The effects of the stimulation of the sympathetic cardiac nerves, the vagal nerves, and the central nervous system (e.g. hypothalamus) on the cardiac output have been investigated by using several different methods. However, there have been controversies on their results. Some of these disagreements were due to the difference of the methods used; no adequate flowmeters had been utilized, before the study of R. E. Shipley and D. E. Gregg, who observed the cardiac response to the stimulation of the stellate ganglia and cardiac nerves with a rotameter in 1945. Mean cardiac output is measured quantitatively and continuously with a rotameter. Although the results of the stimulation of the sympathetic cardiac nerves were essentially similar, there was no agreement on the effects of the reflex hyper- or hypotension on the cardiac output.

In the studies on the nervous and neurohumoral control of the coronary circulation in our department, S. Okinaka et al., M. Ikeda, K. Hashiba, and K. Murata observed that the coronary blood flow followed the changes in blood pressure and in humoral substances, induced by faradic stimulation of the autonomic nerves; coronary flow was also influenced by the direct effect of the nerve stimulation on the coronary arteries. M. Ikeda also reported the functional difference of the right and left cardiac sympathetic nerves.

In this study, the experiments have been designed to find out the relationship between the cardiac output and the coronary blood flow during stimulation of the cardiac autonomic nerves and to determine the effects of the reflex hyper- and hypotension on the cardiac output. Furthermore, experiments have been conducted to determine the mechanism of a biphasic elevation of the systemic blood pressure, after the stimulation of the sympathetic cardiac nerves, especially at the left side.

**Methods**

Thirty dogs weighing 11.6 to 27.0 kg were anesthetized with a relatively small dose of thiopental sodium (50 mg/kg wt, intravenously), so as to maintain the corneal reflex during the experiment. The experimental preparation employed is shown in Figure 1. The chest wall was opened through the fifth intercostal space on the left side; lung was inflated with an intermittent positive pressure respirator was used at the rate of 20 to 25 times per minute. Heparin sodium (5 mg/kg, intravenously) was employed as an anticoagulant, and it was added if necessary. The thoracic aorta was isolated from intercostal arteries and cut at the level of the fifth intercostal space, then the aortic bypass was established. Blood from the proximal end of the cut thoracic aorta was returned through a rotameter (A) to the distal end of the cut thoracic...
aorta and both common carotid arteries. The interruption of aortic blood flow for the cannulation was released within 6 minutes. The cerebral blood flow was kept as sufficient as possible during the carotid artery cannulation. The left coronary artery was also perfused from the bypass through another rotameter (B) with a specially designed glass cannula\(^{10}\). Mean cardiac output and mean left coronary arterial inflow measured and recorded with the two electromagnetic rotameters continuously and simultaneously. The rotameter (A) was one of Hilger's type\(^{20}\). The rotameter (B) was modified from Shipley's type\(^{21}\).

An example of typical calibration curves is shown in Figure 2. Mean cardiac output and means left coronary inflow in millilitres per minute, were calculated by planimetry of the actual recording and the calibration curves. Systemic blood pressure was recorded with a mercury manometer from the left femoral artery and with an electromanometer from the proximal portion of the aortic arch respectively. Blood transfusion was given according to necessity. The experiments, in which the mean systemic blood pressure at the femoral artery was under 60 mm Hg, were excluded from the results. Since the blood pressure measured at the left femoral artery was lower than that of the aortic arch, 60 mmHg at the femoral artery did not show a shock level. Electrocardiogram was also recorded.

The nerve stimulation was conducted as follows (Fig. 3).

A) Peripheral sympathetic cardiac nerve stimulation.

The sympathetic rami to the stellate ganglion were sectioned on the left or right side, care being taken to leave the connections to the heart intact. A bipolar electrode connected to an electric stimulator was placed on the isolated stellate ganglion or its cardiac connection. The

Fig. 2. An example of calibration curves for rotameter A.

Fig. 3. The distribution of cardiac nerves of the dog (H. Kurihara 17)

effect of section of both carotid sinus nerves was investigated in 4 dogs.

B) Afferent cervical vagal nerve stimulation.

Left or right cervical vagosympathetic trunk was sectioned, and the proximal end of the cut vagal nerve was placed in a bipolar electrode connected to an electric stimulator.

Parameters of the electric stimulation to each nerve were adjusted so as to yield the desired intensity of response. (Square wave: Voltage; 4–20 volt, Cycle: 10–80 cycles/sec, Duration: 3–5 millisecond). The stimulation was continued for 10–20 seconds in each experiment.

The total peripheral vascular resistance was calculated by the following formula: TPVR = BP/CO, where TPVR is total peripheral vascular resistance, BP is the mean systemic blood pressure and CO is the mean cardiac output. The cardiac work was calculated as CO×BP, where CW is cardiac work.

RESULTS

1) Effect of the stimulation of the left sympathetic cardiac nerve.

The results on 20 experiments in 10 dogs are summarized in Table 1. A typical case of the experiment is shown in Figure 4.

![Graph showing blood pressure (BP), heart rate (HR), cardiac output (CO), and cardiac work (CW) over time.](image)

Fig. 4. Effect of stimulation of the left sympathetic nerve. (Exp. No. 12, 1) Marked increase in cardiac output is observed.

Blood pressure is elevated biphasiically. Heart rate increases only slightly. The total peripheral vascular resistance is reduced during the initial elevation of blood pressure. In proportion to the increase in cardiac output, cardiac work increased markedly.


![Graph showing time series data for blood pressure (BP), heart rate (HR), cardiac output (CO), and other parameters.](image)

Fig. 5. Effect of stimulation of the left sympathetic nerve. A schematic representation and its actual record. (Exp. 10, 4)

The left coronary inflow is almost in parallel with the rise and fall of systemic blood pressure rather than with the change in cardiac output, but the late increase in coronary inflow is more prominent and longer than the second elevation of blood pressure.

blood pressure rather than with the change in cardiac output, but the late increase in coronary inflow was more prominent and longer than the second elevation of blood pressure (Figure 5).

The total peripheral vascular resistance was reduced by 5% to 23% during the initial elevation of systemic blood pressure. In proportion to the increase in cardiac output, cardiac work increased markedly (51 to 150%).

Experiments after section of both cardiac sympathetic nerves after section of both cardiac sympathetic nerves and both cervical vagosympathetic trunks, showed essentially similar results (Table I, No. 15–18).

Table I Effects of the stimulation of the left sympathetic cardiac nerves

<table>
<thead>
<tr>
<th>No. of Dog Exper.</th>
<th>B.P. mmHg</th>
<th>H.R. per m.</th>
<th>C.O. ml/m.</th>
<th>Elevation of B.P.</th>
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2) Effect of the stimulation of the left sympathetic cardiac nerve, after the section of both carotid sinus nerves.

After section of both carotid sinus nerves, the left sympathetic cardiac nerve was stimulated on 12 experiments in 5 dogs. Representative tracings obtained during experiments before and after section of both carotid sinus nerves in one dog, are reproduced in Figure 6.

The increases in cardiac output before and after section of both carotid sinus nerves were similar. The biphasic elevation of blood pressure was abolished after the section of both carotid sinus nerves; blood pressure rose steadily for about 10 seconds after the onset of the stimulation, showing a single peak, in every case. Four experiments in one dog about the responses of blood pressure before and after the section of both carotid

Cardiac output increased slightly on 5 experiments and increased markedly in one case. Cardiac output showed no change in another case. The increase in cardiac output reached the maximum within 10 to 15 seconds from the beginning of the stimulation, and the rates of the increases ranged from zero to 50% with the average of 17.3% of the previous value. Cardiac output returned to the control level within 75 seconds.

Blood pressure followed the changes in cardiac output, and rose only slightly by 3 to 18 mmHg.

The increase in heart rate was manifest on these experiments; heart rate increased directly after the onset of the stimulation, lasting for about 45 seconds. Heart rate increased by 67% to 118% with average of 92.6% of the control heart rate.

Total peripheral vascular resistance and cardiac work changed slightly in comparison with the stimulation of the left sympathetic cardiac nerve.

4) Effect of the stimulation of afferent cervical vagal nerve.

The stimulation of the proximal end of cut cervical vagosympathetic trunk was performed on 20 experiments in 11 dogs on the left side and 5 experiments in 3 dogs on the right. These stimulations caused pressor responses on 10 experiments, depressor responses on 12 experiments and pressor—depressor responses on 3 experiments.

Usually, it was noted that stimulation of higher voltage and more frequent cycle induced pressor responses, while the stimu-
lation of lower voltage and less frequent cycle caused depressor responses.

Heart rate increased on 5 experiments, did not change in 6 experiments and decreased on 15 experiments.

A) Pressor response.

Although cardiac output increased gradually as blood pressure, rose, the increases cardiac output occurred later than the elevation of blood pressure. In two experiments, cardiac output did not increase (An example of them is illustrated in Figure 9). The rate of increase in cardiac output ranged from 2.0% to 126.0% (average 24.2\%) of the control level, as the blood pressure rose by 7 to 54 mmHg, or by 10.4\% to 103.8\% with the average of 32.6\% of the previous value. The duration of the effect was about one minute. Heart rate increased on 4 experiments. However, the changes in heart rate did not influence the cardiac output. The results are shown in Table 3.

Total peripheral vascular resistance increased with the marked elevation of blood pressure.

![Figure 9](image9.png)  
**Fig. 9.** Effect of stimulation of the proximal end of cut vagal nerve.  
(Pressor response) (Exp. No. 83, 2)  
Cardiac output does not increase.  
Total peripheral vascular resistance increases with the elevation of mean blood pressure. Cardiac work also increases.

![Figure 10](image10.png)  
**Fig. 10.** Effect of stimulation of the proximal end of cut vagal nerve.  
(Pressor response) (Exp. No. 10, 2)  
The increase in cardiac output is observed later than the elevation of blood pressure. The left coronary inflow is almost in parallel with the elevation of blood pressure.

### Table III Effects of the stimulation of afferent cervical vagal nerves

<table>
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<th>A) Pressor response</th>
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<td><strong>No. of</strong></td>
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TABLE IV Effect of the stimulation of afferent cervical nerves

B) Depressor response

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<th>No. of Dog Exper.</th>
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<th>C.O. ml/m.</th>
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</table>

Pressure and cardiac output. Cardiac work also increased. After cardiac denervation on 2 experiments, blood pressure fell under 60 mmHg. These experiments showed similar increases in cardiac output (Table III, No. 1' and 2').

The left coronary inflow was almost in parallel with the elevation of systemic blood pressure (Fig. 10).

B) Depressor response.

Depressor response appeared several seconds after the onset of stimulation, and continued for 30 to 60 seconds. Blood pressure reached the minimal value in 5–25 seconds from the start of stimulation. Cardiac output always increased in the decreasing phase of blood pressure, and had reached at the maximal level before blood pressure fell to the lowest value, and then returned to the control value prior to the restoration of blood pressure. No decrease in cardiac output was observed. Cardiac output increased by 0.2% to 25.5% (average of 12.7%), while the decrease in blood pressure ranged from 6 to 21 mmHg, or from 5.6% to 26.1% of the control level (average of 13.4%). These results are tabulated in Table IV.

The stimulation caused bradycardia on 10 experiments; there was no change in heart rate on two experiments. Increase in cardiac output was observed regardless of the heart rate. Thus the stroke volume increased markedly in brady-cardiac phase. Figure 11 and 12 represent two typical experiments.

**Fig. 11.** Effect of stimulation of the proximal end of the cut vagal nerve.

(Depressor response) (Exp. No. 24, 7) Cardiac output increases in the decreasing phase of blood pressure. Total peripheral vascular resistance decreases markedly.

Total peripheral vascular resistance decreased markedly one very experiment (–5 to –30%). Cardiac work fluctuated in plus minus 15%.

In spite of the increase in cardiac output the left coronary inflow decreased with the fall in blood pressure (Fig. 12).

Cardiac denervation influenced the heart rate, but had no effect on the response of cardiac output and blood pressure.

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Fig. 12. Effect of stimulation of the proximal end of the cut vagal nerve (Depressor response) (Exp. No. 16, 2) In spite of the increase in cardiac output, the left coronary inflow decreases with the fall in blood pressure.

C) Pressor-depressor response.

The data are summarized in Table V.

<table>
<thead>
<tr>
<th>No. of Dog Exper.</th>
<th>B.P. of mmHg</th>
<th>H.R. per m.</th>
<th>C.O. ml/m.</th>
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</table>

Table V: Effects of the stimulation of afferent cervical vagal nerves

C) Pressor-depressor response

Cardiac output increased a little in the pressor phase. In the depressor phase, cardiac output increased more remarkably than in the pressor phase. Results were similar to those obtained in the pure pressor and pure depressor responses.

The left coronary inflow changed in parallel with the blood pressure.

DISCUSSION

The present study reveals the mechanism of the biphasic elevation of the blood pressure after the stimulation of the left sympathetic cardiac nerves, the relationship between the cardiac output and the coronary blood flow during the stimulation of the autonomic nerves, and the effect of the reflex hyper- and hypotension of the cardiac output.

The present study shows that the stimulation of the left sympathetic cardiac nerves has inotropic effect and confirmed the findings of Shipley, Rushmer, Randall, and Saroff. The stimulation of the left sympathetic cardiac nerves excites directly the myocardium and causes forced contraction of the myocardium, and marked increase in cardiac output. The stimulation excites partly the cardiac pacemaker and induces slight tachycardia. The increase in cardiac output contributes to the elevation of blood pressure. As shown in Fig. 6, the late elevation of blood pressure is abolished after the section of both carotid sinus nerves, and blood pressure rises steadily for about 10 seconds after the onset of the stimulation, showing a single peak. The initial rapid elevation of blood pressure stimulates the carotid sinus and causes the depressor response.

However, the surplus depression causes a slight reflex pressor response, and a late elevation of blood pressure. The total peripheral vascular resistance decreases during the first increase in blood pressure and returns to the previous level or increases during the late elevation of blood pressure. As shown in Figure 13, the increase in cardiac output at the late elevation of blood pressure is intermediate between the initial rapid elevation from the stimulation of the left sympathetic nerve and pressor response from the stimulation of the proximal end of the cut cervical vagal nerve. Thus, the late elevation of blood pressure may be induced not only by reflex pressor response but also by the humoral and mechanical factors.

Catecholamine (norepinephrine), induced by the nervous stimulation from myocardial nervous endings in the myocardium,
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![Graph showing the relationship between maximal elevation or depression of the mean systemic blood pressure and the maximal increase in the cardiac output in every experiment. The change is expressed as the percentage of the control value. The increase in cardiac output in comparison with the elevation of blood pressure is most marked in the first peak from the stimulation of the left sympathetic nerve, moderate in the second peak from the stimulation of the left sympathetic nerve, and little in the pressor response from the stimulation of the proximal end of the cut cervical vagal nerve.]

The slight increase in cardiac output, less elevation of blood pressure is observed in the right sympathetic nerve stimulation than in the left. The functional difference of cardiac nerves in the laterality in the previous paper is also supported by the present study. The heart rate in the range of these experiments does not influence the cardiac output (Fig. 8).

The stimulation of the proximal end of cut cervical vagal nerves containing aortic nerves causes pressor and/or depressor response. Previous investigators on the baroreceptor reflexes and cardiac output recognized increases or decreases in cardiac output during pressor or depressor reflex respectively using different methods of experiments. Carotid sinus reflex was often used in studying the effect of the pressor or depressor responses on cardiac output or cardiac performance. Previous investigations showed increases in cardiac output in pressor responses and decreases in depressor responses of cardiac output in depressor responses.

In the pressor response, blood pressure is elevated with the increased total peripheral vascular resistance, mainly the reflex arterial constriction in the splanchnic area. The slight increase in cardiac output follows the blood pressure elevation, and is free from the changes in heart rate. In one of the cases of intact sympathetic cardiac nerves, e.g. No. 3 in Table 3, there is little increase in cardiac output. The elevation of blood pressure is more marked than the increase in cardiac output, in comparison with the experiments of the left sympathetic cardiac nerve stimulation (Fig 13).

On the other hand, denervated cases (No. 1' and 2' in Table 3), shows moderate increases in cardiac output. Therefore, the mechanism of the increase in cardiac output is not the reflex efferent stimulus of sympathetic cardiac nerves. In the first place, the elevated aortic pressure causes a homeometric autoregulation. Secondly, an increase in venous return is caused by a reflex venoconstriction and atrial contraction similar to the reflex due to carotid hypotension.
Rosenblueth described that any increase in cardiac work augmented the amplitude of following ventricular contractions. The increased total peripheral vascular resistance increases the cardiac work and augments the amplitude of following ventricular contractions, and cardiac output.

In the depressor response, mean systemic blood pressure decreases with the decreased total peripheral vascular resistance, mainly from the reflex arterial dilatation in the splanchnic area. As the blood pressure decreases, the cardiac output increases.

The increase in cardiac output is seen in reflex bradycardia or tachycardia. In one of the cases with intact sympathetic cardiac nerves, e.g. No. 4 in Table IV, there is only a slight increase in cardiac output. On the other hand, several cases of semidenervated dogs show moderate increase in cardiac output in the phase of depression of blood pressure (e.g. No. 2 in Table IV).

Increase in cardiac output with the reflex hypotension may be explained by the following mechanism. The reflex hypotension lowers the peripheral vascular resistance, decreases the residual blood volume of the left ventricle, and may increase the venous return by the mechanism of diastolic suction. The change of venous tone and pulmonary vascular bed may also have some relations to the increase in venous return.

The left coronary inflow decreased as the blood pressure decreased in spite of the increase in the cardiac output.

**Summary**

The nervous control of the cardiac output was studied in the anesthetized and hemithoracotomized dogs.

1) The stimulation of the left sympathetic cardiac nerve usually caused biphasic increase in cardiac output as well as biphasic elevation of the systemic blood pressure. Initial marked increase in cardiac output was accompanied with the first elevation of blood pressure during the stimulation. After the stimulation, however, the late elevation of blood pressure was usually accompanied with only a slight increase in cardiac output and marked increase in coronary blood flow.

2) The stimulation of the right sympathetic cardiac nerve caused marked tachycardia with less increase in cardiac output compared with these changes on the left side.

3) The stimulation of the proximal end of the cut cervical vagal nerve caused pressor and/or depressor effect. In the pressor response, cardiac output increased slightly almost in parallel with the systemic blood pressure, while in the depressor response, cardiac output increased with the depression of the blood pressure.

4) The carotid sinus nerves influence particularly the biphasic elevation of the blood pressure by stimulation of the left sympathetic cardiac nerve.

5) The changes of the coronary blood flow were parallel with the blood pressure than with the cardiac output.

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