Pleurodesis and Silver Nitrate

To the Editor:

Vargas and coworkers (September 2000) have concluded that intrapleural silver nitrate was more effective than talc in producing a pleurodesis. Bouros and coworkers (September 2000) have commented that silver nitrate could become the sclerosant of choice, given its wide availability and inexpensiveness.

Brock, in 1948, advocated the production of a chemical pleuritis by injecting into the pleural cavity, through an intercostal tube, 5 to 10 mL of a weak solution (2%) of silver nitrate followed by active suction on the drainage tube to rapidly reexpand the lung. This method was effective but had been condemned by some who maintained that it may have caused a fibrothorax.

Robert R. Shaw, MD, Professor of Thoracic Surgery during my cardiothoracic residency training at the University of Texas Southwestern Medical School at Dallas, TX, gave us his 107-page Surgery for Thoracic Disease: an Outline as a teaching guide, in 1969–1971. Under the tutelage of Dr. Shaw and Donald L. Paulson, MD, in Dallas, we had routinely used dry talc poudrage on the ward via 3/4-inch thoracostomy sites, using a combination of latex chest tubes and sterilized rigid bronchoscopes, for bedside talc poudrage. For highly selected cases, not only for pleural effusions but for classical causes responsible for atrial septal distortion: pneumoanea: a population study in Australian men. Am J Respir Crit Care Med 1995; 151:1459–1465
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Platypnea-Orthodeoxia Syndrome

A Probably Underestimated Syndrome?

To the Editor:

We have read with interest the observation of Acharya and Kartan (September 2000) concerning a case of platypnea and orthodeoxia because of interatrial shunting. As reported by the authors, many causes can be responsible for this syndrome despite no interatrial shunting. We would like to focus on patients with this syndrome and interatrial shunting.

In our experience, we collected 18 patients (mean ± SD age, 71 ± 6 years; range, 59 to 79 years) with similar symptoms. The patients had variable dyspnea and/or hypoxemia, but postural character was present in only 55% of them. In fact, this may be underestimated or difficult to diagnose when patient cannot be weaned off the ventilator. All of them had a persistent foramen ovale (PFO) and/or small atrial septal defect (ASD) with normal right-sided pressures, and symptoms occurred in only two patients after right pneumonectomy.

In our opinion, the interatrial shunting clearly results from a combination of both hemodynamic and flow phenomena. The hemodynamic phenomenon is interatrial pressure gradient favoring a right-to-left shunt across the ASD during early diastole. Mean right atrial pressure equal to or lower than the mean left atrial pressure has also been reported. In fact, a significant amount of blood flow from the inferior vena cava could probably cross a PFO by kinetic energy without the need of a favorable pressure gradient between the right and left atria. In the same way, decreased right ventricular compliance could produce the same effect. The second aspect is a flow phenomenon, which seems to be essential in our experience. This is well demonstrated by injection of contrast material in the inferior vena cava just at the junction, with the right atrium showing a complete filling of the left atrium through a displaced atrial septum. There are classical causes responsible for atrial septal distortion: pneumoanea, lobectomy, and kyphoscoliosis. In our experience, the most frequent cause was enlargement of the aortic root, which is physiologic with age, or can be also observed with an aortic aneurysmal condition. The consequence is horizontalization of the atrial septum, placing the PFO directly on line with the blood flow from the inferior vena cava. A similar flow phenomenon can
occur with the existence of PFO and a large eustachian valve or with high ASD close to the superior vena cava. The amount of shunting and symptoms are dependent on the hydration state. Another aspect of the interatrial shunting is the possibility of paradoxical embolism and/or stroke in this population, as observed in the history of 28% of our patients.

In fact, diagnosis can be erratic and usually pulmonary embolism is suspected. Breathing 100% oxygen is good test, showing usually insufficient rise in the arterial oxygen pressure. Then, the most powerful and simplest examination is contrast transesophageal echocardiography showing the interatrial defect and the right-to-left shunting, sometimes only after Valsalva maneuvers. In the observation, the therapeutic option chosen was surgical closure of the interatrial defect. We clearly recommend transcatheter PFO/ASD closure, as it was performed successfully in all our patients but one. The procedure is safe, simple, and effective without need of general anesthesia. Moreover, a significant number of patients are debilitated or carry a high or prohibitive risk for surgical correction.

We hope that publication of this observation in CHEST will heighten awareness of platypnea-orthodeoxia syndrome. Postural change in dyspnea and/or hypoxemia should lead to transesophageal echocardiography to depict the interatrial right-to-left shunt and to propose subsequent transcatheter closure as the procedure of choice for relief of platypnea-orthodeoxia. In fact, the true incidence of this syndrome remains to be stated, taking into account the high prevalence of PFO in the normal population, and a registry of this condition would be of interest.

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