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Right-sided Aortic Arch

To the Editor:

In the May 1991 issue of Chest, Bose et al report a case of chronic airway obstruction in a 29-year-old woman caused by a right-sided aortic arch. Investigations performed included chest radiography, computed tomography with contrast enhancement, and pulmonary function testing (with methacholine inhalation and intravenous fluid challenges). The authors speculate that the airway compression is due to a vascular "ring" formed by the right aortic arch (by definition coursing to the right of the trachea) and a "tight" ligamentum arteriosum (to the left of the trachea), and conclude that "surgical division of the ligamentum arteriosum should be curative." On the basis of the evidence in the report, these statements could be erroneous.

The adequate delineation of aortic arch anomalies and vascular rings must include not only the side of the arch but also the brachiocephalic vessel and ductus arteriosus/ligamentum arteriosum anatomy. This is highly variable, with specific clinical correlations.

The most common aortic arch malformation is a left aortic arch with aberrant right subclavian artery. Since all vascular structures (including the ligamentum arteriosum) are to the left of the trachea, no ring is formed, and associated symptoms are rare.

Less common are the right aortic arch malformations. Mirror-image brachiocephalic vessel anatomy is frequently encountered, and is nearly always associated with serious congenital heart disease. The ligamentum arteriosum usually connects the proximal left pulmonary artery and the subclavian portion of the left innominate artery, but in these cases the trachea is not surrounded by vascular or fibrous tissue, and no ring results. A vascular ring can be formed when the ligamentum courses posteriorly from the left pulmonary artery to a descending aortic diverticulum, but this is extremely rare.

The most likely cause of symptoms in the reported case is the combination of right aortic arch with aberrant left subclavian artery (which arises as the most distal branch of the arch). A left-sided ligamentum often connects this artery to the proximal left pulmonary artery, and a vascular ring is formed.

Our investigative approach to the patient with a potential vascular ring includes chest radiography, barium swallow study, and echocardiography. Complete anatomic information concerning the arch and brachiocephalic vessels is often obtained through careful ultrasound imaging from the suprasternal notch. Digital subtraction angiography is performed if further information is required.

Knowledge of the ligamentum and brachiocephalic vessel relationships is especially important in planning surgery. The tightness of the ligamentum is only one factor in the formation of a vascular ring. While simple division of the ligamentum is often curative in young patients, dissection of surrounding vascular structures may be necessary in adults. There can be significant degeneration of chronically compressed airway walls, and meticulous postoperative respiratory care is required. Complete symptom relief may take weeks.

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REFERENCES


To the Editor:

We agree with Dr. Giddins and his colleagues completely. With regard to our specific patient, the definitive study would seem to be angiography of the aortic arch and great vessels. Since the patient refused to entertain the idea of surgery, we felt that this procedure was not indicated at the time she was studied.

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Airway Obstruction by a Mucus Ball from a Transtracheal Oxygen Catheter

To the Editor:

We read with interest in the June 1991 issue of Chest the report by Burton et al on fatal airway obstruction due to mucus ball formation from a transtracheal oxygen catheter, apparently induced by transtracheal oxygen therapy (TTO2T).

We report the near-fatal course of mucus ball formation during TTO2T in a 28-year-old Turkish woman, resident in The Netherlands for 15 years. The patient had end-stage pulmonary disease due to pulmonary tuberculosis in childhood, which had been contracted

CHEST / 101 / 6 / JUNE, 1992 1739

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in Turkey; obstructive pulmonary disease and bronchiectasis had developed in later life. Since her arrival in The Netherlands, all sputum cultures had been negative for tuberculosis. She had been evaluated for lung transplantation in another hospital, but this treatment was considered impossible due to severe pleural thickening and calcification (Fig 1). She was referred to us because domiciliary oxygen treatment was impossible due to an extremely high oxygen demand.

On admission, the patient displayed dyspnea and cyanosis at rest, worsening during minimal exercise, and marked finger clubbing. There were coarse crackles over all lung fields, with amphoric breath sounds over the left side of the chest. Arterial blood gas analysis at rest with 12 L of oxygen per minute showed the following values: $P_{O_2}$, 35 mm Hg; $P_{CO_2}$, 56 mm Hg; sodium bicarbonate, 32 mmol/L; pH, 7.37.

Chest radiography, though hampered by tachypnea, revealed destruction of the left lung with severe honeycombing and similar destruction of the right upper lobe; the right lower lobe showed hyperinflation (Fig 1). Sputum production was approximately 100 mL/d. Sputum culture grew *Pseudomonas aeruginosa* in abundance. Ciprofloxacin was administered orally; the regimen was then changed to piperacillin plus tobramycin given intravenously preoperatively. A TTO$_2$I catheter (Johnson TTO$_2$I; Cook, Bloomington, Ind) was inserted with the use of combined local (xylocaine) and intravenous (ketamine) anesthesia without complications. Then TTO$_2$I was started, but the flow rate had to be reduced to less than 3 L/min because of tracheal irritation. A humidifier with acetylcysteine and sodium bicarbonate was used to facilitate sputum clearance.

On the third postoperative day, stridor was heard, and fiberbronchoscopy identified a mucus plug as the cause. Vigorous chest physiotherapy with intratracheal suction and use of an oscillating humidification device was started, resulting in the expectoration of a 5-cm-diameter cast of impacted mucus with a clearly visible hole in the middle, suggesting that the origin of the mucus ball was the area around the tip of the TTO$_2$I catheter. After expectoration of a second mucus ball, the catheter, which had failed to obviate her oxygen demand by face mask (proving nearly fatal on two occasions), was removed without complications.

The patient later died of a non-TTO$_2$I-related cause. Autopsy was refused on religious grounds.

We feel that, on the basis of our experience and that of others,14 TTO$_2$I is absolutely contraindicated if there is substantial sputum production. Catheter types that are subcutaneously tunnelled are even more disadvantageous, since they cannot be cleaned. Therefore, use of TTO$_2$I should remain limited, instead of being freely propagated.1

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**To the Editor:**

As Burton et al noted in their article,1 few serious and no fatal complications have been associated with the use of TTO$_2$I. Recently, we reported our experience in 40 subjects followed up for a total of 629 months while using the SCOOP transtracheal catheter (Transtracheal Systems, Denver).5,6 All subjects produced mucus balls. Most (75 percent) were able to cough them up with use of the standard cleaning technique and therapeutic cough. However, 25 percent developed symptomatic mucus balls that required catheter removal over a wire for resolution (stripping). As has been noted by others,1,2,4 these subjects developed mucus balls when there was no known lapse in catheter cleaning technique or use of humidification.

To prevent large mucus ball accumulation, we monitor all patients by phone during the initial two to three weeks of TTO$_2$I. If they report symptoms of potential mucus accumulation (whistling sound from the humidifier, sudden pop when oxygen tubing is disconnected, increasing or severe cough, dyspnea, or fluid in the tubing), we schedule a clinic visit to strip the catheter. For subjects at high risk (≥4 L/min at rest, large sputum volume, poor cough), we use routine prophylactic stripping, as advocated by Spofford and Christopher,2 in addition to irrigation three or four times a day and humidification. Although the majority of TTO$_2$I patients return to the clinic for the stripping procedure, refractory patients may find

**FIGURE 1.** Posteroanterior chest radiograph depicts severe pleural thickening and calcification, destruction of the left lung with severe honeycombing, similar destruction of the right upper lobe, and hyperinflation of the right lower lobe.