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Case Reports

Cough-Cardiopulmonary Resuscitation in the Cardiac Catheterization Laboratory: Hemodynamics During an Episode of Prolonged Hypotensive Ventricular Tachycardia

Brian Miller, PhD, Edward Lesnefsky, MD, Theresa Heyborne, RN, Betty Schmidt, RN, Kathy Freeman, RN, Susan Breckinridge, RN, Kathleen Kelley, RN, David Mann, MD, and Michael Reiter, MD, PhD

Cough-CPR, a deep rhythmic forceful cough repeated 30–60 times per minute, can be an effective resuscitative technique during emergencies occurring in the cardiac catheterization laboratory. We provide documented evidence on the potential of cough-CPR to maintain adequate systemic arterial blood pressure and consciousness during malignant ventricular arrhythmias, including the longest cough-CPR episode (75–90 sec), with continuous hemodynamics recorded. Results in three patients disclose that 1) mean arterial pressure during cough-CPR was 47–66% of nonarrhythmic baseline at a cough rate of 38–46% of normal sinus rhythm heart rate; 2) mean arterial pressure during hypotensive ventricular tachycardia was 17–60 mm Hg higher with than without cough-CPR; 3) at comparable diastolic pressures (33 vs. 31 mm Hg), systolic arterial pressure during cough-CPR was 40 mm Hg higher than basic CPR; and 4) consciousness can be maintained with cough-CPR during prolonged malignant ventricular arrhythmias. Thus cough-CPR can be a valuable adjunct in maintaining patient stability while definitive therapy for the malignant ventricular arrhythmia is administered.

Key words: cough resuscitation, arrhythmia

INTRODUCTION

Malignant ventricular arrhythmias requiring cardiopulmonary resuscitation can occur in the cardiac catheterization and electrophysiology laboratories. Thirty seconds or more may elapse before therapy restores sinus rhythm and adequate systemic perfusion. Cough-cardiopulmonary resuscitation (cough-CPR), a deep rhythmic forceful cough repeated 30–60 times per minute, may be an effective resuscitative technique during this critical time. Cough-CPR has maintained consciousness for 92 sec during ventricular fibrillation in the coronary care unit [1]. This implies adequate cerebral blood flow, since 8–11 sec of cerebral circulatory arrest causes loss of consciousness [2]. We provide further evidence on the potential of cough-CPR to maintain adequate systemic arterial blood pressure and cerebral perfusion during malignant ventricular arrhythmias, including the longest cough-CPR tracing with continuous hemodynamics recorded.

MATERIALS AND METHODS

Patients

Three patients who used cough-CPR during hypotensive arrhythmia are presented. Patient 1 is a 65-year-old man who underwent cardiac catheterization for unstable angina. Ventricular tachycardia (180 bpm) developed as a Swan-Ganz catheter was withdrawn through the right heart. Intravenous lidocaine was ineffective. Patient 2 is a 66-year-old man who had an inferior myocardial infarction and recurrent sustained ventricular tachycardia. He underwent cardiac electrophysiologic testing, during which sustained ventricular tachycardia was induced. Patient 3 is a 46-year-old woman with a non-Q-wave myocardial infarction and recent onset angina. Coronary spasm and ventricular fibrillation developed during right coronary arteriography. Patient 1 practiced a few coughs before catheterization and was intermittently coached to cough during ventricular tachycardia. Patient 2 was

From the Division of Cardiology, Rose Medical Center and Division of Cardiology, University of Colorado Health Sciences Center, Denver.

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Address reprint requests to Dr. Brian Miller, Division of Cardiology, Rose Medical Center, 4567 East Ninth Avenue, Denver, CO 80220.

TABLE I. Hemodynamic Data

Patient No.	MAP NSR	MAP CCPR	MAP MVA	HR	CR	T
1	97	50	33	73	28	75–90
	137/77	98/33				
2	106	70	10–30	103	42	10
3	64	30	—	83	38	20
	80/52	58/18				

MAP, mean arterial pressure; NSR, normal sinus rhythm; CCPR, malignant ventricular arrhythmia with cough-CPR; MVA, malignant ventricular arrhythmia without cough-CPR; HR, heart rate; CR, cough rate/min; T, duration of malignant ventricular arrhythmia with cough-CPR (sec).

coached on each cough during ventricular tachycardia. Patient 3 was instructed beforehand on the possible need for forceful repetitive coughing during coronary arteriography.

Hemodynamic Recording

Cardiac catheterization and electrophysiologic testing, according to standard techniques, were performed after informed consent was obtained. At least two orthogonal EKG leads and intra-arterial pressure were continuously monitored for all three patients. Pressure transducers were calibrated before all procedures. 'Average' systolic and diastolic pressures were computed as the mean of all recorded beats during cough-CPR. Mean arterial pressure in patients 1 and 3 was computed as the average of arterial pressures sampled every 0.25 sec for the duration of cough-CPR. For patient 2, mean arterial pressure was electronically derived from the transducer signal input to a Hewlett Packard 78205D Monitor. Arterial catheters (all fluid-filled) were located in the central aorta (patients 1 and 3) and in the femoral artery (patient 2).

Mean arterial pressure during hypotensive ventricular arrhythmias with and without cough-CPR, during basic CPR, and in normal sinus rhythm were compared. The duration of cough-CPR during hypotensive arrhythmia was recorded.

RESULTS

Hemodynamic data for the three patients during ventricular arrhythmia alone, ventricular arrhythmia with cough-CPR, and during normal sinus rhythm are presented in Table I.

Patient 1

Cough-CPR (rate = 28/min) was maintained for 75–90 sec[1] during ventricular tachycardia. Cough-CPR was associated with a mean arterial pressure of 50 mm Hg (98/33 mm Hg 'average'). The arrhythmia alone was hypotensive: During a 4 sec pause in cough-CPR, mean arterial pressure was 33 mm Hg and no systolic pressure was generated (Fig. 1). Basic CPR was begun after 75–90 sec of cough-CPR because of decreasing cough systolic pressures and because signs of loss of consciousness were judged to be present. Mean arterial pressure during basic CPR was 41 mm Hg (57/31 mm Hg 'average') with loss of consciousness. Prior to cardioversion (on initial attempt at 360 joules), mean arterial pressure during ventricular tachycardia without cough or basic CPR was 29 mm Hg with no systolic pressure generated. Normal sinus rhythm (heart rate = 73 bpm) and arterial pressure (137/77 mm Hg) were restored after cardioversion.

[1]Hardcopy tracing of cough-CPR during ventricular tachycardia is for 75 sec. However, ventricular tachycardia is likely to have occurred for some time during the previous 15 sec when the hardcopy was off.

Patient 2

A mean arterial pressure of 106 mm Hg was measured during sinus rhythm (heart rate = 103 bpm). Sustained ventricular tachycardia (210 bpm) was induced. During an initial period of 19 sec without cough-CPR, mean arterial pressure was 30 mm Hg. The patient was then coached in cough-CPR for 10 sec, during which mean arterial pressure rose to 70 mm Hg (cough-CPR rate = 42/min). When the patient was instructed to stop coughing, a 12 sec interval of ventricular tachycardia without cough-CPR was associated with a mean arterial pressure of less than 10 mm Hg. Loss of consciousness occurred. Subsequent burst pacing restored sinus rhythm and normal mean arterial pressure (100 mm Hg).

Patient 3

Cough-CPR (rate = 38/min) was maintained for 20 sec until defibrillation. Mean arterial pressure during ventricular fibrillation with cough-CPR was 30 mm Hg (58/18 mm Hg 'average') compared with 64 mm Hg (80/52 mm Hg 'average') in sinus rhythm (heart rate = 83 bpm). Defibrillation (on initial attempt) at 100 joules successfully restored normal sinus rhythm and systemic blood pressure.

DISCUSSION

Cough-CPR can be a valuable resuscitative method in the cardiac catheterization laboratory. Maintenance of arterial pressure and consciousness up to 90 sec can help maintain patient stability while definitive therapy for the malignant ventricular arrhythmia is administered. Forceful coughing may also convert ventricular tachycardia to sinus rhythm. In one investigation [3], 9% (6/68) of monitored patients with sustained ventricular tachycardia (30–90 sec) converted to sinus rhythm with cough. In several records, cough artifact coincided with the mo-

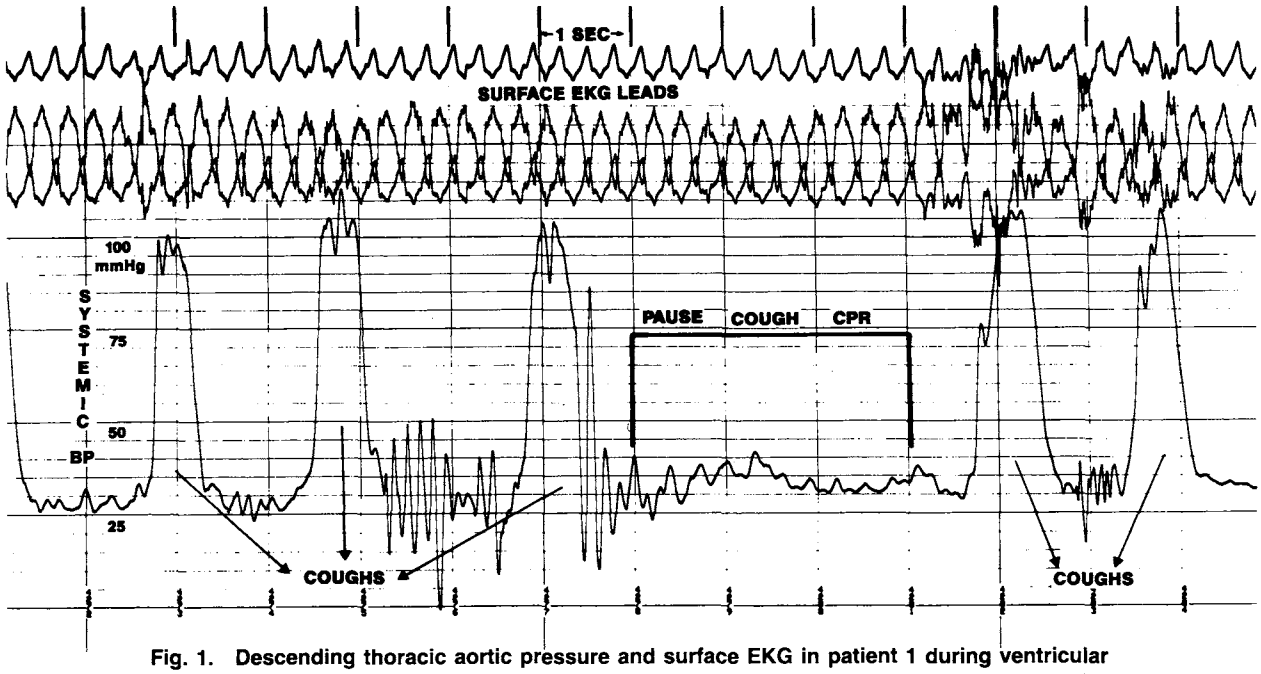

Fig. 1. Descending thoracic aortic pressure and surface EKG in patient 1 during ventricular tachycardia. During a 4 sec pause in cough-CPR, no systolic pressure was generated, with mean arterial pressure being 33 mm Hg.

ment of cardioversion. No ventricular tachycardia patient converted to ventricular fibrillation on cough (Chamberlain, 1989, personal communication).

In our study, an arterial pressure of 98/33 mm Hg during cough-CPR was associated with maintenance of consciousness for 75–90 sec in a patient with hypotensive ventricular tachycardia. The arterial pressure increase during ventricular tachycardia with cough-CPR was almost entirely due to cough systolic pressure rise. For our three patients, mean arterial pressure during cough-CPR was 47–66% of nonarrhythmic baseline at a cough rate 38–46% of normal sinus rhythm heart rate. Mean arterial pressure during ventricular tachycardia with cough-CPR was 17–60 mm Hg higher than that of ventricular tachycardia without cough-CPR. Systolic pressure during cough-CPR was also 40 mm Hg higher than basic CPR in the one patient in whom they could be compared.

There are several advantages of cough-CPR compared with basic CPR. Rib and sternal fractures are avoided with cough-CPR. Cough-CPR can be started immediately, prior to loss of consciousness, and is self-performed, freeing physicians and nurses to concentrate on other aspects of the resuscitative effort.

The mechanism of blood flow during malignant ventricular arrhythmias with cough-CPR in humans is unknown. One recent investigation examined changes in left ventricular chamber size, aortic and mitral valve opening, and flow velocity by two-dimensional echo-doppler during brief (5–10 sec) ventricular asystole with cough-CPR in pacemaker-dependent patients [4]. Left ventricular chamber area was almost the same during coughs as between coughs. The aortic and mitral valves opened up only slightly with low flow velocities during coughs. All patients evidenced presyncope during cough-CPR. However, the average age of study patients was more than 70 years, and cough effort may have been suboptimal. Chest motion during cough can also make accurate determination of echo-Doppler changes in valve area, flow velocity, and left ventricular chamber size difficult.

Experimental animal models of cough-CPR have been developed [1,5–7]. These investigations have shown that only infrequently and for small chest sizes is blood flow during cough-CPR caused by direct compression of the ventricles between the sternum and the spine. Instead there is evidence for a thoracic pump mechanism of antegrade blood flow during Cough-CPR [7,8]. Cough causes a sudden increase in intrathoracic pressure. This pressure increase is transmitted through the arteries, which are valveless, thick-walled, and do not collapse. Cough-induced intrathoracic and right atrial pressure rise is *not* transmitted to the superior extrathoracic veins, such as the jugular vein, because of the venous valves preventing reflux and venous collapse at the superior thoracic outlets [8]. This results in a carotid artery to jugular vein pressure gradient responsible for antegrade cephalad blood flow during cough.

The thoracic pump theory postulates that blood is propelled forward from the most compressible, thin-walled thoracic vessels, the pulmonary venules, using the increased pressure generated during cough. Indeed, indocyanine dye movement and cineangiography studies [7,8] suggest that during experimental cough-CPR thoracic flow emanates from the pulmonary veins. Flow is antegrade through the left atrium and left ventricle, with both mitral and aortic valves opening synchronously during coughs. These thick-walled chambers may not be compressed, but instead act as a noncontracting conduit for blood flow during cough-CPR. Blood flow in the aorta is almost exclusively brachiocephalic during coughs, including retrograde flow from the descending aorta. The latter effect is probably due to the elevated intra-abdominal pressure during cough. Right heart filling occurs primarily between coughs, when intrathoracic pressure is lower, and is greatly augmented by precough inspiration. Antegrade flow in the descending aorta also occurs between coughs.

Cough-CPR may be a valuable method for maintaining blood flow during malignant ventricular arrhythmias [9,10]. However, much still needs to be learned about the physiology of human cough-CPR and whether further modifications in cough-CPR can increase the efficacy of this technique.

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