Background and Aim

Right heart catheterization (RHC) should be performed on all candidates evaluated for cardiac transplantation (CT) since elevated pulmonary vascular resistance (PVR) is associated with right heart failure and mortality after CT. A vasodilator challenge is recommended for those with pulmonary hypertension (PHT), to assess the reversibility of PVR. The effects of inhaled nitric oxide (NO) on pulmonary and systemic hemodynamics have been reported only in small series. Our purpose was to describe the response to NO in this population.

Population and Methods

From 167 right heart catheterization procedures performed between 2010 and 2018, vasodilator challenge with inhaled nitric oxide (NO) was used in 88 patients, of which 60 had end-stage cardiac disease under evaluation for CT (55±11 years old; 72% male gender; 42% with ischemic cardiomyopathy; LVEF 30±12%; peak VO2 of 11.9±3.1; 63% with systolic PAP=50 mmHg). NO was administered through a tight-fitting facial mask at a mean dose of 34±10 ppm, regardless of baseline pulmonary pressure. LV pressures were measured simultaneously in all but 3 cases. Cardiac output was measured using the Fick method. Pressure measurements were made at end-expiration.

Results

There were no relevant side effects of NO administration. Hemodynamic profiles at baseline and after NO are represented in Table 1. In summary, typical response consisted of a reduction in PVR, concordant with an increase in both PCWP and LV end-diastolic pressures and no significant change in mean pulmonary artery pressure (resulting in a lower mean transpulmonary gradient). Cardiac index and systemic vascular resistance were unaffected (p=NS). PVR fell to < 5 Wood units with NO in 11/20 pts with higher baseline PVR; in 2 cases (3.3%) PVR increased paradoxically. Patients with hypertrophic cardiomyopathy (n=13) displayed higher PVR (mean 5.4 ± 1.5; p<0.01), but responded similarly to NO.

Conclusions

Inhaled NO is safe and produces a reasonably predictable response in patients undergoing vasodilator challenge prior to heart transplant listing. As opposed to patients with predominant type 1 pulmonary hypertension, the absence of significant reductions in pulmonary artery pressures may not mean that the PVR is fixed.
Abstract:
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Acute hemodynamic effects of inhaled nitric oxide during right heart catheterization in cardiac transplant candidates with and without pulmonary hypertension

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Topic(s):
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Table 1. Hemodynamic profile at baseline and during inhaled nitric oxide (NO)

<table>
<thead>
<tr>
<th>Variable (mmHg)</th>
<th>baseline</th>
<th>NO</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAP systolic</td>
<td>58.7 ± 18.3</td>
<td>56.3 ± 16.6</td>
<td>0.01</td>
</tr>
<tr>
<td>PAP diastolic</td>
<td>26.6 ± 7.6</td>
<td>26.4 ± 7.7</td>
<td>0.79</td>
</tr>
<tr>
<td>PAP mean</td>
<td>30.6 ± 11.0</td>
<td>30.4 ± 10.5</td>
<td>0.05</td>
</tr>
<tr>
<td>PCWP (mmHg)</td>
<td>26.8 ± 7.5</td>
<td>30.3 ± 9.5</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVEDP</td>
<td>23.3 ± 9.1</td>
<td>25.6 ± 10.4</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Mean TPG</td>
<td>13.5 ± 7.3</td>
<td>8.1 ± 6.0</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>PVRI (Wood Units)</td>
<td>5.0 ± 3.4</td>
<td>2.9 ± 2.5</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Cardiac Index (L/min/m²)</td>
<td>3.6 ± 0.4</td>
<td>1.75 ± 0.6</td>
<td>0.06</td>
</tr>
<tr>
<td>SVRI (dynes<em>cm²/(sec</em>mmHg))</td>
<td>1851 ± 756</td>
<td>1902 ± 772</td>
<td>0.28</td>
</tr>
</tbody>
</table>