A 29-year-old woman presented with severe refractory intraoperative wheezing and airflow limitation that resolved spontaneously. Contrast-enhanced computed tomographic (CT) scan of the thorax confirmed a right-sided aortic arch. Variable intrathoracic large airway obstruction that worsened markedly when the patient was in a supine position and slightly more following intravascular volume expansion was shown on flow-volume studies. We postulate the right-sided aortic arch caused airflow obstruction that worsened intraoperatively because of position and intravenous fluids.

(Chest 1991; 99:1308-10)

Right-sided aortic arch have symptoms from tracheal compression. Most reports are of children; case reports of right-sided arch causing symptoms in adults have been scarce. We present herein the case of a 29-year-old woman in whom severe intraoperative airflow obstruction developed because of a right aortic arch; moderate symptoms, in retrospect, had been present since childhood.

**Case Report**

A 29-year-old woman was referred for evaluation of severe wheezing and airflow obstruction that developed during administration of a general anesthetic for an orthopedic procedure. Wheezing, dyspnea, and airflow obstruction were refractory to nebulized albuterol, nebulized ipratropium, and intravenous aminophylline. These symptoms resolved gradually over several postoperative days with the patient receiving albuterol, ipratropium, aminophylline, and intravenous methylprednisolone sodium succinate (40 mg every 6 hours). At induction of anesthesia, the anesthetist reported the subjective feeling of a "square wave" inspiratory pressure curve.
Figure 2. Predicted (circles), seated, and supine (dotted) maximal expiratory and maximal inspiratory flow-volume curves with no fluid loading.

During the 2.5-hour procedure, 1,700 ml of fluid (1,000 ml of Ringer’s lactate solution and 700 ml of “4′-v′”) were administered intravenously; there was minimal blood loss (<100 ml). Intravenous fluids were administered at 150 to 200 ml/h for 48 hours after surgery.

When examined nine days after surgery, the patient was back to “normal” and was rapidly reducing the doses of inhaled prednisone and using inhaled albuterol intermittently. She gave a lifelong history of moderate wheezing with mild dyspnea during exercise and when in a supine position. She had no swallowing difficulty. Albuterol by inhalation did not improve or prevent wheezing. Symptoms were self-treated by avoiding strenuous exercise and by avoiding lying or sleeping in a supine position. Three general anesthetics (two for cesarean sections, and one for a choledectomy) had been administered in the previous seven years, the first associated with some “postoperative bronchospasm” (mild) and the other two uneventful.

Results of physical examination were normal except for audible inspiratory and expiratory wheezing heard only when in a supine position. Chest roentgenogram showed the absence of the usual left-sided aortic knob raising the suspicion of a right-sided arch; the thoracic spine was straight and the anteroposterior thoracic diameter was small. Contrast-enhanced computed tomographic (CT) scan of the thorax confirmed right-sided arch and showed marked narrowing of the trachea at the level of the carina (Fig 1). Lung volumes and spiromgrams suggested mild restriction. The FEV1 of 67 percent of expected and the FVC was 88 percent expected. Flow volume curves revealed a pattern consistent with variable intrathoracic airways obstruction. The Vmax50 was 45 percent of expected when the patient was seated and fell by 36 percent to 1.32 when the patient was supine (Fig 2). Results of a standardized methacholine inhalation test were normal, ruling out current asthma.

The patient underwent a closely supervised intravenous fluid challenge. One liter of sterile 0.9 percent NaCl was infused as 250-ml boluses as rapidly as possible (about 8 to 10 minutes each). Sitting and supine maximal expiratory flow volume curves were done before and after each bolus. Dyspnea, chest tightness, and audible wheeze increased only after the full 1,000-ml infusion (45 minutes) and only when the patient was in a supine position. At this time, Vmax50 had fallen by 20 percent when she was seated to 1.64 L/s and by 17 percent when she was supine to 1.09 L/s. Flow rates before and during the saline solution infusion are shown in Table 1.

**DISCUSSION**

Congenital anomalies of the great vessels (aortic and pulmonary trunks) are infrequent causes of tracheoesophageal compression. The classic anomaly is the double aortic arch that creates a complete vascular ring surrounding the trachea and esophagus. This causes difficulty swallowing and wheezing dyspnea, with both inspiratory and expiratory wheeze or stridor, and generally presents within the first year of life. Other less common malformations include anomalous origin of the innominate artery and the exceedingly rare anomalous origin of the left common carotid artery; various anomalies of the pulmonary arteries rarely cause tracheal compression.

A right-sided aortic arch, perhaps the most common major malformation of the great vessels, is usually asymptomatic. In normal individuals, the left aortic arch passes in front and to the left of the trachea and the descending aorta is connected to the pulmonary trunk by the ligamentum arteriosum. In the anomalous situation, the aortic arch develops from the right (rather than the left) fourth branchial

| Table 1—Flow Rates Before, During, and After Saline Solution Infusion* |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
|                 | FEV1, L         | FVC, L          | Vmax25, L/s     | Vmax50, L/s     | Vmax75, L/s     | PFR, L/s        |
| Seated          |                 |                 |                 |                 |                 |                 |
| Rest            | 2.17            | 2.83            | 2.97            | 2.05            | 0.84            | 4.34            |
| + 250 ml        | 2.03            | 2.58            | . . .            | . . .            | . . .            | 3.79            |
| + 500 ml        | 1.92            | 2.49            | . . .            | . . .            | . . .            | 3.53            |
| + 750 ml        | 1.95            | 2.59            | . . .            | . . .            | . . .            | 3.35            |
| + 1,000 ml      | 1.80            | 2.48            | 2.51            | 1.64            | 0.66            | 3.08            |
| Supine          |                 |                 |                 |                 |                 |                 |
| Rest            | 1.44            | 2.23            | 1.51            | 1.32            | 0.57            | 2.16            |
| + 250 ml        | 1.4             | 2.10            | . . .            | . . .            | . . .            | 2.07            |
| + 500 ml        | 1.35            | 2.02            | . . .            | . . .            | . . .            | 1.76            |
| + 750 ml        | 1.36            | 2.09            | . . .            | . . .            | . . .            | 2.11            |
| + 1,000 ml      | 1.24            | 1.83            | 1.29            | 1.09            | 0.53            | 1.90            |

*FEV1 = forced expiratory volume in 1 s; FVC = forced vital capacity; Vmax = forced expiratory flow when the lung is at 25 percent, 50 percent, and 75 percent (respectively) of its total lung capacity; and PFR = peak flow rate.
Self-Administered Hyperventilation Cardiopulmonary Resuscitation for 100 s of Cardiac Arrest during Holter Monitoring*

Yoshiaki Harada, M.D.; Hideo Fuseno, M.D.; Toshiyuki Ohtomo, M.D.; Yasuhiro Yamahara, M.D.; and Mitsuo Nakamura, M.D.

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)