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 in mitral stenosis before and after balloon valvotomy. J. Appl. Physiol. 83(4): 1083–1089, 1997.—This study had the purpose of documenting the hemodynamic correlates of effective arterial elastance (Ea; i.e., an accurate estimate of hydraulic load) in mitral stenosis (MS) patients. The main hypothesis tested was that Ea relates to the total vascular resistance (R)-to-pulse interval duration (T) ratio (R/T) in MS patients both before and after successful balloon mitral valvotomy (BMV). High-fidelity aortic pressure recordings were obtained in 10 patients (40 ± 12 yr) before and 15 min after BMV. Ea 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 on heart period (i.e., T) in MS patients.

Aortic pressure (ESAP) to stroke volume (thermodilution). Ea 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 on heart period (i.e., T) in MS patients.

A precise and complete description of LV afterload (i.e., hydraulic load) is provided by the input impedance of systemic circulation (17, 20, 21), but this complex approach is not always feasible in clinical practice. Sunagawa et al. (25, 26) have proposed an alternative assessment of hydraulic load, namely, effective arterial elastance (Ea). In both healthy subjects and hypertensive patients, Ea has been shown to mainly depend on 1) the R-to-T ratio (R/T) (12, 25, 26), where R is the peripheral resistance and T is heart period, and 2) the magnitude of wave reflections (4, 23). Conversely, Ea poorly depends on total arterial compliance (25, 26).

The above-mentioned studies were performed under baseline conditions, and it remains to be established whether acute load manipulations modify the hemodynamic correlates of Ea. The loading conditions of the heart are dramatically modified in MS patients at both baseline and after balloon valvotomy (27, 28), and this may well modify the hemodynamic correlates of Ea. To the best of our knowledge, only one study has documented Ea values in MS patients (14), and none has examined the effects of percutaneous balloon mitral valvotomy (BMV) on arterial load, as reflected in Ea values.

Accordingly, the purpose of our preliminary study was to document the hemodynamic correlates of Ea in MS patients studied both before and after valvotomy. In our patients, we tested the hypothesis that Ea could relate to R, T, R/T, total arterial compliance, and the indexes of wave reflection.

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 narrowed mitral valve orifice (<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 mitral valve 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), digoxis (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 dlorazepate (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 means of a mathematical formula taking into account the effective volume of circulation (25). The theoretical Ea 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, Ea is currently obtained by calculating the steady-state ratio of ESAP to SV (3, 8). Effective Ea

Theoretical background. In the Ea model, the proximal aorta is considered as an elastic chamber, the effective volume elastance Ea (mmHg/mL) of which is the slope of the relationship between ESAP and SV. This model has markedly improved the evaluation of the systemic circulation for two reasons. First, in humans, Ea 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 Ea (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 ESAP-SV relationships in the pressure-volume plane (24–26). Coordinated changes in the Ees-to-Ea ratio, stroke work, and mechanical efficiency have been reported (1, 11, 25, 26).

The concept of Ea is based on the Windkessel model of arterial circulation (25). Theoretical Ea 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, Ea is currently obtained by calculating the steady-state ratio of ESAP to SV (4, 11, 12). The hemodynamic correlates of Ea have been documented in experimental studies and in studies performed on normotensive and hypertensive subjects without valve disease. In this population, Ea 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 Ea (12). Although experimen-

| Subject No. | Age, yr | Gender | Body Surface Area, m² | NYHA Therapy | MVA, cm² |
|-------------|---------|--------|----------------=======|--------------|---------|
| 1           | 32      | M      | 2.07                  | II           | 1.0     |
| 2           | 22      | F      | 2.25                  | II           | 1.0     |
| 3           | 42      | F      | 1.48                  | III          | 0.8     |
| 4           | 35      | F      | 1.55                  | II           | 0.8     |
| 5           | 51      | M      | 2.01                  | III          | 1.0     |
| 6           | 40      | F      | 1.58                  | II           | 1.0     |
| 7           | 37      | F      | 1.37                  | II           | 0.9     |
| 8           | 32      | F      | 1.71                  | II           | 1.0     |
| 9           | 31      | F      | 1.82                  | II           | 1.0     |
| 10          | 68      | F      | 1.92                  | II           | 1.0     |

Mean ± SD 40 ± 12 1.78 ± 0.29 0.92 ± 0.11 1.97 ± 0.26

M, male; F, female; NYHA, New York Heart Association classification; MVA, mitral valve area; Before, before mitral valvotomy; After, after mitral valvotomy; F, furosemide; D, digitalis; AC, anticoagulant therapy; A, amiodarone; FL, flecainide; N, nitrates. * P < 0.01.
nal 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

\[
Ea = ESAP/SV
\]

(2)

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

\[
Ea = R/T
\]

(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

\[
C = SV/K(ESAP - EDAP)
\]

(4)

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

\[
K = \frac{(systolic \ area + diastolic \ area)}{diastolic \ area}
\]

(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 significant (≥2+) aortic 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 the pressure waveform. The time from the foot of the pressure wave to Pi (ΔtP) is because of arterial wave reflection and the relative increase in the height of the mid-to-late systolic peak pressure above the aortic insufficiency (4, 23). 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></th>
<th>Before BMV</th>
<th>After BMV</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>P, 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>219.9 ± 10.5</td>
<td>287.9 ± 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 arterial pressure (SAP) minus diastolic arterial 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.

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

<table>
<thead>
<tr>
<th></th>
<th>Before BMV</th>
<th>After BMV</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ea, mmHg/ml</td>
<td>1.55 ± 0.63</td>
<td>1.85 ± 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>Effective arterial compliance, mmHg/ml</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 = 7 subjects. All data were averaged out over 10 consecutive cardiac cycles. PAP, pulse aortic pressure; systolic arterial pressure (SAP) minus diastolic arterial 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. 2. A: $E_a$ as a function of total vascular resistance ($R$)-to-pulse interval duration ($T$) ratio ($R/T$) at baseline ($n = 10$). $E_a$ was positively related ($r = 0.99, P = 0.0001$) to $R/T$ in accordance with following equation: $E_a = 1.09R/T - 0.01$ (mmHg/ml). B: $R/T$ as an estimate of $E_a$ at baseline. Solid line, mean difference (i.e., $R/T - E_a$); dashed lines, ±2 SD; dotted line, zero axis. There was a positive linear relationship ($r = 0.64, P = 0.046$) between difference and $E_a$: $R/T - E_a = -0.09 E_a + 0.03$.

Effects of BMV

After BMV, $E_a$ increased in eight patients and decreased in two patients (Fig. 1). On average, $E_a$ 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 ($r = -0.31, P = n$-significant (NS)). The decrease in $C$ was not related to the increase in $E_a$ ($r = 0.25, P = NS$) nor to the increase in mitral valve area as induced by BMV ($r = 0.41, P = NS$). $\Delta P/PAP$ increased ($P < 0.001$) (Table 3). This was linked to an increase in $\Delta P$ (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 $E_a$ After BMV

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

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.
DISCUSSION

The present study indicated that $E_a$ depended mainly on $R$ and $T$ in patients with MS studied at baseline. We also observed that $R/T$ was a reasonable estimate of $E_a$ 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. $E_a$ increased after BMV and remained closely dependent on both $R$ and $T$. $E_a$ also related to $C$ and wave reflections, although to a lesser extent.

Comparison with Previous Studies

Before BMV, $E_a$ (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 $E_a$ and $R/T$ in MS Patients at Baseline and After BMV

Importantly, we documented a powerful link between $E_a$ and $R/T$ at baseline.

$$E_a = 1.09R/T - 0.01 \text{ mmHg/ml}$$

On the assumption that $-0.01 \text{ mmHg/ml}$ is so small as to be negligible, the following equation is obtained:

$$E_a = 1.09R/T = 1/T \cdot (R + 0.09R)$$

Thus, for a given $T$, our study indicates that $E_a$ 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 $E_a$. Our study extends to MS patients the previous theoretical study of Sunagawa et al. (25) suggesting that $R/T$ is a reasonable estimate of $E_a$, as well as the study of Kelly et al. (12) indicating that $E_a$ 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 $E_a$ and $R/T$ was ~10% of $E_a$ 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 $E_a$, $C$, and the indexes of wave reflection. In our study, $E_a$ significantly increased after BMV, although BMV did not modify the hemodynamic correlates of $E_a$. We found that

$$E_a = 1.15R/T - 0.13 \text{ mmHg/ml}$$

On the assumption that $-0.13 \text{ mmHg/ml}$ is so small as to be negligible, the following equation is obtained:

$$E_a = 1.15R/T = 1/T \cdot (R + 0.15R)$$

Thus, after BMV, $E_a$ reflected the nonpulsatile component of afterload ($R$) rather than the pulsatile one

<table>
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<tr>
<th>Table 4. Accuracy of the two empirical formulas previously proposed in estimation of end-systolic aortic pressure</th>
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<tr>
<td><strong>(2/3 SAP + 1/3 DAP) – ESAP</strong></td>
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<tr>
<td><strong>Before</strong></td>
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<tr>
<td>Mean error, mmHg</td>
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<tr>
<td>SD</td>
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<tr>
<td>95% CI, mmHg</td>
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<tr>
<td>$r$</td>
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<tr>
<td>$P$</td>
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</table>

Values are means ± SD; n = 10 subjects. DAP, diastolic aortic pressure; ESAP, end-systolic aortic pressure; CI, confidence interval. Mean error, standard deviation, and CI of estimation of ESAP by each formula are calculated over 100 beats (i.e., 10 consecutive beats in each patient) at basal state before BMV, 100 beats 15 minutes after BMV, and 200 beats before and after BMV. $P$ and $r$ are calculated by using a linear regression between ESAP and each formula.
related. The increase in $E_a$ was not related to the increase in mitral valve area as induced by BMV. Because BMV did not modify SV, the increase in $E_a$ 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 $E_a$ concept is based on the Windkessel model, which also takes $C$ into account, experimental studies have shown that $E_a$ is poorly influenced by $C$ (25, 26). In our MS patients studied at baseline, there was a negative linear relationship between $E_a$ 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 $E_a$ induced by BMV and the decrease in $C$ were not related.

A positive relationship between $E_a$ and $\Delta P/PAP$ 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 not 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 $\frac{2}{3} SAP + \frac{1}{3} DAP$, 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 $E_a$ has also been estimated indirectly after having replaced end-systolic pressure by 1) intrabronchial 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: $ESAP = 1.09 \times MAP$.

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 $E_a$ were not documented in our study. Further studies are needed to document the chronic effects of BMV on $E_a$. Second, we studied a limited sample size of MS patients. Despite this, we found an unusually powerful relationship both between $E_a$ and $R/T$, and between ESAP and MAP, and this tends to strengthen the relevance of our results.

Conclusions

$E_a$ depends mainly on $R$ and $T$ in patients with MS studied at baseline or after BMV. The powerful relationship between $E_a$ 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 $E_a$ 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 $E_a$ in populations similar to ours. Last, in patients with MS, and for a given $T$, our study indicates that $E_a$ depends mainly on the steady rather than the pulsatile component of arterial load ($R$), whether before or after BMV.

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