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Increased Pulmonary Artery Pressure Related to Sternal Retraction

To the Editor:—The pulmonary circulation is a highly distensible, low-pressure, and low-resistance system that can accept substantial increases in blood flow with only small changes in pulmonary artery pressure.[1,2] Pulmonary vascular resistance may increase in response to a variety of stimuli, including hypoxia, hypothermia, hypercarbia, and acidosis.[3,4]

A 50-yr-old woman was admitted to the hospital because of fatigue, angina, and dyspnea on exertion. Cardiac catheterization revealed a heavily calcified bicuspid 0.3-cm^2 aortic valve, a mean left ventricular–aortic systolic pressure gradient of 78 mmHg, and a left ventricular ejection fraction of 76%. The patient was scheduled for aortic valve replacement.

In the operating room, an oximetric pulmonary artery catheter, placed through the left internal jugular vein, revealed a mixed-venous hemoglobin oxygen saturation ($S\bar{v}_{O_2}$) of 78%, a cardiac index of 2.2 $l \cdot min^{-1} \cdot m^{-2}$ and a pulmonary artery pressure of 36/18 mmHg. Anesthesia was induced with intravenous fentanyl 50 $\mu g \cdot kg^{-1}$ and midazolam 5 mg. Atraumatic tracheal intubation with a 7.5-mm cuffed tube followed the administration of 10 mg vecuronium bromide. Heparin 20,000 U was administered and cardiopulmonary bypass initiated. The aortic valve was replaced with a 19-mm valve (St. Jude Medical, Inc., St. Paul, MN), with a cross-clamp time of 97 min.

The patient was separated from cardiopulmonary bypass after 2 h while dobutamine 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ was administered. Her blood pressure was 80/40 mmHg and paced heart rate 90 beats per min. Pulmonary artery pressure immediately after bypass was 44/15 mmHg, cardiac index 2.5 $l \cdot min^{-1} \cdot m^{-2}$, and $S\bar{v}_{O_2}$ 83%. Heparin was antagonized with 200 mg protamine given slowly over 10 min. The activated clotting time was 133 s. Twenty minutes after bypass was terminated, the pulmonary artery pressure gradually increased to equal or exceed the systemic arterial pressure of 80/40 mmHg. $S\bar{v}_{O_2}$ was 82–85% (table 1), central venous pressure 10–12 mmHg, *p*Ha 7.34, Pa_{O_2} 497 mmHg, and Pa_{CO_2} 43 mmHg at the time of the increase in pulmonary artery pressure. Visualization revealed normal cardiac contraction, no evidence of right ventricular distension, and no pulmonary torsion.[5] Transducers were checked and recalibrated, and a needle was inserted in the left pulmonary artery to confirm pulmonary artery pressure measurement of 80/60 mmHg. The chest roentgenogram confirmed the pulmonary catheter to be in the right pulmonary artery. The sternal retractor was loosened and removed, and the pulmonary artery pressure immediately decreased to 44/18 mmHg. The patient recovered without further difficulty and was discharged from the cardiac intensive care unit 2 days later.

Direct measurement of pulmonary artery pressure confirmed pulmonary hypertension and precluded measurement artifact. The possibility of a pulmonary hypertensive reaction to protamine was considered but ruled out because enough time had elapsed since the protamine had been administered. Fortunately, right ventricular failure did not occur, as has been reported following allergic reaction to protamine.[6–8] In addition, $S\bar{v}_{O_2}$ and cardiac index remained within acceptable ranges (table 1), which we were unable to explain.

We determined that the elevation of pulmonary artery pressure was caused by the sternal retractor, which may have been opened unusually wide while attempting to gain hemostasis. Stretching and compression of the left atrium or of the pulmonary veins may have compromised pulmonary venous return and caused elevation of the pulmonary artery pressure. In addition, a reaction to protamine was unlikely because this drug was given slowly, and the elevation of pressures occurred later. Hence, when other possible causes of unexplained elevation of pulmonary artery pressures—such as hypoxia, hypothermia, hypercarbia, acidosis, light anesthesia, and drug reaction—have been eliminated, then loosening of the sternal retractor should be considered.

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TABLE 1. Results of Hemodynamic Measurements

	Before Bypass	After Bypass		No Retraction
		Initial	20 min	
Pulmonary artery pressure (mmHg)	34/16	44/15	90/40	44/18
Systemic blood pressure (mmHg)	100/60	80/50	80/40	80/60
Cardiac index ($l \cdot min^{-1} \cdot m^{-2}$)	2.5	2.9	3.0	3.1
$S\bar{v}_{O_2}$ (%)	85	82	83	85
Heart rate (beats)	55	90	90	90
PET_{CO_2} (mmHg)	30	30	30	30

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An Aid in the Diagnosis of Malpositioned Double-lumen Tubes

To the Editor:—We have found that the use of continuous spirometric monitoring, such as that provided by the Datex Capnomac Ultima™ to be a valuable aid in detecting the incorrect positioning of double-lumen endobronchial tubes. Such tubes are commonly used during intrathoracic surgical procedures. Unfortunately, these tubes are often placed incorrectly or may be displaced during patient positioning and surgery.

At our hospital, disposable endobronchial tubes (Sheridan and Rusch) require readjustment using fiberoptic bronchoscopy during 24% of thoracic operations.[1] The use of spirometric monitoring may diagnose an incorrect tube position before a significant clinical event occurs. The Datex monitor uses an in-line sensor at the endotracheal tube connector that incorporates pressure and flow (*via* a Pitot principle) measurements, and gas sampling. Continuous pressure–volume (P–V) or flow–volume ($\dot{V}$–V) loops may be displayed and compared to a loop stored in memory. Expiratory flow obstruction, such as occurs with distal placement of the endobronchial tube, is detectable by a change in the shape of the expiratory limb of the $\dot{V}$–V loop. A leaking endobronchial cuff is detectable as an increased difference between inspiratory and expiratory tidal volumes. Inspiratory obstruction, such as a kinked tube, is best seen as changes in the inspiratory limb of the P–V loop. The following example illustrates the utility of this new monitor.

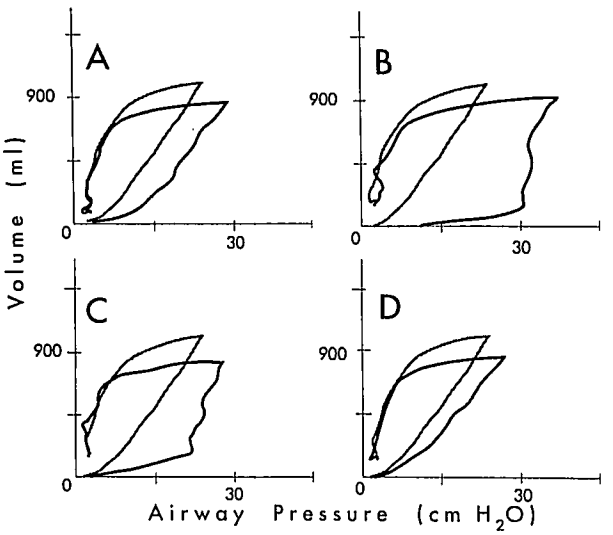

FIG. 1. Pressure–volume loops during two-lung (stippled curve) and one-lung (solid curve) ventilation. *A*: Initial. *B*: Obstruction. *C*: Decreased obstruction as tube is withdrawn. *D*: Return to control state with further withdrawal.

Following insertion of a left-sided endobronchial tube, a 65-yr-old man was placed in the left lateral decubitus position for a right lower lobectomy. Figure 1A shows normal P–V loops for two-lung (stippled curve) and left-lung (solid curve) ventilation. The same two-lung trace is used for comparison in figures 1B–1D. Late in the case an increase in peak pressure was noted, with the waveform shown in figure 1B. This P–V waveform is characteristic of an obstruction that is relieved at greater than normal inspiratory pressure. The $\dot{V}$–V loop (not shown) was unchanged. Fiberoptic bronchoscopy verified that the tip of the endobronchial tube was impacted against the airway wall. Figures 1C and 1D show the effect of slowly withdrawing the endobronchial tube. Figure 1C shows a decrease in peak pressure but persistence of the abnormal waveform; figure 1D shows a return to the normal shape. Of particular importance is the observation in figure 1C that the P–V loop showed an obstructive pattern despite only a slight increase in peak pressure; this degree of obstruction would not have been diagnosed by traditional methods.

It has been our experience since working with this device that subclinical malpositions occur much more frequently than we previously expected, with both left- and right-sided tubes. Early detection of endobronchial tube placement problems may improve the safety of double-lumen tube use and obviate the need for frequent intraoperative flexible fiberoptic bronchoscopy to assess and correct tube position.

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