Feasibility of Routine Pulmonary Arterial Impedance Measurements in Pulmonary Hypertension*

Sandrine Huez, MD; Serge Brimioulle, MD; Robert Naeije, MD; and Jean-Luc Vachiery, MD

**Objectives:** Right ventricular (RV) afterload is best described by a pulmonary arterial impedance (PVZ) spectrum, which integrates pulmonary vascular resistance (PVR), elastance, and wave reflection. We evaluated the feasibility of PVZ determinations in patients with pulmonary arterial hypertension (PAH) during routine right heart catheterization and Doppler echocardiography.

**Design:** Prospective study.

**Setting:** Academic hospital.

**Patients:** Twenty-two patients with PAH.

**Interventions:** Right heart catheterization with a fluid-filled Swan-Ganz catheter, Doppler echocardiography, and administration of inhaled nitric oxide (NO) [10 to 20 ppm; 17 patients], maximum tolerated dose of IV epoprostenol (average, 8.5 ng/kg/min; 5 patients), and IV dobutamine (8 μg/kg/min; 8 patients).

**Measurements and results:** PVZ was calculated from the spectral analysis of synchronized pulmonary artery pressure (Ppa) and flow waves. The mean (±SE) Ppa was 63 ± 3 mm Hg, and the mean PVR was 16 ± 2 Wood units. The PVZ spectrum was markedly shifted to higher than normal pressures and frequencies, with a mean 0-Hz impedance (Z₀) of 1,506 ± 138 dyne·s·cm⁻⁵, and a mean characteristic impedance (Zc) of 124 ± 11 dyne·s·cm⁻⁵, which are in keeping with data from previous studies. Inhaled NO levels decreased Ppa, PVR, Z₀, and Zc without a change in cardiac output. Epoprostenol administration did not affect Ppa, increased cardiac output, and decreased Z₀ and Zc. Dobutamine administration increased cardiac output and Ppa, and decreased PVR and Z₀, without changing Zc.

**Conclusions:** The determination of PVZ to quantify RV afterload is feasible during routine right heart catheterization and Doppler echocardiography. The measurement is sensitive to pharmacologic interventions.

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**Key words:** characteristic impedance; dobutamine; echocardiography; epoprostenol; nitric oxide; pulmonary arterial hypertension; pulmonary vascular impedance; pulmonary vascular resistance

**Abbreviations:** F₀ = first minimum frequency of the ratio of pressure and flow moduli; F₀ cross = frequency of zero crossing of the impedance phase angle; HR = heart rate; NO = nitric oxide; NYHA = New York Heart Association; PAH = pulmonary arterial hypertension; PPH = primary pulmonary arterial hypertension; Ppa = pulmonary artery pressure; Ppao = pulmonary artery occluded pressure; Pfr = right atrial pressure; Psa = systemic artery pressure; PVR = pulmonary vascular resistance; PVZ = pulmonary arterial impedance; Q = cardiac output; RF = reflection factor; RV = right ventricle, ventricular; Z₀ = impedance at 0 Hz; Z₁ = first harmonic impedance; Zc = characteristic impedance

Right heart catheterization with measurements of pulmonary vascular pressures and cardiac output, and the calculation of pulmonary vascular resistance (PVR) is an essential step in the diagnosis of pulmonary arterial hypertension (PAH). These hemodynamic measurements are of prognostic value, with, however, survival being more closely related to flow (cardiac output) than to pressure (ie, pulmonary artery pressure [Ppa]). The clinical state of PAH patients, as assessed by functional class and exercise...
capacity, also appears to be more related to cardiac output than to Ppa. 5,6 Clinical signs of right heart failure are often not clearly related to the progression of pulmonary hypertension, as assessed by Ppa and PVR. 7 These observations may be explained by the fact that heart failure in patients with PAH is the consequence of increased right ventricular (RV) afterload, which is not accurately measured by routine hemodynamic evaluations. 7–9

Ventricular afterload can be defined either by maximum wall tension or by hydraulic load. 10 The measurement of RV wall tension is problematic because of particular geometry and associated volume measurement uncertainties. However, hydraulic load can be assessed from the morphology of Ppa and flow waves. At a given level of PVR, RV hydraulic load may be increased by a decrease in pulmonary arterial compliance and an increase in wave reflection. These changes affect pressure waves by an increased pulse pressure and late systolic peaking, and they affect flow waves by a shortened acceleration time and by late or mid-systolic deceleration. 7,8,10 There are data indicating that Ppa and wave qualitative morphology analysis may be of diagnostic and prognostic value in patients with pulmonary hypertension. 9,11,12

However, a more accurate quantification of RV hydraulic load can be obtained by a calculation of pulmonary arterial impedance (PVZ) from a spectral analysis of Ppa and flow waves. 10 The results of this analysis are expressed as a PVZ spectrum, consisting of a pressure/flow ratio and a phase angle, both of which are expressed as a function of frequency. A PVZ spectrum includes a measure of total PVR, indexes of wave reflection such as the first minimum of the ratio of pressure and flow moduli or low-frequency phase angle, characteristic impedance (Zc), which corresponds to an inerterance to the compliance ratio, and hydraulic load, as evaluated by low-frequency impedance and the amplitude of impedance oscillations. 7,10

There are only a few previous studies 13,14 on PVZ in PAH patients. This is to be explained by the technical difficulties of instantaneous pressure and flow measurements requiring high-fidelity, manometer-tipped catheters and flowmeters. 10,13,14 It has been generally assumed that the frequency response of the fluid-filled thermodilution Swan-Ganz catheters that are used for routine right heart catheterizations would be insufficient for instantaneous pressure measurements but would be acceptable for mean pressure estimations. On the other hand, flow measured by thermodilution using these catheters necessarily covers several cardiac cycles, imposing the steady flow hemodynamic approach. However, the natural frequency of commercially available Swan-Ganz catheters is around 30 Hz, decreasing to only 18 to 20 Hz for the external manometer-tubing-pulmonary catheter system, which actually may be adequate for valid and clinically relevant pressure wave measurements. 15 Instantaneous pulmonary artery flow velocities can be measured by transthoracic pulsed Doppler echocardiography. 16

Therefore, we evaluated the feasibility of performing instantaneous Ppa and flow measurements during a routine right heart catheterization that was combined with Doppler echocardiography in patients with PAH. We compared the obtained PVZ calculation to those values previously reported in PAH patients and evaluated its sensitivity to pharmacologic interventions.

Materials and Methods

Study Population

Twenty-two patients (5 men and 17 women; mean age, 46 years) with PAH gave informed consent to participate in this study, which was approved by the Institutional Review Board of the Erasme University Hospital. PAH was defined by an increase in Ppa values without identifiable cardiac or pulmonary cause, and possibly associated with conditions such as appetite-suppressant intake, connective tissue disease, liver cirrhosis, HIV infection, and congenital left-to-right shunts, as defined by a World Health Organization-sponsored expert consensus conference held in 1998 in Evian. 1, 17 PAH was primary (ie, with no identifiable associated conditions) in 14 patients, and was associated with the intake of appetite suppressants (fenfluramines) in 3 patients, with systemic-to-pulmonary congenital cardiac shunts in 4 patients, and with portal hypertension in 1 patient. The patients were in New York Heart Association (NYHA) functional class III, excepted for two patients who were in NYHA class IV and one patient who was in NYHA class II. The patients had been referred for diagnostic evaluation before the administration of prostacyclin or endothelin receptor blocker therapy. All but 3 patients were receiving conventional treatment, consisting of anticoagulants in 15 patients, diuretics in 13 patients, low-dose nifedipine in 4 patients, and digoxin in 1 patient.

Right Heart Catheterization

Right heart catheterization was performed without premedication, with the patient lying supine and breathing room air. A balloon-tipped, flow-directed thermodilution 7F Swan-Ganz catheter (131HF7; Baxter Healthcare Corp; Irvine, CA) was inserted under local anesthesia into an internal jugular vein and was floated under continuous pressure-wave monitoring into a pulmonary artery to measure Ppa, pulmonary artery occluded pressure (Ppa0), right atrial pressure (Pra), and cardiac output (Q). Systemic arterial pressure (Psa) was determined intermittently by an automated BP cuff. Heart rate (HR) was determined from a continuously monitored ECG lead. Pulmonary vascular pressures were measured using disposable transducers (TruWave; Baxter Healthcare Corp) connected to a bedside hemodynamic and ECG monitoring system (Sirecust 404; Siemens; Erlangen, Germany). The pressure transducers were zero-referenced at midchest, and
vascular pressures were obtained at end-expiration. The static calibration was checked against a water column, and the dynamic response of the catheter-manometer system was checked using the “fast flush” test. Cardiac output was measured using the thermodilution technique as a mean of at least three successive measurements (COM-2, Baxter Healthcare Corp.). The pulmonary vascular pressure signals were sampled at 200 Hz using an analog/digital converter (DAS 8-PCA; Keithley-Metabyte; Taunton, MA), and were stored and analyzed on a personal computer.

**PVZ Acquisition**

Transthoracic Doppler echocardiography was performed (SO-NOS 2000; Hewlett-Packard; Palo Alto, CA) with a 3.5-MHz probe during catheterization with the patient in a dorsal or lateral supine position. Pulsed-Doppler velocity was recorded in the RV outflow tract using the short-axis parasternal view, as previously described. Sampling frequency and gain setting were optimized to obtain the best flow-velocity envelope. In our center, the interobserver variability for pulmonary arterial flow acceleration time and the velocity-time integral is < 5%.

Ppa and flow signals were recorded after the pulmonary catheter was then carefully withdrawn to position its tip just above the pulmonary valve, as close as possible to the pulsed Doppler pulmonary artery flow-velocity sampling site. The signals were visually checked for quality, and then were synchronized by an ECG artifact and recorded on paper at a speed of 100 mm/s using the built-in printing system of the echocardiograph. Pressure and flow-velocity tracings were scanned (Scanjet 4470c; Hewlett Packard), digitized at a sampling rate of 200 Hz, and analyzed on a personal computer. Between three and five heartbeats were used for the analysis during each data collection period. For each patient, a series of thermodilution cardiac outputs and concomitant average Doppler flow velocities were used to calculate a conversion factor for the flow-velocity tracings into volume flow.

The PVZ spectrum was calculated from the Fourier series expressions for pressure and flow signals, as previously described. Zc was calculated as the average of the impedance wave reflection factor (RF) moduli between 2 and 15 Hz. A wave RF was calculated as \( \frac{(Z_0 - Z_c)/(Z_0 + Z_c)}{Z_0} \), where \( Z_0 \) is impedance at 0 Hz. First harmonic impedance \( Z_1 \), the first minimum frequency of the ratio of pressure and flow moduli (\( F_{min} \)), and the frequency of the zero crossing of the impedance phase angle (\( F_0 \) cross) were also determined.

**Protocol**

A complete set of pulmonary hemodynamic measurements and sampling of the instantaneous pressure and flow signals was obtained at baseline, with the patients in a stable state, as assessed by unchanged HR and pulmonary vascular pressures for at least 15 min. The measurements were repeated in 17 patients after 10 min of breathing nitric oxide (NO) [10 to 20 ppm], and epoprostenol administration slowly increased until intolerance (ie, a fall in Psa, facial flush, headache, jaw pain) had occurred to an average of 8.5 ng/kg/min 5 other patients. In addition, after a washout period of 20 min, and a return of Ppa, HR, and Psa values to baseline levels, a complete set of hemodynamic measurements, and a sampling of pressure and flow signals was obtained at the 15th min of an infusion of dobutamine at a dose of 8 μg/kg/min.

The dose of NO had been selected on the basis of findings from previous reports that 10 to 20 ppm allows the maximum possible pulmonary vasodilation in PAH patients. NO was supplied from a pure NO source tank (Oxyhydrine; Machelen, Belgium) and was delivered through a tight facemask. The inspired fraction of NO was monitored by chemiluminescence after calibration against a standard NO concentration (42 chemiluminescence NO-N02-Nox analyser; Thermo Environmental Instruments Inc; Franklin, MA). The dose of epoprostenol was selected on the basis of experimental data showing that this inotropic drug is without flow-independent pulmonary vascular effects at doses of up to 10 μg/kg/min.

**Statistical Analysis**

Results are presented as the mean ± SE. Paired t tests were used to compare the hemodynamic values after different pharmacologic interventions to baseline levels. A p value of < 0.05 was defined as being statistically significant.

**Table 1—Hemodynamic Measurements in 22 Patients With PAH Compared to Previously Reported Measurements in PPH Patients and Healthy Control Subjects***

<table>
<thead>
<tr>
<th>Variables</th>
<th>Healthy Control Subjects</th>
<th>PPH Patients</th>
<th>Present Study</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 10)</td>
<td>PPH Patients (n = 8)</td>
<td>PAH Patients (n = 22)</td>
</tr>
<tr>
<td>PVR, dynes/cm²</td>
<td>73 ± 10</td>
<td>880 ± 158</td>
<td>1,282 ± 160</td>
</tr>
<tr>
<td>Ppa, mm Hg</td>
<td>14 ± 2</td>
<td>50 ± 3</td>
<td>63 ± 3</td>
</tr>
<tr>
<td>Pra, mm Hg</td>
<td>11 ± 1</td>
<td>13 ± 1</td>
<td>13 ± 1</td>
</tr>
<tr>
<td>Ppao, mm Hg</td>
<td>9 ± 3</td>
<td>8 ± 4</td>
<td>12 ± 1</td>
</tr>
<tr>
<td>Q, L/min</td>
<td>7.4 ± 0.5</td>
<td>4.5 ± 0.6</td>
<td>3.5 ± 0.3</td>
</tr>
<tr>
<td>Zo, dynes/cm²</td>
<td>38 ± 4</td>
<td>385 ± 75</td>
<td>1,506 ± 138</td>
</tr>
<tr>
<td>Z₀, dynes/cm²</td>
<td>38 ± 4</td>
<td>385 ± 75</td>
<td>289 ± 23</td>
</tr>
<tr>
<td>Z₁ phase, rad</td>
<td>0.9 ± 0.06</td>
<td>1 ± 0.06</td>
<td>124 ± 11</td>
</tr>
<tr>
<td>Z₁, dynes/cm²</td>
<td>22 ± 4</td>
<td>55 ± 9</td>
<td>6.6 ± 0.4</td>
</tr>
<tr>
<td>F₀ cross, Hz</td>
<td>7 ± 0.7</td>
<td>6.4 ± 0.8</td>
<td>0.84 ± 0.01</td>
</tr>
<tr>
<td>RF</td>
<td>0.33 ± 0.04</td>
<td>0.88 ± 0.06</td>
<td>0.84 ± 0.01</td>
</tr>
</tbody>
</table>

*Values given as mean ± SE. Z₀ phase = first harmonic phase angle; rad = radian.

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RESULTS
Effects of Pulmonary Hypertension

As shown in Table 1, the patients had increased levels of Ppa, Pra, and PVR, a normal Ppao level, and decreased cardiac output, with a PVZ spectrum showing marked increases in Z₀, Z₁, and Zc, a shift of Fₘᵦᵣᵦ to higher frequencies, and a negative first harmonic phase angle, with Z₀ cross-shifted to higher frequencies (Table 1). Representative PVZ spectra with source pressure and flow signals are shown in Figures 1 to 3.

Compared to previously reported primary pulmonary hypertension (PPH) patients, our patients had higher Ppa and PVR values, and lower cardiac output, suggesting a more advanced stage of the disease. The differences were quantitatively similar when only the subgroup of our 14 PPH patients was considered. There were more important increases in Z₁ and Zc, amounting up to seven times normal, but Fₘᵦᵣᵦ and RF were not different (Table 1).

Pharmacologic Interventions

Inhaled NO therapy decreased Ppa and PVR, while Q, HR, and Psa remained unaffected. Z₀, Z₁, and Zc decreased, while Fₘᵦᵣᵦ, phase indexes, and RF were unchanged (Table 2). Epoprostenol therapy decreased PVR and Psa, increased Q, while HR remained unaffected, and decreased Z₀, Z₁, and Zc, while Fₘᵦᵣᵦ, phases indexes, and RF were unchanged (Table 2). Dobutamine therapy increased Q, HR, and Ppa, did not affect Psa, and decreased PVR and Z₀, while Z₁, Zc, Fₘᵦᵣᵦ, and phase indexes

![Graph of PVZ spectra with baseline and NO 20 ppm data](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/22010/)
remained unaffected (Table 2). Figures 1 to 3 illustrate the typical PVZ spectra modifications observed with each intervention.

**DISCUSSION**

The present results show that it is possible to analyze Ppa and flow waves obtained during routine right heart catheterization with thermodilution fluid-filled pulmonary artery catheters and concomitant transthoracic Doppler echocardiography. Derived PVZ spectra are realistic, as they were similar to those previously reported with sophisticated high-fidelity equipment, and were sensitive to pharmacologic interventions.

Right heart catheterization for the diagnosis and therapeutic follow-up of PAH patients in clinical practice relies on measurements of mean flow (by thermodilution or the Fick method) and mean Ppa, and on the derived PVR calculations.1-6 This "steady-flow" hemodynamic approach neglects the natural pulsatility of the pulmonary circulation, and the pressure and wave morphology changes induced by the disease.7-10 The rationale for using the steady-flow hemodynamic approach is its simplicity, the inherent technical limitations of commercially available catheters, and the fact that the pathologic changes in PAH patients are essentially limited to the small resistive pulmonary arteries, to which PVR is sensitive. In addition, the quantification of pressure and waveform morphology requires a mathematical analysis, with results expressed in the frequency domain unfamiliar to clinicians. The frequency response of fluid-filled catheters is thought to be insufficient for accurate instantaneous pressure measurements anyway.

However, PVR is known to be insufficient for the evaluation of all the forces that oppose RV ejection, which result from a dynamic interplay among resistance, elastance, and wave reflection.6 This information is contained in Ppa and flow waves, and can be quantified by a PVZ spectrum calculation.10 The
method provides a measure of hydraulic load by a low-frequency impedance determination ($Z_1$), and by an estimate of compliance and wave reflection using a high-frequency $Z_c$ determination, together with the measurement of $F_{\text{min}}$ and the frequency of phase angle zero crossing. An index of wave RF can be calculated from $Z_0$ and $Z_c$ determinations. In our PAH patients, the major change in the PVZ spectrum was an increase in $Z_0$, or total PVR (that is PVR calculated as $P_{pa}/Q$). $Z_1$ was markedly increased, indicating increased hydraulic load, and $Z_c$, $F_{\text{min}}$, $F_0$ cross, and RF were increased as well, indicating important increases in arterial elastance and wave reflection. These results look comparable to those of the only reported PVZ determinations in eight patients and two patients, except for higher $Z_0$ and $Z_c$ values, which appear to be related to more advanced disease, as assessed by $P_{pa}$, PVR, and cardiac output. Similar changes have been reported in patients with severe pulmonary hypertension secondary to mitral stenosis or in those with advanced left heart failure. We could not consider a direct comparison between Swan-Ganz catheter-derived PVZ spectra and PVZ spectra obtained using high-fidelity, micromanometer-tipped technology because of the prohibitive cost related to single use imposed by the latest European Union regulations.

**Effects of Pharmacologic Interventions**

In our patients, both inhaled NO and IV epoprostenol not only decreased PVR, but also shifted the entire PVZ spectrum to lower pressure/flow ratios. Both interventions are aimed at the small peripheral resistive arterioles. This is particularly true for NO diffusing from alveolar spaces to adjacent vascular structures, which is inactivated by avid uptake by hemoglobin as soon as it enters the bloodstream.

**Figure 3.** Pulmonary vascular impedance spectra with source pressure and flow waves before and during the administration of 8 μg/kg/min dobutamine, in a 55-year-old patient with PPH who was in NYHA class III. The mean $P_{pa}$ was 84 mm Hg and remained unchanged during dobutamine infusion. PVR decreased from 17.3 to 11.2 Wood units because of an increase in cardiac output from 2.9 to 4.3 L/min. Left, A: dobutamine therapy had no significant effect on the PVZ spectrum. Right, B: shows that the predominant effect of dobutamine is a global increase in flow.
**Clinical Implications**

There has been interest in time-domain pressure-wave morphology analysis for the differential diagnosis of pulmonary hypertension. There are data showing earlier wave reflection in patients with chronic thromboembolic pulmonary hypertension compared to that in those with PPH,

These differences were explained by a more proximal site of wave reflection in patients with chronic thromboembolic pulmonary hypertension. To our knowledge, there has been no report on time-domain pulmonary arterial flow wave analysis for the diagnosis of pulmonary hypertension, excepted for the estimation of mean Ppa from acceleration time.16

The determination of a PVZ spectrum presents the advantage of integrating all of the information contained in both the pressure and flow waves. It offers the possibility of quantifying the contributions of resistance, elastance, and wave reflection to RV hydraulic load. Pulmonary hypertension symptomatology and prognosis are essentially dependent on RV tolerance and the adaptation to chronically increased afterload. PVZ, more than PVR, allows for the estimation of RV afterload. Thus, it is possible that PVZ calculations based on the sampling and frequency analysis of routine right heart catheterization and echocardiographic signals will prove useful in assessing responses to therapy and the prognosis of patients with severe pulmonary hypertension. However, the proof of this concept will require a prospective clinical evaluation.

**References**


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**Table 2—Hemodynamic Effects of Pharmacologic Interventions in 22 Patients With PAH**

<table>
<thead>
<tr>
<th>Variables</th>
<th>Baseline</th>
<th>NO†</th>
<th>Baseline</th>
<th>Epoprostenol‡</th>
<th>Baseline</th>
<th>Dobutamine§</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR, ppm</td>
<td>84 ± 3</td>
<td>81 ± 3</td>
<td>86 ± 3</td>
<td>90 ± 4</td>
<td>85 ± 3</td>
<td>101 ± 5</td>
</tr>
<tr>
<td>Psa, mm Hg</td>
<td>88 ± 3.1</td>
<td>92 ± 2.9</td>
<td>81 ± 2.2</td>
<td>59 ± 3.1</td>
<td>90 ± 5.1</td>
<td>87 ± 5</td>
</tr>
<tr>
<td>Ppa, mm Hg</td>
<td>60 ± 2.3</td>
<td>56 ± 2.4</td>
<td>56 ± 1.9</td>
<td>53 ± 3.4</td>
<td>56 ± 3.6</td>
<td>65 ± 4.6</td>
</tr>
<tr>
<td>Q, L/min</td>
<td>3.5 ± 0.22</td>
<td>3.6 ± 0.28</td>
<td>3.7 ± 0.39</td>
<td>5.3 ± 0.51</td>
<td>3.6 ± 0.38</td>
<td>5.1 ± 0.55</td>
</tr>
<tr>
<td>PVR, wood units</td>
<td>15.3 ± 1.4</td>
<td>13.7 ± 1.5</td>
<td>11.7 ± 1.1</td>
<td>8.1 ± 1.3</td>
<td>17.2 ± 2.3</td>
<td>14.1 ± 1.5</td>
</tr>
<tr>
<td>Zc, dynes/cm²</td>
<td>1,463 ± 138</td>
<td>1,345 ± 141</td>
<td>1,208 ± 103</td>
<td>820 ± 124</td>
<td>1,376 ± 221</td>
<td>1,097 ± 165</td>
</tr>
<tr>
<td>Zf, dynes/cm²</td>
<td>292 ± 23</td>
<td>255 ± 22</td>
<td>238 ± 25</td>
<td>190 ± 20</td>
<td>267 ± 26</td>
<td>262 ± 35</td>
</tr>
<tr>
<td>Z phase, rad</td>
<td>0.93 ± 0.06</td>
<td>1.03 ± 0.06</td>
<td>0.9 ± 0.1</td>
<td>0.9 ± 0.1</td>
<td>0.9 ± 0.1</td>
<td>0.5 ± 0.1</td>
</tr>
<tr>
<td>Zc* phase, rad</td>
<td>124 ± 14</td>
<td>103 ± 10</td>
<td>90 ± 6</td>
<td>73 ± 10</td>
<td>99 ± 11</td>
<td>104 ± 15</td>
</tr>
<tr>
<td>Fmin, Hz</td>
<td>6.5 ± 0.5</td>
<td>6 ± 0.3</td>
<td>5.9 ± 0.4</td>
<td>5.7 ± 0.5</td>
<td>6.5 ± 0.8</td>
<td>6.7 ± 0.5</td>
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<tr>
<td>Fc crossing, Hz</td>
<td>7 ± 0.9</td>
<td>6.5 ± 0.7</td>
<td>5.5 ± 0.8</td>
<td>4.6 ± 0.6</td>
<td>7.9 ± 1.6</td>
<td>6.1 ± 0.7</td>
</tr>
<tr>
<td>RF</td>
<td>0.857 ± 0.001</td>
<td>0.848 ± 0.012</td>
<td>0.859 ± 0.009</td>
<td>0.836 ± 0.012</td>
<td>0.834 ± 0.017</td>
<td>0.819 ± 0.02</td>
</tr>
</tbody>
</table>

*Values given as mean ± SE. See Table 1 for abbreviations not used in the text.
†n = 17.
‡n = 5.
§n = 8.

*p < 0.05 compared to baseline values.