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An Unusual Cause of Sudden Complete Airway Obstruction

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The causes of subacute or acute partial or total occlusion of the airways are numerous and generally well known. However, we recently encountered an unusual case, which is described below.

REPORT OF A CASE

A 5½-year-old boy who was severely cyanotic, with transposition of the great arteries and pulmonary hypertension, was scheduled for a Baffes' procedure [1] because of deterioration of his condition and gradual increases in hemoglobin (24.6 g/100 ml) and hematocrit (71 per cent). At the age of 2½ months, a Blalock-Hanlon procedure [2] had been performed successfully. Since the pulmonary vascular resistance was practically equal to the systemic vascular resistance, total correction [3] of the abnormality was considered impossible. Cardiac catheterization two months earlier had disclosed the pressures and hemoglobin saturations shown in figure 1. The pressures in systemic and pulmonary vessels were nearly equal, in the absence of a ventricular septal defect. Angiocardiograms confirmed the diagnosis of transposition of the great arteries without ventricular septal defect or pulmonary stenosis. An open ductus arteriosus could not be demonstrated.

After induction of anesthesia, the trachea was intubated and the patient was connected to an Engström respirator. Breath sounds were audible over both lungs, slightly less on the left than on the right. This did not change after readjustment of the endotracheal tube. The right pulmonary artery, right main bronchus, and inferior vena cava were dissected through a right posterolateral incision. On proceeding, we found that we could not cross-clamp both the right pulmonary artery and the right main bronchus, as ventilation then became totally impossible. Cardiac arrest occurred. After removal of the clamp the patient was successfully resuscitated.

Since these events might have been caused by inadvertent right endobronchial intubation (although it was unlikely, since after removal of the clamp breath sounds were audible over the left hemithorax), the endotracheal tube was withdrawn under direct vision and immediately reinserted, the tip now being about 1 cm below the vocal cords. Breath sounds again were audible over the left lung, and the right lung was seen to ventilate well. Once again, an attempt was made to cross-clamp the right pulmonary artery and the right main bronchus at the same time. Again, it immediately became impossible to ventilate the lungs, and the clamp was again removed.

The presence of blood draining from the site of operation into the left main bronchus could have been the causative factor (a very small amount of mucosanguineous secretion was aspirated from the endotracheal tube), but good breath sounds were audible over the left lung and remained so when only the right main bronchus was occluded. This indicated passage of gases through the left main bronchus. Cross-clamping of the right main bronchus only was well tolerated by the patient, but when the right pulmonary artery was cross-clamped as well, the breath sounds on the left disappeared. This effect could be reproduced. Unfortunately a large tear occurred in the right pulmonary artery. Massive hemorrhage followed, with intractable cardiac arrest from which the patient could not be resuscitated in spite of vigorous efforts.

At autopsy the pertinent findings were: 1) transposition of the great arteries; 2) extreme dilatation of the pulmonary trunk, especially the right branches; 3) narrow aortic arch; 4) patent ductus arteriosus; 5) pulmonary vasculopathy, consisting of intimal fibrosis (right more than

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left), duplication of elastica interna (right more than left), and organized thrombi (left more than right). No indication of decreased patency of the left main bronchus was found.

Discussion

Analysis of the operative events made it evident that cross-clamping the right pulmonary artery had led to complete obstruction of the left main bronchus. This might seem strange, considering the anatomic relations of the normal hilar structures (fig. 2), but it is not an unreasonable event if one considers the changes in the anatomic relationship of the various hilar structures owing to pulmonary hypertension with dilatation of the pulmonary arterial system (fig. 3).

While the pathogenesis of the pulmonary hypertension in this patient need not be discussed, the role of persistent polycythemia in patients with congenital cyanotic cardiac defects leading to intravascular thrombosis and consequent diminution of the total pulmonary vascular bed should be mentioned.[4] The invariable presence in the pulmonary arterial vasculature of severe intimal fibrosis in patients more than 2 years old who have transposition of the great arteries and pulmonary hypertension[5,6] should also be mentioned, since these abnormalities were also found at autopsy in our case.

As indicated in figure 3 (modified after Stanger),[7] the gross anatomic changes in the left hilum due to pulmonary hypertension and dilatation of the pulmonary arterial system are (arrow *a*) compression of the anterosuperior area of the left main bronchus where it crosses the stem of the left pulmonary artery, and (arrow *b*) compression of the posterior wall of the left upper lobe bronchus where it is caught between the pulmonary artery supplying the left lower lobe and the last portion of the main left pulmonary artery. Compression of the left side of the trachea owing to craniomedial shift of the aorta by the dilated pulmonary arterial system (fig. 3, arrow *c*) is another possibility which may be revealed by bronchography. Although the compressive action of these anatomic changes on ventilation occurred in the left lung in our case, the same may hold true for the right lung in the absence of transposition of vessels in certain patients, such as those in whom partial obstruction of a bronchus may lead to lobar emphysema and total obstruction to atelectasis after sudden cross-clamping of a pulmonary artery. This maneuver will naturally cause a sudden decrease in the outflow drainage of pulmonary arterial blood, which invariably will cause further increases in pressure in the main and left pulmonary arteries. This, in turn, may cause further distention of these vessels, leading to further compression of the bronchial structures. Complete obstruction may occur, as in our case. This is the more likely to happen if there already seems to be a preferential flow of blood to the right lung with increased pulmonary vascular obstruction to the left lung. These factors were both present in our patient.

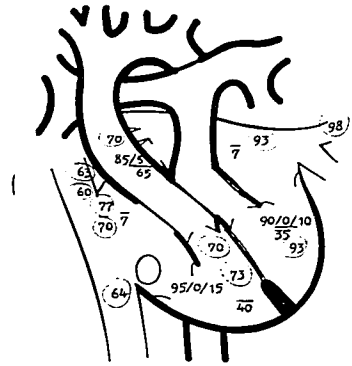

Fig. 1. Data obtained by cardiac catheterization, indicating nearly equal pressures in the ventricles in the absence of a ventricular septal defect and of pulmonary stenosis. Encircled figures indicate hemoglobin saturations; the other figures indicate pressures.

The patent ductus arteriosus, not clinically recognized by us, certainly was an important factor in the development of pulmonary hypertension and pulmonary arterial dilatation. Furthermore, the fact that a decreased lumen in the left main bronchus was not readily demonstrable at autopsy does not rule out the possibility of bronchial compression during life, since the static conditions of the former are not comparable to the dynamic conditions

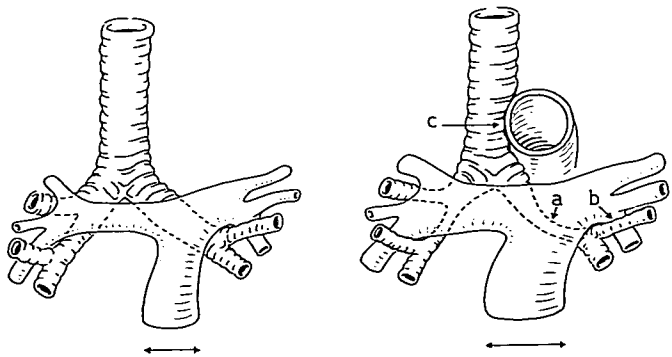

FIG. 2 (*left*). Interrelationship of bronchial and pulmonary arterial structures in the hilar areas under normal circumstances.

FIG. 3 (*right*). Anatomic changes in hilar areas in pulmonary hypertension and subsequent dilatation of pulmonary arterial system. Note compression of the main bronchi and the left-upper-lobe bronchus. Arrow *c* indicates indentation of the left lateral aspect of the trachea by the aorta secondary to craniomedial displacement of the aorta by the dilated pulmonary arterial system.

of the latter. In our patient, however, the caliber of the left pulmonary artery was smaller than that of the right pulmonary artery, and diffuse organized pulmonary vascular thrombosis was more pronounced in the left than in the right lung. These factors suggest that there must have been preferential flow of blood to the right lung. Occlusion of this relatively large flow by cross-clamping the right pulmonary artery must have dilated the left pulmonary artery enormously to obstruct the left main bronchus completely.

If complete cross-clamping of the right hilar structures is surgically necessary in such a case, the anesthesiologist could attempt to introduce a much smaller endobronchial tube into the left main bronchus prior to surgical cross-clamping of the right pulmonary artery. In children the technique certainly is not simple, and it would be largely a matter of trial and error to determine the size of the endobronchial tube. This approach to the problem seems to be the only one theoretically feasible. The danger of endobronchial hemorrhage with this technique is real in such a patient with an abundance of hypertrophied vessels of the bronchial circulation.

SUMMARY

A case of acute complete obstruction of the left main bronchus after cross clamping of the right pulmonary artery in a patient with a transposition of the great arteries and pulmonary hypertension is presented. A technique to circumvent this difficulty is suggested. Changes on chest roentgenograms suggestive of the presence of these abnormalities are described.

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Unusual Failure of an Oxygen Flowmeter

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Erratic behavior in the action of the oxygen flowmeter was noticed in a Model 64-134 anesthesia machine manufactured by the Chicago Anesthesia Equipment Company. At indicated oxygen flow rates of less than 4–5 l/min, the rotometer bobbin behaved normally. When the flowmeter needle valve was adjusted to increase oxygen flow rates, the flowmeter bobbin bounced up and down erratically. A delivered flow of more than 6–8 liters could not be obtained consistently.

The flowmeter was dismantled and the rotometer tube cleaned with ether. The needle valve was also checked for dirt or obstruction. However, after reassembly and leveling, there was no improvement in the behavior of the flowmeter. It was then apparent that there must be some obstruction distal to the oxygen flowmeter. The metal couplings and tubing leading from the flowmeter were sequentially disassembled. At the first metal coupling connecting the output of the oxygen flowmeter to the internal copper tubing system, a small metal sphere resembling a BB shot was found. The origin of this foreign body was not immediately apparent. However, it was evident that the shot was acting as an intermittent ball valve. At low oxygen flow rates, it would allow the stream of oxygen to pass. At higher flow rates, the turbulence of the gas in the coupling would propel the shot into the central gas stream, where it would impinge upon the central opening in the tubing coupling. This opening was too small to permit the shot to pass through it, but did act as an effective valve seat.

In an attempt to locate the source of this foreign body, the other flowmeters on the same machine were disassembled. Other rotometer bobbins were found to have identical shots cemented within their hollow interiors. Figure 1 is a photograph of the bobbin-sepa-

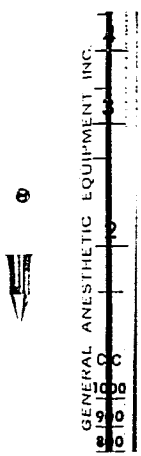

FIG. 1. Exploded view of the bobbin and separated shot.

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