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－Case Report－

Successful Resuscitation from Ventricular Fibrillation during Jogging in a Young Patient with Hypertrophic Cardiomyopathy

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A 15-year-old girl, who was previously in good health, suddenly collapsed while jogging. Immediate cardiopulmonary resuscitation (CPR) was initiated, and she arrived at our hospital 13 minutes later. The ventricular fibrillation (VF) on admission was reverted to sinus rhythm 18 minutes after collapse by the second cardioversion. The echocardiogram revealed hypertrophic nonobstructed cardiomyopathy (HNOCM), although the 24hr ambulatory electrocardiographic, electrophysiologic and exercise stress tests could not define the exact cause of VF. Exercise-induced ischemia with sustained mild hypokalemia was suspected to be the cause of VF. The patient recovered consciousness three days after admission, and followed an uneventful course of treatment with oral atenolol not associating with disabling neurological deficit.

Immediate basic life support and delivery of automatic external defibrillator on the spot is needed to rescue patients with out-of-hospital cardiac arrest.

Key Words: cardiopulmonary resuscitation (CPR), cardioversion, automatic defibrillator, cardiac arrest

Introduction

Hypertrophic cardiomyopathy (HCM) is a heterogeneous disease genotypically, phenotypically, pathophysiologically and therapeutically[1,2]. Sudden death is a common complication of HCM, especially in adolescents and young adults. Although the annual mortality rate from sudden death is 2 to 3% in adults, it is even higher (approximately 6%) in young patients[3]. Unexpected sudden cardiac death (SCD) of young athletes occurs in approximately 1 of 200,000 high school athletes per academic year[4]. Even if electrocardiographic abnormalities were detected at annual school physical examinations, some young individuals reportedly had SCD during school physical education classes. This was due to delayed or lack of further investigation, and requires precise evaluation systems. However, the authority of permitting each young patient with hypertrophic nonobstructed cardiomyopathy (HNOCM) to perform exercise is dependent on individual physician judgement. Such judgement sometimes may differ since some patients sometimes show neither severe symptoms nor significant pressure gradient of the left ventricular outflow tract.

Ventricular fibrillation (VF) is the most common mechanism of SCD, and some of the conditions facilitating VF include bradycardia, long QT syndrome, electrocution, electrolyte imbalance, drugs, sympathetic stimulation and myocardial ischemia. If young, cardiac arrest survivors were rescued with longstanding cerebral damages, their quality of life would be aggravated. We herein report a case with dramatic, successful resuscitation from cardiac arrest remaining no cerebral damage in a young asymptomatic female with previous unknown HCM.

Case Report

A 15-year-old, high school student, who had been well and playing volleyball during middle high school days, had an electrocardiographic abnormality noted on April 22, 1998. It showed negative T wave in leads II, III, $_aV_f$, V_1 through V_6 and high amplitude of R wave in V_1 through V_3 without QT interval prolongation (QTc interval=0.41 seconds)(Fig. 1). She was scheduled for admission to a university hospital on

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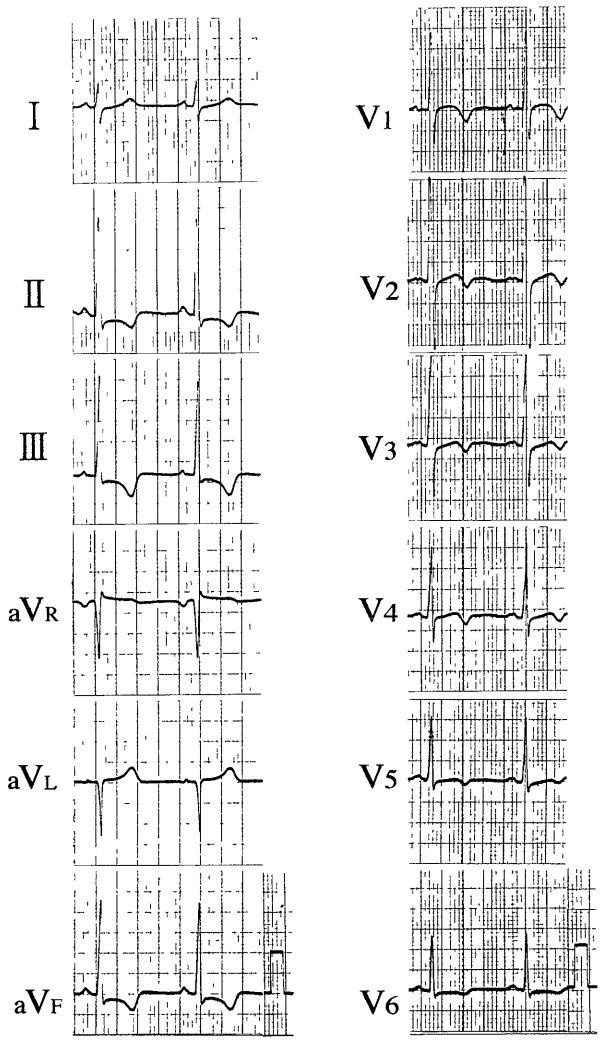


Fig. 1, Electrocardiogram on April 22, 1998.
Negative T wave in leads II, III, $_aV_f$, V_1 through V_6 and high amplitude of R wave in V_1 through V_3 were shown. QTc interval was 0.41 seconds.

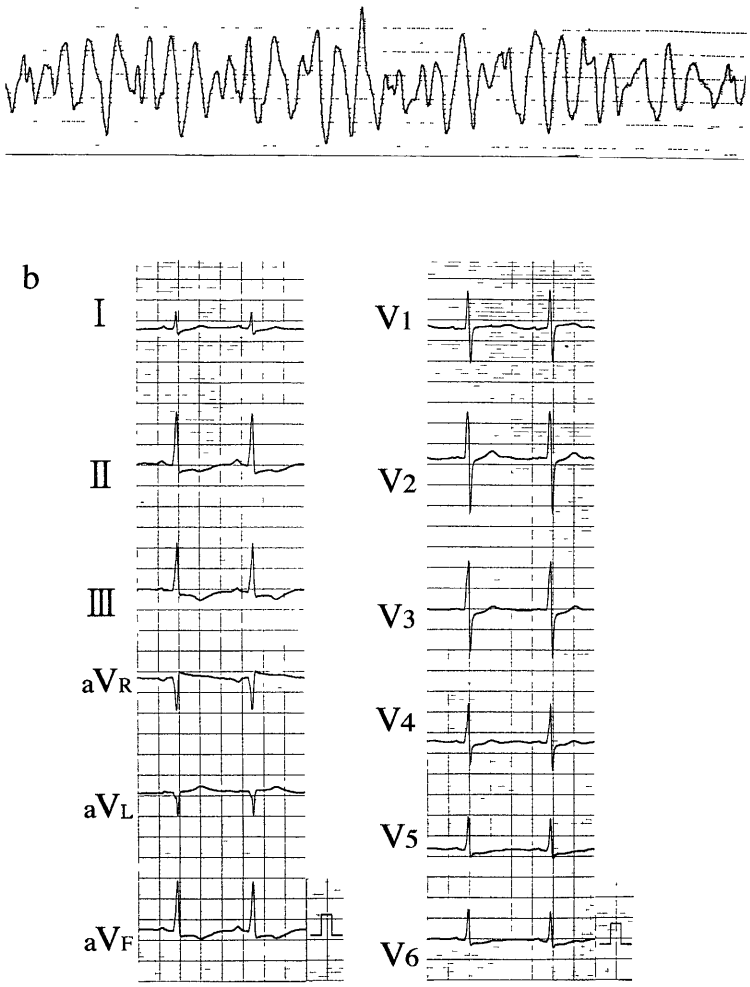


Fig. 2. Electrocardiogram on June 3, 1998.
Ventricular fibrillation was reverted to sinus rhythm after DC cardioversion. (a: before cardioversion ; b: after cardioversion)
DC cardioversion : direct current cardioversion

June 4, 1998 for further diagnostic investigation. She had been suffering from diarrhea for two months owing to irritable colitis. Her grandfather and uncle had left ventricular hypertrophy without definite diagnosis. There was no family history of sudden death, congestive heart failure and lethal heart diseases.

At 10: 35 a.m. on June 3, 1998, the patient collapsed suddenly on the school grounds while jogging approximately 300 meter in distance during physical education. Basic life support was started immediately by a teacher, and a first-responding fire fighter vigorously initiated CPR two minutes later. The patient was brought to our hospital by ambulance 13 minutes after collapse, with mydriasis, weak spontaneous respiration and E-1, V-1 and M-1 of the Glasgow Coma Scale (GCS), or III-300-A of Japan Coma Scale (JCS). The initial rhythm was VF, and the second direct current (DC) cardioversion reverted VF to sinus rhythm 18 minutes after collapse (Fig. 2-a, b). A clinical time table is shown in Table 1. The arterial blood gas, complete blood cell count and serum biochemical analysis on admission to our hospital are shown in Table 2. Echocardiographycally, the wall thickness of the interventricular septum (IVS) was 22.5mm ; anterior left ventricular wall 19.3mm ; lateral left ventricular wall 14.0mm ; posterior left ventricular wall 11.3mm (Fig. 3). Neither left ventricular outflow tract obstruction nor systolic anterior motion (SAM) of the anterior mitral valve leaflet was present. Our diagnosis was HNOCM with mild mitral regurgitation.

Since her coma scale was improved (E1, V1, M4 of GCS or III-100 of JCS) with hemodynamic stability (Table 1), the patient was transferred to the university hospital for hyperbaric oxygen therapy (HOT).

Table 1. Clinical Time Table on June 3, 1998

Time	Clinical Course	GCS
10:35 a.m.	Collapse on the school ground	E1, V1, M1
10:40 a.m.	Ambulance's arrival in the shool ground	E1, V1, M1
10:48 a.m.	Patient's arrival at hospital; BP: unmeasurable	E1, V1, M1
10:52 a.m.	Intratrcheal intubation The first delivery of 360J of DC cardioversion	E1, V1, M1
10:53 a.m.	The second delivery of 360J of DC cardioversion	E1, V1, M1
10:56 a.m.	BP : 104/-mmHg	E1, V1, M2
11:15 a.m.	BP : 120/56mmHg	E1, V1, M4
12:50 a.m.	BP : 150/88mmHg	E1, V1, M4
01:00 p.m.	Transfer to the university hospital	E1, V1, M4

BP: blood pressure
DC cardioversion: direct current cardioversion
GCS: Glasgow Coma Scale

However, the patient did not require HOT; due to improved cerebral function, and received glycerol and potassium L-aspartate intravenously for five days to correct hypokalemia (K=2.7mEq/L in the university hospital on June 3). Three days after admission, the patient recovered to E-4, V-4 and M-6 of GCS or I-1 of JCS without neurological deficits, and the value of potassium was corrected to 4.0mEq/L.

On June 17, 1998, left ventriculography and biventriculography demonstrated hypertrophic IVS with normal coronary arteriogram and slightly elevated left ventricular end-diastolic pressure (LVEDP) (Fig.4, 5, Table 3). There was no pressure gradient between the left ventricular outflow tract and the aorta.

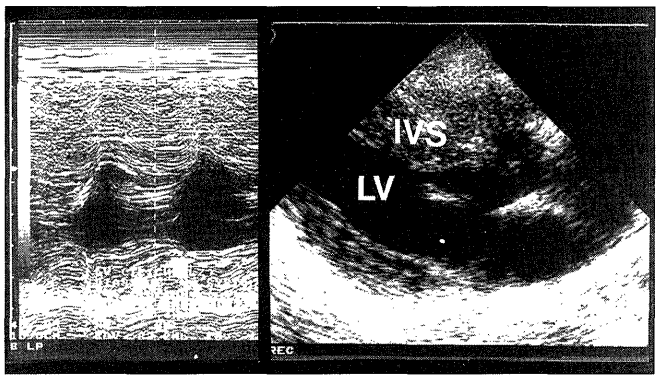
Table 2. Blood Gas, Complete Blood Cell Count and Serum Biochemical Data at 10:54 a.m. on June3, 1998

pH	6.98pH units	AST	36U/ℓ
PCO_2	35.7mmHg	ALT	25U/ℓ
PO_2	306.4mmHg	LDH	342U/ℓ
(with 10 ℓ/min oxygen)		CK	95U/ℓ
HCO_3-	8.4mmol/ℓ	Na	140mE/ℓ
Base Excess	−23.6mmol/ℓ	K	3.7mEq/ℓ
WBC	9900/μℓ	Cl	95mEq/ℓ
RBC	520x10⁴/μℓ	BUN	121mg/dℓ
Hgb	17.1g/dℓ	Creatinine	1.0mg/dℓ
Plt	14.7x10⁴/μℓ	CRP	0.4mg/dℓ

Table 3. Hemodynamic Variables

		Systole	Diastole	Mean	EDP
PAWP	mmHg	*	*	10	*
PA	mmHg	25	11	16	*
RV	mmHg	29	1	*	6
RA	mmHg	*	*	3	*
LV	mmHg	90	9	*	17
Ao	mmHg	100	60	80	*

Ao: aorta
EDP: end-diastolic pressure
PA: pulmonary arterial pressure
PAWP: pulmonary arterial wedge pressure
RA: right atrium
RV: right ventricle
*: not available



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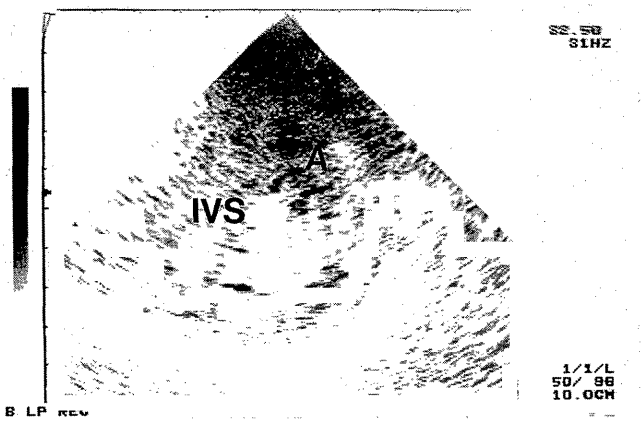

b

Fig. 3. Echocardiogram on June 3, 1998.
Echocardiogram showed hypertrophy of the interventricular septum and the anterior left ventricular wall (a : long axis view ; b: short axis view)
A : anterior left ventricular wall
IVS : interventricular septum
LV : left ventricule

Ventricular tachycardia (VT), VF and supraventricular tahcycardia (SVT) were not induced by programmed ventricular stimulation, isoproterenol provacation and by exercise stress test. The 24hr ambulatory electrocardiogram (24hr ECG) also failed to identify any arrhythmias.

The patient was given 25mg/day of atenolol orally, and was discharged without any neurological sequelae on June 9, 1998. She has been following an uneventful course without worsening her daily quality of life.

Discussion

Coronary artery disease is the most common cause of sudden death in those over the age of 35 years. In contrast, structural cardiovascular abnormalities, including HCM (36%), unexplained increased cardiac mass ("possible HCM") (10%), aberrant coronary arteries (13%), other coronary anomalies (6%), ruptured

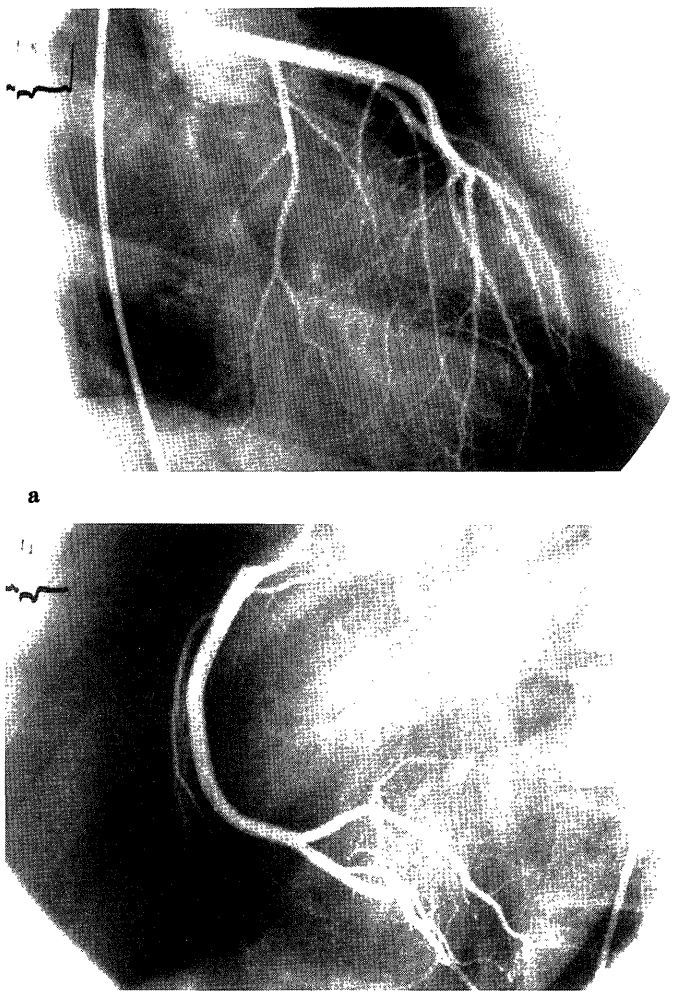

Fig. 4. Coronary angiogram on June 17, 1998.
Coronary angiogram showed no significant stenosis of the left (a) and the right coronary arteries.

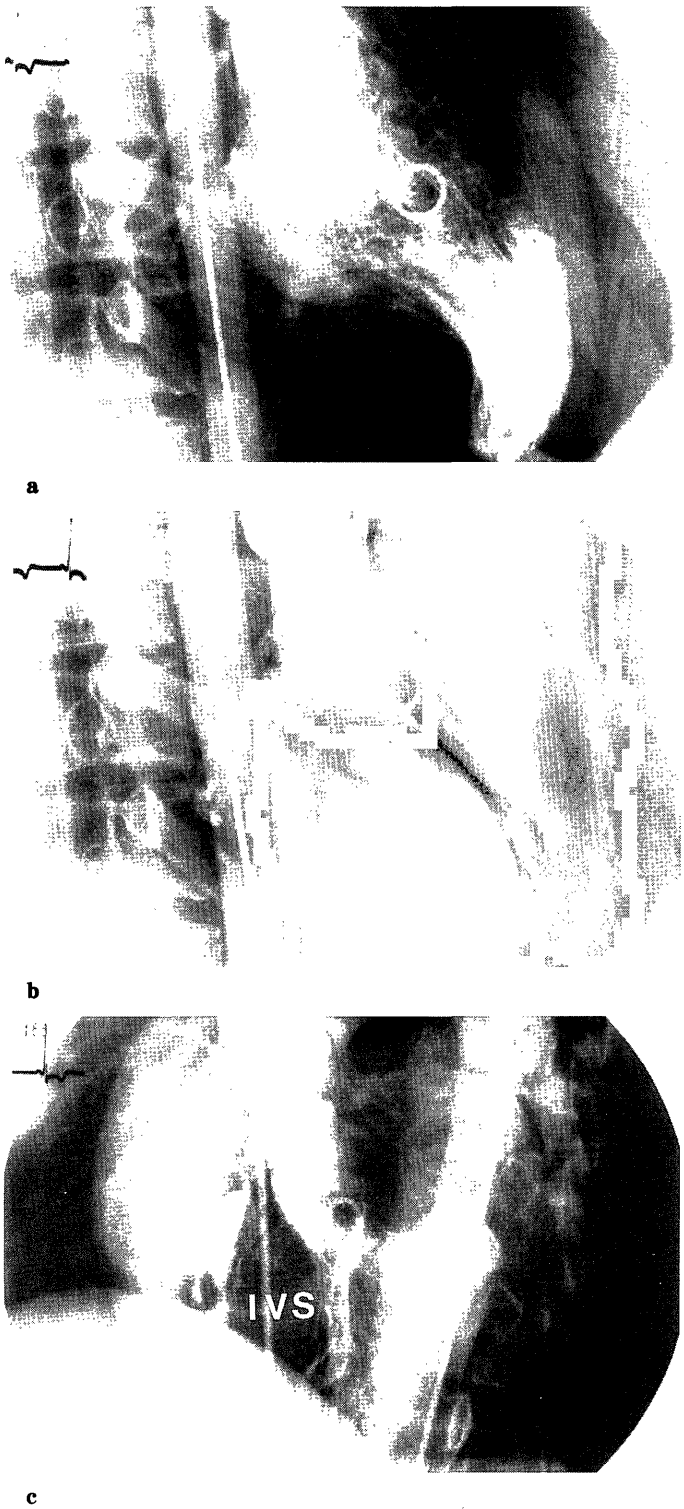

Fig. 5. Left ventriculography (a: end-diastole ; b : end-systole) and biventriculography (c) on June 17, 1998.
Marked hypertrophy of the interventricular septum was demonstrated.
IVS : interventricular septum

aortic aneurysm(5%), tunneled LAD(5%) and aortic valve stenosis(4%) are the major causes of sudden death in young athletes(< age 35)[5]. HCM is present in about 2 of 1,000 young adults and its prevalence in men and women is 0.26 : 0.09%[6]. Sudden death is often the first manifestation in an asymptomatic patient with HCM, and occurs during or immediately after moderate or heavy physical activity[7]. Although the precise mechanisms by which exercise precipitates SCD in person with HCM is unclear, potential mechanisms are as follows : (1) VT or SVT arise in the disarrayed muscle or in ischemic areas of small vessel disease ; (2) sudden hemodynamic instability occurs involving dynamic increase in the left ventricular outflow tract obstruction ; (3) exercise-induced systemic hypotension occurs by activating ventricular baroreceptor reflex, resulting in withdrawal of sympathetic tone[8], or abnormal vascular response to exercise occurs due to increased sensitivity of arterial baroreflexes[9] ; (4) VF arising from VT is induced by ischemia due to shortening of diastole or hypotension during exercise-induced tachycardia and (5) impairment of coronary flow or aggravation of the left ventricular filling occurs during exercise, or mental excitement[10]. We could not identify the definite cause of cardiac arrest and VF, because neither sustained VT nor nonsustained VT were detected with electrophysiologic study, 24hr ECG and exercise stress testing.

In a series of 115 patients immediately after resuscitation from out-of hospital VT, resuscitated patients revealed that admission mean serum potassium value was 3.70±0.72(SD) mEq/L, compared with 4.09±0.66 mEq/L in acute myocardial infarction, and 4.17±0.35 mEq/L in patients with coronary heart disease[11]. Exercise-induced ischemia with sustained mild hypokalemia owing to irritable colitis may be a cause of VF in our case.

The mainstay treatment of HCM is drug therapy using beta-blockers, calcium antagonists and other drugs including disopyramide, diuretics and antivitamin K agents[12]. The benifits of beta-blockers are significant reduction in nonfatal cardiac arrest in the short term trials and sudden cardiac death in long term trials, which are likely due to relief ischemia, reduction of heart rate and maintenance of favorable autonomic nervous system balance[12]. Amiodarone is reported to improve symptoms and to prevent sudden death in patients with HCM. In the study of amiodarone treatment refractory to conventional drug therapy(beta-blockers and calcium antagonist), eight out of 50 patients treated with amiodarone died during mean follow-up period of 2.2± 1.8 years, and the survival rate of patients with VT was significantly worse than that of patients without VT[13]. Decrease in peak left ventricular filling rate within 10 days of amiodarone therapy was associated with subsequent sudden death[13]. While recent studies have indicated that nonsustained ventricular tachycarida in asymptomatic patients without additional risk factors, such as a positive family history of sudden death or syncope should not be treated prophylactically with amiodarone, and symptomatic patients with sustained ventircular tachycardias and/or syncope related to ventricular arrhythmias should undergo implantable cardioverter-defibrillator (ICD) implantation[14]. Elliott et al. showed that ICD was probably superior to low dose amiodarone in patients with HCM who survived a cardiac arrest owing to an episode of VT/VF[15]. Kron et al. also emphasized that the ICD represented an effective treatment approach for young patients less than 20 years old with life-threatening ventricular tachyarrhythmias[16]. The patient of our case will need to implant ICD when a VT/VF episode occurs in the future.

Weaver et al. reported that both the period from collapse until initiation of basic life support and the duration of basic life support before delivery of the first defibrillatory shock were shorter in patients who survived compared with those died (3.6±2.5 versus 6.1 ±3.3minutes and 4.3±3.3 versus 7.3±4.2 minutes)[17]. Mortality increased by 3% each minute until CPR was begun and by 4% a minute until the first shock was dilivered[18]. Survival from VF can be improved by shortening the delay of CPR initiation and to defibrillation. Therefore, it is necessary to increase the number of qualified persons who can use an automatic external defibrillator and to authorize them to attempt it without the permission of a physician in some urgent cases. Furthermore, many doctors should have interest in CPR, and actively educate and enlighten the general citizen on the basic life support maneuvers required to decrease morbidity and mortality from serious diseases.

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