Postoperative Improvement in Blood Lactate Threshold during Exercise in a Patient With Pulmonary Arteriovenous Fistula*

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A 13-year-old female patient with a pulmonary arteriovenous fistula (PAVF) of 24 percent shunt fraction (SF) underwent resection of segments 8 and 9 of the left lower lobe. Three months after the operation, the shunt became insignificant and PaO2 was normalized. In terms of the maximum work rate, maximum VO2 and the VO2-blood lactate relationship, this patient showed a remarkable postoperative improvement in the exercise capacity.

(Chest 1993; 103:289-91)

CO = cardiac output; PAVF = pulmonary arteriovenous fistula; SF = shunt fraction

We report a patient with pulmonary arteriovenous fistula (PAVF), whose blood lactate threshold in the incremental exercise test improved remarkably after surgery.

CASE REPORT

A 13-year-old girl was referred to our hospital for consultation regarding an abnormal shadow on a chest roentgenogram. Although she complained of exertional dyspnea, her development was normal with 151 cm in height and 40 kg in weight. Bruit was audible on the left side of the chest, but there was no cyanosis, finger clubbing, or telangiectasia.

A pulmonary arteriogram (Fig 1) demonstrated a large PAVF in segment 9 of the left lower lobe.

The hemoglobin level was 15.6 g/dL. In the sitting position, pH was 7.462, PaO2 was 52.5 mm Hg, PaCO2 was 28.4 mm Hg, and SaO2 was 88.8 percent. The routine pulmonary function parameters were within the normal ranges, ie, VC was 89 percent; FEV1, was 96 percent, MVV/BSA was 73.6 L/min/m2, and Deo2 was 80 percent.

The incremental exercise test was performed on a bicycle ergometer (Monark, Sweden) at a pedal frequency of 50 rpm and the outputs shown below. Using an electronic/mechanical calibration device consisting of an electronic motor and a system of movable weights on an extended bar, the ergometer had been calibrated. A Swan-Ganz catheter was introduced into the pulmonary artery so that mixed venous blood was sampled for gas analysis. After 15 min of rest, the work rate was increased from 0 (unloaded) to 25, 37.5, 50, 67.5, and 75 W every 3 min until the breaking point. VO2 was measured by a computerized on-line breath-by-breath system (System RM-300, Minato Medical Science; Osaka, Japan). The VO2 over the last 1 min of each work rate was adopted, and the VO2 at the highest completed work rate was defined as the maximum VO2.

During the last 30 s of exercise at each work rate, arterial blood was sampled from a cannula in the radial artery for gas analysis and lactate determination. The serum lactate concentration was determined (Determiner LA kit, Kyowa Medex, Tokyo, Japan) by the oxidase method.

Prior to conducting the protocol, written informed consent was obtained from the patient and her guardians.

Cardiac output (CO) and right-to-left shunt fraction (SF) were calculated as follows:

\[
CO = \frac{\dot{V}O_2}{(CaO_2 - CvO_2)} \\
SF = \frac{(CvO_2 - CaO_2)}{(CvO_2 - CvO_2)}
\]

where CaO2 = arterial oxygen content, CVO2 = mixed venous oxygen content, and CvO2 = end-capillary oxygen content. The end-capillary oxygen tension was regarded as being identical to the alveolar oxygen tension.

Under the incremental workload, VO2 increased from 182 ml/min at rest to 1,002 ml/min at 50 W (Table 1). PaO2 did not change during exercise, while PaCO2 decreased to reach 18.0 mm Hg at the maximum work rate. Despite a gradual increase in CO, SF, which was 24 percent at rest, declined as the work rate increased.

On August 30, 1989, segments 8 and 9 of the left lower lobe were resected. The patient was discharged from the hospital on the 11th

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FIGURE 1. Pulmonary arteriogram showing a large arteriovenous fistula in segment 9 of the left lower lobe. A = pulmonary artery; V = pulmonary vein; F = arteriovenous fistula.

FIGURE 2. Relationship between oxygen uptake (\(\dot{V}O_2\)) and blood lactate concentration during incremental exercise test. Closed circles = before operation; open circles = after operation.
Table 1 — The Results of Preoperative and Postoperative Incremental Exercise Tests

<table>
<thead>
<tr>
<th></th>
<th>Rest*</th>
<th>Unloaded</th>
<th>25 W</th>
<th>37.5 W</th>
<th>50 W</th>
<th>67.5 W</th>
<th>75 W</th>
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<tbody>
<tr>
<td>VO₂, ml/min</td>
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<tr>
<td>Pre</td>
<td>182</td>
<td>553</td>
<td>650</td>
<td>810</td>
<td>1,002</td>
<td></td>
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<tr>
<td>Post</td>
<td>218</td>
<td>455</td>
<td>672</td>
<td>885</td>
<td>1,048</td>
<td>1,225</td>
<td>1,470</td>
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<tr>
<td>PaO₂, mm Hg SaO₂, %</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>52.5</td>
<td>(88.8)</td>
<td>52.8</td>
<td>(88.6)</td>
<td>50.1</td>
<td>(86.5)</td>
<td>51.1</td>
</tr>
<tr>
<td>Post</td>
<td>111.0</td>
<td>(98.1)</td>
<td>102.7</td>
<td>(97.7)</td>
<td>96.3</td>
<td>(97.1)</td>
<td>99.9</td>
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<tr>
<td>PVO₂, mm Hg (SVO₂, %)</td>
<td>26.2</td>
<td>(58.3)</td>
<td>23.4</td>
<td>(42.0)</td>
<td>20.7</td>
<td>(34.6)</td>
<td>19.2</td>
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<tr>
<td>Blood lactate, mmol/L</td>
<td></td>
<td></td>
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<tr>
<td>Pre</td>
<td>1.2</td>
<td>1.6</td>
<td>1.8</td>
<td>3.1</td>
<td>NA</td>
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<tr>
<td>Post</td>
<td>1.2</td>
<td>1.3</td>
<td>1.3</td>
<td>1.6</td>
<td>2.3</td>
<td>4.0</td>
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<td>CO₂, L/min</td>
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<tr>
<td>Pre</td>
<td>2.6</td>
<td>5.4</td>
<td>6.2</td>
<td>6.5</td>
<td>NA</td>
<td></td>
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<tr>
<td>Post</td>
<td>2.5</td>
<td>5.8</td>
<td>6.5</td>
<td>7.7</td>
<td>8.3</td>
<td>9.0</td>
<td>9.9</td>
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<td>SF, %</td>
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<td>1</td>
<td>1</td>
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*At sitting position. NA = not available; Pre = before operation; Post = three months after operation.

The postoperative day. The exertional dyspnea disappeared completely after the surgery.

Three months after the operation, PaO₂ was normalized. In the exercise test, she was able to tolerate up to a 75-W work rate, when the maximum VO₂ was 1,470 ml/min. PaO₂ was 87.3 mm Hg, and PVO₂ was 20.1 mm Hg (Table 1). The shunt became insignificant and SF did not exceed 4 percent.

The relationship between VO₂ and the blood lactate level is shown in Figure 2. The VO₂-blood lactate curve shifted markedly rightward after the operation, indicating improvement in the blood lactate threshold during exercise.

**Discussion**

In the present case, PaO₂, which was as low as 52.5 mm Hg (SaO₂: 88.8 percent) due to 24 percent SF preoperatively, was normalized with the diminution of the shunt after the operation. In addition, the postoperative exertional dyspnea disappeared completely.

Concerning the decrease in SF during preoperative exercise, we previously discussed this in another patient with PAVF: the pulmonary vascular resistance, except for in the fistula, may drop during exercise in response to increased CO by the mechanism of recruitment and distention of the pulmonary capillaries, while the resistance in the fistula may not change.

As to the reason for steady decrease in PaO₂ during postoperative exercise, we considered that some minor mismatching of V̇A/Q due to operative lung damage would still exist and so the blood flow to comparatively low V̇A/Q areas might increase as the work rate increased.

After removal or mechanical obstruction of PAVF, improvement in the exercise capacity has been reported inconstantly from the standpoint of prolongation of exercise duration, improvement in the maximum work rate, and increase in the maximum VO₂. The present case also showed improvement in the maximum work rate from 50 W to 75 W, and an increase in the maximum VO₂ from 1,002 to 1,470 ml/min. In addition, we evaluated the VO₂-blood lactate curve. This curve shifted markedly to the right side postoperatively, indicating an improvement in the blood lactate threshold. Although the blood lactate threshold is influenced by oxygen delivery to the working muscles as well as by the characteristics of the muscle fiber and the clearance of lactate from the blood, it is improbable in this patient that any factors other than the oxygen delivery had changed after operation. Oxygen delivery consists of three factors, i.e., CO, hemoglobin, and SaO₂. In this study, the postoperative CO was at the same level as the preoperative CO at the same level of VO₂, and the postoperative hemoglobin level, 14.8 g/dl, was rather lower than the preoperative level (15.6 g/dl). Consequently, the improvement in SaO₂ from 88.8 percent to 98.1 percent after surgery appeared to be the main factor that could explain the postoperative rightward shift of the VO₂-blood lactate curve. Our result is consistent with the reports of Hughes et al., Limnarsson et al., and Lundin and Ström who showed that the blood lactate formation during exercise is influenced by the concentration of inspired oxygen and consequently the oxygen saturation.

**References**

Diagnosis of Circumferential Dissection of the Ascending Aorta by Transesophageal Echocardiography*

Michel G. Farah, M.D.,† and Randeep Suneja, M.D.‡

A 28-year-old woman with Marfan's syndrome presented with chest pain; transesophageal echocardiography showed circumferential dissection of the ascending aorta. Both aortic angiography with digital subtraction and computed tomography scanning with contrast were negative for dissection. Circumferential dissection of the ascending aorta was confirmed by surgery at which time replacement of the aorta and aortic valve were performed. Transesophageal echocardiography may become the most practical and reliable procedure for the diagnosis of aortic dissection.

(Chest 1993; 103:291-92)

Acute aortic dissection is associated with a high mortality rate.1,2 Clinical diagnosis is usually difficult and early confirmation of the diagnosis in suspected patients is essential for improved survival. Although angiography is considered the gold standard to confirm such a diagnosis, noninvasive tests such as transesophageal echocardiography, computed tomography scanning, and magnetic resonance imaging may be as accurate yet less costly and more practical.3,4 We describe a patient with circumferential dissection of the ascending aorta detected by transesophageal echocardiography while angiography and computed tomography scanning failed to show evidence of dissection.

CASE REPORT

A 28-year-old woman was transferred to University Hospitals of Cleveland from another hospital for evaluation of shoulder and chest pain. She began to experience severe shoulder pain associated with severe diaphoresis and generalized weakness one day prior to hospital admission. On the morning of hospital admission, she also complained of pleuritic chest pain. Her mother had undergone aortic valve replacement for severe aortic regurgitation secondary to aortic aneurysm with Marfan's syndrome five years ago. Pertinent physical findings at the time of hospital admission included a tall woman with marfanoid features. Blood pressure was 90/62 mm Hg in the right arm and 110/60 in the left. Pulses were also diminished in the right arm. Auscultation revealed a grade 4/6 decreasing diastolic murmur heard best at the left sternal border. No abnormal diastolic sounds were present. The lungs were clear and resonant. Neither venous distention nor peripheral edema was present.

The electrocardiogram was normal. Chest roentgenogram was normal with no widening of the mediastinum. M-mode and two-dimensional echocardiography revealed the aortic root to be dilated to 6 cm with a possible intimal flap and mild aortic regurgitation. Mitral valve prolapse with mild mitral regurgitation was present. Left ventricular size and function were normal. Aortic angiogram with digital subtraction confirmed dilatation of the aortic root but no evidence of dissection was found. The coronary arteries were not visualized. Computed tomography of the thorax with contrast showed aortic dilatation but no evidence of dissection.

Transesophageal Echocardiography

Transesophageal echocardiography was performed using a 5-MHz transducer and an echograph (Sono 1000, Hewlett Packard) after sedation with midazolam. Imaging of the ascending aorta showed a large and very mobile intimal flap consistent with circumferential dissection (Fig 1). The flap had an S pattern of motion; the origin of the right coronary artery appeared to be involved in the area of the false lumen. No entry site was detected. The flap was seen all the way to the origin of the aortic arch; the descending aorta was normal. Mild aortic regurgitation was present.

Cardiac catheterization was performed but the coronary arteries could not be cannulated nor could the catheter be advanced into the left ventricle suggesting catheter presence in the false lumen. At operation, complete circumferential dissection of the ascending aorta from the aortic root to the origin of the innominate artery was confirmed with a large intimal tear. In addition, the dissection had involved the ostium of the right coronary artery with evolution of its origin. The ascending aorta was replaced with a 30-mm graft and the aortic valve was replaced by a St. Jude prosthesis. In addition, saphenous vein graft from the ascending aorta to the distal right coronary artery was placed. The patient was well at six months of follow-up. Pathologic findings confirmed circumferential dissection with cystic medial necrosis of the ascending aorta.

DISCUSSION

Trans thoracic two-dimensional echocardiography and Doppler echocardiography are helpful in the diagnosis of acute aortic dissection of the ascending aorta and may show the intimal flap in about 60 percent of the cases.1 False-positive and false-negative studies are reported and this conventional echocardiographic procedure is limited by its inability to visualize adequately parts of the ascending and descending aorta. It is very helpful in detecting complications of dissection such as pericardial effusion, cardiac tamponade, and aortic regurgitation.

Transesophageal echocardiography, using the transesophageal approach to imaging of the aorta, overcomes the above limitations because of the proximity of the aorta to the esophagus with excellent quality images. Transesophageal echocardiography, performed in the awake patient, involves