Retrograde Flow in the Thoracic Aorta in Patients With Systemic Emboli*

A Transesophageal Echocardiographic Evaluation of Mobile Plaque Motion

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Study objectives: Blood flow in the aorta is complex and incompletely characterized. Mobile aortic plaques (MAPs), moving freely with the pulsatile aortic flow, in fact represent natural tracers that reflect the flow pattern itself. Our aim was to use MAP motion on transesophageal echocardiography (TEE) in order to characterize flow patterns in the atheromatous thoracic aorta of patients with systemic emboli.

Design and patients: The study group was recruited from 250 patients referred for TEE to evaluate recent embolism. Among them, 22 patients (14 men and 8 women; mean ± SD age, 66.3 ± 7.2 years; 16 patients with cerebrovascular and 6 patients with peripheral emboli) with MAPs of ≥ 3 mm in length formed the study group. The longest amplitudes of three spatial components of mobile lesion motions were measured: x (antegrade/retrograde [A/R]), y (up/down [U/D]), and z (right/left [R/L]).

Results: A total of 33 mobile lesions were detected: 3 in the ascending aorta (1 patient), 13 in the arch (10 patients), and 17 in the descending aorta (11 patients). The length of mobile plaque components ranged from 3 to 13 mm; amplitudes of A/R, U/D, R/L, and retrograde flow motions ranged from 3 to 26 mm, from 1 to 16 mm, from 1 to 17 mm, and from 1 to 13 mm, respectively. Systolic rotational motion was clockwise in six patients (27%), counterclockwise in five patients (23%), incomplete (semicircle) in six patients (27%), and alternate clockwise/counterclockwise in five patients (23%). Diastolic rotational motion was clockwise in 5 patients (23%), counterclockwise in 6 patients (27%), and incomplete (semicircle) in 11 patients (50%). There were 18 multiple MAPs in seven patients: in all these cases, simultaneous rotations of MAP in different directions (as a marker for the presence of multiple vortices) were found. In nine patients with cerebral embolism, MAPs on the distal part of aortic arch solely were found; in five of them, all alternative potential sources of stroke were excluded. Therefore, retrograde cerebral embolism from distal aortic plaques in these patients is highly probable.

Conclusions: Retrograde and rotational blood flow in the thoracic aorta probably exists in all patients with systemic emboli and mobile protruding aortic atheromas. Therefore, retrograde cerebral embolism from distal aortic plaques is theoretically possible.

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Key words: blood flow; echocardiography; embolism; stroke; thoracic aorta

Abbreviations: A/R = antegrade/retrograde; MAP = mobile aortic plaque; R/L = right/left; TEE = transesophageal echocardiography; U/D = up/down

Blood flow in the thoracic aorta is complex and as yet incompletely characterized. During the last 2 decades, the presence of vortex, helical, and reversed flow in aortic models and in both animals and humans has been recognized.1–5 However, there are very limited data regarding discrepancies between the flow pattern in normal subjects and in patients with varying clinical conditions.5 All previously used methods, like Doppler ultrasound, MRI, and invasive procedures, presented significant limitations.6–8 The sites of deposition of atherosclerotic plaques are considered to be influenced by flow patterns that cause regions of altered shear stress on the aortic wall,3,9,10 whereas more unstable flow predisposes to
atherogenesis. In turn, aortic plaquing impairs aortic distensibility and causes lumen encroachment, influencing blood flow.\textsuperscript{2,11,12}

Protruding aortic plaques—especially with mobile properties on transesophageal echocardiography (TEE)—have been established as an important potential source for stroke and systemic emboli in elderly patients.\textsuperscript{13–18} Mobile aortic plaques (MAPs) moving freely back and forth with the pulsatile aortic flow in fact represent natural tracers that reflect the flow pattern itself. Motion patterns of MAP are not fully understood. Therefore, the purpose of the present study was to use MAP motion on TEE to characterize flow patterns in the atheromatous thoracic aorta of elderly patients with stroke and systemic emboli.

**Materials and Methods**

**Patients**

The study group was recruited from 250 consecutive patients referred for TEE to evaluate recent embolism. The criterion for inclusion was the presence of at least one plaque in the thoracic aorta with a mobile component of $\geq 3$ mm in length moving freely with the aortic flow. Patients presenting with significant aortic insufficiency (grade $>2$) were excluded.

Of these 250 patients, 45 patients (18\%) had MAPs of various sizes and morphology. Among them, 22 patients (14 men and 8 women; 16 patients with cerebrovascular emboli and 6 patients with peripheral emboli) fulfilled admission criteria and constituted the study group. Mean $\pm$ SD age was $66.3 \pm 7.2$ years (range, 50 to 85 years).

**TEE**

All studies were performed with a Hewlett-Packard Sonos 1000, 1500, or 2500 (Hewlett-Packard; Andover, MA) ultrasound system employing 5-MHz multiplane transducers. Light sedation with midazolam and lidocaine 10\% topical spray were used. All visible regions of the thoracic aorta were fully explored in every patient, including the proximal ascending aorta beginning at the aortic valve. The descending aorta and the arch were visualized in multiple views, including both longitudinal and transversal axes. All studies were recorded on a tape recorder (Panasonic 6200 Super VHS; Matsushita Electric; Osaka, Japan). The frequency and distribution of mobile plaques in different regions of the thoracic aorta (ascending, arch, descending) were analyzed. In patients where multiple mobile lesions were detected, only the longest one was selected for the following quantitative analyses. However, the interrelationship between all detected multiple mobile lesions during the cardiac cycle was analyzed separately in this subgroup of patients.

For the purposes of analysis, each plaque was divided into fixed and mobile parts. The mobile part of atheroma length was defined as the longest measured distance between the fixed/mobile parts interface and the external free side of the mobile lesion.

The longest amplitudes of three components of mobile lesion motion during the cardiac cycle were measured: $x$ (antegrade/retrograde [A/R], longitudinal TEE view [Fig 1]), $y$ (up/down [U/D], both longitudinal and transverse views) and $z$ (right/left [R/L], transverse view).

Since the anatomic interrelationship between the esophagus and the thoracic aorta is complex in the elderly, and dependent on individual variations and aortic tortuosity, it was not possible to establish true spatial coordinates on TEE, except for A/R direction. Therefore, the $y$ axis and $z$ axis in this article refer to U/D and R/L screen-oriented directions, respectively, on longitudinal and transverse TEE views. In addition, the longest amplitude of retrograde mobile component motion was calculated as the
distance between the proximal fixed/mobile parts interface and
the point of maximal free edge of the mobile lesion back position.
The presence of rotational flow (screen oriented clockwise and
counterclockwise motion) was determined on transverse view. In
the subgroup of patients with multiple mobile lesions, multiple
vortices were suggested to be present when simultaneous rota-
tions in different directions were found. All measurements were
made off-line from the recorded images.

Statistical Analysis

Continuous variables were expressed as a mean value (±1 SD)
and compared using paired t test. Pearson correlation coefficients
(\(r\)) between the mobile part of atheroma length and amplitudes
of the motion in various directions were calculated. A p value of
< 0.01 was considered significant.

RESULTS

Of 22 patients, 15 patients (68%) exhibited single
MAPs and 7 patients (32%) had multiple MAPs
(ranging from two to five per patient). A total of 33
mobile lesions were detected: 3 in the ascending
aorta (1 patient), 13 in the arch (10 patients), and 17
in the descending aorta (11 patients). There was no
significant difference between the arch and the
descending aorta with respect to the frequency of
the mobile plaques in these regions, whereas the
ascending aorta presented the lowest prevalence for
atheromatosis.

The length of mobile plaque components ranged
from 3 to 13 mm; amplitudes of A/R, U/d, and R/L
motions ranged from 3 to 26 mm, from 1 to 16 mm,
and from 1 to 17 mm, respectively (Table 1). There
were very close correlations among amplitudes of
motion in all directions and length of mobile com-
ponents (Table 2).

Diastolic retrograde flow was observed in all pa-
tients, and its amplitude ranged from 1 to 13 mm. Of
16 patients with stroke, 7 patients had MAPs located
proximally or between the origins of cervical arteries
and 4 patients had MAPs distally from the origin of
left subclavian artery, but concomitant possible rea-
sons for stroke were also present: atrial fibrillation in
2 patients and significant carotid stenosis in 2 pa-
tients. The remaining five patients exhibited MAPs
located on the distal part of aortic arch just distally to
the origin of left subclavian artery (within 1 cm), and
all alternative potential sources of stroke (such as
proximal aortic atheroma on TEE and CT, atrial
fibrillation, carotid stenosis, intracardiac thrombi,
and patent foramen ovale) were excluded in these
patients. Since retrograde aortic flow was registered
in all of them, retrograde cerebral embolism from
distal aortic plaques could theoretically be present.

Rotational components in MAP motion could be
suggested in all patients. Systolic motion was clock-
wise in six patients (27%), counterclockwise in five

<table>
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<th>Patient</th>
<th>Length, mm</th>
<th>Mul MAP</th>
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<th>D Rot</th>
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<th>U/d</th>
<th>R/L</th>
<th>RF, mm</th>
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<tr>
<td>Mean</td>
<td>66 ± 7.2</td>
<td>7.3 ± 3.3</td>
<td>11.5 ± 6.3</td>
<td>5.5 ± 3.6</td>
<td>5.7 ± 4.0</td>
<td>4.9 ± 2.9</td>
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*Data are presented as No. or mean ± SD. Mul MAP = presence (+) or absence (-) of multiple MAPs; G = gender; Loc = location; D Rot =
diastolic rotation; S Rot = systolic rotation; RF = retrograde flow; C = clockwise; CC = counterclockwise; M = male; F = female; A = alternate;
I = incomplete; Asc = ascending aorta; Desc = descending aorta.
patients (23%), incomplete (semicircle) in six patients (27%), and alternate clockwise/counterclockwise in five patients (23%). Diastolic motion was clockwise in 5 patients (23%), counterclockwise in six patients (27%), and incomplete (semicircle) in 11 patients (50%). In addition, periodically chaotic irregular multidirectional MAP motions were registered in all patients. There were 18 multiple MAPs in seven patients: in all these patients, simultaneous rotations of MAPs in different directions (highly suggestive for the presence of multiple vortices) were found (Fig 2).

**Table 2—Pearson Correlation Coefficients (r) Between the Mobile Component of Atheroma Length and Amplitudes of their Motion in Various Directions**

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<tr>
<th>MAP Motion</th>
<th>r</th>
<th>p Value</th>
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<tr>
<td>A/R</td>
<td>0.94</td>
<td>0.0001</td>
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<tr>
<td>U/d</td>
<td>0.70</td>
<td>0.0001</td>
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<tr>
<td>R/L</td>
<td>0.76</td>
<td>0.0001</td>
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<tr>
<td>Retrograde flow</td>
<td>0.80</td>
<td>0.0001</td>
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</table>

**Discussion**

The main finding of our study is direct evidence (obtained from TEE analyses of MAP motion) that diastolic retrograde flow and probably both systolic and diastolic rotational flows occur everywhere in the atheromatous thoracic aorta among elderly patients with stroke and systemic embolism, regardless of the presence of significant aortic regurgitation. Therefore, our data suggest that retrograde systemic embolism from distal aortic plaques is theoretically possible.

The presence of helical and retrograde flows in the aorta has been observed previously using magnetic resonance,4,8,19 and color Doppler ultrasound velocity mapping.2,5 However, these data were obtained mainly in healthy volunteers and considerable limitations have been reported.7,8 There are very limited data regarding discrepancies between the flow pattern in normal subjects and in patients with ischemic heart disease.5 Frazin et al described one case of systolic clockwise motion of a large mobile component in a patient with an atherosclerotic aorta. To the best of our knowledge, the present study is the first report using a systematic analysis of MAP motion on TEE as a method to characterize flow patterns in atheromatous thoracic aorta of elderly patients with stroke and systemic embolism. Freely mobile aortic plaques exposed to the pulsatile aortic flow field in fact represent partial flow tracers, indicating the vector of force that results from the spatial components at any given time.

**Retrograde and Rotational Flow in the Thoracic Aorta**

Flow in the thoracic aorta is extremely complex and incompletely characterized. The partial analyses of the variables influencing the aortic fluid dynamics should deal with the following points: arch curvature, flow pulsatility, elasticity of the aorta, protruding aortic atheromatosis, and diastolic coronary flow. The interactions among these factors have important effects: retrograde flow20–22 and rotational flow19,23, as well as creation of vortices, become very likely. Among our population, in all cases where multiple mobile lesions were present, simultaneous rotations of MAP in different directions—highly suggestive for the presence of multiple vortices—were found.

In our study, patients presenting with significant aortic insufficiency (grade > 2) were excluded. Obviously, aortic insufficiency can lead to an additional augmentation of the retrograde flow.

**Aortic Plaques and Systemic Embolism**

Stroke and systemic embolism from atheromas in the thoracic aorta were previously thought to be rare,
Both calcified and soft plaques in these upper part of the ascending aorta and the proximal column in the trachea and the left bronchus, TEE thoroughly elucidated; due to interposition of the air mas among patients with embolism has not yet been reported with increasing frequency. A number of studies have evaluated the significance of the mobile lesions are associated with an increased risk for stroke and peripheral embolism. The majority of these studies suggest that retrograde cerebral embolism and mobile protruding aortic atheromas in association with embolic events. The true prevalence of protruding aortic atheromas among patients with embolism has not yet been thoroughly elucidated; due to interposition of the air column in the trachea and the left bronchus, TEE frequently does not allow full visualization of the upper part of the ascending aorta and the proximal arch. Both calcified and soft plaques in these important regions around the origin of the cervical arteries may be undetected on TEE. Therefore, the role of these atheromas as a source of embolism may be underestimated at present, especially in patients with an undetermined cause of the event.

Furthermore, the protruding atheromas situated just distally after the origins of cervical arteries in stroke patients are considered as unrelated to cerebral embolism and consequently remain untreated. We suggest the possibility of retrograde cerebral embolism from distal aortic plaques in stroke patients in whom all alternative potential sources of stroke (as proximal aortic atheroma on TEE and CT, atrial fibrillation, carotid stenosis, intracardiac thrombi, and patent foramen ovale) are excluded.

In a recent study, Dressler et al demonstrated impressive benefits from warfarin treatment in patients with aortic plaques and stroke. Therefore, our concept regarding retrograde sources of ischemic stroke may have significant implications in the study of the pathogenesis and management of embolic events.

The length of the mobile component of the plaque among our patients ranged from 3 to 13 mm; amplitude of retrograde MAP motion ranged from 1 to 13 mm. There was a very close correlation between amplitude of retrograde MAP motion and length of mobile component \((r = 0.80)\). Our analysis has established the presence of retrograde aortic flow in all study patients, but could not determine the true extent of this flow because the movement of the mobile plaque was limited by its own “rein.”

**Conclusion**

Retrograde and rotational blood flow in the thoracic aorta probably exists in all patients with systemic emboli and mobile protruding aortic atheromas. Therefore, retrograde cerebral embolism from distal aortic plaques is theoretically possible. This issue may have significant implications in the study of atherosclerosis development and the pathogenesis of embolic events.

**References**

patterns in the human aorta studied by magnetic resonance. Br Heart J 1987; 58:316–323

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