction velocity and the late recovery for 16 weeks.

MATERIALS AND METHODS

Sixty mature white rabbits, unselected as to sex and weighing 2.5-3.5 kg, were used as experimental animals. Penthotal was injected to an ear vein of the rabbit and general anesthesia was induced. Hair was removed from both legs and four extremities were tied to the table for immobilization. Basic studies such as nerve conduction velocity and electromyography was checked preoperatively to know the normal state and to get the control values.

Wick catheter was introduced to the central-upper portion of the deep posterior compartment of one hind leg of a rabbit. After connecting to barometer with three-way stopcock, tissue pressure was measured. Three-inches tourniquet was applied to the thigh and inflated lightly to 30 mm Hg. Medicut was inserted to the central-lower portion of the deep posterior compartment and the dextran was infused and the necessary levels of the tissue pressure were maintained by monitoring the tissue pressure (Fig. 1).

The levels and durations of pressure applied in 6 groups of rabbits were as follows:

Group 1: 80 mm Hg 6 hours = 10 rabbits
Group 2: 120 mm Hg 6 hours = 10 rabbits
Group 3: 160 mm Hg 6 hours = 10 rabbits
Group 4: 80 mm Hg 12 hours = 10 rabbits
Group 5: 120 mm Hg 12 hours = 10 rabbits
Group 6: 160 mm Hg 12 hours = 10 rabbits

The time when the tissue pressure was elevated to the desired level was set to time zero. Nerve

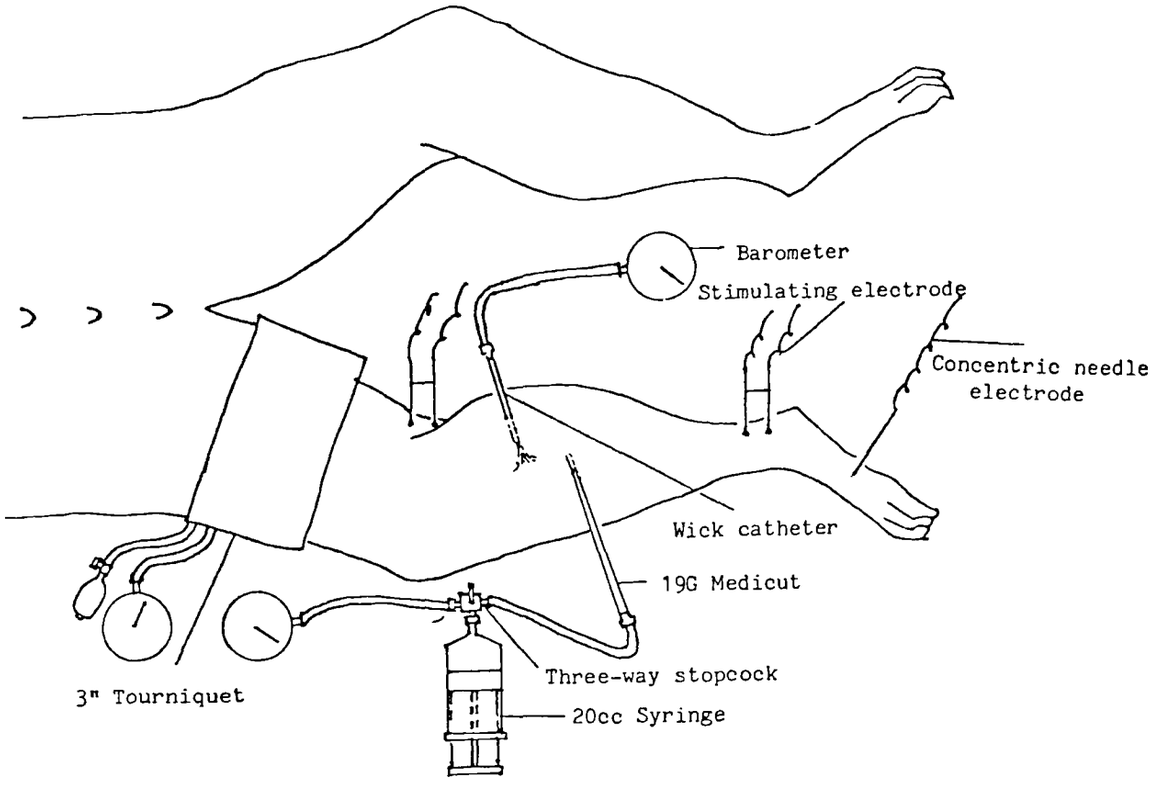

Fig. 1. Schematic model of experiment.

conduction velocity was measured every one hour for the first ten hours, then every two hours till twenty-four hours, and then on the second day, fourth day, first week, second, third, fourth, sixth, eighth, tenth, twelfth and sixteenth week. Nerve conduction velocity was measured by TECA 42 type electromyography machine with conventional measuring method of motor nerve conduction velocity. Concentric needle electrode was inserted to the plantar muscle of the rabbit, after confirming the muscle by electromyography, electrical stimulation was applied on the medial aspect of the ankle joint and the posterior aspects of the knee joint. Two graphs were taken from the electromyography machine and the time interval was checked. The distance between the two points of electrical stimulation was measured with a string ruler, and the nerve conduction velocity was calculated by dividing the distance into the time interval (m/sec).

RESULTS

1. Reduction of Nerve Conduction Velocity with the Progress of Time

In 80 mm Hg groups (Group 1 and 4) nerve conduction velocity was reduced 6.2 per cent in 1 hour, 25.5 per cent in 2 hours, 48.0 per cent in 3 hours and 95.8 per cent in 4 hours, and in 4 hours nerve conduction was blocked in 17 cases out of 20 cases. At 5 hours nerve conduction was blocked in all cases.

In 120 mm Hg group (Group 2 and 5) nerve conduction velocity was reduced 47.5 per cent in 1 hour, 93.4 per cent in 2 hours, and in 2 hours nerve conduction was blocked in 16 cases out of 20 cases. At 3 hours nerve conduction was blocked in all cases.

In 160 mm Hg group (Group 3 and 6) nerve conduction velocity was reduced 95.5 per cent in 1 hour, and at that time nerve conduction was blocked in 15 cases out of 20 cases. At 2 hours nerve conduction was blocked in all cases (Fig. 2).

2. Recovery of Nerve Conduction Velocity with the Progress of Time after Removal of Pressure

In Group 1 (80 mm Hg-hours), after removal of pressure, nerve conduction velocity was recovered to 56.8±7.1 per cent in 1 hour, 84.4±6.2 per cent in 6 hours, 93.8±5.7 per cent in 1 week and 99.1±5.1 per cent in 16 weeks. There was no difference with the level of 95 per cent statistical significance (t=0.35)(Fig. 3).

In group 2 (120 mm Hg—6 hours), after removal of pressure, nerve conduction velocity was recovered to 33.0±23.2 per cent in 1 hour, 65.7±4.9 per cent in 6 hours, 77.4±8.9 per cent in 1 week, 83.2±7.9 per cent in 16 weeks. It could not be considered as complete recovery since there was difference with the level of 99 per cent statistical significance (t=5.36)(Fig. 3).

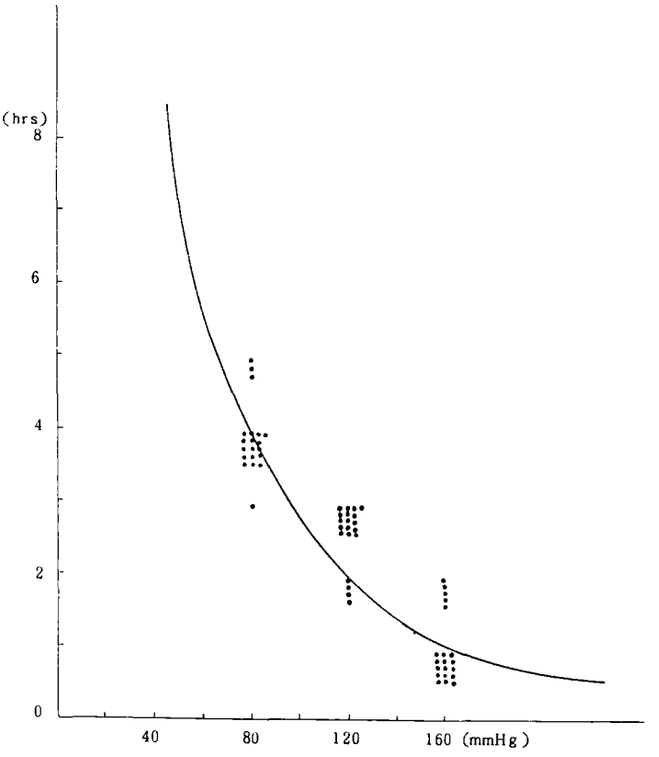

Fig. 2. Corelation between the pressure applied and the duration required for total loss of nerve function.

In group 3 (160 mm Hg—6 hours), nerve conduction velocity was recovered to 12.1±12.6 per cent in 3 hours, 22.8±14.0 per cent in 6 hours, 60.0±15.0 per cent in 1 week and 71.3±15.6 per cent in 16 weeks. It was not complete recovery since there was difference with the level of 99 per cent statistical significance (t=5.39)(Fig. 3).

In Group 4 (80 mm Hg—12 hours), after removal of pressure, nerve conduction velocity was recovered 4.8±14.4 per cent in 6 hours, 33.2±332. per cent in 1 week and 66.7±28.0 per cent in 16 weeks. It was not complete recovery since there was difference with the level of 99 per cent statistical significance (t=3.58). In one case nerve conduction was not recovered in 16 weeks (Fig. 4).

In Group 5 (120 mm Hg—12 hours), after removal of pressure, nerve conduction velocity was recovered 5.6±16.8 per cent in 1 week, for the first time and 48.7±35.0 per cent in 16 weeks. Much difference was observed in 16 weeks com-

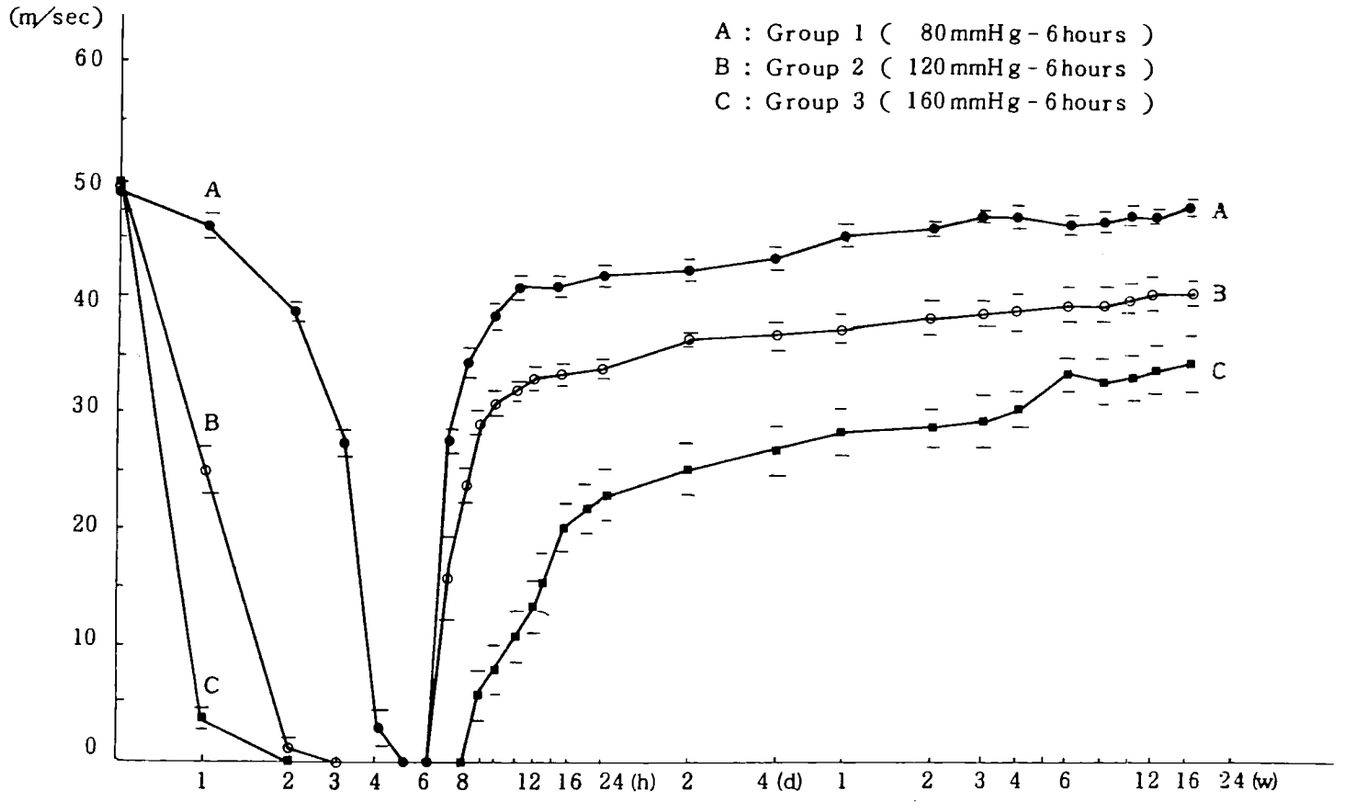

Fig. 3. Sequential changes of nerve conduction velocity after muscle compartments were pressurized for 6 hours

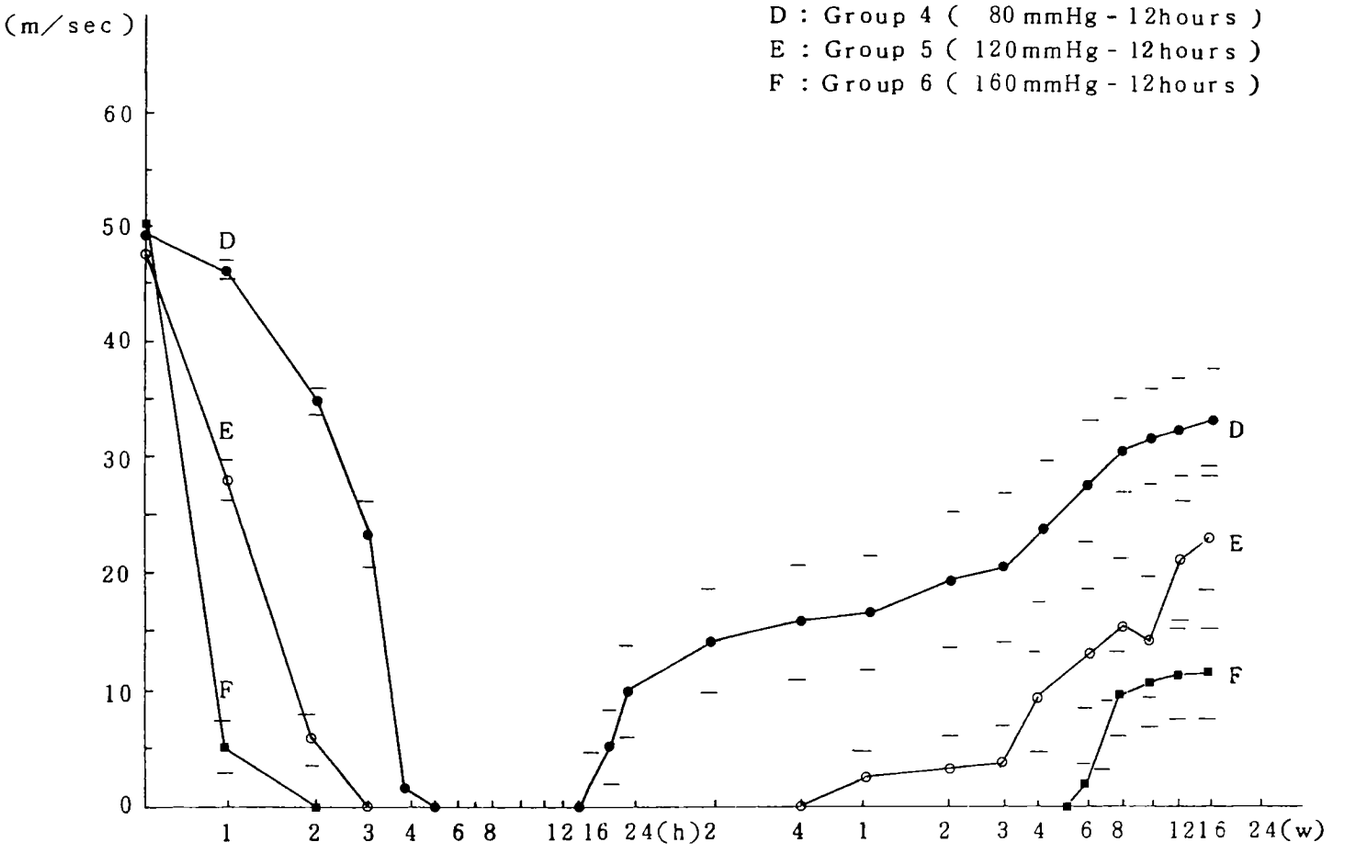

Fig. 4. Sequential changes of nerve conduction velocity after muscle compartments were pressurized for 12 hours.

Table 1. Statistical analysis of nerve conduction velocity (T-test)

Group	X_1	X_2	S_1	S_2	t	Difference
1	48.56	48.11	3.22	2.47	0.35	−
2	48.91	40.70	3.02	3.75	5.36	+
3	48.52	34.61	3.00	7.55	5.39	+
4	49.28	32.86	4.14	13.80	3.58	+
5	47.81	23.30	3.34	16.71	4.52	+
6	49.52	11.32	1.89	14.11	8.43	+

X_1: Means of nerve conduction velocity at preop.
X_2: Means of nerve conduction velocity at postop. 16 weeks.
S_1: Standard deviations at preop. S_2: Standard deviations at postop. 16 weeks.
95% level of significance: $t>t_{.05}=2.10$ 99% level of significance: $t>t_{.01}=2.88$

pared to preoperative state($t=4.52$) and in 3 cases nerve conduction was not recovered in 16 weeks (Fig. 4).

In comparison with the preoperative state, almost complete recovery was observed only in Group 1 with the level of 95 per cent statistical significance and in other five groups there were differences with the level of 99 per cent statistical significance (Table 1). The difference of recovery according to the pressurized time, between 6-hour groups (Group 1,2 and 3) and 12-hour groups (Group 4, 5 and 6), was statistically significant ($X^2=17.73$) (Table 2). In consideration of the difference of recovery according to pressure comparing 80 mm Hg groups (Group 1 and 4) with 120 Hg groups (Group 2 and 5), there was no difference with the level of 95 per cent statistical significance ($X^2=2.10$). In the case of 120 mm Hg groups and 160 mm Hg groups (Group 3 and 6) there was also no difference with the level of 95 per cent statistical significance ($X^2=5.66$). But in the case of 80 mm Hg groups and 160 mm Hg groups there was difference with the level of 99 per cent statistical significance ($X^2=14.84$)(Table 3).

Table 2. Statistical analysis of recovery according to the pressurized time

Recovery	6 hours	12 hours	Total
-40%	0	10	10
40 -80%	9	13	22
80%-	21	7	28
Total	30	30	60

* Data; numbers of rabbits $X^2=17.73>X^2_{.99}=9.21$

Table 3. Statistical analysis of recovery according to the applied pressure

Recovery	80 mm Hg	160 mm Hg	Total
-40%	1	6	7
40 -80%	4	11	15
80%-	15	3	18
Total	20	20	40

* Data; numbers of rabbits $X^2=14.84>X^2_{.99}=9.21$

DISCUSSION

There were many experimental models of tissue pressure elevation such as vein occlusion (Jepson 1926), fluid infusion (Rorabeck and Clarke 1978), balloon insertion (Sheridan and Matsen 1975) and air splint (Matsen *et al.* 1976), *etc.* In this experiment, fluid infusion method was used and proximal migration of fluid was prevented by three-inch tourniquet.

There are several objective methods of determining degrees of nerve injuries such as histopathological methods to demonstrate structural changes and nerve conduction study and electromyography to demonstrate functional changes. Histopathological method can be used for research purposes such as animal experiments but it has only limited use in human being clinically. Nerve conduction velocity measurement can be performed easily, noninvasively and control values may be easily obtained from the opposite limb. Although serves are more resistant to permanent injury from ischemia than muscles, they demonstrate functional abnormalities before abnormal muscle function is detectable. Most compartments contain nerves,

and nerve conduction velocity reflects the physiological status of intracompartmental tissue.

Matsen *et al.* (1977) and Mubarak *et al.* (1978) reported that acute compartment syndrome could occur when tissue pressure was over 40 mm Hg. Rorabeck and MacNab(1978) elevated the tissue pressure of the anterior compartment of dogs by infusing plasma and found that nerve conduction velocity was recovered to normal value when fasciotomy was performed after 4 hours but nerve conduction velocity was not recovered to normal value within 6 hours when fasciotomy was performed after 12 hours. Hargens *et al.* (1979) reported that the minimum pressure which caused complete nerve block was 50 mm Hg which took 300 minutes. In case of 120 mm Hg complete nerve block occurred after 50 minutes and in case of 30-40 mm Hg only incomplete nerve block was observed.

Although Lundborg(1970) insisted that pressure was the primary cause of peripheral nerve injury, there were many authors who emphasized ischemia for the main cause of the reduction of nerve conduction velocity in tissue pressure elevation. Whitesides *et al.* (1975) applied tourniquet to the thigh of dogs with the pressure of 600 mm Hg and found that nerve conduction was maintained for 70-75 minutes, but excitability of muscle was maintained for 180 minutes.

In case of tissue pressure elevation it was different from above mentioned occasion. Mubarak *et al.* (1978) proposed ischemia to be only a hypothesis and Lundborg(1970) and Matsen *et al.* (1977) proposed the ischemia-pressure complex hypothesis. According to the recovery of nerve injury after tissue pressure elevation Matsen *et al.* (1977) reported that contracture was noticed in case of complete ischemia for 4-12 hours. Rorabeck and Clarke (1978) reported that complete nerve block was not caused after 12 hours in case of 40 mm Hg but the reduced velocity was not recovered to normal value after 6 hours.

Hargens *et al.* (1979) experimented with anterior compartments of dogs and they found near complete recovery in case of 160 mm Hg when fasciotomy was performed after 4 hours, but light microscopic studies of nerve tissue which was pressurized above 60 mm Hg for 8 hours showed a spectrum of peripheral neuropathy at 3 weeks. At pressure of 120 mm Hg for 8 hours, various stages of axonal degeneration and demyelination were encountered. Electron microscopic studies showed pathological alterations consistent with widespread Wallerian degeneration.

Rorabeck *et al.* (1978) made a similar experiment and reported that when a pressure of 80 mm Hg was introduced and maintained from time zero, the conduction velocity fell to 0 m/sec after 4 hours. If fasciotomy was carried out at 4 hours, these changes in the conduction velocity was completely reversible. If fasciotomy was delayed for 8 hours, however, the conduction velocity was reversible but 3 hours was required for its return to normal. If fasciotomy was further delayed for 12 hours at 80 mm Hg pressure, irreparable damage to the nerve often occurred and the conduction velocity did not return to normal. At higher pressure of 120 or 160 mm complete block occurred within the first hour. Regardless of the timing of fasciotomy, *i.e.,* at 4, 8, or 12 hours, irreversible damage of the nerve occurred and the conduction velocity did not return to normal resting values for a period of 6 hours following fasciotomy.

Sheridan *et al.* (1975) treated 66 cases of acute compartment syndrome by faschiotomy. Fasciotomy performed less then 12 hours after the onset of the compartment syndrome, resulted in normal function in 68 per cent of cases. Only 8 per cent of those having fasciotomy more than 12 hours had normal function.

REFERENCES

Hargens AR, Romine JS, Sipe JC, Evans KL, Mubarak SJ, Akeson WH. Peripheral nerve-conduction block by high muscle compartment pressure. J. Bone and Joint Surg. 1979, 61±A: 192-200

Jepson PN. Ischemic contracture. Experimental study. Ann. Surg. 1926, 84: 785-795

Lundborg G. Ischemic Nerve Injury. Scandinavian J. Plastic Surg. 1970, Supplement 5

Matsen FA III. Compartment syndrome. A unified concept. Clin Orthop. 1975, 113:8-14

Matsen FA III, Sheridan GW, Krugmire RN Jr. Monitoring of intramuscular pressure. Surg. 1976, 79:702-709

Matsen FA III, Mayo KA, Krugmire RB, Sheridan GW, Kraft GH. A model compartmental syndrome in man with particular references to the quantification of nerve function. J. Bone and Joint Surg. 1977, 59-A: 648-653

Mubarak SJ, Hargens AR, Owen CA, Akeson WH, Garetto LP. The wick technique for measurement of intramuscular pressure: A new research and clinical tool. J. Bone and Joint Surg. 1976, 58-A: 1016-1020

Mubarak SJ, Owen CA, Hargens AR, Garetto LP, Akeson WH. Acute compartment syndromes: Diagno-

sis and treatment with aid of the wick catheter. J. Bone and Joint Surg. 1978, 60-A: 1091-1095

Reneman RS. The anterior and the lateral compartment syndrome of the leg due to intensive use of muscles. Clin. Orthop 1975, 113: 52-57

Rorabeck CH, MacNab I. The pathophysiology of the anterior tibial compartmental syndrome. An experimental investigation. J. Trauma 1978, 18:299-304

Sheridan GW, Matsen FA III. An animal model of the compartmental syndrome. Clin. Othop. 1975, 113:36-42

Suk SI, Hahn MS, Kim KW, Lee EY. Clinical analysis on ischemic myositis associated with carbon monxide poisoning. Journal of Korea Orthop. Ass. 1971, 6:335-360

Whitesides TE Jr., Haney TC, Hirada H, Holmes HE, Morimoto K. A simple method for tissue pressure determination. Arch. Surg. 1975, 110:1311-1313

Wiggins HE. The anterior tibial compartmental syndrome: A complication of the Hauser procedure. Clin. Orthop. 1975, 113:90-94

= 국문초록 =

조직내압 상승이 신경전도 속도에 미치는 영향

서울대학교 의과대학 정형외과학교실 및 재활의학교실*

석세일 · 한태륜* · 이상훈

성숙한 백색가토를 6군으로 나누고 하퇴부 심후방구획에 조직내압을 상승시켜 각 군에 따라 각각 압력을 80, 120 및 160 mm Hg 3가지로, 시간을 6시간과 12시간 2가지로 유지시킴으로써 신경손상을 유발하고 이로 인한 신경전도 속도의 변화를 16주간 관찰하여 다음과 같은 결론을 얻었다.

1. 80 mm Hg의 압력에서는 4시간에 95.8%, 120 mm Hg의 압력에서는 2시간에 93.4%, 160 mm Hg의 압력에서는 1시간에 95.5%의 신경전도 속도의 감소를 보여 압력이 증가될수록 신경속도의 조속한 감소가 일어남을 알 수 있었다.

2. 제1군 (80 mm Hg-6시간)만이 95%의 유의 수준에서도 실험전과 유의한 차가 없는 거의 완전한 회복 소견을 보였다.

3. 신경전도 회복에 미치는 영향은 시간 요소와 압력요소가 다같이 중요한 것으로 나타났으며, 압력 적용시간이 길수록 또 압력이 높을수록 회복이 덜 되는 것으로 나타났다.

4. 조직내압 상승시 신경전도의 감소가 조기에 나타났으며, 이는 구획증후군 진단에 의의가 큰 것으로 생각되었다.