Kobe University Repository : Kernel

PDF issue: 2026-11-02

Response of Sympathetic Nerve Activity to Cardiac Arrest

Kujime,Kenchi
Sawatari,Hisayuki
Su,Michiyo
Ueba,Yasuyuki

(Citation)
Bulletin of allied medical sciences Kobe : BAMS (Kobe),4:27-32

(Issue Date)
1988-12-26

(Resource Type)
departmental bulletin paper

(Version)
Version of Record

(URL)
https://hdl.handle.net/20.500.14094/80070288

KOBE UNIVERSITY

Response of Sympathetic Nerve Activity to Cardiac Arrest

Kenchi Kujime[1], Hisayuki Sawatari[2], Michiyo Su[3] and Yasuyuki Ueba[1]

Cardiac arrest is an emergency state and should be treated as fast as possible. Although catecholamines are usually used for the treatment, very little is known as to how the sympathetic nervous system plays its role in this condition.
In anesthetized rabbits, cardiac arrest was dereloped by ventricular fibrillation (Vf) or air embolism. When blood pressure fell abruptly, three types of sympathetic nerve activity recorded from the renal, cardiac or cervical nerve cervical nerve showed biphasic increased pattern in different ratios.
The deneration of baroceptor fibers 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 a higher autonomic center such as the hypothalamus.
The peak of the second phase was approximately at the point of two minutes after the development of cardiac arrest. The nerve activity decreased gradually and continued until about five minutes after the occurrence of cardiac arrest. This increase in sympathetic nerve activity stimulates contraction of the vascular system and may be determined as restoration phase from a fall in blood pressure. This mechanism is called "self-resuscitation".
When spontaneous recovery from Vf occurred, blood pressure showed marked elevation. Because this phenomenon suggests excessive catecholamine secreation in the blood, it is indicated that additional catecholamine administration may result in more arrhythmogenetic heart condition.
Thus, when cardiac arrest developed, sympathetic nerve activity markedly increased to cope with the fall in blood pressure. Because the continuation of this period was approximately five minutes after cardiac arrest, the resuscitation must be started within this period. Additional catecholamines may not be needed within five minutes after cardiac arrest and may become harmful to induce arrhythmias.

Key Words
Cardiac arrest,
Cerebral ischemia,
Sympathetic nerve activity,
Catecholamines,
Self-resuscitation.

School of Allied Medical Sciences, Kobe University[1], Takasago Municipal Hospital[2], Department of Internal Medicine, Division I, School of Medicine, Kobe University[3], Kobe, Japan.

INTRODUCTION

Cardiac arrest (1–3) is an emergency state and should be treated as fast as possible. Because cardiac arrest is due to the stop of effective cardiac contraction, the pulse and blood pressure become unobtainable. The two most common causes of cardiac arrest are ventricular fibrillation and asystole. A third cause of cardiac arrest has been termed electromechanical dissociation.

In this condition, whatever the causes, 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 (4), are given to victims in terms of advanced cardiopulmonary resuscitation. However, very little is known of the mode of changes in sympathetic nerve activity in the course of cardiac arrest and its modification by the treatment.

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 (5,6). Therefore, the resuscitation procedure must be started within this period.

The central nervous system stops the activation within 30 seconds and pupillary dilatation begins approximately 45 seconds after cessation of cerebral blood flow. This discrepancy indicates alert state of the sympathetic nervous system after loss of consciousness.

In a physiological condition, a fall in blood pressure increases 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 sympathetic nerve activity plays its role in pathologic condition of, in this report, cardiac arrest.

From above points of view, our main purpose of this report is to clarify the changes in sympathetic nerve activity and to obtain suggestion of the treatment for the caridac arrest.

MATERIALS AND METHODS

1. Surgical Procedures

Adult rabbits 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.

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°C 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 bipolar platinum electrodes. The right cardiac nerve was exposed by cutting the sternum. The left cervical nerve activity was also recorded. One of these three electrical activities was recorded by a high gain amplifier simultatneously with other parometers.

3. Development of Cardiac Arrest

Cardiac arrest was developed by ventricular fibrillation associated with the coronary ligation of left anterior

descending branch. In some rabbits, air emblism in the pulmonary artery was induced by venous injection of air (10ml). Nerve activity and blood pressure were recorded until significant activities were disappeared.

RESULTS

1. Effects of Cardiac Arrest on Sympathetic Nerve Activity

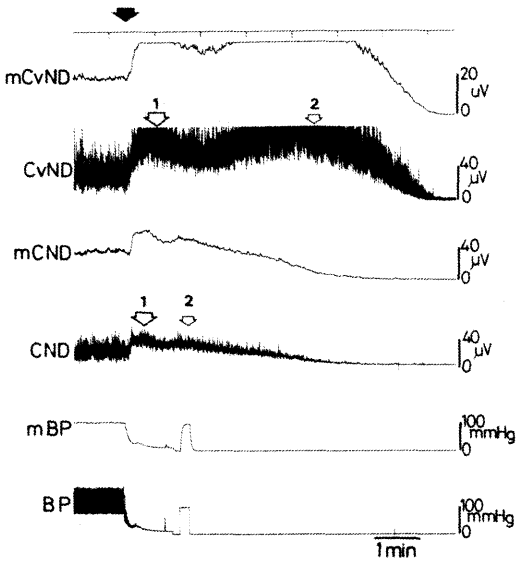


Figure 1. Effect of Cardiac Arrest on Sympathetic nerve Activities and Hemodynamics.

Cardiac arrest was induced by air embolism at the arrow of the top of the panel. Each arrow noted as 1 in the figure demonstrates the first phase. Each arrow noted as 2 demonstrates the second phase.

Following abbreviations were used: CvND: cervical sympathetic nerve discharge CND: cardiac nerve discharge BP: blood pressure m: mean.

Figure 1 demonstrates the changes in cervical sympathetic nerve activity (CvND) and cardiac sympathetic nerve activity (CND) with simultaneous recording of blood pressure. At the point of an closed arrow on the top of the panel, cardiac arrest was induced by air embolism.

When blood pressure fell down abruptly by cardiac arrest, sympathetic nerve activity increased by two to four times more in the comparison of the control value. The increase pattern usually can be classified into two phases as shown by open arrows in Fig. 1. As clearly observed in this record, the time to the peak of the first phase, the peak of the second phase and the disappearance of the nerve activity are quite different between the two nerve activities. Cervical sympathetic nerve activity continues longer than those of cardiac sympathetic nerve activity does.

2. 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 Fig. 2, even in a rabbit with baroceptor denervation, the first phase and second phase were observed. This indicates that the baroceptor reflex plays no significant role in this phenomenon.

3. Statistical Analysis

When the time to the peak of phase 1, phase 2 and disappearance of the each nerve activity were compared statistically in table 1. The lengths of phase 1, phase 2 and time to dis-

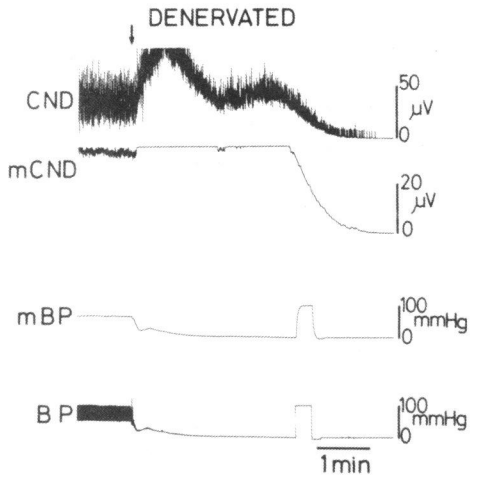


Figure 2. Effect of Baroceptor Denervation on Cardiac Arrest-Induced Increase in Sympathetic nerve Activity. Cardiac arrest was induced by air embolism. Nerve activity was recorded from the left cardiac nerve. Same abbreviations are used as in Fig. 1.

appearance of the CvND showed significantly longer value than other two activity. On the other hand, the shortest continued activity was cardiac sympathetic nerve activity. There were no statistical differences between groups of intact baroceptor and baroceptor denervation.

4. Effects of Cardiac Compression

In order to observe the effects of cardiac compression, ventricular fibrillation (Vf) was developed by myocardial infarction.

Figure 3 depicts the changes in EKG, BP (blood pressure) and cardiac nerve dischange (CND). The top panel shows irreversible ventricular fibrillation. The middle panel shows spontaneous recovery from Vf. The bottom panel shows effects of cardiac compression

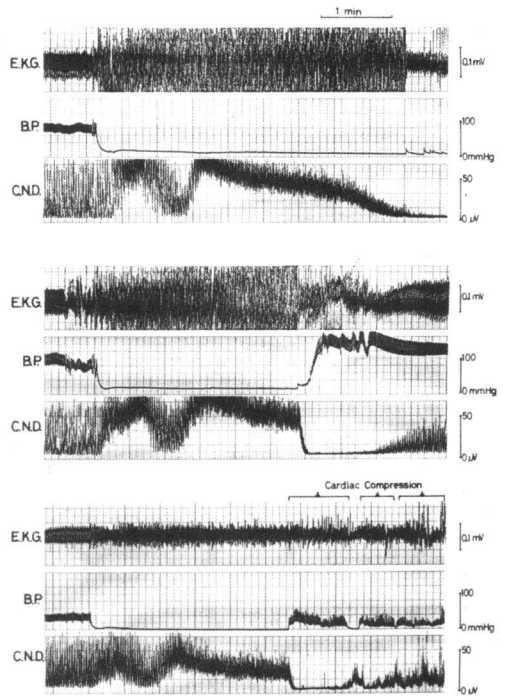


Figure 3. Effect of Spontaneous Recovery and Cardiac Compression.

Cardiac arrest was induced by ventricular fibrillation. Top of the panel shows the changes in cardiac nerve discharge in the condition without recovery. Middle of the panel shows the effect of spontaneous recovery from ventricular fibrillation. Bottom of the panel show effect of cardiac compression.

during Vf.

When blood pressure was risen after spontaneous recovery from ventricular fibrillation in the middle panel or cardiac compression in the bottom panel, sympathetic nerve activity diminished almost completely. When the heart beat rhythmically again as seen in middle panel, because catecholamines secreted from sympathetic nerve endings massively during Vf, blood pressure rose up very markedly.

Table 1. Statistical Analysis of the Phase 1, Phase2, Total Length.
Statistical analysis of phase 1, phase 2 and time to disappearance (end) of three nerve activities in 6 rabbits.
N: number S. D.: standard deviation S. E.: standard error.

(MINUTES)

	CvND			CND			RND		
	Phase 1	Phase 2	END	Phase 1	Phase 2	END	Phase 1	Phase 2	END
N	6	6	6	6	6	6	6	6	6
MEAN	0.73333	3.3	6.63333	0.53333	1.61666	4.78333	0.66666	2.58333	5.03333
S. D.	0.12110	0.35777	0.81158	0.12110	0.28577	0.45789	0.12110	0.39200	0.46332
S. E.	0.04944	0.14605	0.33132	0.04944	0.11666	0.18693	0.04944	0.16003	0.18915
1	0.6	3.8	7	0.4	1.4	5.6	0.6	3	5
2	0.8	2.8	6.5	0.6	2	4.6	0.6	2.4	4.4
3	0.9	3	8	0.7	1.8	4.2	0.8	2.6	5.8
4	0.8	3.5	6.2	0.4	1.3	4.8	0.8	2.8	5.2
5	0.7	3.4	6.5	0.5	1.4	4.8	0.5	2.8	5
6	0.6	3.3	5.6	0.6	1.8	4.7	0.7	1.9	4.8

CvND:Cervical sympathetic nerve discharge
CND :Cardiac sympathetic nerve discharg
RND :Renal nerve discharge

DISCUSSION

1. Biphasic Increase in Sympathetic Nerve Activity

The central nervous system stops the activation within 30 seconds and pupillary dilatation begins approximately 45 seconds after cerebral blood flow cessation (5,6). 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 seemes to survive longer to play its role after fainting due to cardiac arrest than the higher nervous center. This mechanism we call "self-resuscitation". The same mechanism is reported as cough-induced cardiac compression (9).

Because the first phase was still remained after baroceptor denervation, the first phase must not be produced by baroceptor reflex. However, the first phase may be produced by direct ischemia of the vasomotor center. The second phase produced by the higher autonomic center such as the hypothalamus or others because EEG diminished usually within 30 seconds (5).

Three different types of nerve activity showed different peak and continuation. This may be due to indicate fragility of the nerve center to the ischemia or spinal mechanism.

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 (5,6).

These results indicates the resuscitation must started while the activity of the autonomic nervous system is alive

and continues self-resuscitaion by secretion of catecholamines.

3. Necessity of Additional Catecholamines

As shown in middle panel of Fig. 3, after the recovery from cardiac arrest, blood pressure increased markedly. This suggested that excessive catecholamine secretion was occurred by accentuated sympathetic nerve activity. In this condition of what catecholamines were filled in the body, it may be harmful to give more catecholamines because catecholamines stimulates heart muscle and increase arrhythmogenesity (10).

However, beyond 5 minutes after the development of cardiac arrest (top panel of Fig. 3) or during cardiac compression (bottom panel of Fig. 3), sympathetic nerve activity decreased and may be effective to give additional dose of catecholamines (4).

CONCLUSION

Thus, when cardiac arrest was developed, sympathetic nerve activity increased markedly and showed biphasic pattern. This mechanism we call self-resuscitation. Although the continuation of this period was approximately five minutes and the peak of the second phase appeared two minutes after the arrest, the resuscitation must be started within this period. Because of marked increase in sympathetic nerve activity, in some circumstances, additional catecholamines may become harmful.

REFERENCES

1. Standards for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC) JAMA 227(suppl): 837-866, 1974.
2. Barber JM, Budassi SA: Mosby's manual of emergency care, CV Mosby Saint Louise 1979.
3. Goldberger E, Wheat NW Jr: Cardiac arrest in treatment of cardiac emergencies. CV Mosby Saint Louis 1977.
4. Zoll PM: Rational use of drugs for cardiac arrest and after cardiac resuscitaion. Am J Cardiol 27: 645, 1971
5. Binnie CD et al :Electroencephalographic prediction of fatal anoxic brain damage after resuscitation from cardiac arrest. Br Med J 4: 265, 1970.
6. Ames A, Wright RL, Kowada M et al: Cerebral ischemia, II. The no-reflow phenomenon. Am J Pathol 52: 437, 1968.
7. Ninomiya, I. and Irisawa, H.: Summation of baroceptor reflex effects on sympathetic nerve activities. Am. J. Physiol. 216: 1330-1336, 1969.
8. Kujime K, Sawatari H, Ueba Y, et al: Digitalis toxicity and baroreceptor in rabbits. Bull Allied Med Sci (Kobe) 1: 21-30, 1985.
9. Criley JM, Blaufuss AH, Kissel GL: Cough-induced cardiac compression: self-administration form of cardiopulmonary resuscitation. JAMA 236: 1246, 1976.
10. Kujime K, Natelson BH: Effects of stellectomy on cardiac rhythm disturbance induced by ouabain in guinea pigs. J Pharmacol Exp Ther 229: 113-117, 1984.