arch, and passes to the right and behind the trachea. The ligamentum arteriosum completes a “ring” to the left of the trachea, and in circumstances where the ligamentum arteriosum is short or taut, compression of the trachea and esophagus can occur. Only a minority of patients with right-sided aortic arch have development of symptomatic tracheal or tracheoesophageal compression. As with all anomalies of the great vessels, symptoms generally occur in infancy or childhood.

Our patient had right-sided aortic arch and moderate resting airflow obstruction. Although presenting at age 29 years, in retrospect, moderate symptoms had been present since childhood and had been treated by an altered lifestyle. The expiratory airflow obstruction was likely the result of her right aortic arch. Since the aorta crosses in front of the vertebral bodies while going behind the trachea, it is possible that the narrow anteroposterior thoracic diameter further accentuated tracheal compression. The worsening of symptoms and airflow obstruction when in a supine position were likely due to increased compression of the trachea between the heart and great vessels above, and the descending aorta and vertebral bodies below. Increased intravascular volume while in a supine position might create some engorgement of the great vessels (particularly the pulmonary artery) and could further exacerbate airflow obstruction. Modest fluid challenge, 1 L of isotonic saline solution over 45 minutes, did worsen airflow obstruction and increase symptoms and signs of airflow obstruction, only in the supine position.

We hypothesize that our patient had development of severe intraoperative airflow obstruction secondary to position and a moderate to large intravascular fluid challenge. If this hypothesis is correct, symptoms should have improved with a brisk diuresis. As it was, they resolved spontaneously while she was receiving vigorous asthma therapy, accompanied by vigorous hydration (150 to 200 ml/h) for 48 hours after surgery.

Surgical division of the ligamentum arteriosum should be curative. Our patient has declined this procedure. She is well educated as to the nature of the problem, and it is hoped that with this knowledge, future incidents can be prevented or treated more appropriately.

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Self-Administered Hyperventilation Cardiopulmonary Resuscitation for 100 s of Cardiac Arrest during Holter Monitoring*
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An 80-year-old man remained conscious due to vigorous deep breathing during 100 s of ventricular arrest which was recorded on a Holter ECG. Arterial blood flow is considered to have been maintained by changes in intrathoracic pressure produced by deep respiratory movements. This case may represent a pure model of the “thoracic pump” mechanism. (Chest 1991; 99:1310-12)

CPR = cardiopulmonary resuscitation

There are two distinct mechanisms for the generation of forward blood flow during CPR. One is the “cardiac pump mechanism; the other is the “thoracic pump” mechanism. The patient may have been conscious due to the effect of repeated large amplitude movements of the thoracic cage during cardiac arrest. This case may represent a pure model of the “thoracic pump” mechanism.

CASE REPORT
An 80-year-old man had suffered from occasional syncope for two years and had frequented the outpatient department of his local hospital. After disopyramide therapy was started by his private physician for premature ventricular contractions, he complained of urine retention and was admitted to our urology service. After urinary catheterization, he lost consciousness for several seconds and was transferred to the medical ward for a complete investigation of his syncope.

At the time of physical examination, there were no significant abnormalities. The pulse was regular at a rate of about 80 beats per minute and supine blood pressure was 130/70 mm Hg. A complete neurologic examination disclosed no abnormalities.

The complete blood cell count and routine electrolyte, hepatic and renal biochemical tests were within normal limits. The urinalysis was positive for protein and the sediment contained 8 to 15 red blood cells and numerous white blood cells per high-power field. A chest x-ray film showed a normal cardiothoracic ratio, a normal cardiac silhouette and clear lung fields. The ECG showed sinus rhythm, first-degree atrioventricular block with a PR interval of 0.26 s, complete right bundle branch block, anterior divisional block and transient atrial fibrillation with a ventricular rate of about 150 beats per minute. There were occasional paired premature ventricular contractions.

Because of paired premature ventricular contractions, therapy with aprindine (a class 1b antiarrhythmic agent) was begun on the second hospital day accompanied by ECG monitoring.

On the afternoon of the eighth hospital day, while sitting on the bed with his legs crossed, the patient suddenly had a premonition of syncope and began vigorous deep breathing. He called a nurse by pressing a nurse-call alarm button and said a few unintelligible

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Self-administered Hyperventilation Cardiopulmonary Resuscitation (Harada et al)
words over a loudspeaker communication system. The nurse noted that the ECG monitor revealed a cardiac arrest. When she arrived at the bedside, the patient was fully conscious, and she noted deep respirations with short straining at each end-expiration. This type of breathing continued for about 5 min. Conventional CPR was not performed. A temporary pacing catheter was inserted immediately and right ventricular pacing was started. The entire episode was recorded on a Holter ECG which was being used on that day. The recording from the Holter monitor showed that the ventricular arrest followed a pair of premature ventricular complexes, which occurred during an episode of atrial fibrillation at a rate of 150 beats per minute. The arrest continued for 100 s and ended with idioventricular rhythm at a rate of 88 beats per minute (Fig I). After this episode a permanent pacemaker was implanted, and there have been no more episodes of syncope during a follow-up period of one year.

**Discussion**

It is interesting that the patient remained fully conscious despite 100 s of ventricular arrest. In 1976, Criley et al reported that three patients undergoing coronary arteriography were successfully resuscitated by repeated coughing. They called this method "cough-induced CPR." Niemann et al reported four additional cases of cough-induced CPR in which coughing maintained consciousness for 92 s.

The mechanism of cough-induced CPR has been described as due to blood flow secondary to an increase in intrathoracic pressure. Several studies in dogs have shown that during cardiac arrest, chest compression produces equal rises in pressure in all intrathoracic structures but is not raised equally in all extrathoracic vessels. That is because venous valves at the thoracic inlet and perhaps venous collapse due to their thin walls causes a pressure gradient between the noncollapsible thick-walled carotid arteries and the thin-walled collapsible jugular veins. Therefore, forward flow into the systemic circulation can occur during the period of high intrathoracic pressure. Jugular venous flow into the chest occurs on release of the high intrathoracic pressure as airway pressures and intrathoracic vascular pressures decrease rapidly to atmospheric pressure. Blood then flows into the chambers of the right side of the heart and the lungs. In the human subject, two-dimensional echocardiograms have shown that the mitral and aortic valves remain open during the compression phase of resuscitation. Therefore, during periods of high intrathoracic pressures the chambers of the left side of the heart function only as a passive conduit. During periods of low intrathoracic pressure, the tricuspid and pulmonary valves are open and the chambers of the right side of the heart act as a passive conduit. The pulmonary valve may close during periods of high pressure and prevent retrograde flow from the lungs.

Our patient apparently performed vigorous, rhythmic respirations with short straining at end-expiration during his cardiac arrest. This forceful expiration against a closed glottis could produce a high intrathoracic pressure, simulating the effect of manual chest compression. Therefore, this is the first report of deep respiration-induced CPR and serves as a model of the pure "thoracic pump" mechanism.

We considered the possibility that the patient actually did lose consciousness and had retrograde amnesia due to the long duration of the cardiac arrest. However, there are several factors that make this unlikely:

1. There were three witnesses in the same four-bed ward who noticed nothing unusual in the patient's state of wakefulness at the time of arrest.
2. The nurse, after receiving his call for help, noticed the cardiac standstill on the nursing station monitor and was in the patient's room within 10 to 15 s, at which time she noted a conscious hyperventilation. Therefore, the patient could not have called for help after recovering from the cardiac arrest.

During the cardiac arrest, the Holter recordings showed complete ventricular standstill with only the f waves of atrial fibrillation. The ventricular arrest was apparently precipitated by a pair of premature ventricular contractions. Ventricular beats were resumed with an accelerated idioventricular rhythm, strongly suggesting that the ventricular arrest was due to advanced atrioventricular block.

It is commonly said that a transient advanced atrioventricular block is rare in cases of atrial fibrillation with a moderate ventricular rate. Rebello and Brownlee et al reported that intermittent atrioventricular block commonly
may occur in patients with controlled atrial fibrillation who complain of dizziness or syncope. Our patient probably had an incomplete trifascicular block (right bundle branch and anterior divisional block plus a long P-R interval) and aprindine might have depressed fascicular conduction and produced complete trifascicular block following the paired premature ventricular contractions.

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