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Neurophysiological Study on the Effect of Joint Movement on the Regulation of Cardiovascular Function

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This experiment was performed to determine the causes of circulatory activities during muscular work, to evaluate the effect of various kinds of painful stimuli to the knee joint on the cardiovascular system, and to compare the effect of joint pain on the cardiovascular system with that of skeletal muscle pain on the cardiovascular system. In this experiment, blood pressure, heart rate and the impulse discharges of cardiac sympathetic efferents were recorded to evaluate the changes of circulatory activities in the baroreceptor of denervated cats anesthetized with α-chloralose.

The results are summarized as follows;

1. Rhythmic flexions and extensions of a knee joint in its physiological working range have an influence on circulatory activities in 11 out of 14 units. This effect disappeared after the cutting of the posterior and medial articular nerve.
2. Inward and outward rotations were extended into the noxious range, significant increase in the circulatory activities were elicited. The increases in circulatory activities induced by noxious outward rotation exceeded those elicited by noxious inward rotation.
3. Injection of KCl into the knee joint triggered circulatory reflexed that may indicate a nociceptive response in 17 out of 22 units. An injection of bradykinin into the knee joint elevated the circulatory activities in 11 out of 23 units, it produced biphasic responses or a fall in blood pressure, and on the other hand, it increased heart rate and impulse discharges of cardiac sympathetic efferents.
4. The increases in circulatory activities induced by the injection of KCl and bradykinin into the gastrocnemius muscle exceeded those induced by the injection of KCl and bradykinin into the knee joint.
5. A 1V stimulus to the medial articular nerve, which could not excite the Group IV units, induced increases in circulatory activities at a 20V stimulus, which could excite all medial articular nerve afferents.

It can be conclued that passive movements of the knee joint activate a sympathetic outflow to the cardiovascular system as various kinds of painful stimuli (mechanical, chemical, electrical) in baroreceptor denervated cats anesthetized with α-chloralose.

INTRODUCTION

Cardiovascular changes occur during exercise when the cardiovascular control center is stimulated by nerve impulses descending from the superior motor center and those ascending from the reflex mechanism of peripheral sensory nerves.

So far studies on the reflex mechanism of peripheral sensory nerves in connection with cardiovascular reflex have placed emphasis on skeletal muscle receptors (Mitchel, 1977 ; Mitchel et al., 1977 ; Kaufman et al., 1983) while on articular sensory receptors only partial studies have been conducted (Sato et al., 1985).

In order to investigate how articular receptors work on the cardiovascular system and what their mechanisms are like, they were stimulated mechanically, chemically, electrically, and then changes in blood pressure, heart rate and efferent impulse discharges of inferior cardiac nerve were observed. By examining the differences between the cardiovascular reflexes arising from muscle receptors and the reflexes from articular receptors, this study attempted to determine how the articular receptors, which are activated during exercise, affect the cardiovascular reflex mechanism.

METHODS

In this study, adult cats (2.5~4kg) were anesthetized by an intramuscular injection of α-chloralose (10mg/kg), Additional dose of 10mg/kg/hr. were used to maintain a level of anesthesia throughout the experiment. A polyethylene cannula was inserted into the left antebrachium vein to inject anesthetics and other chemicals.

A polyethylene cannula inserted to record the heart rate, was connected to the pressure transducer (p23 XL, Statham) and heart rate was recorded continuously by the physiograph (7PIG, Grass), maintaining mean arterial pressure at 80~120mmHg. To maintain air way, the tracheostomy was performed on all the test animals and a artificial respirator (Model 645, Harvard) was connected to them. The respirator was controlled to keep end-expiratory carbon dioxide at 29~36 mmHg when measured by the capnometer (Model 2200, TMN). During the incision of chest, dyspnea, which usually happens, was prevented by an intravenous injection of pancuronium bromide (mioblock) 0.5~1.0 mg/hr and artificial respirator. To keep the body temperature at 37~38℃ during the experiment, the retal

temperature was monitored and connected to a homeothermal blanket control unit (Model 50~7129, Harvard). Prior to the experiment the vagus nerve and left carotid sinus nerve were removed to see if the results were caused by the sympathetic nerve and also to remove the reflex mechanism of blood pressure through pressure receptors.

1. Experiment

The midline incision was done on arterior chest to 5th posterior ribs were removed, the inferior cardiac nerve (ICN) was separated from surroudning tissue, and the nerve was soaked in paraffin oil to keep it from getting dry (Fig. 1). In order to apply electrical and chemical stimuli to the knee joint nerve, the sartorius muscle was removed exposing the branch of artery leading to the medial articular nerve and knee joint. The exposed medial articular nerve was separated from surrounding tissue and an electrode was installed (Fig. 2). Fig 3 shows the recording and analysis system of the results of this experiment.

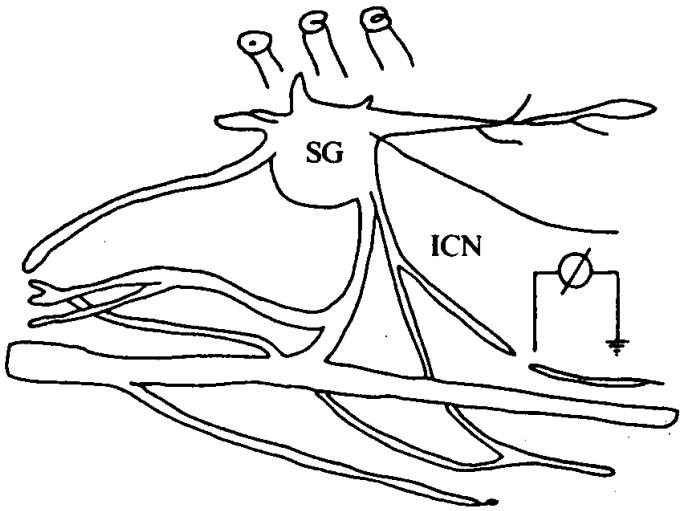

Figure 1. Schematic drawing of the preparation and recording system

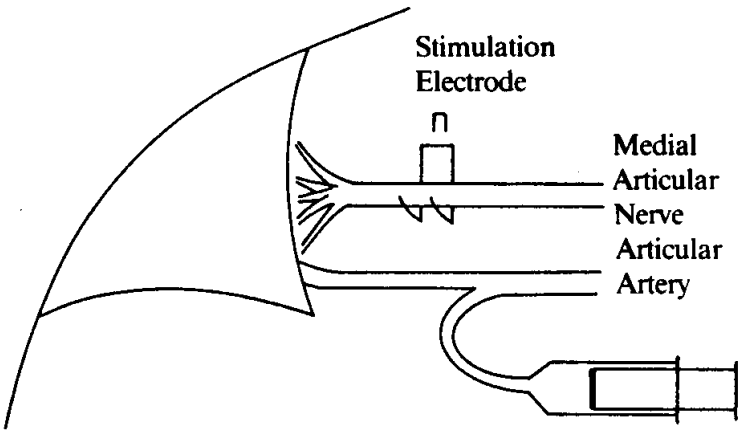

Figure 2. Schematic drawing of the preparation and recording system

2. Stimulation

(1) Mechanical stimulation

In order to induce the same stimuli in the knee joint as are induced during exercise, the thigh and ankle were fixed and were made to move passively twice per sec for 30 secs at a normal angle using the knee joint as an axis. To ascertain whether the change which occurred was caused by the knee joint sensory nerve fibers, the medial and posterior articular nerves were cut and the same test was repeated to compare the results to those of the test before the cutting of the nerves. In order to stimulate the knee joint, noxious inward and outward rotation movements were made passively at an over-strained angle and the changes were observed.

(2) Chemical stimulation

After checking the branch of artery leading to the knee joint, a micro-polyethlene cannula was inserted through which pain-inducing KCI (0.3m, 0.26ml) and bradykinin (26μg) was injected. To prevent iniected chemicals from infiltrating tissue other than that of the knee joint, the branches of the artery in other areas were eigated. Evans blue dye (T-1824) was injected and the area of infiltraion was ascertained before the experiment. To induce noxious stimuli in skeletal muscles, a micro-polyethlene cannula was inserted into the branch of the artery leading to the gastrochemius-soleus muscle and the result was ascertained in the same way as was done with the knee joint.

(3) Electrical stimulation

A platinum electrode was placed on the medial articular nerve and

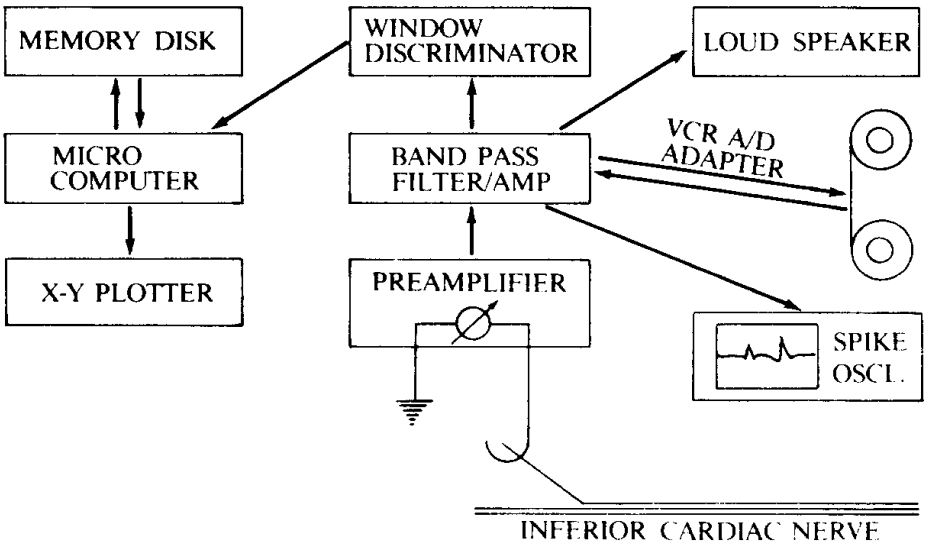

Figure 3. Experimental arrangement of the data recording system

responses were oberved after stimulating the knee joint nerve for 30 secs with a voltage (1v, 2ms) which cannot activate the unmyelinated nerve and then after stimulating it 10 times per sec (10Hz) for 30 secs with a voltage which can activate all the unmyelinated nerve fibers.

RESULTS

1. Mechanical Stimulation

(1) Passive movements

The thigh and ankle of the cat anesthetized with α-chloralose were fixed and forced to make passive movements twice per sec for 30 secs. As a result, 11 out of 14 units showed increases : 12.9± 2.3mmHg (mean± S.E., $p<0.01$, as in Figs. 4 and 5) in blood pressure ; 6.4± 1.7 impulses/min (mean± S.E., $p<0.01$, as in Figs. 4 and 6) in impulse discharges of the cardiac sympathetic efferents ; 3+1.2 hearts/min (mean± S.E., as in Figs. 4 and 7) in heart rate. These Changes were significantly decreased after the medial and posterior articular nerves were cut. The other 3 units showed little or no change.

(2) Noxious inner rotation

When the cat's knee was twisted inwardly at an over-strained angle for 30 secs all of 14 units showed increases : 39.4± 7.0mmHg (mean± S.E., $p<0.01$, as in Figs. 4 and 5) in blood pressure ; 23.7± 8.4 impulses/min (mean± S.E., $p<0.05$, as in Figs. 4 and 6) in impulse discharges of the cardiac sympathetic efferents ; 7+0.3 hearts/min (mean± S.E., $p<0.01$, as in Figs. 4 and 7) in heart rate.

(3) Noxious outer rotation

When the cat's knee was twisted outwardly at an over-strained angle, all of the 14 units showed increases : 40.9± 8.4mmHg (mean± S.E., $p<0.01$, as in Figs. 4 and 5) in blood pressure ; 23.7+8.4 impulses/min (mean± S.E., $p<0.05$, as in Figs. 4 and 6) in impulse discharges of the cardiac sympathetic efferents ; 7.7± 0.3 beats/min (mean± S.E., $p<0.01$, as in Figs. 4 and 7) in heart rate.

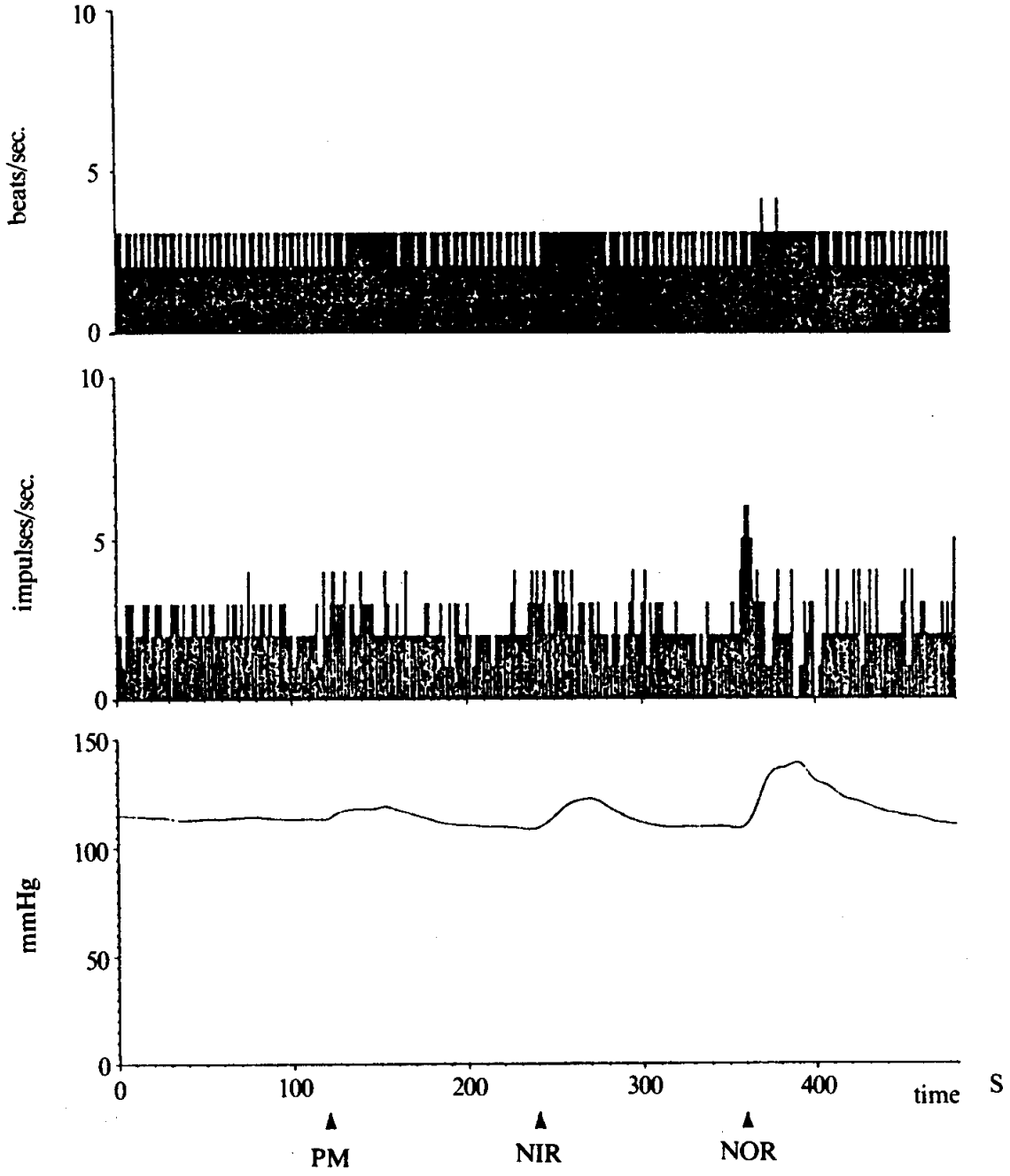

Figure 4. Cardiovascular changes induced by passive movements (PM), noxious inward rotation(NIR), and noxious outward rotation(NOR) of lower limb. Upper panel, heart rate ; middle panel, impulse discharges of cardiac sympathetic efferents ; lower panel, mean arterial pressure.

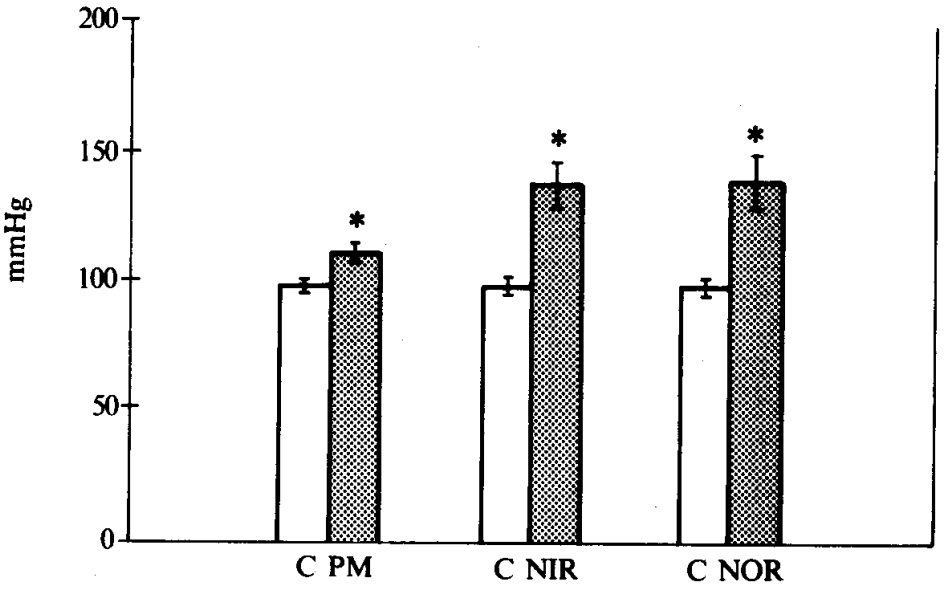

Figure 5. Mean arterial pressure increases (mean± S.E.) induced by the indicated movements. Open columns, control value before movements ; crosshatched columns, value following movements. *Significant increase ($p<0.01$) from the control value. PM, passive movement; NIR, noxious inward rotation ; NOR, noxious outward rotation.

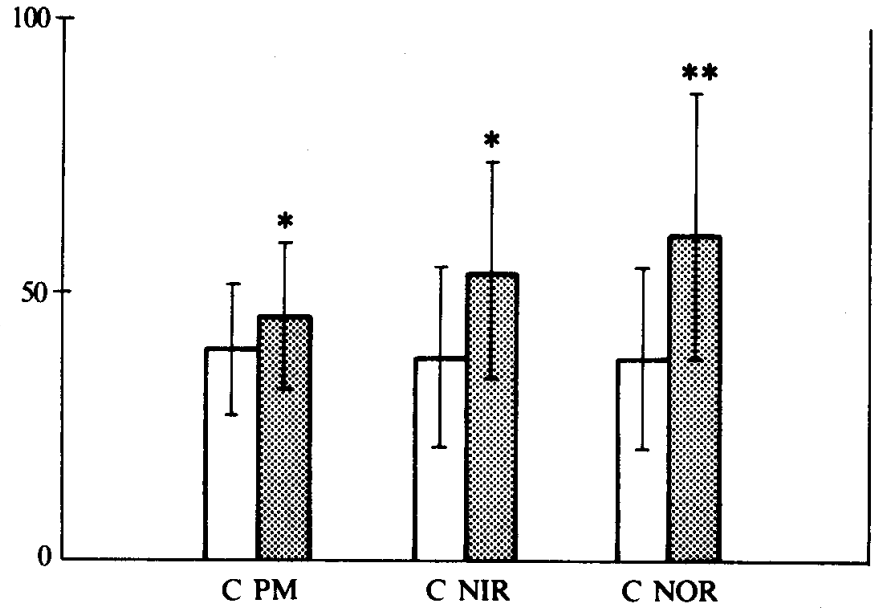

Figure 6. Impulse discharges of cardiac sympathetic efferents increase (mean ± S.E.) induced by the indicated movements. Open columns, control value before movements ; Crosshatched columns, value following movements. *Significant increase ($p<0.01$) from the control value ; **Significant increase ($p<0.05$) from the control value. PM, passive movement ; NIR, noxious inward rotation ; NOR, noxious outward rotation.

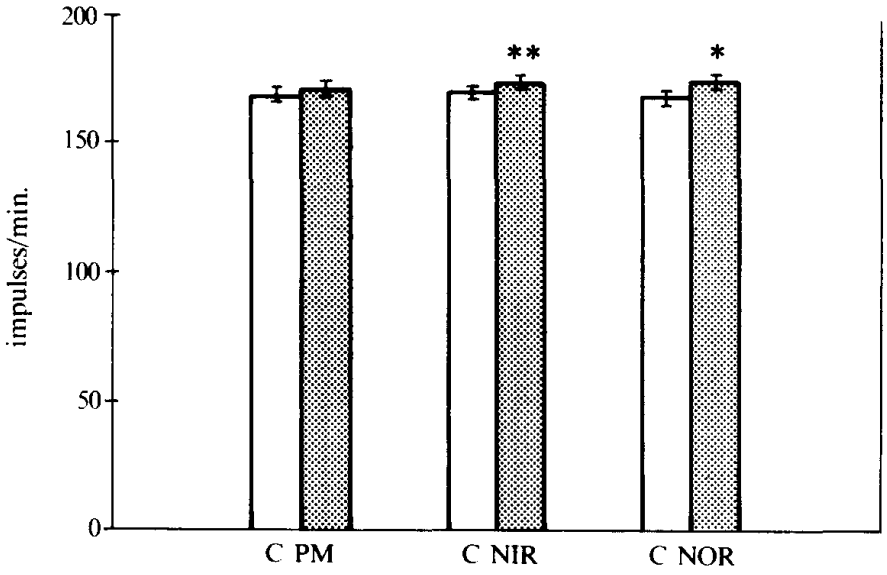

Figure 7. Heart rate increases (mean± S.E.) induced by the indicated movements. Open columns, control value before movements; Crosshatched columns, value following movements. *Significant increase ($p<0.01$) from the control value; **Significant increase ($p<0.05$) from the control value. PM, passive movement; NIR, noxious inward rotation; NOR, noxious outward rotation.

2. Chemical stimulation

(1) KCI

When 0.3M KCI 0.26ml was injected into a branch of the articular artery, 17 out of 22 units showed increases; 15.8± 2.5mmHg (mean± S.E., $p<0.01$, as in Figs 8 and 9) in blood pressure; 23.3± 4.1 beats/min (mean± S.E., $p<0.01$, as in Figs 8 and 11) in heart rate. The other 5 units showed neither any increasing nor decreasing tendency.

(2) Bradykinin

When bradykinin 26μg was injected into a branch of the articular artery, 11 out of 23 units showed increases; 13.8± 2.2mmHg (mean± S.E., $p<0.01$, as in Figs 8 and 9) in blood pressure; 61.4± 12.1 impulses/min (mean± S.E., $p<0.01$, as in Figs 8 and 10) in impulse discharges of the cardiac sympathetic efferents; 33.5± 3.5 beats/min (mean± S.E., $p<0.01$, as in Figs 8 and 11) in heart rate. The other 12 units, after injection, showed biphasic blood pressure changes or decreases in blood pressure, but they showed increases; 22± 3.2 impulses/min (mean± S.E., $p<0.05$) in impulse discharges of the sympathetic efferents and 10.2± 3.3 5 beats/min (mean± S.E.) in heart rate.

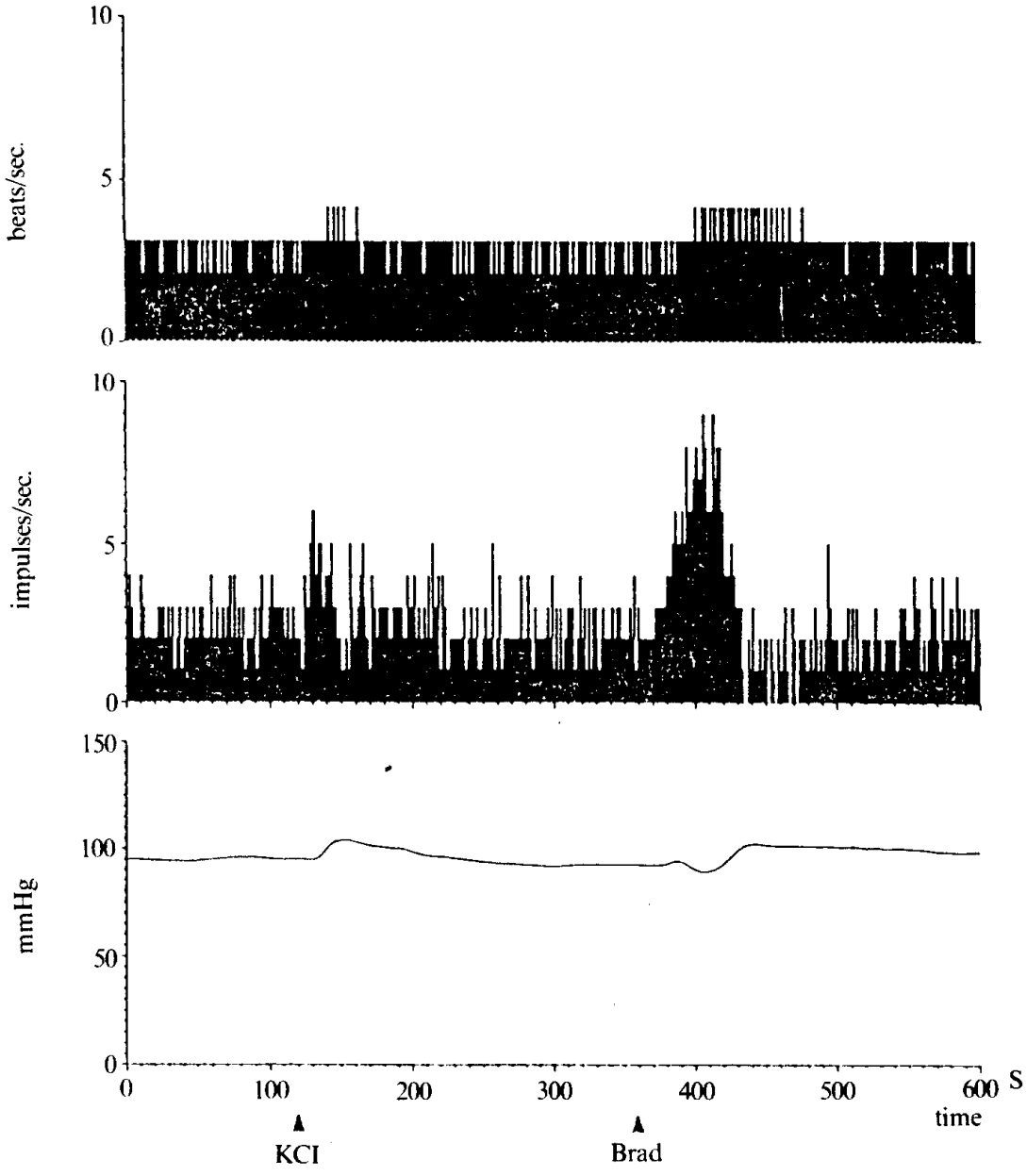

Figure 8. Cardiovascular changes induced by injection of KCI (0.26ml of 0.3M) and bradykinin (brad. 26μg) into knee joint. Upper panel, heart rate ; middle panel, impulse discharges of cardiac sympathetic efferents ; lower panel, mean arterial pressure.

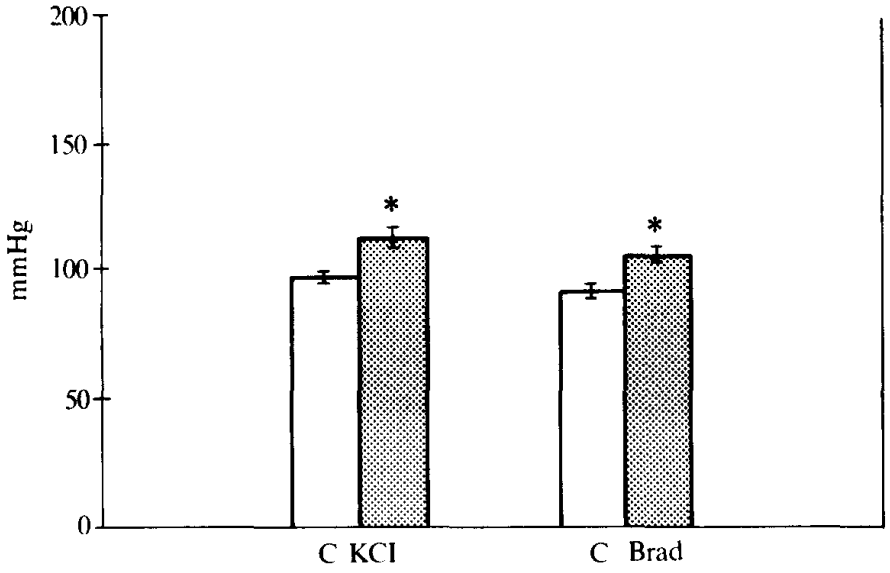

Figure 9. Mean arterial pressure increases (mean± S.E.) induced by the indicated movements. Open columns, control value before injection; Crosshatched columns, value following injection. *Significant increase ($p<0.01$) from the control value. KCl (0.26ml of 0.3M). Brad. (bradykinin 26μg).

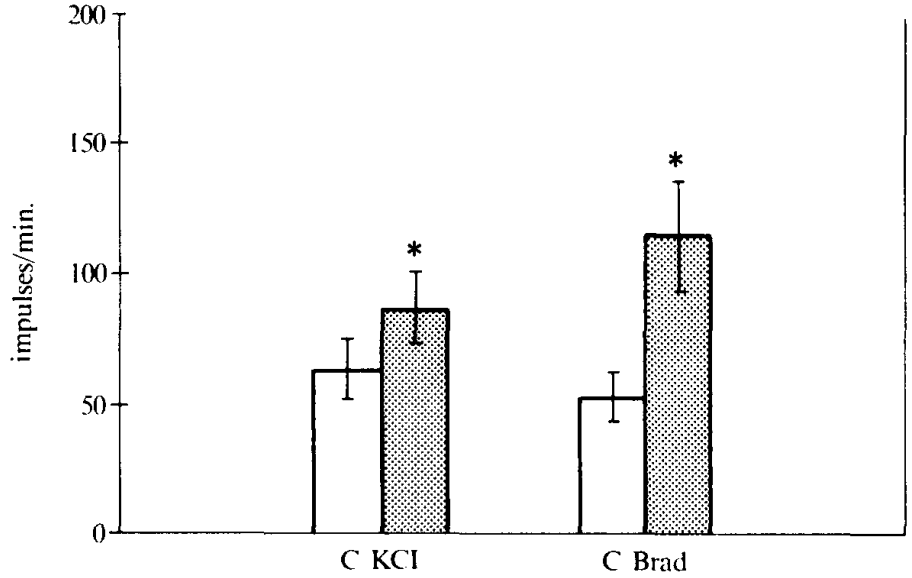

Figure 10. Impulse discharges of cardiac sympathetic efferents increases (mean± S.E.) induced by the injection of the analgesic agents. Open columns, control value before injection.; crosshatched columns, value following injection. *Significant increase ($p<0.01$) from the control value. KCl (0.26mg of 0.3M). Brad. (bradykinin 26μg).

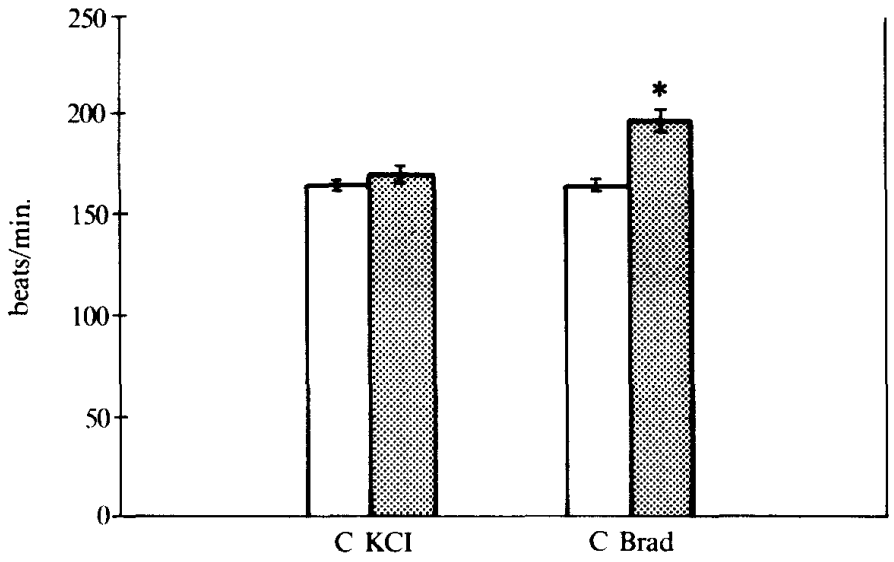

Figure 11. Heart rate increases (mean± S.E.) induced by the injection of the analgesic agents. Open columns, control value before injection; crosshatched columns, value following injection. *Significant increase ($p<0.01$) from the control value; KCI (0.26ml of 0.3M). Brad. (bradykinin 26μg).

(3) A comparison between articular and muscle sensory nerves

When the knee joint pain and skeletal muscle pain was induced by an injection of KCI and bradykinin into a branch of the artery leading to the knee joint and a branch of the sural artery, 8 out of 10 units showed that changes induced by noxious stimuli in the skeletal muscle were greater than those by noxious stimuli in the knee. They also showed increases: 10± 5. 6mmHg (mean± S.E., $p<0.01$, as in Figs 12 and 13) in blood pressure; 10. 9± 2.1 impulses/min (mean± S.E., $p<0.01$, as in Figs 12 and 14) in impulse discharges of the cardiac sympathetic efferents; 1.2± 1.7 beats/min (mean± S. E., as in Figs 12 and 15) in heart rate. Out of 10 units injected with bradykinin, 6 of whose cardiovascular function was enhanced showed that the changes induced by noxious stimuli to the skeletal muscle were greater than those by noxious stimuli to the knee. They also showed increases; 13. 2± 1.6mmHg (mean± S.E., $p<0.01$, rmfla 15, rmfla 16) in blood pressure; 7. 3± 2.3 impulse/min (mean± S.E., $p<0.05$, as in Figs 12 and 14) in impulse discharges of the cardiac sympathetic efferents; 4.4± 1.6 beats/min (mean± S. E., as in Figs 12 and 15) in heart rate.

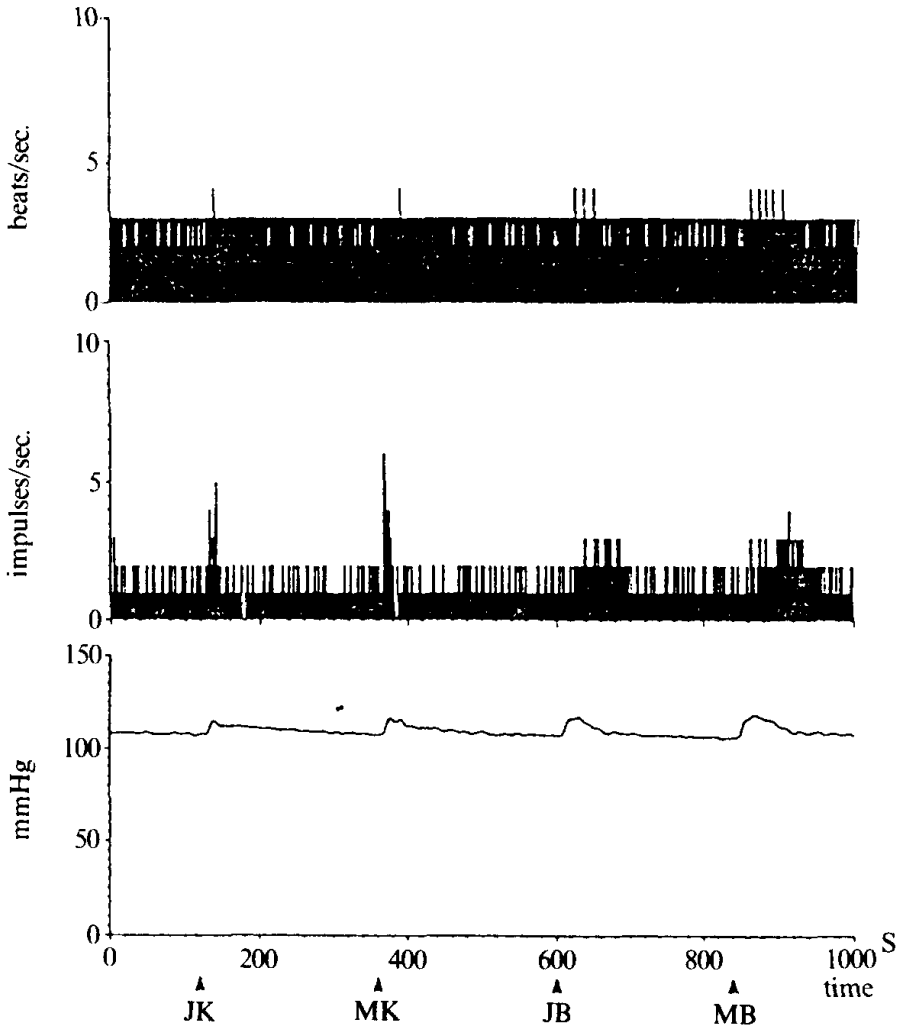

Figure 12. Comparison of the effect of knee joint pain on the cardiovascular system with that of gastrocnemius muscle pain on the cardiovascular sysetem. Above show pain induced by injection of KCI (0.26ml of 0.3M) or bradykinin (26μg). Upper panel, heart rate ; middle panel, impulse discharges of cardiac sympsthetic efferents ; lower panel, mean arterial pressure. JK, injection of KCI into knee joint ; MK, injection of KCI into gastrcnemius muscle ; JB, injection of bradykinin into knee joint;MB injection of bradykinin into gastrocnemius muscle.

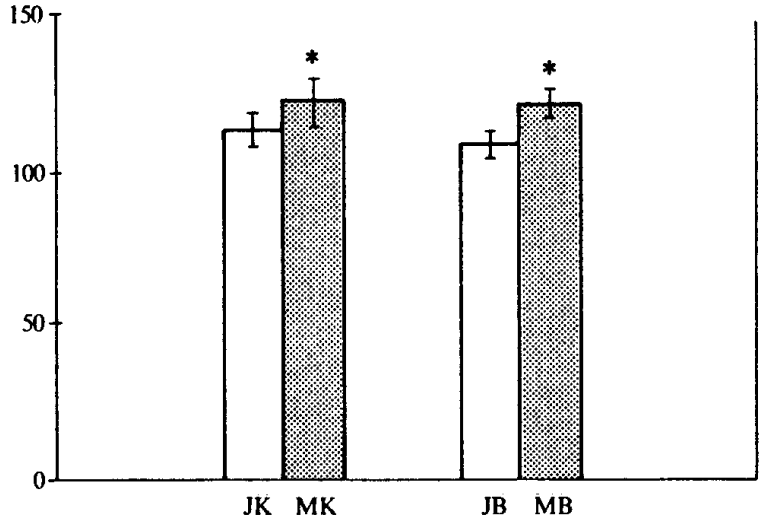

Figure 13. Comparison of the effect of knee joint pain on mean arterial pressure with that of gastrocnemius muscle pain on mean arterial pressure. Above show pain induced by injection of KCI (0.26ml of 0.3M) or bradykinin (Brd. 26 μg). *Significant increase ($p<0.01$) from the corresponding value. JK, injection of KCI into knee joint; MK, injection of KCI into gastrocnemius muscle; JB, injection of bradykinin into knee joint; MB, injection of bradykinin into gastrocnemius muscle.

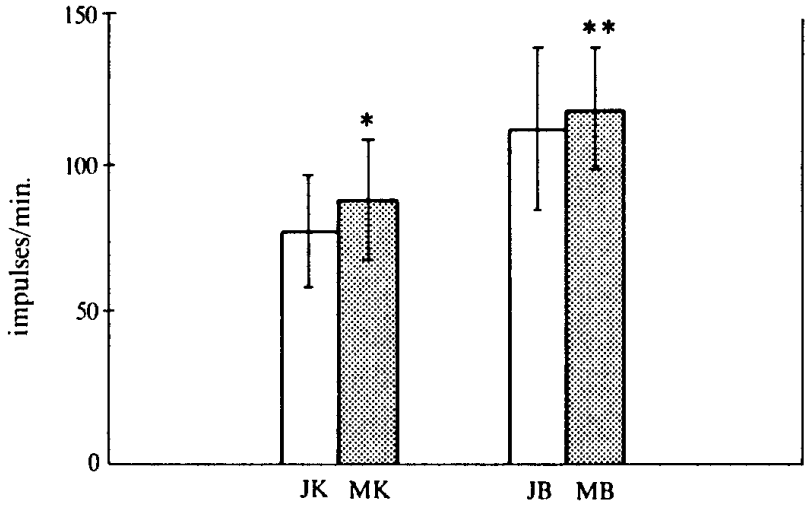

Figure 14. Comparison of the effect of knee joint pain on impulse discharges of cardiac sympathetic efferents with that of gastrocnemius muscle pain on impulse discharges of cardiac sympathetic efferents. Above show pain induced by injection of KCI (0.26ml of 0.3M) or bradykinin (Brad. 26μg). *Significant increase ($p<0.01$) from the corresponding value; **Significant increase ($p<0.05$) from the corresponding value. JK, injection of KCI into knee joint; MK, injection of KCI into gastrocnemius muscle; JB, injection of bradykinin into Joint knee;MB, injection of bradykinin into gastrocnemius muscle.

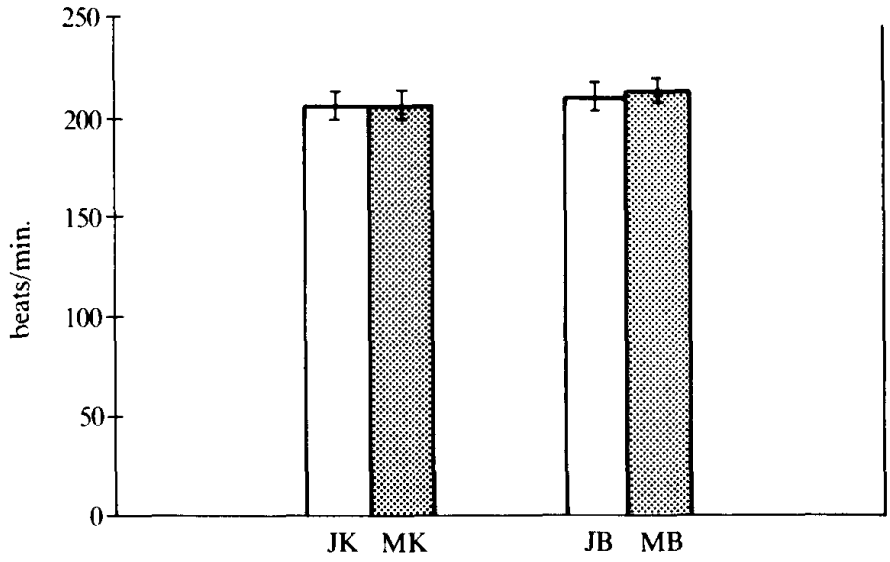

Figure 15. Comparison of the effect of knee joint pain on heart rate with that of gastroonemius muscle pain on heart rate. Above show pain induced by injection of KCI (0.26ml of 0.3M) or bradykinin (Brad. 26μg). *Significant increase ($p<0.01$) from the corresponding value ; *Significant increase ($p<0.05$) from the corresponding value. JK, injection of KCI into knee joint ; MK, injection of KCI into gastrocnemius muscle ; JB, injection of bradykinin into knee joint ; MB, injection of bradykinin into gastrocnemius muscle.
stimulation. increae stimulation.

3. Electrical Stimulation

When stimulated by 1V which is not strong enough to excite the unmyelinated nerves, 12 out of 15 units showed increases : 13.6± 4.5 mmHg (mean± S.E., $p<0.05$, as in Figs 16 and 18) in blood pressure ; 18.7± 7.2 impulse/min (mean± S.E., $p<0.05$, as in Figs 16 and 19) in impulse discharges of the sympathetic nerves ; 3.2± 1.9 beats/min (mean± S.E., $p<0.05$, as in as in Figs 17 and 20) in heart rate. When stimulated by 20V, all of the 15 units showed increases ; 20.2± 5.3mmHg (mean± S.E., $p<0.01$, as in Figs 17 and 18) in blood pressure ; 67.8± 27.1 impulse/min (mean± S.E., $p<0.05$, as in Figs 16 and 19) in impulse discharges of the sympathetic nerves ; 6.4± 1.0 beats/min (mean± S.E., $p<0.05$, as in Figs 17 and 20) in heart rate.

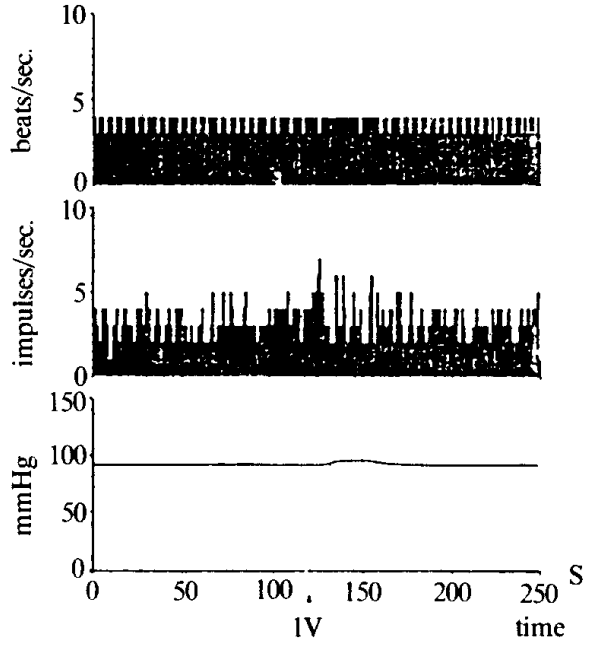

Figure 16. Cardiovascular changes induced by exciting the medial articular nerve for 30 secs (10Hz) with 1V stimuli, which are not strong enough to activate the group 1V units. Upper panel, heart rate ; middle panel, impulse discharges of cardiac sympathetic efferents ; lower panel, mean arterial pressure.

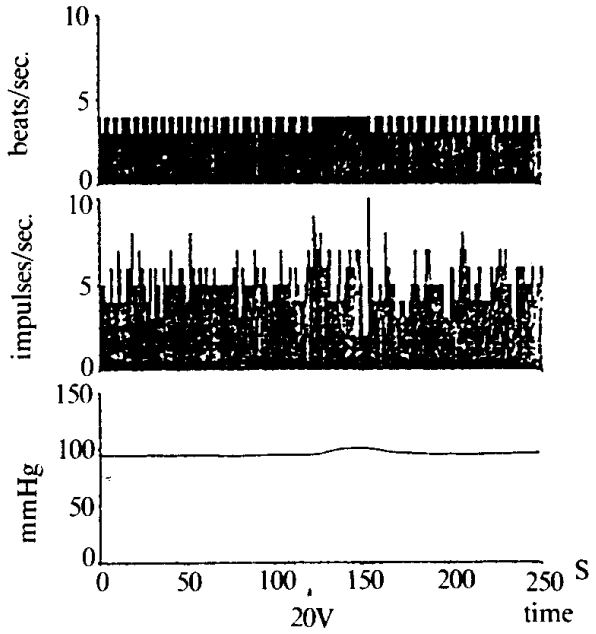

Figure 17. Cardiovascular changes induced by exciting the medial articular nerve for 30 secs (10Hz) with 20V stimuli, which can activate all afferents.

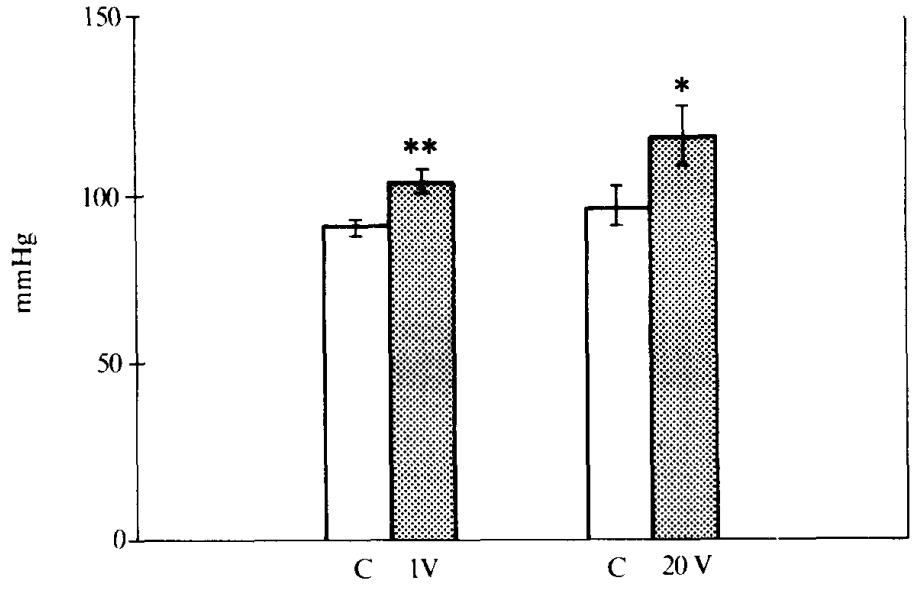

Figure 18. Mean arterial pressure increases (mean± S.E.) induced by electrical stimulation (1V, 20V) of the medial articular nerve. Open columns, control value before stimulation; crosshatched columns, value following stimulation. *Significant increas ($p<0.01$) from the control value; **Significant increase ($p<0.05$) from the control value.

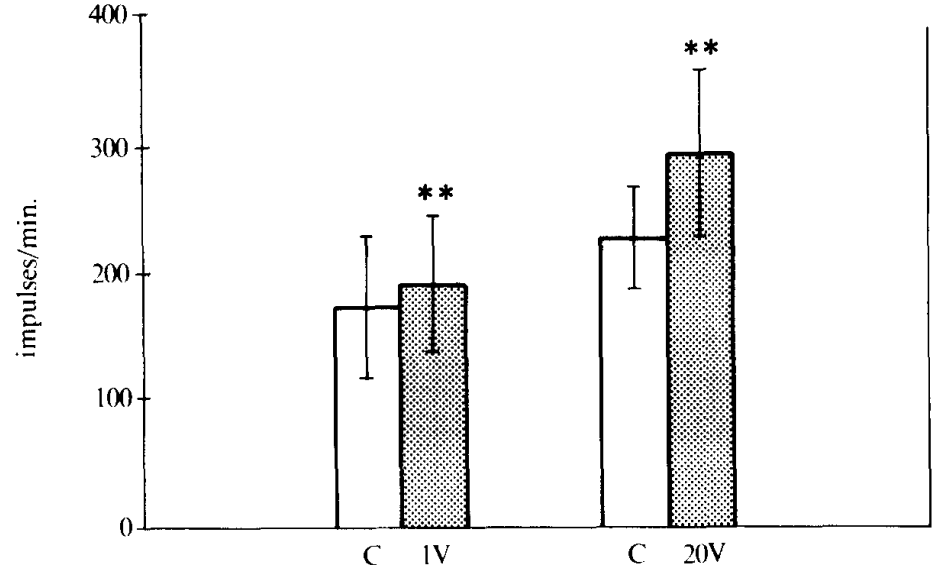

Figure 19. Increases in impules discharges of cardiac sympathetic efferents (mean± S.E.) induced by electrical stimulation (1V, 20V) of the medial articular nerve. Open columns, control value before stimulation; crosshatched columns, value following stimulation. **Significant increase ($p<0.05$) from the control value.

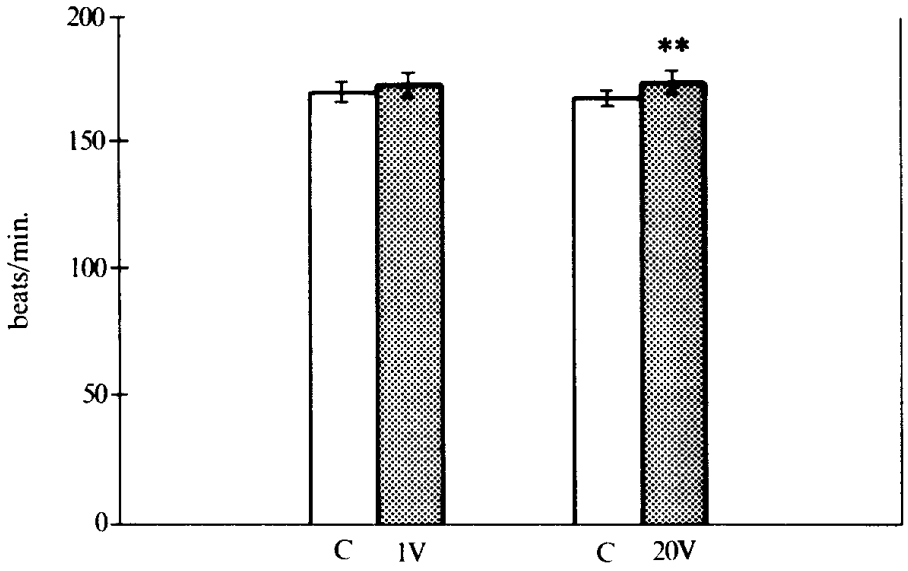

Figure 20. Heart rate increases (mean± S.E.) induced by electrical stimulation (1V, 20V) of the mdedial articular nerve. Open columns, control value before stimulation ; crosshatched columns, value following stimulation. **Significant increase ($p<0.05$) from the control value.

DISCUSSION

In the initial experiment of passive rotational movements, 11 out of 14 units showed significant increases in heart rate, blood pressure and impulse discharges of the inferior cardiac efferents, but 3 units showed either no change or decrease. The discontinuance of increases after the cutting of the medial and posterior nerves may indicate the increases were reflexes by the articular nerves. This also indicates that cardiovascular function can be reflexively activated even by passive movements of the knee joint. Schiable et al., (1987) reported that thick (Group II) myelinated nerve fibers and a considerable portion of thin (Group III) myelinated nerve fibers were activated by passive knee joint movements at a normal angle and even Group Ⅳ nonmyelinated nerve fibers could be activated sometimes by passive rotational movements. In the test conducted with 74 cats the researchers found that 20% of all Groups III and Ⅳ nerve fibers were activated by normal knee joint movements; particularly in normal passive rotational movements reflexive activation was greater and Group III was better activated than Group Ⅳ. This report indicates there is a strong possiblity that the information of movements can flow into the central

nervous system, which supports the findings of this study.

However, Sato et al., (1984) reported that the inferior cardiac nerve was not affected by passive knee joint rotation made within a normal range of movement, which conflicts with the findings of this study. The report said that the blood pressure and heart rate changes reflexively elicited by normal passive movements were so irregular that they were statistically insignificant. But their passive movements were made a every 2 secs unlike ours and it seems that information which flew into the central nervous system was not sufficient while ours was.

In the 2nd experiment, the knee joint was forced to make noxious inward and outward rotations using the knee joint as an axis to investigate the cardgoraswlar reflexes elicited by articular nerves activated by chemical noxious stimuli and all 14 units demonstrated significant increases in blood pressure, heart rate and impulse discharges of cardiac sympathetic efferents; outward rotations produced more increases than inward rotations. The results confirmed that the stimuli elicited by passive rotational movements at an over-strained angle triggered greater reflexes in the cardiovascular nerves by exciting Groups III and IV nerve fibers.

Schaible et al., (1987) reported in the expercment conducted with 74 cats that all the medial articular (Group III) nerve fibers and approximately 16% of Group IV nerve fibers responded to inward and outward rotations made in a normal range of movement and the responses increased as the rotations shifted from normal to strained. They also reported that approximately 33% did not respond at all when inward and outward rotations were made within a normal range; they responded only when strained rotations were made. These reports are considered to support the findings of this study.

In the 3rd experiment, KCI was injected into a branch of the artery to investigate the responses of the articular nerves to pain-producing chemicals and 17 out of 22 units showed increases in blood pressure, heart rate and impulse discharges of cardiac fiber efferents, but 5 of them showed either no change or decrease. It is known that blood pressure increases when the somatic nerve is excited by electrical stimuli strong enough to activate unmyelinated nerves (Mense, 1977; Mense and Schmidt, 1974). Chemical stimulation induces similar responses (Sato et al., 1979; 1982). Intraarterial injection of potassiumion, one of the pain-producing chemicals, into small intestine stimulates pain receptors and reflexively excites sympathetic nerve fiber efferents and resulting in blood pressure increase (Khayutin et al., 1969). It is reported that the increase of blood pressure by potassium ion injection into the skeletal muscle indicates that Group III nerve fibers, which are

myelinated nerves, and Group Ⅳ nerve fibers are involved in these responses (Sato et al., 1982) However, pain does not always stimulate the cardiovascular system. Ranson et al., (1916) said that the changes in blood pressure elicited by excitation of muscular afferents is determined according to which of the pressor and depressor cardiac centers is connected with muscular afferents. Sato et al., (1982) said that around 10 secs after injection of potassium ion into the skeletal mucle, the heart rate either increased or decreased because there were two different kinds of afferent skeletal nerve fibers; one could increase heart rate and the other could decrease it.

Though the reason is not clear why the remaining 5 units showed a decrease in the 3rd test, the probability of the depressor mechanism by fomrmation of inhibitory reflex pathway should not be disregarded. Bradykinin is known to relax blood vessels and induce pain. However, a dose of bradykinin injected may bring biphasic results: blood pressure may increase by reflex mechanism of pain or decrease owing to vascular relaxation. The determining dose of bradyinin varies according to the kinds of animals and organs tested, which makes it difficult to determine if the changes are evoked only by a reflex mechanism. For example, it was reported that in an unanesthetized dog blood vessels dilated and blood pressure was lowered by an injection of bradykinin 3μg into the coronary artery. When a sufficient dose of bradykinin to cause pain is injected, blood pressure rises owing to the cardiovascular reflex of pain. Yet a probable fall in blood pressure by rasodilatation cannot be totaly disregarded (Pagani, et al., 1981). Therefore, it is judged that changes in blood pressure elicited by bradykinin pain have biphasic responses occurred in which blood pressure and heart rate either increased or decreased when they injected a certain dosage of bradykinin into the artery leading to the lower limbs. In this experiment 26μg of bradykinin was injected and the changes in blood pressure, heart rate and impulse discharges of the sympathetic efferents were observed. All 23 units showed significant increases in heart rate and impulse discharges of sympathetic efferents, but in blood pressure only 11 out of 23 units showed increases while 12 units produced biphasic responses to bradykinin or showed decreases. Blood pressure was reduced in half of the tests while heart rate and impulse discharges of the sympathetic efferents increased in all of the tests, which may suggest that the results occurred owing to vasodilatation by bradykinin. Judging from the facts that the somatic nerves excited by chemical stimuli strong enough to activate myelinated nerves produced a rise in blood pressure(Sato et al., 1979; 1982) and that lactic acid, metabolite produced during exercise, activated groups Ⅲ and Ⅳ nerve fibers (Rotto and

Laufman, 1988), it is clear that the joint has nociceptors and various noxious chemicals injected into joint could elicit cardiovascular reflex by activating the nociceptors in joint.

In the 4th experiment, an attempted comparison between the effect of muscle sensory nerves and than of articular sensory nerves on the cardiovascular system was not successful because of experimental difficulties. When KCI and bradykinin were injected into gastrocnemius musde and the knee joint, in 8 out of 10 units the injection into gastrocnemius muscle showed higher blood pressure, heart rate and impulse discharges of the cardiac sympathetic efferents than into the knee joint, and in the remaining 2 units the result was not clear. In 6 out of 10 units injected with bradykinin injectea into gastrocnemius muscle showed higher blood pressure, heart rate and impulse discharges of sympathetic efferents and in the remaining 4 units blood pressure was reduced and the result was not certain. This seems to suggest that the sensory nerve of the gastrocnemius muscle has greater influence than the knee joint sensory nerve. It is clear that the information from the knee joint affects the cardiovascular center just as the information from the skeletal muscles during exercise does.

The medial articular nerves of a cat consist of 200 myelinated nerve fibers, 400 unmyelinated nerve fibers and 400 sympathetic postganglionic fibers (Langford and Schmidt, 1983). The threshold of group III nerve fibers is 160mV-1.5V ; 500mV is about 70% ; the threshold of group III nerve fibers is 150mMV-5.2V ; less than 2V is 90% ; if stimulus strength is set at 1V, all group II nerve fibers and half of group lll can be excited while group III nonmyelinated nerves are not excited at all (Sato et al., 1983).

In the 5th experiment, when the knee articular nerves were electrically stimulated respectively by 1V, which cannot activate nonmyelinated nerves, and by 20V, which can activate all nerve fibers, 12 out of 15 units showed significant increases in blood pressure, heart rate and impulse discharges of sympathetic efferents but 3 units showed no change. In 15 units which were stimulated by 20V, blood pressure, heart rate and impulse discharges of sympathetic efferents all increased significantly. The enhancement of cardiovascular function stimulated by 1V may suggest that the sensory nerve of an anesthetized knee joint can influence cardiovascular function during normal exercise.

It is concluded from the results that during normal knee movements the articular nerves can enhance cardiovascular function and noxious chemicals produced during exercise can also facilitate cardiovascular function by stimulating articular receptors.

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