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Regional Left Atrial Stasis During Atrial Fibrillation and Flutter: Determinants and Relation to Stroke

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Objectives. **This study sought to 1) determine the location of left atrial stasis during atrial arrhythmia; 2) define the degree of stasis associated with significant risk of stroke; and 3) identify clinical or transthoracic echocardiographic data useful for predicting left atrial stasis.**

Background. **Prior studies suggest that stroke during atrial arrhythmia is related to stasis in either the body of the left atrium or the appendage. Recent data indicate that appendage stasis is associated with appendage thrombus formation, but stroke during atrial arrhythmia occurs frequently in the absence of appendage stasis.**

Methods. **Blood flow velocity was measured in multiple sites in the body of the left atrium and in the appendage by transesophageal pulsed wave Doppler echocardiography in 89 patients with atrial fibrillation or flutter. Regional velocities were related to the frequency of probable embolic stroke and to clinical and transthoracic echocardiographic variables.**

Results. **The lowest velocity region was either the posterior left atrium or the appendage. Stroke frequency increased progressively and steeply with velocity <15 cm/s in either region; this cutoff value had an 87% sensitivity and 40% specificity for stroke. Factors related to stasis were low left atrial ejection fraction, mitral regurgitation <3+, fibrillation (vs. type I flutter), left ventricular dilation and mitral valve area <2.0 cm^2.**

Conclusions. **Posterior left atrial stasis appears to be as important as appendage stasis for the risk of stroke, which increases steeply with lower blood flow velocity in either region. Patients likely to have severe stasis during atrial arrhythmia are those with left ventricular dilation and low atrial ejection fraction accompanying left atrial dilation. Direct measurement of atrial velocity by transesophageal echocardiography appears to be useful for the identification of patients at risk for stroke during atrial arrhythmia.**

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Many studies suggest a strong association between cardioembolic stroke during atrial arrhythmia and stasis of blood in the left atrium (1–8). Stroke-related factors include mitral orifice obstruction, left atrial dilation and left ventricular dysfunction, whereas factors thought to protect against stroke include significant mitral regurgitation and type I or "classical" flutter (9–11). Transesophageal echocardiographic data have strengthened the relation of stasis to stroke by linking stroke to spontaneous contrast in the atrium. Although patients with embolism only infrequently have thrombi visualized, when thrombus is seen it is almost always in the appendage. Recent studies have established an association of low appendage velocity with appendage thrombus (4,6,8,12–18). Surprisingly, these same studies found appendage stasis or thrombosis, or both, to be either weakly related or unrelated to cardioembolism (13,15,17). This paradox raises the possibility that factors other than appendage stasis may lead to stroke during atrial arrhythmia.

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Several observations point to stasis in the body of the atrium as important in the pathogenesis of stroke. Mitral regurgitation reduces spontaneous contrast in the atrial body and is thought to reduce stroke, yet it appears to have little or no effect on stasis or thrombosis in the appendage (17,18). Appendage stasis has not been reported during typical flutter, yet stroke does occur during this arrhythmia. Appendage stasis has been associated with stroke-related factors, such as atrial dilation, in some studies (14,16,17) but not in others (12,15,17), and in no study has it been related to left ventricular dysfunction.

In many studies, stroke has been strongly associated with spontaneous contrast in the body of the left atrium, in contrast to the weak or absent association of stroke with appendage stasis (4,6,8). Although thrombi are seen much less often in the atrial body than in the appendage, this could be explained by thrombus persistence in the appendage due to its specific anatomy. Thus, the available data suggest that stasis in the atrial body may lead to stroke, perhaps as a result of transient thrombi shed from the atrial walls or the surfaces of valve leaflets.

Although velocity values accompanying appendage thrombosis have been reported (17,18) (8 to 15 cm/s), no velocity criterion for stasis in either the atrial body or appendage has been related to stroke. This deficiency impedes efforts to

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Table 1. Clinical Characteristics of 89 Study Patients

Age (yr)	
Mean	67
Range	47–85
Men	84 (94%)
Women	5 (6%)
Arrhythmia	
Fibrillation	61 (69%)
Type I flutter	16 (18%)
Type II flutter	12 (14%)
No known heart disease	20 (23%)
Coronary artery disease	47 (53%)
LVEF <50%	37 (42%)
Pulmonary disease	26 (29%)
Mitral stenosis	5 (6%)
Mitral prosthesis	4 (5%)
3–4+ MR	25 (28%)
Arrhythmia duration (days)	
Mean	211
Range	1–721
Arrhythmia duration <7 days	8 (9%)
Stroke during arrhythmia*	15 (17%)

*Probable embolic stroke. Data presented are number (%) of patients, unless otherwise indicated. LVEF = left ventricular ejection fraction; MR = mitral regurgitation.

identify and quantitate clinical and echocardiographic features identifying patients likely to have significant stasis. Such a criterion could be useful to guide the selection of patients with atrial arrhythmia for antithrombotic therapy. Therefore, the purposes of this study were to 1) demonstrate by transesophageal echocardiography where stasis occurs in the left atrium during atrial arrhythmias; 2) determine the velocity value representing the degree of stasis related to stroke; and 3) identify data obtainable clinically or by transthoracic echocardiography that would be useful in identifying patients at risk for stasis and stroke.

Methods

Subjects. Eighty-nine patients with atrial fibrillation or flutter of diverse etiologies were studied (Table 1). All patients gave written informed consent, and the protocol was approved by the Human Research Review Committee of the University of New Mexico.

Stasis quantitation. Blood flow velocity in multiple sites in the left atrial body and appendage was recorded by transesophageal pulsed wave Doppler. Monoplane (49 patients) and multiplane (40 patients) transesophageal echocardiography were used to obtain the standardized four-chamber view through the left atrium. The Doppler sample volume length was 1 cm, and the low frequency filter was set to the lowest setting (50 Hz). Velocities were recorded at sites in the left atrial body and appendage, as shown in Figure 1, A and C. To ensure that velocities in the sites shown in Figure 1A were representative of those elsewhere in the left atrial body, additional sites were measured in a subset of 16 patients in the two-chamber view, as shown in Figure 1B.

The probability of thrombosis was assumed to be inversely related to the maximal velocity during the cardiac cycle. Systolic and diastolic maximal velocities were measured, whether above or below the baseline of the spectral display, and averaged for five to seven cardiac cycles. The highest velocity, whether systolic or diastolic, was used as the measure of stasis.

On the basis of an analysis of velocity concordance between sites (Shively BK, unpublished data), velocities in sites 1 to 11 were grouped into regions: *anterior* (nearest the mitral valve, sites 1 and 7), *center* (sites 6 and 8 to 10), *posterior* (furthest from the valve, sites 2 to 5) and *appendage* (site 11). The velocities of the sites within each region were averaged for further analyses. Representative velocity recordings in each region are shown in Figure 2. The velocity concordance analysis also demonstrated that velocities in sites 12 to 19 (longitudinal plane) were similar to those of the corresponding regions sampled in the horizontal plane.

Mitral regurgitation. Severity of mitral regurgitation was graded on a scale of 0 to 4+ by an independent observer using previously described transesophageal echocardiographic criteria (19,20). To permit identification of localized atrial stasis despite varying degrees of mitral regurgitation, sites demonstrating effects of the jet were excluded from analysis. The type of jet effect depended on the proximity of the pulsed wave sample volume to the jet and was either 1) an aliased high velocity signal limited to systole, or 2) rapidly fluctuating partially aliased or nonaliased velocities during late systole and early or mid-diastole. Regional velocities were calculated from only those sites within the region not demonstrating these jet effects. A region was treated as missing data if all sites within that region were omitted due to jet effects.

Arrhythmia. The atrial rate was the mean of five consecutive appendage-emptying velocity pulses. *Atrial rhythm* was classified as either type I or "classical" atrial flutter, type II flutter or fibrillation. *Type I flutter* was defined prominent inverted f waves on electrocardiographic leads II, III and aVF and a rate of 240 to 320 beats/min. All other flutter was classified as type II (21). Patients with atrial fibrillation or type II flutter, 0 to 2+ mitral regurgitation and a mitral valve area >2.0 cm^2 were classified as having nonvalvular atrial fibrillation (none of these patients had rheumatic valve disease).

Additional factors related to atrial stasis. Stroke volume, mitral valve area, left atrial diameter and volume and left ventricular volumes and ejection fraction were calculated by established methods (22–26). Left atrial ejection fraction was calculated as the stroke volume divided by left atrial volume. Mitral annular calcification was graded on a 0 to 4+ scale by an independent observer. Clinical factors were age, diabetes mellitus, hypertension, congestive heart failure, prior embolic event, current smoking and coronary disease or prior myocardial infarction.

Spontaneous contrast and thrombus. Spontaneous contrast in the atrial body and appendage was graded on a scale of 0 to 4+, and the presence of thrombus was reported by an independent observer. Contrast was classified as follows: 1+ if

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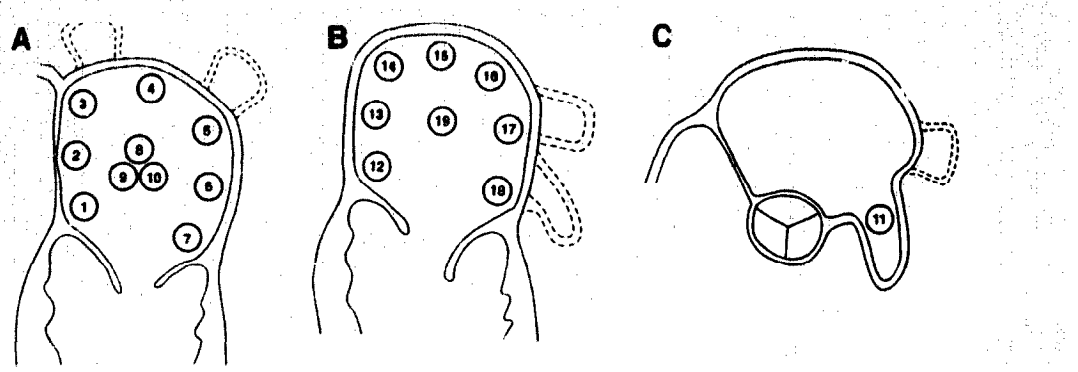

Figure 1. **A,** Schematic of horizontal transesophageal image plane of the left atrium (a portion of the four-chamber view) showing sites of velocity measurement. Site 8 is obtained with the transesophageal probe in position to record the four-chamber view. Site 9 is obtained by advancing the transesophageal probe 1 cm and site 10 by withdrawing 1 cm from the four-chamber view position. Sites 1 and 7 were combined as anterior; 6 and 8 to 10 as center; and 2 to 5 as posterior on the basis of location and concordance analysis (see Methods). **B,** Schematic of longitudinal transesophageal image plane of the left atrium (a portion of the two-chamber view) showing sites of velocity measurement. Sites 12 and 18 are combined as anterior; 13, 17 and 19 as center; and 14 to 16 as posterior on the basis of location and concordance analysis (see Methods). **C,** Schematic of transesophageal short-axis view of the aortic valve, showing velocity sampling site 11 located 1 cm inside the ostium of the left atrial appendage.

swirling reflectors were barely discernible; 2+ if swirling reflectors were immediately apparent, but concentrations of contrast dissipated in ≤1 s; 3+ if any contrast concentrated into strands or clumps dissipated in up to ~5 s; and 4+ when nearly stationary or rapidly oscillating strands or clumps were observed.

Stroke. The diagnosis of probable cardioembolic stroke was made in 15 patients utilizing clinical and computed tomographic scan criteria for cardioembolism of the National Institute of Neurological and Communicative Disorders and Stroke Pilot Stroke Data Bank (27). All patients had stroke during atrial arrhythmia and a deficit documented by neurologic examination. All but one patient had a cerebral infarction by computed tomography or magnetic resonance imaging matching the neurologic deficit. Twelve of the 15 patients had carotid duplex scans, and findings were negative in all (stenosis category no more than 16% to 49%); 4 additional patients with stroke were previously excluded because of positive carotid scan findings (stenosis ≥50%). Except for two patients with stroke after velocity measurements, the mean interval between stroke and measurement of atrial velocities was 21.6 weeks (range 0 to 68). The percent of patients with stroke with hypertension (50%), diabetes (31%), peripheral vascular disease (6%) and smoking (19%) was similar to that of patients without stroke. Two patients with stroke were taking warfarin, but levels were subtherapeutic at the time of the event; 39 (53%) patients without stroke were taking warfarin.

Interobserver and intraobserver variability in atrial velocity measurements. *Interobserver variability* in the measurement of velocity in atrial sites 1 to 11 was assessed by calculation of mean percent difference between results obtained by two trained observers for 12 randomly selected patients. Selection of cardiac cycles for measurement of maximal velocity was made independently by the observers. Mean variability ranged between ±12% for site 1 and ±19% for site 4. Variability for site 11 (appendage) was ±13%. *Intraobserver variability* was assessed by remeasurement of data for 12 randomly selected patients at least 1 month after initial measurement. Mean variability ranged from ±5% for site 1 to ±11% for site 5. Variability for site 11 was ±8%.

Statistical analysis. Results are presented as mean value ± SD, unless otherwise specified. Stroke frequencies at different velocity ranges and spontaneous contrast categories were tested for significance using the Mantel-Haenszel chi-square test. Other differences in velocities between two or more regions or patient groups were tested by two-tailed Student *t* test or one-way analysis of variance (Newman-Keuls test). Associations between continuous variables were tested using Pearson or Spearman correlations as appropriate. Chi-square or Fisher exact tests were used for sensitivity and specificity data. A p value < 0.05 was considered significant. Stepwise regression analyses were performed to select variables predictive of regional velocity at $p = 0.15$. Analyses were performed using SAS version 6.08 for personal computers (SAS, Inc).

Results

Regional atrial velocities. The lowest velocities in the atrial body were found in the posterior region and the widest range in the appendage (Fig. 3). Velocities in regions of the atrial body were highly correlated ($r = 0.61$ to $r = 0.77$, $p < 0.0001$) but less well correlated with appendage velocity ($r = 0.46$ with posterior velocity). Because of the occurrence of the lowest velocities in either the posterior atrium or appendage in 84%

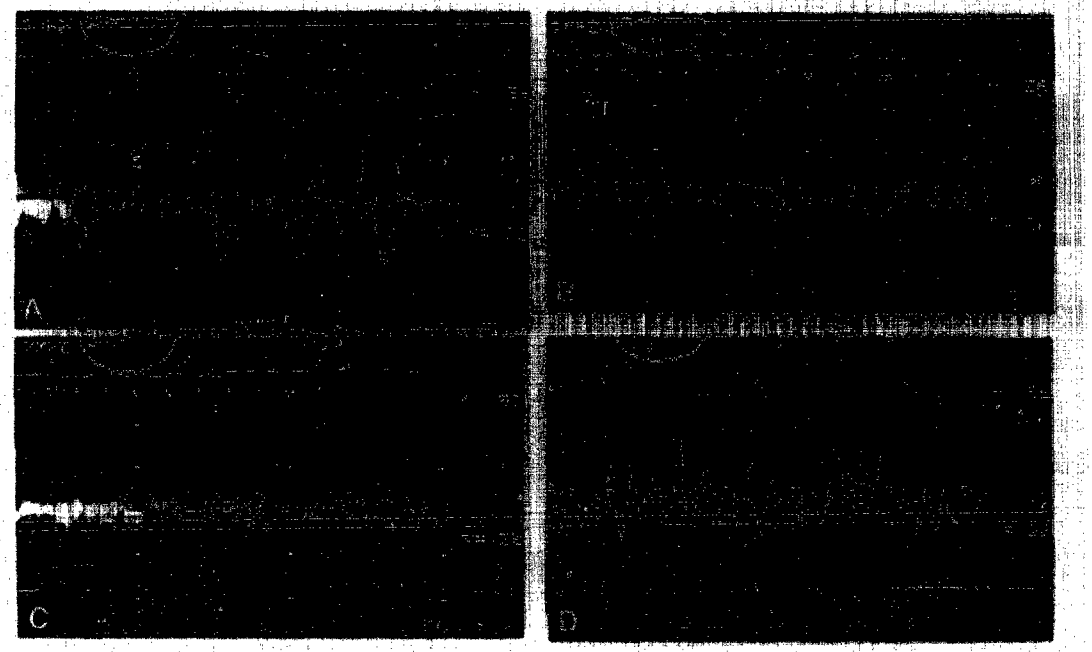

Figure 2. Velocity recordings from the atrial regions of a representative patient. The anterior region recording (A) demonstrates the prominent early diastolic maximal velocity, which is less apparent in the lower velocity center region (B) and often not apparent in the lowest velocity posterior region (C). The atrial appendage recording (D) demonstrates distinct velocity pulses due to atrial contraction, almost always present and usually of greater amplitude during diastole.

of patients, velocities in these two regions were used for subsequent analyses.

Correlation between stasis and stroke. There was a progressive, steep increase in stroke frequency with lower velocity in the posterior region ($p < 0.01$) and a trend to increasing stroke frequency for the appendage ($p = 0.05$) (Fig. 4). Of 15 patients with cardioembolic stroke, 13 had a velocity ≤15 cm/s in either the posterior atrium or the appendage, or both (sensitivity 87%). The estimate of specificity for this cutoff value (40%) was erroneously low because of anticoagulation therapy in patients without stroke. One of 16 patients with typical flutter and 1 of 25 patients with 3+ to 4+ mitral regurgitation had a stroke during atrial arrhythmia.

In a significant minority of patients, discordance of low velocity was observed between the posterior atrium and the

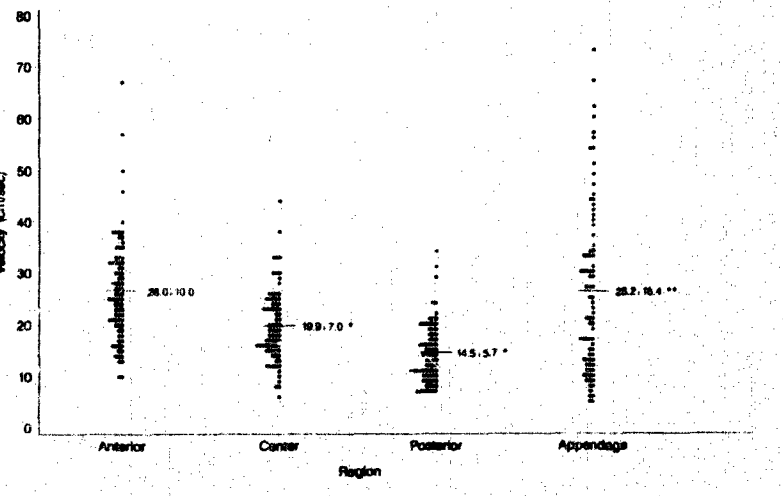

Figure 3. Plot of velocity data from all patients for each atrial region, showing decreased velocity toward the posterior atrium and the wide range of anterior and appendage velocities. Values shown are mean ± SD. *$p < 0.01$ versus anterior region. **$p < 0.01$ versus center and posterior regions.

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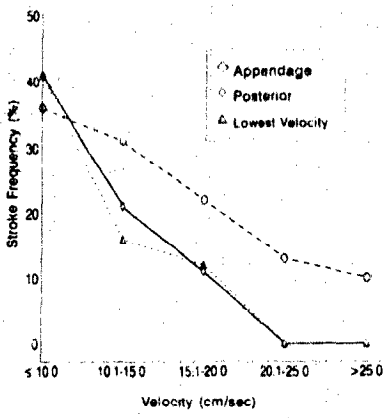

Figure 4. Correlation of stroke frequency to atrial velocity. **Triangles** = velocity of the site with the lowest velocity (either posterior **[diamonds]** or appendage **[circles]**); $p < 0.01$ for the relation of stroke frequency to velocity in the posterior and lowest velocity regions, and $p = 0.05$ for the appendage (Mantel-Haenzel test).

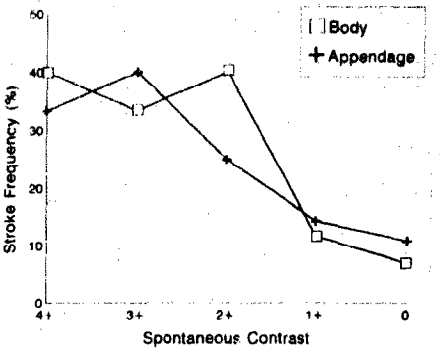

Figure 5. Correlation of stroke frequency to presence and grade of spontaneous contrast in the posterior atrium and appendage ($p < 0.01$ for both [Mantel-Haenzel test]).

appendage. For patients with a posterior velocity ≤15 cm/s, appendage velocity was >15 cm/s in 41 (46%), whereas in patients with an appendage velocity ≤15 cm/s, posterior velocity was >15 cm/s in 13 (15%).

Correlation between spontaneous contrast and stasis and stroke. Spontaneous contrast was significantly associated with low velocity ($p < 0.01$) and appeared at similar mean velocities of 10.6 and 13.5 cm/s in the posterior atrium and appendage, respectively (p = NS). In patients with spontaneous contrast in the body of the atrium, 18 (21%) did not have contrast in the appendage, whereas 23 (27%) of those with contrast in the appendage did not have contrast in the body of the atrium.

Stroke frequency was increased in the presence of 2+ or greater spontaneous contrast in either the atrial body or appendage but did not show the clear, quantitative correlation to stroke frequency that was apparent for velocity (Fig. 5).

Correlation between stasis and visualized thrombosis. Atrial thrombi were visualized in five patients, all in the appendage. The mean appendage velocity in these patients was 9 cm/s (range 5 to 11), significantly lower than that for patients without appendage thrombi (27 cm/s, range 6 to 72, $p < 0.05$).

Effect of rhythm, mitral regurgitation and mitral obstruction on atrial velocity. Velocities were significantly higher in patients with typical flutter than those with atypical flutter or fibrillation (Table 2). Velocities were not significantly different between the latter two. Significant mitral regurgitation was associated with higher posterior atrial velocity, probably due to the effects of regurgitant volume as well as incomplete elimination of influence of the jet. By contrast, 3+ to 4+ mitral regurgitation had no effect on appendage velocity. Mitral valve area <2.0 cm^2 was associated with significantly lower velocities in both the posterior atrium and appendage.

Effect of atrial and ventricular factors. Low posterior velocity was significantly associated with large left atrial ($r = -0.31$, $p < 0.01$) and left ventricular volumes ($r = -0.35$, $p < 0.01$) and a low ventricular ejection fraction ($r = 0.29$, $p < 0.01$). Low posterior velocity was somewhat more strongly associated with low left atrial ejection fraction ($r = 0.41$, $p < 0.001$). Correlations of these factors with appendage velocity were similar, with the exception of left ventricular ejection fraction, which was not correlated with appendage velocity. Correlations were also similar for patients with nonvalvular fibrillation.

Variables unrelated to velocity. No correlation was found between atrial stasis and mitral annular calcification, left ventricular wall thickness, age, history of congestive heart failure, prior myocardial infarction, angina, hypertension, diabetes mellitus, smoking or duration of atrial arrhythmia.

Multivariate analysis of factors related to atrial velocity. Stepwise regression analysis of factors related to posterior velocity yielded a model accounting for 49% of the variability (Table 3). The independent factors were left atrial ejection fraction, grade of mitral regurgitation, atrial rhythm, left ventricular end-diastolic volume index and mitral valve area. The regression analysis for appendage velocity accounted for 56% of the variability with the following factors: atrial rhythm (alone accounting for 41% of the variability), left atrial ejection fraction, mitral valve area and grade of mitral regurgitation. In patients with nonvalvular atrial fibrillation, regression

Table 2. Effect of Arrhythmia and Valve Disease on Regional Atrial Velocities

	Arrhythmia		MR		MVA	
Region	Fibrillation or Type II Flutter	Type I Flutter	1+ to 2+	3+ to 4+	≤2.0	>2.0
Posterior	13.3	18.6*	13.6	18.5†	9.3	14.9‡
Appendage	20.9	48.8*	26.2	26.0	10.9	27.4‡

*$p < 0.01$ versus fibrillation. †$p < 0.05$ versus 1+ to 2+. ‡$p < 0.05$ versus ≤2.0. Data presented are mean velocity (cm/s). MR = mitral regurgitation; MVA = mitral valve area.

Table 3. Stepwise Regression Analysis of Factors Related to Atrial Velocity

Factor	Cumulative R^2 Value
Posterior atrium	
LAEF	0.17
MR grade	0.28
Atrial rhythm	0.38
MVA	0.44
LVEDVI	0.49
Appendage	
Atrial rhythm	0.41
LAEF	0.47
MVA	0.52
MR grade	0.56

LAEF = left atrial ejection fraction; LVEDVI = left ventricular end-diastolic volume index; other abbreviations as in Table 2.

analysis yielded only left ventricular end-diastolic volume index as the factor most related to posterior velocity and left atrial volume index as that most related to appendage velocity.

Prediction of low atrial velocity from transthoracic echocardiography. Table 4 summarizes the sensitivity and specificity of selected variables from the regression analysis for the presence of stasis. Data for the widely used measurement of left atrial diameter are also shown. Cutoff values for left atrial ejection fraction and left atrial diameter were selected to maintain sensitivity, given the clinical importance of not missing patients with stasis. Separate analyses for patients with nonvalvular atrial fibrillation yielded similar results for left atrial ejection fraction and left atrial diameter.

Discussion

There are three important results of the present study: 1) The risk of stroke during atrial fibrillation or flutter increases steeply with decreasing atrial velocity, especially ≤15 cm/s. 2) Stroke appears to be strongly related to stasis in the body of the left atrium, with a lesser association with stasis in the appendage. 3) Left atrial stasis can be predicted with good sensitivity but limited specificity by data from transthoracic echocardiography, in particular with a left atrial ejection fraction ≤0.70 or a left atrial diameter >4.0 cm.

Table 4. Sensitivities and Specificities of Factors for Regional Stasis

	Region					
	Posterior Velocity ≤15 cm/s			Appendage Velocity ≤15 cm/s		
Factor	Sens	Spec	p Value	Sens	Spec	p Value
Fibrillation and type II flutter	91	32	0.05	100	28	0.01
MR 0–2+	93	30	0.01	84	18	NS
LAEF						
≤0.7	88	40	0.01	96	30	0.05
≤0.6	77	54	0.05	92	45	0.01
LA diam						
>4.0 cm	85	42	0.05	93	32	0.05
>4.5 cm	50	58	NS	75	63	0.01

LA diam = left atrial diameter; Sens = sensitivity; Spec = specificity; other abbreviations as in Tables 2 and 3.

Comparison with previous studies. Several previous studies have addressed the relation between atrial stasis during atrial fibrillation or flutter and cardioembolism (13–18). In the study by Verhorst et al. (15), patients with possible embolism had a lower mean appendage velocity (25 cm/s) than those without possible embolism (39 cm/s, $p < 0.05$), but the difference was only significant if patients with sinus rhythm were included, and 47% of events occurred in patients with an appendage velocity >25 cm/s. Embolism was associated with a "low flow profile" of the appendage in the report of Mugge et al. (16). Studies by Fatkin et al. (17) and Santiago et al. (18) identified the relation of low appendage velocity to appendage thrombus (mean 15 and 8 cm/s, respectively), but the relation of appendage velocity to embolism was either not mentioned (18) or found to be weak (17). The data of Fatkin et al. showed that embolic events were actually less frequent in patients with the pattern of appendage function associated with low velocity than in several other patterns with significantly higher appendage velocities and were not at all related to appendage thrombus.

By contrast, the present study demonstrates a quantitative relation between stroke and low velocity in the posterior atrium and appendage. The results of previous studies combined with the present data support the concept that velocity ≤15 cm/s represents clinically important stasis. The significance of this value is further supported by the appearance of spontaneous contrast at ~15 cm/s in the present study as well as in the data of Fatkin et al. (17) and Santiago et al. (18). The possibility that patients with atrial velocity ≤15 cm/s during sinus rhythm are at increased risk of stroke warrants further investigation.

Posterior versus appendage stasis. The results of the present study support the concept that posterior atrial stasis as well as appendage stasis may lead to stroke. In addition to data here and in previous studies showing embolism in the absence of appendage stasis, stroke-related factors such as left ventricular systolic dysfunction or dilation, or both, were more strongly related to posterior atrial stasis than to appendage stasis.

Determinants of stasis. In the present study, left atrial ejection fraction (stroke volume/left atrial volume, or atrial flow/beat per unit atrial volume) emerged as the most important independent determinant of posterior atrial and appendage stasis. This variable is measurable by transthoracic echocardiography and appears to be the best single factor to consider when assessing a patient's risk for stasis. The importance of atrial ejection fraction found here suggests that the previously reported statistical relation of stroke to left ventricular dysfunction may be in part due to reduced stroke volume. The additional independent contribution of increased left ventricular end-diastolic volume to atrial stasis further sup-

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ports the view that factors in addition to atrial size play a role in promoting stasis.

Study limitations. Certain patients with stroke in the present study may not have cardioembolism. In addition, stroke during atrial fibrillation preceded measurement of velocities by 5 months on average (not including two patients with stroke after measurement). The possibility that velocities were different at the time of stroke cannot be excluded. However, preliminary data suggest that atrial velocities are stable over a similar time span. A subset of five patients in this study had repeat velocity measurements after a mean interval of 32 weeks. At the second measurement, posterior velocity and appendage velocity were essentially unchanged (mean 0.3 and 5.4 cm/s higher, respectively, p = NS). A study of the relation between atrial velocity and stroke preferably would not include patients taking warfarin or aspirin; however, current standards of treatment for these arrhythmias make this a difficult limitation to overcome.

The estimate for the sensitivity of a cutoff value of 15 cm/s for stroke (87%) was not influenced by anticoagulant therapy because all patients with stroke either had no therapy or had subtherapeutic levels at the time of stroke. Specificity will be underestimated due to anticoagulant therapy in ~50% of patients without stroke. Low specificity for this cutoff value could also be explained by stasis being a necessary but insufficient condition for thrombosis, with other factors such as blood coagulability playing a role.

For patients with 3 to 4+ mitral regurgitation, the mean number of sites omitted per patient was 7.8 (of a total of 11), whereas for patients with 0 to 2+ mitral regurgitation, the mean number of sites omitted was 2.5.

True maximal velocity may be underestimated if the direction of flow deviates from an axial relation to the transesophageal Doppler beam. True appendage velocity may be underestimated in this and previous studies because in many patients, the direction of flow is likely to deviate significantly from alignment with the Doppler beam. Flow in the atrial body almost always is toward the mitral valve in early diastole, as can be observed when spontaneous contrast is present. Because in this study, maximal velocities were diastolic in 82% to 98% of patients (depending on the site), the velocities measured are of flow approximately axial to the Doppler beam. Despite the potential limitation of velocity underestimation, the degree of which is not determinable by available methods, the data here still provide a basis for comparison of patients and quantitation of the relation of stasis to stroke frequency.

Clinical implications. Identification of patients at highest risk for embolism during atrial fibrillation or flutter remains an important goal. The present study shows that the risk of stroke is quantitatively related to the degree of left atrial stasis and becomes substantial when atrial velocity is ≤15 cm/s. A large proportion of patients with atrial arrhythmia in this study had velocities in this range (65%), including those with nonvalvular atrial fibrillation (72%). Thus, the results of this study are consistent with data from the control groups of recent randomized trials of warfarin, namely, that most patients with nonvalvular atrial fibrillation are at substantial risk of stroke (7).

The quantitative analysis of atrial stasis presented here demonstrates several additional points that are useful when assessing potential benefit from anticoagulation therapy. Patients with normal-sized left atria (diameter <4 cm) are very unlikely to have atrial stasis during atrial fibrillation or flutter. Additionally, the risk of atrial stasis appears to be highest when a low stroke volume and left ventricular dilation accompany left atrial dilation. Furthermore, appendage stasis may occur with marked atrial dilation due to mitral regurgitation, and posterior atrial stasis may occur even in the presence of type I flutter. Unfortunately, transthoracic echocardiographic data seem to offer limited specificity for the presence of stasis.

Transesophageal echocardiographic measurement of posterior atrial and appendage velocities offer important advantages over the determination of the presence and grade of spontaneous contrast. Comparison of Figures 4 and 5 suggests a clearer quantitative relation of stroke frequency to velocity rather than to spontaneous contrast, which may be helpful when estimation of the magnitude of the stroke risk is clinically important. Thus, direct measurement of atrial velocity in the posterior atrium and appendage by tranesophageal echocardiography offers the optimal identification of atrial stasis-related stroke risk during atrial arrhythmia.

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