Journal of Cardiography 14: 597-604, 1984

A catheter-induced syncopal attack in a case of hypertrophic obstructive cardiomyopathy

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Summary

A 48-year-old man with hypertrophic obstructive cardiomyopathy (HOCM) was studied by serial cardiac catheterization during incidentally induced syncope.

His hospital admission was for repeated syncopal attacks and chest pain. His electrocardiogram showed giant negative T waves (>10 mm) in V_3, V_4 and V_5 leads, and his M-mode echocardiogram disclosed typical asymmetric septal hypertrophy, systolic anterior movement of the mitral valve, and a midsystolic semiclosure of the aortic valve. During cardiac catheterization, we incidentally induced syncope and recorded the serial pressure changes. During syncope, systemic blood pressure dropped without appreciable changes in pulmonary arterial and right ventricular pressures. Although blood pressure was maintained by administering etilefrine and hydrocortisone, syncope persisted. After administration of propranolol, he recovered from syncope. He was on sinus rhythm throughout the examinations. The ejection time (ET) obtained from the aortic pressure curve was extremely short (160 msec) during syncope and prolonged (300 msec) after recovery without significant change in the heart rate.

We believe that the prompt intravenous administration of propranolol was very effective in relieving myocardial spasm as a possible cause of syncope.

Key words

Hypertrophic obstructive cardiomyopathy　Syncope　Left ventricular function　Propranolol

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Received for publication December 13, 1983 (Ref. No. 27-38)

Introduction

There have only been sporadic reports of mechanisms of syncope in hypertrophic obstructive cardiomyopathy (HOCM). Its genesis has been attributed to arrhythmias, such as atrial fibrillation[1], asystole[2,3], ventricular tachycardia and ventricular fibrillation[4].

Medical treatment with propranolol has been used for more than 15 years, but long-term effects of beta-blocking agents[5-8] remain controversial. Moreover, patients with HOCM have recently been treated with calcium-antagonists, including verapamil and nifedipine. However, there have been few reports concerning left ventricular function during syncopal attacks in HOCM and the pharmacological effects on syncope.

We report here a case of HOCM in which myocardial spasm as a possible cause of syncope could be relieved by the prompt intravenous administration of propranolol.

Case report

A 48-year-old man was hospitalized because of repeated syncopal attacks and chest pain. At 42 years of age, the patient had an abnormal electrocardiogram, but remained well until 45 years of age. In 1980, he experienced palpitation, exertional dyspnea and chest pain on exertion. The symptoms gradually worsened. He frequently experienced syncopal attacks for more than 15 minutes after an excessive alcoholic intake during 3 months prior to admission. He was diagnosed as having HOCM by electrocardiography and echocardiography. There was no history of sudden deaths in his family, and he had never been diagnosed as having hypertension before.

Physical examination disclosed a blood pressure of 128/74 mmHg and a regular pulse of 66 per minute. The lungs and abdomen were unremarkable. The third and fourth heart sounds were audible and the latter was palpable. These was a grade 2/6 midsystolic high-pitched murmur.

Chest radiography showed no congestion or other abnormalities in the lung fields. The cardio-thoracic ratio was 47 percent (**Fig. 1**).

An electrocardiogram demonstrated high voltage R waves, giant negative T waves above 10 mm in V_3, V_4 and V_5 leads, and negative T waves in I, II, aV_L and V_6 leads (**Fig. 2**).

An M-mode echocardiogram demonstrated abnormal systolic movement of the mitral valve[9], asymmetric septal hypertrophy[10] (ratio: 22/14= 1.6), decreased septal motion, and a midsystolic semiclosure of the aortic valve[11] (**Fig. 3**).

In the two-dimensional echocardiogram, a long-axis view suggested midventricular narrowing in systole (**Fig. 4B**). On the apical view, there was obstruction at the midportion of the left ventricle during systole, and the left ventricular cavity had the shape of a figure '8' (**Fig. 4D**).

As shown in **Table 1**, cardiac catheterization revealed slightly elevated left ventricular end diastolic pressures. Cardiac index and stroke index measured by the thermodilution method were decreased.

The serial changes in hemodynamic parameters before, during and after syncope were as follows (**Fig. 5A~K**):

Before syncope, the heart rate was 77 beats per minute and regular. The aortic (AoP),

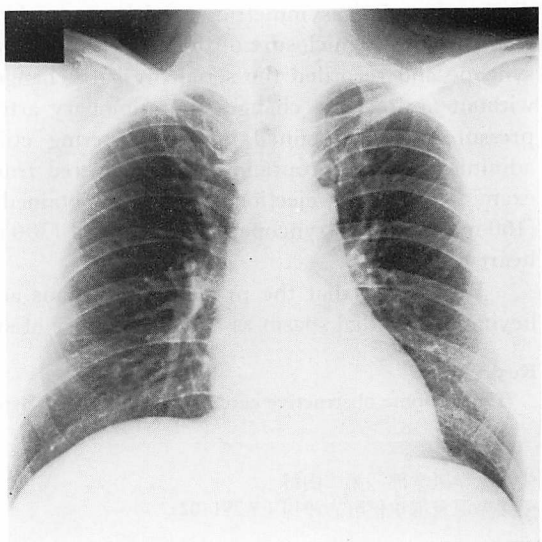

Fig. 1. Chest radiograph on admission.

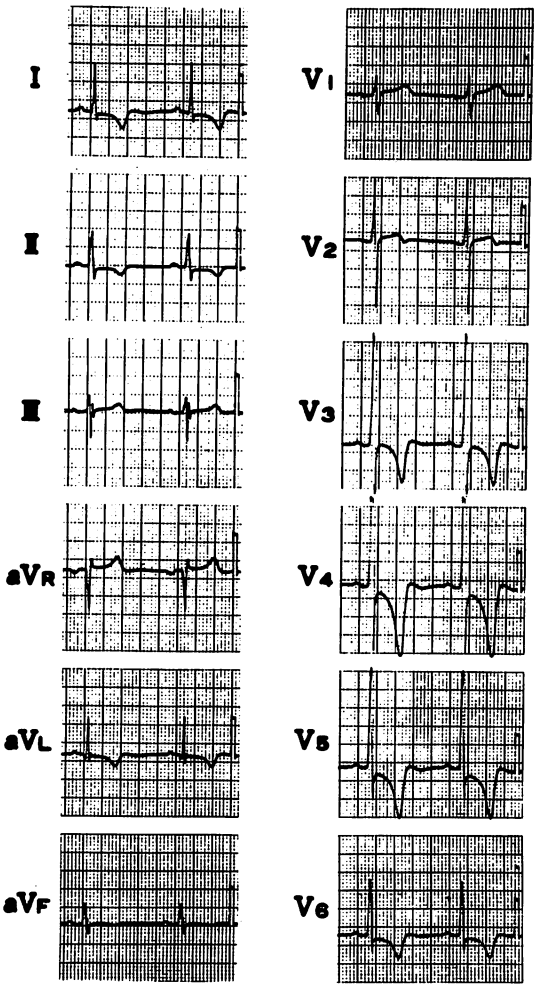

Fig. 2. Electrocardiogram on admission.
Giant negative T waves and high voltage R waves are noted in leads V_3, V_4, and V_5.

pulmonary arterial (PAP) and left ventricular systolic pressures and the ET were 132/74 mmHg, 24/10 mmHg, 128 mmHg, and 252 msec, respectively (**Fig. 5A, B**).

When we inserted a catheter into the apex of the left ventricle, ventricular premature beats occurred, and the patient lost consciousness (**Fig. 5C**). During syncope, the AoP fell to 78/46 mmHg (**Fig. 5D**) but the electrocardiogram showed a sinus rhythm and a rate of 120 per minute. Pulmonary arterial and right ventricular systolic pressures, 27/10 mmHg and 25 mmHg, respectively, did not change throughout the syncopal attack. However, the ET was markedly shortened to 102 msec (**Fig. 5D, E**).

Table 1. Hemodynamic parameters

(mmHg)

	Systolic	Diastolic	Mean	EDP
RV	24			5
PA	24	10	13	
PCW			8	
AO	128	72		
LV	132			14
CI	2.83 *l*/min/m²			
SI	40 m*l*/m²			

Abbreviations: RV=right ventricle; PA=pulmonary artery; PCW=pulmonary capillary wedge; AO=aorta; LV=left ventricle; CI=cardiac index; SI=stroke index.

Twenty seconds after the onset of syncope, the heart rate spontaneously returned to 66 per min, but the AoP remained 88/46 mmHg, and the ET remained short (160 msec) (**Fig. 5F**).

We promptly administered 5 mg etilefrine and 1,000 mg hydrocortisone intravenously. The AoP increased to 120/58 mmHg. The electrocardiogram showed a regular sinus rhythm with 74 per minute, but the ET remained short (160 msec) during the syncopal attack (**Fig. 5G**).

After placing a temporary pacemaker in the right ventricle, we again administered 2 mg propranolol at a rate of 0.4 mg per minute. Abruptly the ET was lengthened to 300 msec and the patient recovered from his syncope (**Fig. 5H~K**).

Discussion

The annual mortality in HOCM is 3 or 4 percent[7,12,13] and about half of the deaths occur suddenly. Many investigators hava proposed a possible relationship of syncope or sudden death to arrhythmias. HOCM is characterized by outflow tract obstruction with pressure gradi-

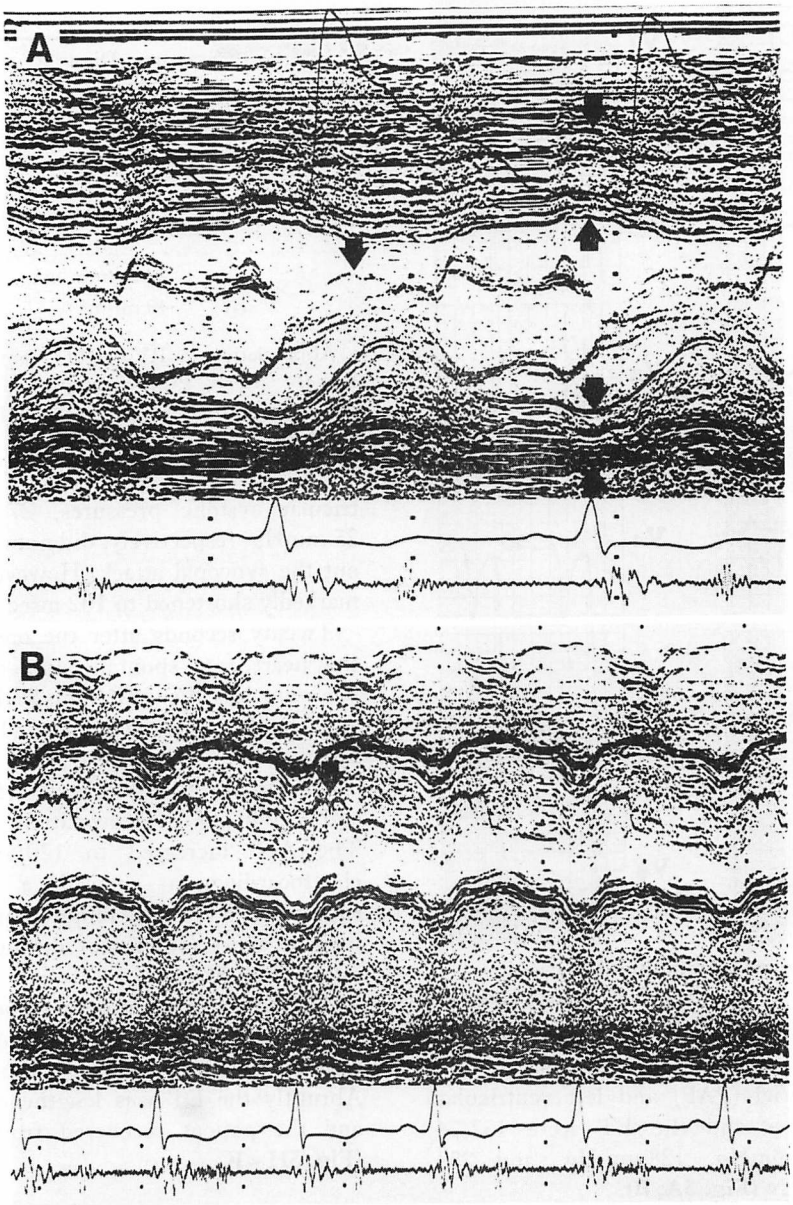

Fig. 3. M-mode echocardiograms.

In the upper panel (A), arrows indicate systolic anterior movement of the mitral valve (left) and asymmetric septal hypertrophy (right). Thickness of the interventricular septum is 22 mm and thickness of the left ventricular posterior wall is 14 mm. In the lower panel (B), an arrow indicates a midsystolic semiclosure of the aortic valve.

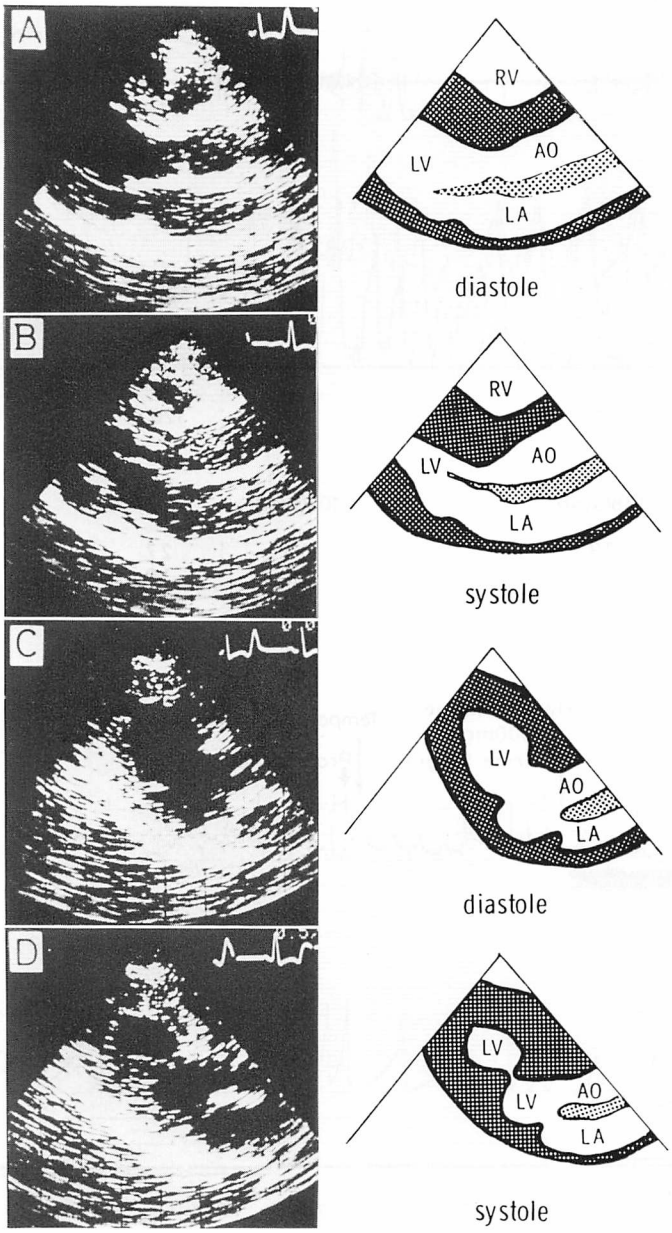

Fig. 4. Two-dimensional echocardiograms in the long-axis view (A, B) **and apical view** (C, D).

A and C show end-diastolic phase, B and D, end-systolic phase, respectively. In the long-axis view, the left ventricular cavity has mid-ventricular narrowing in systole (B). In the apical view, the left ventricular cavity is obliterated at the midportion in systole (D) and has the shape of a figure ' 8 '.

Abbreviations: RV=right ventricle; LV=left ventricle; LA=left atrium; AO=aorta.

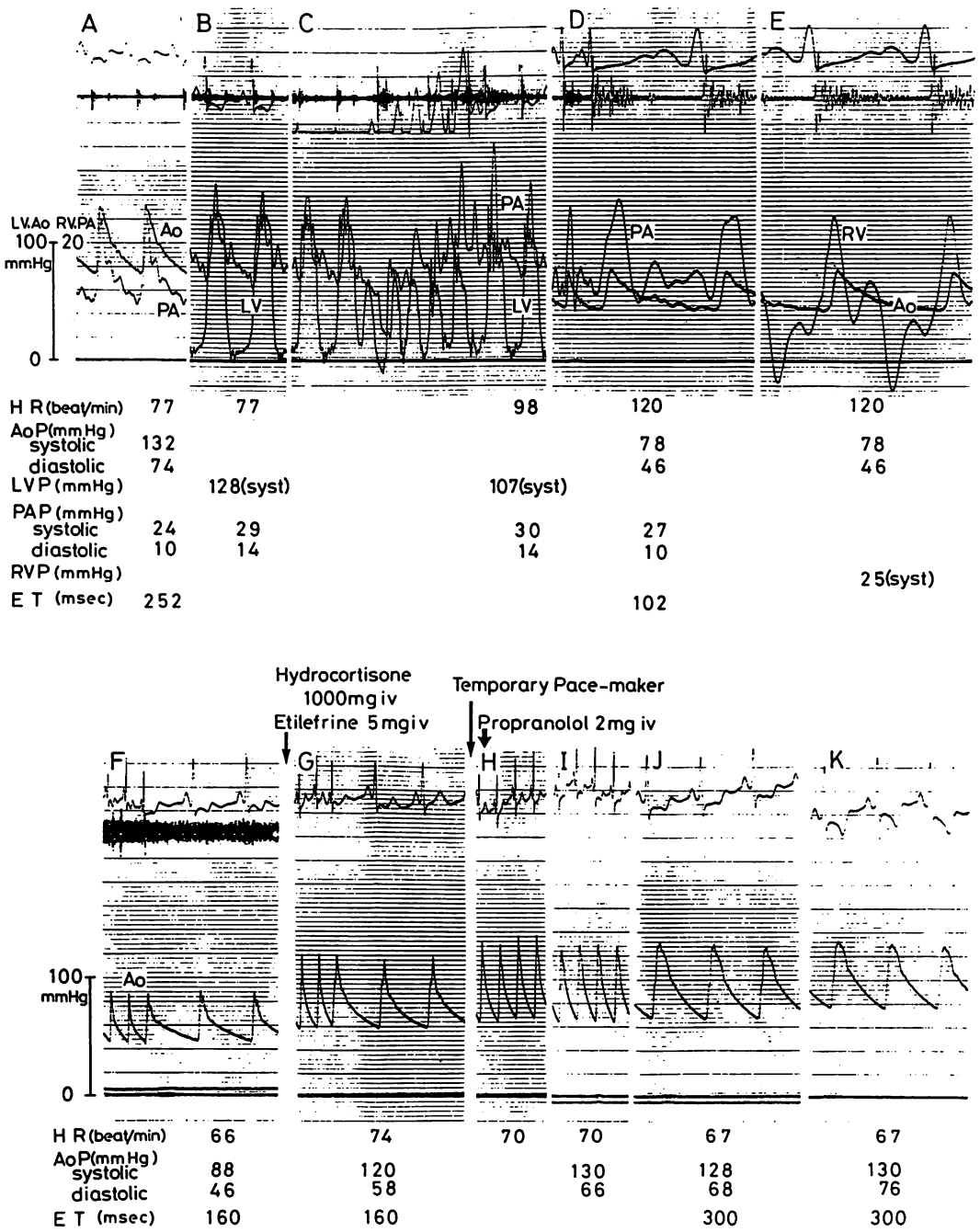

Fig. 5. Serial changes of hemodynamic parameters before, during and after syncope.
Abbreviations: HR=heart rate; AoP=aortic pressure; LVP=left ventricular pressure; PAP=pulmonary artery pressure; RVP=right ventricular pressure; ET=ejection time.

ent[14] and decreased compliance. As Goodwin reported[15], exercise and emotion may precipitate tachycardia and a decrease in stroke volume and blood pressure, which secondarily may lead to arrhythmia. Such a case was reported by Nishimura et al[16], however, it has never been proved whether arrhythmia is a cause or result.

We encountered a very rare case of HOCM in which syncope continued without arrhythmia.

Initially, pulmonary arterial and right ventricular pressures showed no remarkable changes in the syncopal attack. Therefore, so-called shock was an unlikely cause. Following propranolol administration, the ET was abruptly lengthened to 300 msec without change in heart rate and the patient promptly recovered from his syncope. The rhythm was always regular sinus rhythm. The mechanism of action of propranolol is not known, but it may act directly on the myocardial cells and produce a decrease in myocardial contractility.

As suggested by Cumming et al[17], propranolol administered intravenously quickly relieve anoxic spells in tetralogy of Fallot[18]. Johnson also reported that the mechanism underlying the anoxic attack was due to excessive myocardial tonus or right ventricular infundibular spasm[19]. In the present case, myocardial spasm or an increase in the obstruction led to disturbances of left ventricular relaxation and decrease of stroke volume and blood pressure, resulting in the syncopal attack.

We hypothesize myocardial spasm as a cause of syncope and sudden death in HOCM. Administration of a bolus of propranolol is mandatory to relieve a catastrophic syncopal attack.

心カテーテル検査中に失神発作をおこした肥大閉塞性心筋症の1例

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今回，我々は，失神発作中の血行動態を観察しえた HOCM 例を経験したので報告した．

主訴は繰り返す失神発作と胸部圧拍感である．心電図上，巨大陰性 T 波を V_3-V_5 誘導に認め，心エコー図では典型的な ASH, SAM, 大動脈弁の semiclosure が存在した．心臓カテーテル検査中，偶然，失神発作が誘発され，その時の大動脈圧は低下したが，肺動脈圧，右心室圧には変化を認めなかった．Hydrocortisone, etilefrine の静注により，大動脈圧は上昇したが，駆出時間は 160 msec と著明に短縮しており（心拍数 66/分），失神は持続した．続いて propranolol を静注したところ，駆出時間は 300 msec と著明に延長し（心拍数 67/分），それとともに失神から回復した．経過中，心電図はすべて洞調律であった．

今回我々が経験した失神は，心筋のスパスムによるものと思われ，propranolol の静注が著効を示した．

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