tages compared with the JOSTENT in cases of aneurysms with very long longitudinal axis that are located in vessels with angulations/tortuosities or in those with a large reference diameter, since the JOSTENT has higher flexibility, and is available in longer and larger sizes.

The present case was technically challenging due to the length of the aneurysm and its location after an angulation of the vessel, as well as the presence of two calcific stenoses proximally and distally. The combination of guiding catheter deep-intubation and buddy wiring finally allowed the positioning of the long Symbiot stent.

Despite the excellent angiographic and early clinical outcome of the procedure in this patient, questions remain concerning the use of this new PTFE-coated stent. The incidence of stent thrombosis is not known, and the optimal medical therapy to prevent complications following the use of the stent has not been well-defined. For this reason, we chose to use a very aggressive antithrombotic medical regimen following the placement of the long Symbiot stent.

Despite the excellent angiographic and early clinical outcome of the procedure in this patient, questions remain concerning the use of this new PTFE-coated stent. The incidence of stent thrombosis is not known, and the optimal medical therapy to prevent complications following the use of the stent has not been well-defined. For this reason, we chose to use a very aggressive antithrombotic medical regimen following the placement of this stent. More experience with the use of the Symbiot stent will allow the refinement of the medical regimen needed to assure good long-term results with the use of this stent.

CONCLUSIONS

The Symbiot stent may be successfully used to exclude large coronary aneurysms involving a long segment of the native coronary artery allowing for an expansion of the anatomic indications for percutaneous interventions in coronary aneurysms.

REFERENCES


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Functional Assessment of Pulmonary Vein Stenosis Using Radionuclide Ventilation/Perfusion Imaging*

Kumaraswamy Nanthakumar, MD; James M. Mountz, MD, PhD; Vance J. Plumb, MD; Andrew E. Epstein, MD; and G. Neal Kay, MD

Pulmonary vein (PV) stenosis following catheter ablation of atrial fibrillation (AF) is a new clinical syndrome. The optimal method of assessing this syndrome is not known. We evaluated radionuclide perfusion imaging, anatomic imaging, and direct measurements of PV-left atrial (LA) pressure gradients in patients suspected of having PV stenosis after catheter ablation for the treatment of AF. The study included 11 consecutive patients who were referred to a tertiary referral center for the evaluation of symptoms suggesting or imaging evidence of PV stenosis following catheter ablation for AF. All patients underwent anatomic imaging of their PVs with direct pulmonary venography or CT scanning as well as radionuclide perfusion imaging. PV stenosis (> 50% diameter) was diagnosed by venography in 6 of the 11 patients and in 16 of 44 PVS. All six patients with PV stenosis had perfusion defects in the af-

*From the Division of Cardiovascular Medicine (Drs. Nanthakumar, Plumb, Epstein, and Kay), University of Alabama at Birmingham, Birmingham, AL; and the Department of Radiology (Dr. Mountz), Division of Nuclear Medicine, University of Pittsburgh, Pittsburgh, PA.

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Correspondence to: K. Nanthakumar, MD, University of Alabama at Birmingham, 1670 University Blvd, B140 Volker Hall, Birmingham, AL 35294-0019; e-mail: kn@crml.uab.edu

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Figure 1. Right coronary angiogram before treatment (left, a), after implantation of the Symbiot stent, which did not cover the proximal edge of the aneurysm (middle, b), and after implantation of the JOSTENT followed by postdilatation of both stents (right, c).
fectected pulmonary lobe. In contrast, all of the patients without anatomic evidence of PV stenosis had normal perfusion. There were 14 PVs with stenoses of > 80% of the luminal diameter, all of which had a corresponding perfusion abnormality ascertained by perfusion scanning. In all 14 PVs with a resting PV-LA gradient of > 5 mm Hg, there was a corresponding perfusion defect. PV stenosis results in decreased perfusion in the affected lobe when the resting PV-LA pressure gradient is at least 5 mm Hg or when there is 80% luminal stenosis. A perfusion scan may serve as an effective screening tool for PV stenosis and may be most useful in assessing the hemodynamic significance of an anatomic PV stenosis.

(PCHEST 2004; 126:645–651)

Key words: atrial fibrillation; catheter ablation; perfusion imaging; pulmonary vein stenosis

Abbreviations: AF = atrial fibrillation; LA = left atrium, atrial; LLL = left lower lobe; LPO = left posterior oblique; LUL = left upper lobe; PV = pulmonary vein; RLL = right lower lobe; RPO = right posterior oblique; RUL = right upper lobe; V/Q = ventilation/perfusion

Pulmonary vein (PV) stenosis is a potentially devastating complication of catheter ablation for the treatment of atrial fibrillation (AF).1,2 This has recently been recognized as an emerging new clinical syndrome.2 The clinical manifestations of PV stenosis are quite variable, including chest pain, dyspnea, cough, hemoptysis, recurrent lung infection, and pulmonary hypertension.2,3 In other cases, PV stenosis may produce no symptoms.4,4 Although physicians performing catheter ablation have recognized that the PVs are vulnerable to thermal injury5 and strategies have been designed to limit the application of ablative energy to the ostium of the PVs,6–8 an increasing number of centers are now performing this procedure. Inasmuch as experienced centers have reported this complication, as this procedure disseminates in the community the actual prevalence of PV stenosis may rise due to the inherent learning curve. The exact incidence of PV stenosis after catheter ablation is unknown since the diagnosis is dependent on the imaging modality used to assess PV anatomy and physiology, and on the rigor with which patients are monitored on the imaging modality used to assess PV anatomy and physiology, and on the rigor with which patients are monitored.

The optimal method for screening patients after catheter ablation for the development of PV stenosis has not been determined. Stenosis of a PV may be assessed by both anatomic and functional imaging. A functional test that detects reduced perfusion to a segment of the lung is likely to add information to that provided by an anatomic image alone. Noninvasive diagnostic tests that assist in the diagnosis and functional characterization of the significance of anatomic imaging evidence of PV stenosis have not been evaluated.

Several centers have used either CT scanning or MRI for routine screening after PV ablation procedures.2,4,15 While either of these techniques provides information regarding PV and lung anatomy, they are not without limitations. For example, the resolution of these techniques depends on the intervals between scanned images as well as the angle in which the PVs are viewed. In addition, although an anatomic stenosis may be identified, the physiologic significance of a given lesion may be more difficult to determine. Transesophageal and intracardiac Doppler echocardiography provide means to measure PV flow velocity16,17 but are less specific than direct measurements such as PV venography. Thus, an imaging technique that is noninvasive and widely available, and that provides physiologic information regarding pulmonary blood flow would be ideal for the management of patients suspected of having PV stenosis. This report details the results of radionuclide ventilation/perfusion (V/Q) imaging in patients who are suspected of having PV stenosis, and correlates the perfusion defects with anatomic imaging and direct measurements of the PV-left atrial (LA) pressure gradient.

### Table 1—Patient Characteristics*

<table>
<thead>
<tr>
<th>Patient/Age, yr/Sex</th>
<th>Ablation</th>
<th>Time to Symptom</th>
<th>PV Stenosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/56/M</td>
<td>Linear</td>
<td>1 mo Cough</td>
<td>No</td>
</tr>
<tr>
<td>2/68/F</td>
<td>Segmental</td>
<td>1 wk Cough</td>
<td>No</td>
</tr>
<tr>
<td>3/64/F</td>
<td>Segmental</td>
<td>12 mo Exertional SOB</td>
<td>Yes</td>
</tr>
<tr>
<td>4/35/F</td>
<td>Linear</td>
<td>2 mo SOB</td>
<td>Yes</td>
</tr>
<tr>
<td>5/53/M</td>
<td>Linear</td>
<td>7 mo SOB</td>
<td>Yes</td>
</tr>
<tr>
<td>6/43/M</td>
<td>Segmental</td>
<td>1 wk Hemoptysis</td>
<td>Yes</td>
</tr>
<tr>
<td>7/46/M</td>
<td>Segmental</td>
<td>2 mo SOB</td>
<td>No</td>
</tr>
<tr>
<td>8/24/F</td>
<td>Segmental</td>
<td>1 mo Cough</td>
<td>No</td>
</tr>
<tr>
<td>9/65/F</td>
<td>Segmental</td>
<td>NA None</td>
<td>Yes</td>
</tr>
<tr>
<td>10/56/F</td>
<td>Segmental</td>
<td>4 mo Hemoptysis + cough</td>
<td>No</td>
</tr>
<tr>
<td>11/60/M</td>
<td>Segmental</td>
<td>NA None</td>
<td>Yes</td>
</tr>
</tbody>
</table>

*M = male; F = female; NA = not applicable; SOB = shortness of breath.

The optimal method for screening patients after catheter ablation for the development of PV stenosis has not been determined. Stenosis of a PV may be assessed by both anatomic and functional imaging. A functional test that detects reduced perfusion to a segment of the lung is likely to add information to that provided by an anatomic image alone. Noninvasive diagnostic tests that assist in the diagnosis and functional characterization of the significance of anatomic imaging evidence of PV stenosis have not been evaluated.

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![Figure 1](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/22013/ on 06/25/2017)
Materials and Methods

Study Population

The study population included 11 consecutive patients who were referred to the University of Alabama at Birmingham for evaluation of possible PV stenosis. These patients had symptoms or imaging evidence that was suggestive of PV stenosis after being treated for AF with catheter ablation (Table 1). AF had been paroxysmal in eight patients and permanent in three patients. All patients had received either radiofrequency catheter ablation to isolate the PVs (eight patients) or linear LA ablation to create conduction barriers linking the PVs (three patients). This study was approved by the Institutional Review Board for Research Involving Human Subjects at the University of Alabama at Birmingham.

Imaging of the PVs

Two different anatomic imaging techniques were used to evaluate the PVs. The PVs were imaged with spiral CT scanning in four patients and retrograde biplane pulmonary venography in eight patients. Transeptal access was obtained with standard techniques, and pulmonary venography was performed with hand injections of iodinated contrast via a 6F multipurpose angiographic catheter in each of the PVs during biplane cineangiography. The degree of PV stenosis was calculated as the ratio of the minimum diameter of the stenotic region compared with the diameter of the nearest normal segment of PV. PVs with stenoses of ≥50% were diagnosed to have PV stenosis for the purpose of this study. When possible, the simultaneous PV-LA pressure gradient was measured with two fluid-filled transducers that were simultaneously zeroed and calibrated using a 6F multipurpose angiographic catheter with side holes positioned distal to the stenosis in the PV and an 8F transeptal sheath in the LA positioned near the PV ostium.

Radionuclide V/Q Imaging

Ventilation images were first performed either after inhalation from a nebulizer containing 45 mCi 99mTc diethylenetriaminepentaacetic acid or after inhalation of 15 mCi 133Xe from an administration system (ie, with washing, equilibrium, and washout phases). Second, the perfusion lung scans were performed by the standard technique of obtaining six views (ie, anterior, posterior, left anterior oblique, left posterior oblique [LPO], right anterior oblique, and right posterior oblique [RPO]), after the IV injection of 5 mCi 99mTc macroaggregated albumin). The images were interpreted by a radiologist who was blinded to the diagnosis and the clinical symptoms.

To quantify the degree of the perfusion defect, the RPO and LPO projections of each lung were analyzed, since the major lobes (ie, right upper lobe [RUL], right middle lobe, right lower lobe [RLL], lingula, and left lower lobe [LLL]) could be separated from other lobes of the same lung, and from the counts from the contralateral lung, as illustrated in Figure 1. Using a normal perfusion scan, the uninvolved segment of lung or lobe was count-normalized to obtain an expected perfusion percent-

Table 2—Anatomic Stenosis and Perfusion Abnormality*

<table>
<thead>
<tr>
<th>Patient</th>
<th>Anatomic Imaging</th>
<th>RSPV</th>
<th>RIPV</th>
<th>LSPV</th>
<th>LIPV</th>
<th>RUL</th>
<th>RLL</th>
<th>LUL</th>
<th>LLL</th>
</tr>
</thead>
<tbody>
<tr>
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<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>2</td>
<td>Venography</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>3</td>
<td>Venography</td>
<td>80</td>
<td>N</td>
<td>50</td>
<td>80</td>
<td>Abn</td>
<td>N</td>
<td>N</td>
<td>Abn</td>
</tr>
<tr>
<td>4</td>
<td>Venography</td>
<td>80</td>
<td>100</td>
<td>90</td>
<td>90</td>
<td>Abn</td>
<td>Abn</td>
<td>Abn</td>
<td>Abn</td>
</tr>
<tr>
<td>5</td>
<td>Venography</td>
<td>90</td>
<td>80</td>
<td>90</td>
<td>80</td>
<td>Abn</td>
<td>Abn</td>
<td>Abn</td>
<td>Abn</td>
</tr>
<tr>
<td>6</td>
<td>Venography</td>
<td>100</td>
<td>N</td>
<td>100</td>
<td>N</td>
<td>Abn</td>
<td>N</td>
<td>Abn</td>
<td>N</td>
</tr>
<tr>
<td>7</td>
<td>Venography</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>8</td>
<td>CT scan</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>9</td>
<td>CT scan</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>90</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Abn</td>
</tr>
<tr>
<td>10</td>
<td>CT scan</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
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<td>N</td>
<td>N</td>
</tr>
<tr>
<td>11</td>
<td>Venography</td>
<td>60</td>
<td>N</td>
<td>80</td>
<td>N</td>
<td>Abn</td>
<td>N</td>
<td>Abn</td>
<td>N</td>
</tr>
</tbody>
</table>

*Abn = perfusion defect; N = normal perfusion; RSPV = right superior PV; RIPV = right inferior PV; LSPV = left superior PV; LIPV = left inferior PV.

Table 3—Pressure Gradient and Perfusion Abnormality*

<table>
<thead>
<tr>
<th>Patient</th>
<th>Mean Gradient, mm Hg</th>
<th>Decrease in Perfusion, %</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RSPV</td>
<td>RIPV</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>4</td>
<td>21</td>
<td>Occ</td>
</tr>
<tr>
<td>5</td>
<td>21</td>
<td>&gt;20</td>
</tr>
<tr>
<td>6</td>
<td>Occ</td>
<td>N</td>
</tr>
<tr>
<td>9</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>11</td>
<td>3</td>
<td>N</td>
</tr>
</tbody>
</table>

*NM = not measured (90% stenosis by CT scan); Occ = occluded PV preventing pressure measurement; Abn = Normalized to this abnormal segment. See Table 2 for other abbreviations not used in the text.
The degree of perfusion reduction was calculated as follows: (normal lobe perfusion/affected lung perfusion)/total lung perfusion. This yielded a percentage of lobar perfusion reduction that was correlated with location and the degree of PV stenosis, as measured on the pulmonary venogram, CT scan, and hemodynamic measurement. The counts were normalized to the most normal segment of the lung in patients with multiple lobar involvement. In patients with multiple perfusion defects, the reductions were relative to the most normal segment of the involved lobes.

**Results**

The clinical characteristics of the patients are shown in Table 1. Linear LA ablation was performed in three patients, and segmental PV isolation was performed in eight patients. Of the three patients evaluated with suspected PV stenosis who underwent linear ablation, two had stenosis and presented with shortness of breath. Of the eight patients who had PV isolation with segmental approach and were investigated for possible stenosis, four had PV stenosis. In this cohort of patients, the interval between catheter ablation and the evaluation for possible PV stenosis ranged from 1 week to 12 months. Symptoms were reported by four of six patients with significant PV stenosis. The anatomic and perfusion assessments of these patients are shown in Table 2. PV stenosis was diagnosed by direct PV venography in 6 of the 11 patients. In all six of these patients, lobar radionuclide perfusion defects were demonstrated in the affected region of the lung. None of the patients without PV stenosis had abnormal perfusion on V/Q scanning. In the 11 patients studied, 16 of the 44 PVs had anatomic evidence of stenosis. Of the 16 PVs with anatomic evidence of >50% stenosis, 14 PVs had stenosis of >80%, 1 PV had stenosis of 50%, and another PV had stenosis of 60%. All 14 PVs with stenosis of >80% had a corresponding radionuclide perfusion defect. There was no perfusion defect in any of 29 PVs.
with < 60% stenosis. The patient with the 60% stenosis did have a corresponding perfusion defect.

The relation of hemodynamic pressure gradient and perfusion imaging is shown in Table 3. Of the 16 veins with anatomic evidence of PV stenosis, 3 were occluded and in another the pressure gradient could not be measured. In all 13 PVs with a resting PV-LA gradient of > 5 mm Hg, there was a corresponding perfusion defect ascertained by nuclide imaging. Only one patient with a resting PV-LA gradient of < 5 mm Hg had abnormal perfusion. Pulmonary venography demonstrated a 60% stenosis of this vessel with a resting pressure gradient of 3 mm Hg.

Figure 2 illustrates a perfusion abnormality in the left lung of patient 3. The left superior PV had a 50% stenosis and a pressure gradient of 4 mm Hg that was not associated with a radionuclide perfusion abnormality. The left inferior PV had stenosis of 80% and a pressure gradient of 6 mm Hg, with a corresponding radionuclide perfusion abnormality. Figure 3 illustrates a perfusion abnormality in the right superior PV of the same patient with an 80% stenosis of the right superior PV by venography and a 7 mm Hg pressure gradient. The right inferior PV was widely patent.

Figure 4 demonstrates the perfusion abnormality of patient 6, who had bilaterally occluded superior PVs. The perfusion scan demonstrated a significant reduction in perfusion in both the RUL and the left upper lobe (LUL). The gradient in these vessels could not be measured since a multipurpose catheter could not be advanced distal to the occlusions.

The anatomic imaging (ie, high resolution CT scanning and MRI) performed in these patients did not reveal the presence of a pulmonary arterial thromboembolism in any patient. In two patients with severe PV stenosis (patients 4 and 5), high-resolution selective pulmonary arteriograms were performed, which showed no obstruction to the pulmonary arteries.

**DISCUSSION**

Among 11 consecutive patients with suspected PV stenosis, nuclear perfusion scanning identified a lobar perfusion deficit corresponding to every PV with > 80% stenosis and a PV-LA gradient of > 5 mm Hg. No patient without PV stenosis demonstrated a defect in perfusion imaging. This report of radionuclide imaging in patients with PV stenosis following AF ablation confirms the complementary contributions of physiologic and anatomic imaging methods. This finding may prove to be useful in the follow-up of patients after AF ablation, allowing the hemodynamic significance of a narrowed PV to be assessed noninvasively.

Radionuclide perfusion imaging is more widely available and much easier to interpret and quantitate than are CT scans or MRI images of the PVs. This makes perfusion...
significant lesions after the ablation procedure is low.3,16,17 Cardiographic techniques for the prediction of clinically significant PV flow velocity. However, the specificity of these echocardiographic techniques in the management of patients undergoing ablation procedures for the treatment of AF, and they can be used to estimate the PV-LA pressure gradient by determining PV flow velocity. However, the specificity of these echocardiographic techniques for the prediction of clinically significant lesions after the ablation procedure is low.3,16,17

The Use of Functional Imaging in PV Stenosis

There has been no systematic evaluation of nuclide imaging of patients with suspected PV stenosis. A recent study by Saad et al2 demonstrated significant improvement in the perfusion abnormality after dilatation of the stenosed PV, which is concordant with the physiologic significance of our findings. While no anatomic “gold standard” exists, current tests utilized in the diagnosis of PV stenosis include selective pulmonary venography, CT scanning, and MRI. All of these tests aim to image the PVs and to define the degree of anatomic stenosis by measuring vein size. They are limited by the difficulties in accurately defining the dimensions of the PVs in a beating heart and are highly technique-dependent. Transesophageal and intracardiac echocardiography are useful modalities in the management of patients undergoing ablation procedures for AF, and they can be used to estimate the PV-LA pressure gradient by determining PV flow velocity. However, the specificity of these echocardiographic techniques for the prediction of clinically significant lesions after the ablation procedure is low.3,16,17

Mechanism of Perfusion Abnormality

The impedance of the pulmonary circulation is usually low with a transpulmonary pressure gradient in healthy individuals that is usually <12 mm Hg. The postcapillary barrage created by the PV stenosis leads to an increase in impedance in the affected region. Due to the low impedance in the pulmonary circulation, even a subtle increase in impedance in the region drained by a stenotic PV probably leads to preferential flow to unaffected regions with lower resistance. Lung perfusion scanning is highly sensitive for the detection of regional abnormalities of blood flow. Since a fixed radionuclide dose will be distributed in proportion to the relative blood flow through the pulmonary lobes, a major defect in the perfusion scan is detected.15 The absence of arterial emboli from CT scans and MRI images, in addition to the fact that perfusion defects were observed only where there was a PV obstruction, and that the finding that pulmonary arteriograms performed in two patients showed no obstruction to the pulmonary arteries, are strong evidence that pulmonary arterial obstruction is not the cause of the abnormal radionuclide-perfusion scan finding.

Variability in Clinical Manifestation

The numbers of patients studied in this series preclude firm conclusions correlating imaging findings with clinical manifestations of PV stenosis. Given this limitation, the two patients who were asymptomatic in this series each had only a single vein that had stenosis of >80%. The finding that symptoms are likely in patients with multiple-vein involvement compared to single-PV involvement has been described previously1 and is concordant with our findings.

Our data do not really allow us to provide conclusions regarding the site of energy delivery and the appearance of PV stenosis. However, previous studies1,5 have clearly demonstrated the importance of limiting the application of ablative energy as proximal as possible to the PV ostium to prevent PV stenosis. The time to the onset of symptoms is rather variable in our series. Patient 6, who had occlusions of both upper lobes, presented with hemoptysis within 1 week. It is possible that acute occlusion of the PV, with no time course for the development of compensatory mechanisms in the pulmonary circulation, would manifest sooner than in those patients with slowly progressing stenosis. The development of collateral circulation may be variable among patients and may explain partly the variability in presentation. Genta et al19 described the significance of the development of collateral flow in the event of PV occlusion. These authors demonstrated PV thrombosis after surgery by the absence of flow in the RUL PV by transesophageal echocardiography. Pulmonary angiography performed 35 days after surgery showed preferential contrast flow to the left lung. The RUL PV was not visualized in the venous phase. Instead, the venous return was seen through the intercostal veins. The authors reported that 18 months after surgery the patient was in excellent condition without any restriction to physical activity, demonstrating the importance of collateral and compensatory mechanisms in the pulmonary circulation.
**Limitations**

Although all patients underwent either PV venography or spiral CT scanning, the degree of stenosis detected by these techniques may vary. Second, radionuclide perfusion images are planar images and represent an aggregate over time, so potential lobar segments may overlap on imaging. Since 99mTc MAA is a first-pass pulmonary perfusion agent, the time integration is nearly equal for contrast, when the degree of stenosis was a lobar perfusion defect was identified in all patients. In addition, while a resting PV-LA pressure gradient of > 5 mm Hg was highly correlated with a defect in radionuclide perfusion imaging, it remains to be determined whether imaging during exercise would better define more subtle degrees of PV stenosis. In patients with multiple perfusion defects, the reductions are relative to the most normal segment of the involved lobes. This method would tend to underestimate the degree of perfusion reduction, but, however, it provides further evidence that these areas were indeed abnormally perfused.

**CONCLUSION**

Nuclide perfusion imaging is noninvasive and widely available, and it provides physiologic information regarding pulmonary blood flow in patients suspected of having PV stenosis. When the degree of PV stenosis was > 80%, a lobar perfusion defect was identified in all patients. In contrast, when the degree of stenosis was < 50%, there were no perfusion defects noted. The technique is simple to perform and interpret, and may be especially suitable for centers without experience in CT scanning or MRI of PVs.

**REFERENCES**


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