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Origin of Biphasic Pattern of Sympathetic Nerve Activity at Cardiac Arrest

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Cardiac arrest is an emergency state and should be treated as fast as possible. As we reported before, cardiac arrest induced the biphasic pattern of sympathetic nerve activity. Although we speculated that the origin of this phenomenon was in the central nervous system, it is not clear yet.
In anesthetized rabbits, cardiac arrest was produced by air embolism. When blood pressure fell abruptly, renal nerve activity was exaggerated markedly and showed a biphasic pattern.
To elucidate the origin of the biphasic pattern, the denervation of baroceptor fibers were performed. However, this procedure did not abolish the biphasic pattern. This result suggested that the first phase was developed by the vasomotor center and the second phase was developed by a higher autonomic center such as the hypothalamus. For further examination, the posterior hypothalamus was stimulated repeatedly. The stimuli increased sympathetic nerve activity which lasted as long as the presence of increased sympathetic nerve activity during the cardiac arrest. This result suggested that the second phase was developed by the hypothalamus.
Thus, the biphasic pattern of sympathetic nerve activity was not related to baroreflex and this may be partly emanated from the posterior hypothalamus. This phenomenon to help sympathetic nerve activity markedly increases to cope with the fall in blood pressure. Since the continuation of this period lasted approximately 5.4 minutes after cardiac arrest, the resuscitation must be started within this period. The increased phase might indicate the the function of the hypothalamus is still alive.

Key Words

Cardiac arrest,
Cerebral ischemia,
Sympathetic nerve activity,
Posterior hypothalamus,
Biphasic increase of sympathetic nerve activity.

INTRODUCTION

Cardiac arrest (1–3) is an emergency state and should be treated as fast as possible. Because cardiac arrest is due to the cessation of effective cardiac contraction, the pulse and blood pressure become unobtainable. As we reported before (4), the fall in blood pressure at cardiac arrest induced biphasic pattern of sympathetic nerve activity.

In this condition, cardiac compression and artificial respiration are performed as basic life support (1). When obtainable, various kinds of drugs, such as bicarbonate, catecholamines and so on (5), are given to victims in terms of advanced cardiopulmonary resuscitaion.

However, as we reported before (4), when blood pressure fell abruptly, three different types of sympathetic nerve activity were increased in each different

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mode. Because the increased sympathetic nerve activity has an action of the contraction of the artery, sympathetic nervous system had an action of life support which we called self-resuscitation. We also noticed the harmfulness of catecholamines in overdose because of large activation of sympathetic nerve activity.

In a physiological condition, a fall in blood pressure increase sympathetic nerve activity by an action of the baroceptor reflex (7, 8). This mechanism maintains blood pressure at a certain level. However, it is still obscure how the increased sympathetic nerve activity play its role in the setting of, in this report, cardiac arrest.

In some condition (9) the higher autonomic center has prominent action to increase sympathetic nerve activity partly through the posterior hypothalamus. However, it is not clear how long the hypothalamus works after the start of cardiac arrest and what role the hypothalamus plays in cardiac arrest by increasing the sympathetic nerve activity (10).

From above points of view, our first purpose of this report is to clarify the origin of biphasic pattern of increased sympathetic nerve activity at cardiac arrest. The second purpose is to clarify the active length of the higher autonomic nerve center.

METHOD AND MATERIAL

Thirteen adult rubbits of unselected as to sex and ranging from 2.5 to 3.0 kg in weight were used. They were anesthetized with urethane (1200mg/kg) administered intraperitoneally. After a tracheotomy was performed, each rabbit was ventilated on room air using a Harvard respirator pump. Gallamine (0.2mg/kg, i.m.) was injected initially to prevent movement and was repeated during the course of the experiment as required. Blood gas was measured on occasion and kept within the physiological range with supplement of bicarbonate or oxygen as necessary.

(1) Surgical Procedures

The right femoral vein was cannulated to produce air embolism. A cannula was also introduced into the left femoral artery for recording arterial blood pressure or sampling arterial blood. Blood pressure and electrocardiogram were recorded on chart paper continuously. The rectal temperature was also monitored and maintained at 38 ℃ with a heat pad. A lead II electrocardiogram were used to monitor cardiac rhythm throughout the experiment.

(2) Recording of Nerve Activity

The left renal nerve was exposed through the flank with fine forceps under a binocular microscope and placed on biopolar platinum electrodes. The electrical activities was recorded by a high gain amplifier simultaneously.

(3) Development of Cardiac Arrest

Cardiac arrest, which was developed by air embolism in the pulmonary artery was induced by venous injection of air (10ml). Nerve activity and blood pressure were recorded until significant activities were disappeared.

(4) Baroceptor Denervation

In five rabbits, total baroceptor denervation(7) was completed by severing bilateral vagoaortic nerves and carotid sinus nerves.

(5) Posterior Hypothalamus Stimulation

In eight rabbits, the posterior hypothalamus was stimulated for 10 seconds by each one minute. The needle electrical stimulation was inserted to the posterior hypothalamus following Saywer's figure (10).

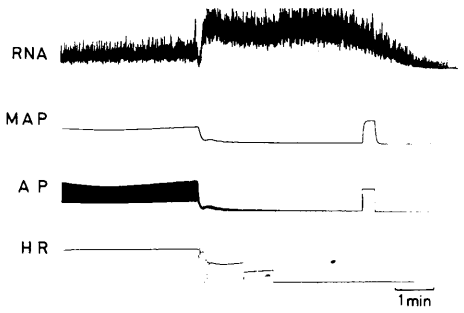


Figure 1. Effect of baroceptor denervation on cardiac arrestinduced increase in sympathetic nerve activity
The figure shows a typical result which obtained in a rabbit with baroceptor denervation. Cardiac arrest was induced by air embolism. Sympathetic nerve activity was recorded from the left renal nerve. Notice the biphasic pattern is still remained. Following abbreviations were used: RNA: renal nerve activity MAP: mean arterial pressure Ap: arterial pressure HR: heart rate

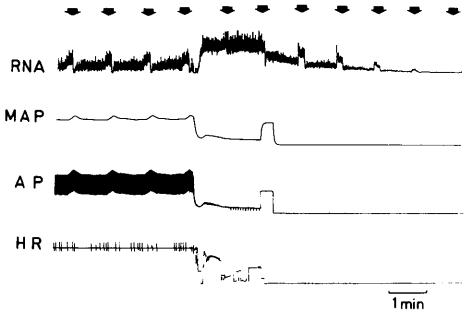


Figure 2. A tripical results of electrical stimulation to the posterior hypothalamus.
By each arrow on the top of the figure, electrical stimulation was given to the posterior hypothalamus. RNA and AP were increased concomitantly. After the development of cardiac arrest, because of the marked increase in RNA, the effect of the stimulation was not seen clearly. However, after the start of the second phase, the effect of electrical stimulation was clearly observed and lasted to the end of the second phase. Same abbreviations were used in Fig. 1.

RESULTS

(1) EFFECTS OF BAROCEPTOR DENERVATION

To clarify the role of baroceptor in the fall of blood pressure, baroceptor denervation was performed by dissecting bilateral carotid sinus and vagoaortic nerves. As shown in a typical result of Fig. 1, even in a rabbit with baroceptor denervation, the first phase and second phase were observed. This indicates the baroceptor plays no significant role in this phenomenon. The similar result was obtained in five rabbits.

(2) EFFECTS OF STIMULATION OF POSTERIOR HYPOTHALAMUS

As shown in a typical results of Fig. 2, the electrical stimulation (arrows in the figure) of the posterior hypothalamus increased renal sympathetic nerve activity.

When cardiac arrest was developed, the overwhelmingly increased sympathetic nerve activity was overlapped the increased activation by electrical stimulation. However, as the activated nerve potential diminishing, the effect of electrical stimulation appeared clearly again and lasted for 5.4 ± 0.2 mitutes in eight rabbits.

DISCUSSION

(1) THE ORIGIN OF BIPHASIC INCREASE IN SYMPATHETIC NERVE ACTIVITY

Initial three minutes of the cardiac

arrest is sometimes called as "golden time", because higher cerebral center does not suffer from irreversible changes in this period (11, 12). Therefore, the resuscitation procedure must be started within this period.

The higher central nervous system stops the activation within 30 seconds and pupillary dilatation begins approximately 45 seconds after cerebral blood flow cessation (11, 12). It is reported (2) that the centers that control corneal and pupillary reflexes can survive 10 minutes and the neurons of the medulla may survive 20 or more minutes. Thus, the autonomic nervous system seemed to play its role after fainting due to cardiac arrest than the higher nervous center. This mechanism we call "self-resuscitation".

Because the first phase was still remained after baroceptor denervation, the first phase must not be produced by baroceptor reflex. The first phase may be produced by direct ischemia of the vasomotor center because of its short lag to activation. Furthermore, because effects of the electrical stimulation was diminished during the first phase, it is less probable that the posterior hypothalamus plays important role in the first phase.

The second phase produced by the higher autonomic center such as the hypothalamus or others because EEG, which is the activity of the higher central nervous system, diminished usually within 30 seconds (11). Because effects of stimulation of the posterior hypothalamus lasted to the same span as the second phase, the posterior hypothalamus should be related to the second phase.

Thus, the biphasic pattern of the renal sympathetic nerve activity was produced in the brain stem and the hypothalamus and two phases may play different role in cardiac arrest.

(2) THE IMPORTANCE OF GOLDEN TIME IN CARDIAC ARREST

The peak of second phase of the increase in sympathetic nerve activity was appeared at 2 minutes after abrupt fall in blood pressure. The neurons of the cerebral cortex undergo irreversible changes approximately 3 minutes after cardiac arrest (11, 12).

When stimulus was given to the posterior hypothalamus, the effects was diminished within 8 minutes. This suggested the area which controls the autonomic activity was alive at least 5.2 minutes after cardiac arrest.

As we reported before, the activation of the sympathetic nerve activity may have a action of resuscitation which we call self-resuscitaion (4, 13). According to our present results, the active time of the posterior hypothalamus was equal to the activation of renal nerve activity which was active for 5.2 minutes. This suggested the self-resuscitation may depend on the action of the hypothalamus.

These results indicates the resuscitation must started while the activity of the autonomic nervous system is alive and continues self-resuscitation by secretion of catecholamines.

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