Interatrial Right-to-Left Shunting in the Absence of Elevated Right-Sided Pressures After Major Trauma

To the Editor:

It was with considerable interest that I read the recent report of Smeenk and Postmus\(^1\) describing 23 patients who, after pulmonary resections, developed hypoxemia secondary to right-to-left (R-L) shunting in the absence of elevated right-sided heart pressures. Several years ago, I was responsible for the care of an individual with a similar problem.

This 40-year-old white man was in excellent health before the night of admission when he was involved in a severe motor vehicle accident resulting in multiple fractures (Pelvic, tibia, fibula, ribs, and femur), flail chest, pulmonary contusion, respiratory failure, and head trauma. During his prolonged hospitalization, the patient’s medical problems gradually resolved with the exception of persistent and severe hypoxemia. Initially, it was thought that the hypoxemia was a result of atelectasis and pulmonary contusion. Two months after the accident, however, the patient continued to have persistent dyspnea that was worse in the upright position, and decreased exercise tolerance and hypoxemia requiring supplemental oxygen. Therefore, the patient underwent further evaluation. Physical examination at that time revealed an alert patient in no acute distress. His head, ears, eyes, nose and throat examination was normal. His lungs were clear to auscultation and percussion with no wheezing, rhonchi, or rales. Cardiovascular examination was normal with no murmurs or gallops. Abdominal examination was benign and there was no clubbing, cyanosis, or edema. With the exception of multiple healed rib fractures, the chest radiograph was normal. Electrocardiogram was normal. Routine laboratory studies including a CBC were normal. Pulmonary function tests were normal except for mild obstructive disease and a mild decrease in the diffusing capacity. Chest computed tomography scan was unremarkable. Arterial blood gases on room air revealed a pH of 7.48, a Po\(_2\) of 48, a Pco\(_2\) of 28, a bicarbonate of 21, and a saturation of 87 percent. Multiple blood gases confirmed severe hypoxemia on room air. Arterial blood gases on 100 percent oxygen while the patient was supine revealed a Po\(_2\) of 124 while the Po\(_2\) was only 92 while the patient was in the sitting position.

Ventilation and perfusion scan of the lungs was normal with no evidence of embolic disease or early appearance of counts over the kidneys. Pulmonary angiography was normal with no evidence of emboli. Mean pulmonary artery pressure was 6. Contrast echocardiography showed a R-L shunt at the atrial level. Cardiac catheterization revealed a right atrial pressure of 5, a right ventricular pressure of 16/9, and a pulmonary artery pressure of 10/2 with a mean of 5. Pulmonary capillary wedge pressure was 8. Left atrial pressure was 7, left ventricular pressure was 126/10, left ventricular end diastolic pressure was 9, and aortic pressure was 120/80 with a mean of 97. Cardiac index was 3.6 L/min/m\(^2\).

All chambers, and cardiac valves were normal. Coronary arteries were also normal. There was evidence of a R-L shunt at the atrial level with a ratio of 1.3 to 1. Subsequently, the patient underwent thoracotomy, and a 1 cm atrial septal defect was located and closed with a Gore-Tex patch. At the time of surgery, there were no unusual atrial abnormalities that could account for the R-L shunting despite normal right-sided pressures. Postoperatively, the patient did exceptionally well with a return to his previous strenuous occupation as a laborer. He was able to discontinue supplemental oxygen and his room air arterial blood gases revealed a Po\(_2\) of 76. He had a Po\(_2\) of 492 while breathing 100 percent oxygen. He has not experienced any difficulties in the past 4 years since his surgery.

This is a unique and interesting case in many respects. This previously healthy patient had platypnea and orthodeoxia (dyspnea and arterial desaturation accentuated by the upright position) following major trauma. Platypnea-orthodeoxia almost always is reported in association with major pulmonary disorders such as after resectional surgery, after pulmonary embolic disease, with a paralyzed diaphragm, or with parenchymal lung disease. This patient did not have evidence of any chronic lung disorder, and trauma represents an intriguing predisposing factor for the development of platypnea and orthodeoxia. This case is also unique in that it describes a patient with a R-L shunt through an atrial septal defect with normal right-sided pressures after major trauma. Although it is difficult to understand the pathophysiology, it seems likely that this patient did not shunt from right to left until after his severe motor vehicle accident. Despite working at a very strenuous construction job, he had not noted any symptoms of dyspnea before the accident. Although it is possible to speculate that the motor vehicle accident resulted in a traumatic pulmonary arterial-venous fistula contributing to his problem, it was not identified and the severe hypoxemia resolved after closure of the atrial septal defect. This case represents another clinical situation (major trauma) in which a R-L shunt can occur causing significant disability that can be reversed with surgical intervention.

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REFERENCE
1 Smeenk FWJM, Postmus PE. Interatrial right-to-left shunting developing after pulmonary resection in the absence of elevated right-sided heart pressures: review of the literature. Chest 1993; 103:528-31

To the Editor:

We would like to thank Dr. Pacht for his valuable comment on our article. In his comment, he describes a patient who developed a serious right-to-left (R-L) shunt in the absence of
elevated right-sided heart pressures through a patent foramen ovale after a major thorax trauma resulting in platypnea-orthodeoxia. Before this thorax trauma, his heart was asymptomatic, so it is not likely that serious R-L shunting at that time would be present. To my knowledge, this complication occurring after a major thorax trauma has not yet been reported in the literature.

It would be interesting to speculate why a R-L shunt developed in this particular patient. As we mentioned in our article (Chest 1993; 103:528-31) two explanations could be given for the development of a R-L shunt in the absence of elevated right-sided heart pressures after a pulmonary resection.

First, because of a shift of the mediastinum, anatomic relations could be altered resulting in a "preferential" flow from the inferior caval vein through the patient foramen ovale into the left atrium. Second, after resection of pulmonary tissue, the afterload of the right ventricle (RV) could be raised. This in turn could be responsible for elevation of RV end diastolic pressures, thereby lowering the compliance of the RV. It is known that in the presence of an opening in the atrial septum, flow across this septum will occur mainly during the diastole (during the contraction of the atria) and that the direction of this flow is determined by the difference in the afterload between the right and left atria, i.e., the difference in the compliance between the RV and left ventricle (LV). The direction of this flow will be toward the ventricle with the highest compliance (i.e., lowest afterload). In normal circumstances, the compliance of the RV will be higher than the compliance of the LV. In the presence of a patent foramen ovale, however, this will not result in a L-R shunt because of the valve mechanism. In circumstances (for example pulmonary resection) which lower the RV compliance enough, a R-L shunt through this patent foramen ovale could occur.

Reviewing the case report described by Dr. Pacht this second mechanism (i.e., lowering of the compliance of the RV) could be a possible explanation for the development of a R-L shunt through a previously asymptomatic patent foramen ovale in his patient. An indication for a low compliance of the RV in his patient is the somewhat high end diastolic RV pressure he found. Maybe the thorax trauma could have lead to a severe (right) myocardial contusion as well, eventually resulting in a lowering of the compliance of the RV and thereby explaining the development of a R-L shunt in this patient. It would be interesting to know whether echocardiography or serial measurement of myocardial enzymes or both were done in this patient which could confirm this theory.

We would like to stress that in every patient with an unexplained severe hypoxemia that a R-L shunt should be considered. A 100 percent oxygen test is a relatively simple and safe test to strengthen this (as was also shown in this case by Dr. Pacht). It is our belief that every pathophysiologic condition that could result in a lowering of the compliance of the RV can also result in a R-L shunt in the presence of a patent foramen ovale or atrial septum defect.

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REFERENCE

Polyarteritis Nodosa Presenting as Recurrent Myocardial Infarction

To the Editor:

Coronary involvement in polyarteritis nodosa (PAN) is usually silent.1-3 We report a case of PAN presenting as angina and recurrent myocardial infarction (MI). The atypical presentation and lack of vasculitic changes on catheterization led to a delayed diagnosis.

The patient was a 44-year-old man who presented with 2 days of increasing angina, after having been treated elsewhere 4 weeks previously for deep venous thrombosis and MI. On admission, cardiac enzyme values and ECG changes were compatible with anterolateral MI. Cardiac catheterization showed occlusion of the left anterior descending coronary artery, mild intimal irregularity in the circumflex and right coronary arteries, an ejection fraction of 52 percent, and no aneurysms.

The patient remained pain free with conservative treatment, but developed increasing numbness and weakness in the extremities. Angiography showed multiple renal and hepatic arterial aneurysms, consistent with PAN. He was treated with intravenous methylprednisolone.

Within days chest pain developed again with inferolateral transmural MI, and the patient died in cardiogenic shock. Postmortem findings included the vasculitis of PAN in many organs, including the heart and coronary arteries (Fig 1), with extensive recent and old MIs.

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REFERENCES
3 Griffith GC, Vural IL. Polyarteritis nodosa: a correlation of clinical and postmortem findings in seventeen cases. Circulation 1951; 3:481-91

Figure 1. Cross-section of the left anterior descending coronary artery showing disruption of the arterial wall, with necrotizing vasculitis and fibrinoid necrosis (hematoxylin-eosin; original magnification X140).