Hemodynamic correlates of effective arterial elastance in mitral stenosis before and after balloon valvotomy

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Colin, Patrice, Michel Slama, Alec Vahanian, Yves Lecarpentier, Gilbert Motte, and Denis Chemla. Hemodynamic correlates of effective arterial elastance (Ea) in mitral stenosis (MS) patients. In this preliminary study, the authors aimed to document the hemodynamic correlates of Ea in MS patients both before and after balloon mitral valvotomy (BMV). A group of consecutive patients undergoing BMV was treated with furosemide, digitalis, and amiodarone. The purpose of the study was to document the hemodynamic correlates of Ea in MS patients both before and after BMV. High-fidelity aortic pressure recordings were obtained in 10 patients (40 ± 12 yr) before and 15 min after BMV. Ea value was calculated as the ratio of the steady-state end-systolic aortic pressure (ESAP) to stroke volume (thermodilution). Ea increased after BMV (from 1.55 ± 0.63 to 1.83 ± 0.71 mmHg/ml; P < 0.05). Throughout the procedure, there was a strong linear relationship between Ea and R/T: Ea = 1.09R/T - 0.01 mmHg/ml, r = 0.99, P = 0.0001. This ultimately depended on the powerful link between ESAP and mean aortic pressure (MAP; r = -0.99, 95% confidence interval for the difference (MAP - ESAP) from -18.5 to +4.5 mmHg). Ea was also related to total arterial compliance (area method) and to wave reflections (augmentation index), although to a lesser extent. After BMV, enhanced and anticipated wave reflections were observed, and this was likely to be explained by decreased arterial compliance. The present study indicated that Ea depended mainly on the steady component of hydraulic load (i.e., R) and heart period (i.e., T) in MS patients.

METHODS

Patients

From November 1994 to October 1995, 10 consecutive patients who underwent BMV were included in the study after informed consent was obtained. We studied eight women and two men. Characteristics of the study population are listed in Table 1. All the included patients had MS with a combined mitral valve orifice of <1.5 cm² on echocardiographic examination and were NYHA class II (8 of 10) or III (2 of 10). Echocardiography was performed the day before and between 24 and 48 h after BMV and was considered as the reference method for valvular area measurement. Patients were excluded from the study if they had moderate (2+) or severe (3+) mitral regurgitation, significant (<2+) aortic valve insufficiency, or any degree of aortic stenosis, significant calcification of the mitral valve, evidence of left atrial thrombus on transesophageal echocardiography, or a previous history of coronary artery disease. Seven patients had normal sinus rhythm. Three patients were in atrial fibrillation and were given oral anticoagulant therapy (n = 3), digitalis (n = 3), furosemide (n = 1), and amiodarone (n = 1). Six patients were undergoing diuretic therapy.
Catheterization Technique and BMV Procedure

Patients were studied at baseline, at least 12 h after previous intake of their usual medication according to our routine protocol (27, 28). Patients were sedated by using midazolam (10 mg). Aortic pressure was measured by using an 8 Fr single-lumen catheter equipped with a high-fidelity transducer (Sentron/ Cordis, Roden, The Netherlands) (8). The catheter was advanced from the left femoral artery to the aortic root. Routine right-heart catheterization was performed by using the Seldinger technique through the left femoral vein. Before BMV, right heart pressures were obtained and cardiac output was measured in triplicate in all patients by using the thermodilution technique. Stroke volume (SV) was calculated as the cardiac output-to-heart rate ratio. Left ventriculography was performed in the 30° right anterior oblique projection. LV volumes were calculated by using the area-length method, taking care not to include either ventricular premature beats or postextrasystolic beats. Transseptal catheterization was then performed from the left femoral vein by using the Brockenbrough technique, and patients were then given heparin (4,000 IU iv). Balloon dilatation of the mitral valve was performed on all patients by using a stepwise technique and transthoracic echocardiographic monitoring. Optimal results were defined as a final mitral valve area >1.5 cm² without appearance or worsening of mitral valve regurgitation of >1+. Final hemodynamic variables and cardiac output were obtained with the balloon catheter across the transeptal puncture site so as to reduce the error caused by atrial septal defect blood flow. Fifteen minutes after BMV, hemodynamic pressures and flow measurements were repeated. Left ventriculography was then repeated in the right anterior oblique projection to evaluate the severity of mitral regurgitation. Oxymetry was performed after BMV to evaluate left-to-right shunting. Before BMV, three patients had no mitral regurgitation, and seven patients had 1+ mitral regurgitation. Immediately after BMV, seven patients showed no change in the degree of mitral regurgitation, and three patients had 1+ increase (from 1+ to 2+). No left-to-right shunt was detected after BMV.

High-Fidelity Pressure Recordings

High-fidelity pressure data were computed throughout the procedures on a Toshiba 6400-SX portable computer with customized software (sampling rate = 1,000 Hz) as previously described (3, 8). Pressure recordings were obtained at the aortic root level. Mean aortic pressure (MAP) was defined as the area under the pressure curve divided by pulse interval duration (T). T (in ms) was defined as the time between two consecutive aortic pressure upstrokes. Aortic dicrotic notch pressure (ESP), i.e., aortic end-systolic pressure, was defined as the trough of the incisura (dicrotic notch). We measured systolic aortic pressure (SAP), initial diastolic aortic pressure (DAP), end-diastolic aortic pressure (EDAP), and pulse aortic pressure (PAP = SAP – DAP). MAP and PAP reflect the steady and pulsed components of aortic pressure, respectively (21). We also calculated two previously proposed estimates of ESP, namely, 0.9 SAP and 2/3 SAP + 1/3 DAP (12). Total vascular resistance (R; mmHg·ms·ml⁻¹) was calculated according to the following formula

\[ R = \frac{(MAP - T)/SV}{T} \]  

(1)

Effective $E_a$

Theoretical background. In the $E_a$ model, the proximal aorta is considered as an elastic chamber, the effective volume elastance $E_a$ (mmHg·ml⁻¹) of which is the slope of the relationship between SV and pressure. This model has markedly improved the evaluation of the systemic circulation for two reasons. First, in humans, $E_a$ provides a reasonable characterization of arterial load in the time domain (12). Second, the LV can also be considered as an elastic chamber, the end-systolic elastance (Ees) i.e., the slope of the LV end-systolic pressure-volume relationship of which is of similar dimension to $E_a$ (24). The operating point of the coupled equilibrium between LV and the arterial system is located at the intersection of LV end-systolic pressure-volume and ESP-SV relationships in the pressure-volume plane (24–26). Coordinated changes in the Ees-to-$E_a$ ratio, stroke work, and mechanical efficiency have been reported (1, 11, 25, 26).

The concept of $E_a$ is based on the Windkessel model of arterial circulation (25). Theoretical $E_a$ values are obtained by means of a mathematical formula taking into account the intrinsic properties of circulation, namely, total peripheral resistance (R), total arterial compliance (C), and systolic and diastolic time intervals. Because the mathematical model fits with experimental (25) and clinical (12) data, $E_a$ is currently obtained by calculating the steady-state ratio of ESP to SV (4, 11, 12). The hemodynamic correlates of $E_a$ have been documented in experimental studies and in studies performed on normotensive and hypertensive subjects without valve disease. In this population, $E_a$ depends mainly on both R and heart period (i.e., T) (12, 25, 26), in such a way that $R/T$ is a reasonable approximation of $E_a$ (12). Although experimen-
Clinical studies have shown that Ea is poorly influenced by C (25, 26), recent studies have demonstrated a relationship between Ea and the extent of pressure wave reflection from periphery to the heart (4, 23).

Calculation of Ea. Ea (mmHg/ml) was calculated according to the following steady-state formula

$$E_a = \frac{ESAP}{SV}$$  \hspace{1cm} (2)

Given that ESAP is close to MAP, Sunagawa et al. (25, 26) have suggested that Eqs. 1 and 2 yield the following approximations

$$E_a = \frac{R}{T}$$ \hspace{1cm} (3)

Estimated total arterial C. C (ml/mmHg) was estimated by using the area method (15), assuming a two-element Windkessel model of systemic circulation and a linear pressure-volume relationship (15). This method has been proved to give reliable estimates of C (15). C is given by the following formula

$$C = \frac{SV}{K (ESAP - EDAP)}$$ \hspace{1cm} (4)

where the area coefficient (K) is a dimensionless coefficient given by

$$K = \frac{\text{systolic area} + \text{diastolic area}}{\text{diastolic area}}$$ \hspace{1cm} (5)

Systolic and diastolic areas were defined as the area under systolic and diastolic waveform, respectively. Because the area method requires zero flow in diastole, patients with mitral insufficiency were excluded from the study.

Wave reflection and augmentation index. The human aortic pressure waveform exhibits an inflection point (Pi), indicating the end of the forward (or incident) wave and resulting from peak flow input into the vasculature previous to the effects of wave reflection (20, 21). The relative increase in the height of the mid-to-late systolic peak pressure above the Pi shoulder (ΔP) is because of arterial wave reflection and the early return of pressure wave from the lower body (13, 16, 20). The backward or reflected wave cumulates with the incident wave, resulting in a mid-to-late increase in SAP. The ratio of ΔP to PAP defines a so-called “augmentation index” (ΔP/PAP). The time from the foot of the pressure wave to Pi (Δtp) is thought to represent the travel time of the pulse wave to peripheral reflecting sites and its return. According to Murgo et al. (20), Δtp is a reasonable estimate of 1/2 fmin, where fmin corresponds to the frequency of the minimum impedance spectra modulus. In three patients, Pi could not be clearly individualized. Thus the values of Pi, ΔP, ΔP/PAP, and Δtp were recorded and averaged out over 10 consecutive cycles in 7 of 10 patients only.

Statistical Analysis

Data are expressed as means ± SD. Data were averaged out over 10 consecutive beats. Linear regression was obtained by using the least squares method. Comparisons between Ea and R/T were performed by using the Mann-Whitney U-test; we also calculated the 95% confidence intervals (CI) for the difference (2). The same was performed for comparisons between ESAP and each of three estimates of ESAP, namely, MAP, 0.9 SAP, and 2/3 SAP + 1/3 DAP. A P < 0.05 was considered statistically significant.

RESULTS

Hemodynamic data before and after BMV are listed in Tables 2 and 3 and in Fig. 1.

### Table 2. Hemodynamic data before and after BMV

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before BMV</th>
<th>After BMV</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulse interval, ms</td>
<td>843 ± 107</td>
<td>809 ± 149</td>
<td>0.41</td>
</tr>
<tr>
<td>Stroke volume, ml</td>
<td>67 ± 23</td>
<td>67 ± 23</td>
<td>0.17</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>55 ± 11</td>
<td>56 ± 13</td>
<td>0.62</td>
</tr>
<tr>
<td>Mean aortic pressure, mmHg</td>
<td>87.0 ± 22.5</td>
<td>96.7 ± 20.8</td>
<td>0.007</td>
</tr>
<tr>
<td>Total vascular resistance, mmHg·ms·m⁻¹</td>
<td>1.167 ± 0.351</td>
<td>1.343 ± 0.441</td>
<td>0.062</td>
</tr>
<tr>
<td>Mitral valve area, cm²</td>
<td>0.92 ± 0.11</td>
<td>1.98 ± 0.26</td>
<td>0.0001</td>
</tr>
<tr>
<td>Mitral valve gradient, mmHg</td>
<td>10.2 ± 4.4</td>
<td>5.3 ± 2.4</td>
<td>0.006</td>
</tr>
<tr>
<td>Mean pulmonary artery pressure, mmHg</td>
<td>24.7 ± 7</td>
<td>22.4 ± 5</td>
<td>0.22</td>
</tr>
<tr>
<td>End-systolic aortic pressure, mmHg</td>
<td>93.6 ± 24.4</td>
<td>103.7 ± 24.9</td>
<td>0.02</td>
</tr>
<tr>
<td>Effective arterial elastance, mmHg/ml</td>
<td>1.55 ± 0.63</td>
<td>1.83 ± 0.71</td>
<td>0.049</td>
</tr>
<tr>
<td>R/T, mmHg/ml</td>
<td>1.43 ± 0.57</td>
<td>1.70 ± 0.61</td>
<td>0.032</td>
</tr>
<tr>
<td>Estimated total arterial compliance, ml/mmHg</td>
<td>1.83 ± 0.98</td>
<td>1.47 ± 0.79</td>
<td>0.022</td>
</tr>
</tbody>
</table>

Values are means ± SD; n = 10 subjects. BMV, balloon mitral valvotomy; R/T, total vascular resistance; T, pulse interval.

### Hemodynamic Correlates of Ea in MS Patients at Baseline

Ea was 1.55 ± 0.63 mmHg/ml. There was no relationship between Ea and age, MAP, ESAP, or mitral valve area. There was a negative linear relationship between Ea and T (r = −0.72, P < 0.01). Ea was closely related to the heart (4, 23).

### Table 3. Indexes of wave reflection before and after BMV

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Before BMV</th>
<th>After BMV</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PAP, mmHg</td>
<td>41.5 ± 13.6</td>
<td>46.6 ± 17.1</td>
<td>0.05</td>
</tr>
<tr>
<td>Pi, mmHg</td>
<td>106.9 ± 21.5</td>
<td>112.5 ± 20.4</td>
<td>0.029</td>
</tr>
<tr>
<td>ΔP (SAP-Pi), mmHg</td>
<td>10.8 ± 8.3</td>
<td>15.6 ± 12.0</td>
<td>0.0001</td>
</tr>
<tr>
<td>ΔP/PAP, %</td>
<td>21.9 ± 10.5</td>
<td>28.7 ± 12.2</td>
<td>0.0001</td>
</tr>
<tr>
<td>Δtp, ms</td>
<td>143 ± 32</td>
<td>131 ± 28</td>
<td>0.0001</td>
</tr>
</tbody>
</table>

Values are means ± SD; n = 7 subjects. All data were averaged out over 10 consecutive cardiac cycles. PAP, pulse aortic pressure [systolic aortic pressure (SAP) minus diastolic aortic pressure]; Pi, shoulder height of aortic pressure waveform; ΔP, height above shoulder of late systolic peak of aortic pressure waveform; tP, time from foot of pressure wave to foot of late systolic peak.

### Fig. 1. Effects of balloon mitral valvotomy (BMV) on effective arterial elastance (Ea). Values are means ± SD; n = 10 subjects. Ea increased in 8 patients and decreased in 2. *P < 0.05 vs. before BMV.
to R (r = 0.96, P = 0.0001). There was a strong linear relationship between Ea and R/T (Ea = 1.09 R/T - 0.01 mmHg/ml, r = 0.99, P = 0.0001) (Fig. 2). R/T slightly but significantly underestimated Ea (Table 2, Fig. 2), especially at high Ea values. There was also a negative linear relationship between Ea and C (r = -0.85, P < 0.01). After the influence of SV was taken into account, Ea and C were still significantly related (partial correlation coefficient (r') = -0.66, P < 0.05). There was no relationship between Ea and ΔP/PAP (r = 0.61, P = 0.15).

**Effects of BMV**

After BMV, Ea increased in eight patients and decreased in two patients (Fig. 1). On average, Ea increased (P < 0.05), whereas C decreased (P = 0.02) (Table 2). MAP increased (from 87.0 to 96.7 mmHg, P = 0.0001), but changes in C were not related to increases in MAP (r = -0.31, P = not significant (NS)). The decrease in C was not related to the increase in Ea (r = 0.25, P = NS) nor to the increase in mitral valve area as induced by BMV (r = 0.41, P = NS). ΔP/PAP increased (P < 0.001) (Table 3). This was linked to an increase in ΔP (from 13.5 ± 8.2 to 18.7 ± 12.7 mmHg, P < 0.001) that was proportionally more marked than the increase in PAP (from 49.3 ± 11.8 to 53.7 ± 16.2 mmHg, P < 0.001).

**Hemodynamic Correlates of Ea After BMV**

Ea was related to R (r = 0.94, P = 0.0001) but not to heart period (r = -0.30). There was a strong linear relationship between Ea and R/T (Fig. 3), and R/T underestimated Ea (Table 2, Fig. 3). There was a negative linear relationship between Ea and C (r = -0.85, P < 0.01). After the influence of SV was taken into account, Ea and C were no longer related (r' = -0.56, P = NS). Increases in Ea were not related to increases in mitral valve area as induced by BMV (r = -0.42, P = NS). There was a positive relationship between Ea and ΔP/PAP (r = 0.82, P = 0.025).

**Evaluation of MAP as an Estimate of ESAP**

There was a powerful linear relationship between ESAP and MAP both before and after BMV in patients either in sinus rhythm or in atrial fibrillation (Fig. 4). When MAP was taken as an estimate of ESAP, MAP underestimated ESAP (P < 0.001) (Fig. 4). There was a negative linear relationship between the MAP-ESAP difference and ESAP, such that the higher the ESAP, the more negative the difference. Table 4 indicates the accuracy of the two empirical formulas (ESAP = 2/3 SAP + 1/3 DAP; and ESAP = 0.9 SAP), both of which significantly overestimated ESAP.
also observed that R/ on R and

Accuracy of the two empirical formulas previously proposed in estimation of end-systolic aortic pressure

Table 4.

DISCUSSION

The present study indicated that Ea depended mainly on R and T in patients with MS studied at baseline. We also observed that R/T was a reasonable estimate of Ea in the study population. These results extended to MS patients the primary results obtained by Sunagawa et al. in animals (25, 26) and by Kelly et al. (12) in human subjects without valve disease. The short-term effects of BMV were also studied. Ea increased after BMV and remained closely dependent on both R and T. Ea also related to C and wave reflections, although to a lesser extent.

Comparison with Previous Studies

Before BMV, Ea (1.55 ± 0.63 mmHg/ml) was lower than the value previously reported (3.1 ± 1.1 mmHg/ml) (14), and this may be explained by the lower ESAP and the higher SV in our study. The higher SV in our study (67 vs. 38 ml in Ref. 14) could be explained by differences in body surface area (1.8 ± 0.3 vs. 1.5 ± 0.12 m²). In our study, the 67-ml SV value was consistent with the 58-ml value previously reported (29, 30). SV did not significantly change after BMV, as reported in some studies (7, 19). Others have reported that SV increased after BMV (14, 29). These disparities may be related to differences in the incidence and severity of mitral regurgitation and atrial shunts after BMV. Alternatively, LV end-diastolic pressure increases after BMV, and Yasuda et al. (30) reported that SV was either increased or unchanged, depending on the capacity of the LV to increase end-diastolic volume.

Relationship Between Ea and R/ T in MS Patients at Baseline and After BMV

Importantly, we documented a powerful link between Ea and R/ T at baseline

\[ Ea = 1.09R/T - 0.01 \text{ mmHg/ml} \]  \hspace{1cm} (6)

On the assumption that −0.01 mmHg/ml is so small as to be negligible, the following equation is obtained

\[ Ea = 1.09R/T = 1/T \cdot (R + 0.09R) \]  \hspace{1cm} (7)

Thus, for a given T, our study indicates that Ea mainly reflects the steady component of afterload (R). One hypothesis could be that the 0.09 R reflects the influence of the unsteady (i.e., pulsatile) component of afterload on Ea. Our study extends to MS patients the previous theoretical study of Sunagawa et al. (25) suggesting that R/T is a reasonable estimate of Ea, as well as the study of Kelly et al. (12) indicating that Ea slightly but consistently overestimates R/T in healthy subjects and aged or hypertensive patients. Recently, Cohen-Solal et al. (4) have shown that the difference between Ea and R/T was ~10% of Ea in both normotensive and hypertensive subjects; the results obtained in MS patients (Eq. 7) are in keeping with that approximation.

To the best of our knowledge, no study has so far documented the effects of BMV on arterial load, as reflected in Ea, C, and the indexes of wave reflection. In our study, Ea significantly increased after BMV, although BMV did not modify the hemodynamic correlates of Ea. We found that

\[ Ea = 1.15R/T - 0.13 \text{ mmHg/ml} \]  \hspace{1cm} (8)

On the assumption that −0.13 mmHg/ml is so small as to be negligible, the following equation is obtained

\[ Ea = 1.15R/T = 1/T \cdot (R + 0.15R) \]  \hspace{1cm} (9)

Thus, after BMV, Ea reflected the nonpulsatile component of afterload (R) rather than the pulsatile one.
(0.15 R), for a given T. The increase in Ea was not related to the increase in mitral valve area as induced by BMV. Because BMV did not modify SV, the increase in Ea was mainly explained by the significant increase in both ESAP and MAP in all patients. In two previous studies (6, 29), MAP was not significantly modified by BMV, but it must be noted that patients were premedicated with atenolol in the study of Wisenbaugh et al. (29), and this may have minimized reflex changes in aortic pressure.

Although the Ea concept is based on the Windkessel model, which also takes C into account, experimental studies have shown that Ea is poorly influenced by C (25, 26). In our MS patients studied at baseline, there was a negative linear relationship between Ea and C both before and after BMV. After the effects of SV were taken into account, this relationship was no longer observed after BMV. Furthermore, the increase in Ea induced by BMV and the decrease in C were not related.

A positive relationship between Ea and ∆P/PAP has been previously reported in normotensive and hypertensive patients (23), a finding also observed in our MS patients after but not before BMV.

Effects of BMV on C and Wave Reflections

Estimated C significantly decreased after BMV. Even though MAP significantly increased, relative changes in C were not related to relative increases in MAP. The decrease in C was related to the increase in mitral valve area induced by BMV. After BMV, PAP significantly increased, and this was consistent with the observed decrease in C (21). The values of PAP, Pi, SAP – Pi, and (SAP – Pi)/PAP significantly increased, thus attesting to enhanced wave reflection, whereas the decreased ∆Pi suggested anticipated timing of wave reflection. A similar hemodynamic pattern has been attributed to decreased C in aged and hypertensive subjects (12, 16, 20, 21). Thus increased and anticipated wave reflection are probably explained by decreased C.

Clinical Implications: End-Systolic Pressure Estimated From Peripheral Arterial Pressure Recordings in MS Patients

Systolic arterial pressure increases from aorta to periphery, according to the so-called "pulse wave amplification" phenomenon. The magnitude of the pulse wave amplification phenomenon varies markedly from one individual to another, depending on body size, sex, age, arterial pressure, and arterial compliance (13, 16, 20). Thus the two formulas previously proposed (12) as estimates of ESAP, namely, 0.9 SAP and 2/3 SAP + 1/3 DAP, are more relevant to central pressure recordings than to noninvasive peripheral pressure recordings. Furthermore, these formulas significantly overestimated ESAP in MS patients (Table 4).

Effective Ea has also been estimated indirectly after having replaced end-systolic pressure by 1) intrabrachial dicrotic notch pressure recorded invasively (1); 2) carotid dicrotic notch pressure measured by using external tonometry (23); and 3) cuff-determined systolic blood pressure (10). The cannulation of the brachial artery is an invasive procedure and therefore not routinely repeatable. The external tonometry technique is not available in all research laboratories, and the accuracy of carotid dicrotic notch pressure as an estimate of central end-systolic pressure, although probable, remains to be validated (23).

Numerous studies and physiological textbooks have reported that one key property of systemic circulation is that mean arterial pressure remains almost constant along the arterial tree, the drop in mean pressure between the ascending aorta and a large peripheral artery being <3 mmHg (21). We have found a powerful relationship between ESAP and MAP in MS patients, as also recently observed in children (22) and in adults without valve diseases (8). Thus, in patients with MS at baseline, one implication of our study is that ESAP could be reasonably estimated by using cuff-determined mean arterial pressure, rather than systolic arterial pressure, according to the following formula: ESAP = 1.09 mean peripheral arterial pressure. Further studies are needed to confirm this.

Limitations of the Study

The limitations of our study need to be discussed. First, given our invasive study design, clinical implications are limited by its short-term aspect. We judged it unethical to perform a left-sided catheterization in MS patients 1 mo after BMV, such that the long-term effects of valvotomy on Ea were not documented in our study. Further studies are needed to document the chronic effects of BMV on Ea. Second, we studied a limited sample size of MS patients. Despite this, we found an unusually powerful relationship both between Ea and R/T, and between ESAP and MAP, and this tends to strengthen the relevance of our results.

Conclusions

Ea depends mainly on R and T in patients with MS studied at baseline or after BMV. The powerful relationship between Ea and R/T observed in our study extends to MS patients the primary results of Sunagawa et al. (25, 26) and Kelly et al. (12). The Ea vs. R/T relationship ultimately depends on the powerful link between MAP and ESAP in MS patients. Given that mean arterial pressure remains constant along the arterial tree, this result may have clinical implications for the noninvasive assessment of Ea in populations similar to ours. Last, in patients with MS, and for a given T, our study indicates that Ea depends mainly on the steady rather than the pulsatile component of arterial load (R), whether before or after BMV.

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