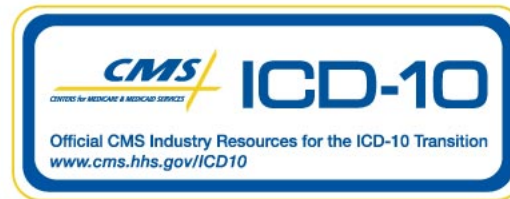


**GET HELP FROM
CMS HERE**



Role of left atrial appendage obliteration in stroke reduction in patients with mitral valve prosthesis: A transesophageal echocardiographic study

Miguel Angel García-Fernández, Esther Pérez-David, Juan Quiles, Juan Peralta, Ismael García-Rojas, Javier Bermejo, Mar Moreno, and Jacobo Silva

J. Am. Coll. Cardiol. 2003;42:1253-1258

doi:10.1016/S0735-1097(03)00954-9

This information is current as of February 9, 2012

The online version of this article, along with updated information and services, is located on the World Wide Web at:

<http://content.onlinejacc.org/cgi/content/full/42/7/1253>

JACC

JOURNAL of the AMERICAN COLLEGE of CARDIOLOGY



Role of Left Atrial Appendage Obliteration in Stroke Reduction in Patients With Mitral Valve Prosthesis

A Transesophageal Echocardiographic Study

Miguel Ángel García-Fernández, MD, PhD, Esther Pérez-David, MD, Juan Quiles, MD, Juan Peralta, MD, Ismael García-Rojas, MD, Javier Bermejo, MD, Mar Moreno, MD, Jacobo Silva, MD
Madrid, Spain

OBJECTIVES	The aim of our study was to assess whether left atrial appendage (LAA) ligation in patients undergoing mitral valve replacement is associated with the risk of future embolisms.
BACKGROUND	Previous studies show that the LAA plays an important role in the development of intracardiac thrombus. According to this decisive role, LAA surgical closure in patients undergoing cardiac surgery may be an attractive choice for reducing stroke.
METHODS	We retrospectively studied 205 patients with previous mitral valve replacement and referred for echocardiography study. Patients were excluded if other causes of systemic embolism were found. The main outcome measure was the occurrence of an embolic event.
RESULTS	Ligation of LAA was performed in 58 patients. However, an incomplete ligation was verified in six patients. During a median time from valve replacement to echocardiography study of 69.4 months (1 to 329), 27 patients had an embolism. Multivariate analysis identified the absence of LAA ligation (odds ratio [OR] 6.7 [95% confidence interval {CI} 1.5 to 31.0]; $p = 0.02$) and the presence of left atrial thrombus as the only independent predictors of occurrence of an embolic event. Moreover, when the identification of an incomplete LAA ligation was considered together with the absence of LAA ligation, risk of embolism increased up to $11.9 \times$ (OR 11.9 [95% CI 1.5 to 93.6]; $p = 0.02$).
CONCLUSIONS	Our study shows that LAA ligation during surgery of mitral valve replacement, performed in a high-risk population, is consistent with a reduction of the risk of late embolism and supports this technique if a mitral valve replacement is indicated. (J Am Coll Cardiol 2003;42:1253-8) © 2003 by the American College of Cardiology Foundation

The left atrial appendage (LAA) plays a fundamental role in the formation of atrial thrombus in patients with atrial fibrillation (AF). In patients with nonrheumatic AF, LAA is the origin of at least 90% of all left atrial (LA) clots (1-3), and the resulting systemic emboli cause approximately 25% of all strokes (4,5). Moreover, in patients in sinus rhythm in whom an LA thrombus was diagnosed with standard echocardiography, LAA location was involved in 95% of the cases (6).

See page 1259

Transesophageal echocardiography (TEE) is the gold-standard diagnostic method for the detection of thrombus in the LAA. The size of the LAA, the reduction of LAA flow velocities (7-11), and the detection of spontaneous echo contrast in either the LA or the LAA have been suggested as markers of increased embolism risk (12-16).

According to the decisive role of LAA in thrombus formation, LAA surgical ligation in patients undergoing cardiac surgery may be an attractive choice for reducing stroke risk (17,18). In addition, LAA closure can be now achieved through different approaches such as thoracoscopic

LAA ligation (19) and, more recently, percutaneous LAA occlusion (20,21).

However, to our knowledge, no prior study in a large and homogeneous population has analyzed the role of LAA ligation in the prevention of strokes or systemic embolic phenomena after a surgical intervention. As a result, intermittent and unsystematic LAA ligation is performed by surgeons worldwide. We hypothesized that LAA ligation would be associated with lower stroke risk and designed a retrospective study of patients undergoing mitral valve replacement. The aim of our study was to assess whether LAA ligation is associated with the risk of future embolism.

METHODS

Study population. A total of 242 patients with prior mitral valve replacement, referred to our echocardiography laboratory from February 1996 to October 2001, were included for the study. Patients were referred for echocardiography by their physician and were both studied with transthoracic and TEE. The indications for referral were 82 patients for quantification of mitral valve regurgitation, 48 patients that were investigated for suspected mitral valve endocarditis, 42 patients for investigation of an embolic source, 30 patients for evaluation of right or left ventricular function, 25 patients for further evaluation of a detected increase in mitral prosthesis diastolic gradient, and 15 patients were studied for miscellaneous indications.

From the Sección de Cardiología No Invasiva, Servicio de Cardiología, Hospital General Universitario Gregorio Marañón, Madrid, Spain. Presented, in part, at the 75th Scientific Session of the American Heart Association, Chicago, November 2002.

Manuscript received February 7, 2003; revised manuscript received April 22, 2003, accepted May 9, 2003.

Abbreviations and Acronyms

AF	= atrial fibrillation
LA	= left atrial/atrium
LAA	= left atrial appendage
LASEC	= left atrial appendage spontaneous echo contrast
TEE	= transesophageal echocardiography

Patients were excluded if other potential causes of systemic embolism were found in the echocardiographic study: thrombotic occlusion of the prosthetic valve (n = 11), confirmed endocarditis (n = 9), aortic dissection (n = 1), and if an extracardiac source were found to be the origin of the embolism (n = 16).

The final study group consisted of 205 patients (130 women) with a mean age of 62.4 years. The reason for the mitral valve replacement was rheumatic valve disease in 170 patients, mitral valve endocarditis in 10 patients, severe ischemic regurgitation in 6 patients, and mitral valve prolapse in 19 patients. All patients with mechanical prostheses underwent anticoagulation treatment with acenocumarin, and those with biological valves were either anticoagulated or received antiplatelet therapy.

Study flow. Retrospective analysis of the patients after mitral valve replacement was supported by a review of patient records. Main outcome measure was the occurrence of an embolic event, considering only those appearing after 48 h of the surgery. An embolic event was defined by the presence of clinical signs and the verification with at least one of the following diagnostic tools: computed tomography (neurological events) or angiography and/or surgery (peripheral artery events).

The clinical diagnosis of an embolic transient ischemic attack, or stroke, was made by a neurologist. Those patients with deficits in the posterior cerebral circulation territory or with lacunar strokes were excluded.

Echocardiography. Conventional transthoracic echocardiography was performed with either a Sonos 5500 (Philips Technologies, Andover, Massachusetts) or an Acuson Se-

quoia 256 (Siemens Technologies, Mountain View, California) using a phased array transducer. Transesophageal echocardiography was performed using an omniplane probe in 182 patients and with a biplane TEE probe in the remaining patients.

Cardiac structures and the aorta were visualized from multiple esophageal and transgastric views. Prosthetic mitral valve area was estimated using the pressure half-time method (22). Mitral regurgitation jet was visualized using color Doppler flow and graded to determine the severity of mitral regurgitation by a previously validated method (23). Maximal LAA areas were measured by tracing a line from the top of the upper pulmonary vein limbus along the entire endocardial LAA border (8).

The LA and LAA were closely inspected for the presence of thrombi and spontaneous echo contrast. The severity of LAA spontaneous echo contrast (LASEC) was graded from 0 to 4 following the classification of Fatkin et al. (24). Surgical ligation of the LAA was clearly identified by the lack of any anatomical structure between the mitral valve base and the upper left pulmonary artery (25) (Fig. 1A and 1B). Incomplete ligation was diagnosed by color Doppler flow, demonstrating a jet traversing the separation between LAA and the LA body (25) (Fig. 2).

Statistical analysis. Data are expressed as mean \pm SD. Proportions were compared with the chi-square test. If any of the cells from a 2×2 table had an expected count below 5, then a Fisher exact test was applied. Quantitative variables were normally distributed and compared with a Student *t* test. The independent contribution of the potential factors involved in embolism was analyzed with a logistic regression. In the multiple regression analysis, the dependent variable was the occurrence of an embolic event, and the independent variables were those that showed, on univariate analysis, a significant correlation with the occurrence of an embolic event as well as variables that showed a trend ($p < 0.25$) and those with well-known clinical relevance. Differences were considered to be statistically significant if the null hypothesis could be rejected with

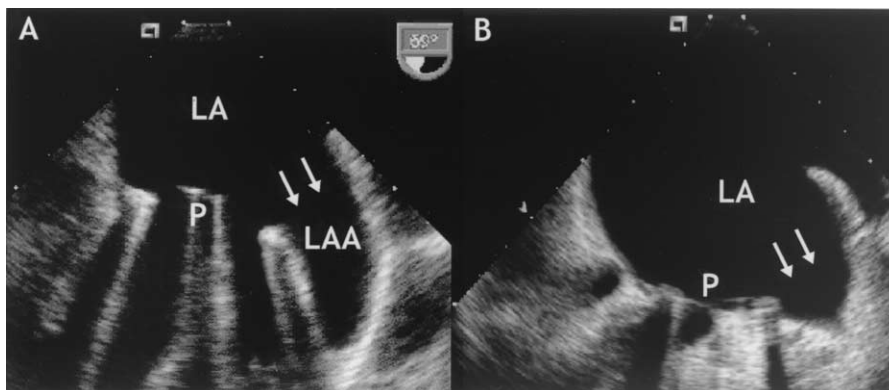


Figure 1. (A) Transesophageal echocardiogram in a patient with Saint Jude medical prosthesis in mitral position and without ligation of the left atrial appendage (LAA) (arrows). (B) Postoperative transesophageal echocardiographic study showing the absence of the LAA with complete obliteration of the cavity (arrows). LA = left atrium; P = prosthesis.

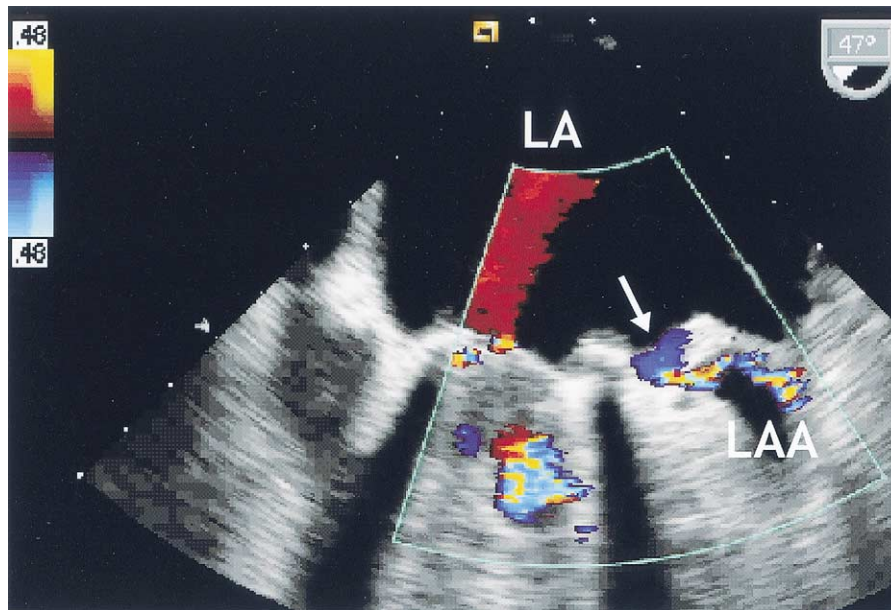


Figure 2. Transesophageal echocardiographic study. Transverse view of an incompletely ligated left atrial appendage (LAA). Color-coded flow transverses the communication between the LAA and the left atrium (LA) body (arrow).

>95% confidence interval. Two different multivariate analyses were carried out. The first one was an intention-to-treat analysis including as independent variable the surgical closure of the LAA, and, in the second analysis, only the effective closure of LAA assessed by TEE was considered. The SPSS 10.0 statistical software package (SPSS Inc., Chicago, Illinois) was used for all calculations.

RESULTS

A total of 205 patients was included in the study. Only 29 patients (14.1%) were in sinus rhythm. Mechanical prostheses were placed in 187 patients (71 Carbomedics [Carbomedics, Inc., Austin, Texas], 50 SJ Medical [St. Jude Medical, Inc., St. Paul, Minnesota], 27 Björk-Shiley [Shiley Laboratories, Inc., Santa Ana, California], 14 Medtronic Hall [Medtronic, Inc., Minneapolis, Minnesota], and 26 miscellanea). All other patients were treated with biological prostheses. Mild valve regurgitation was found in 29 patients (14.1%), moderate in 24 (11.7%), and severe in 22 patients (10.7%).

Complete ligation of the LAA was achieved in 52 patients and incomplete in six patients (10.3% of patients with LAA ligation). Remaining patients had no LAA ligation performed during surgery.

The mean time from valve replacement until the echocardiographic study was performed was 69.4 ± 67 months (1 to 329). During this time, peripheral arterial embolization was identified in 27 patients. In 24 patients, the embolic event occurred within one week before performing the TEE, and in three cases within two weeks. Among patients with embolisms, 19 suffered an ischemic stroke, five peripheral embolism, and three patients a transient ischemic attack. Of these, three demonstrated an LAA thrombus (including one patient with incomplete LAA ligation), and one patient had an LA thrombus. In addition, LA thrombi were observed in another six patients without embolism (located in the LAA in two patients, in the LA main cavity in two patients, and in both locations in another two patients).

Table 1. Characteristics of Patients With LAA Ligation at Surgery Versus No LAA

	LAA Ligation (n = 58)	No LAA Ligation (n = 147)	p Value
Age (yrs)	63 ± 12	62 ± 10	0.8
Female gender	44 (75.9%)	89 (58.5%)	0.02
LA size (cm)	60 ± 12	57 ± 11	0.07
LA or LAA thrombus	3 (5.2%)	7 (4.8%)	0.9*
Mean LVEF (%)	56 ± 10	57 ± 13	0.7
Moderate-severe LVEF depression	8 (13.8%)	25 (17%)	0.6
Severe mitral prosthetic regurgitation	4 (6.9%)	18 (12.2%)	0.3
Sinus rhythm	5 (8.6%)	24 (16.3%)	0.2
Mean time from MVR to TEE (months)	83 ± 73	64 ± 63	0.07
Recent embolism	2 (3.4%)	25 (17%)	0.01

*Fisher test was performed instead of chi-squared test. Data are expressed as mean ± SD or number (percentage).

LA = left atrium; LAA = left atrial appendage; LVEF = left ventricular ejection fraction; MVR = mitral valve replacement; TEE = transesophageal echocardiography.

Table 2. Characteristics of Patients With Embolic Events Versus No Embolic Event

	Embolic Event (n = 27)	No Embolic Event (n = 178)	p Value
Age (yrs)	62 ± 12	62 ± 10	0.9
Female gender	18 (66.7%)	112 (62.9%)	0.7
Mean time from MVR to TEE (months)	73.8 ± 60.9	68.6 ± 68.2	0.7
Mechanical prosthesis	24 (88.9%)	163 (91.6%)	0.6
LA size	55 ± 10	58 ± 12	0.2
LA or LAA thrombus	4 (14.8%)	6 (3.4%)	0.03*
Mean LVEF	59 ± 14	56 ± 12	0.5
Sinus rhythm	4 (14.8%)	25 (14%)	0.9
LAA ligation	2 (7.4%)	56 (31.5%)	0.01
Severe mitral prosthetic regurgitation	2 (7.4%)	20 (11.2%)	0.7*
LASEC grade 3 or 4	16 (64%)	26 (21.3%)	< 0.001†

*Fisher test was performed instead of chi-squared test. †In the analysis of this variable, only patients without LAA ligation were included.

LASEC = left atrial appendage spontaneous echo contrast. Other abbreviations as in Table 1.

Table 1 shows the characteristics of patients according to LAA ligation. Left atrial appendage ligation was performed more frequently in women (75.9% of the patients with LAA ligation compared with only 58.5% of the patients with no ligation; $p = 0.02$). The occurrence of a systemic embolism was significantly more frequent in patients without LAA ligation compared with patients with LAA ligation (17% vs. 3.4% respectively; $p = 0.01$.) Only two of the 27 embolic events occurred in patients with LAA ligation. In addition to the absence of LAA ligation, the presence of LA or LAA thrombus (14.8% vs. 3.4%; $p = 0.03$) and LASEC grade 3

or 4 (64% vs. 21.3%; $p < 0.001$) was also more frequent in patients with an embolic event compared with patients without systemic embolism (Table 2).

Multivariate analysis identified the absence of LAA ligation (odds ratio [OR] 6.7 [95% CI 1.5 to 31.0]; $p = 0.02$) and the TEE finding of an LA or LAA thrombus (OR 5.8 [95% CI 1.2 to 27.3]; $p = 0.03$) as the only independent predictors of occurrence of an embolic event after mitral valve replacement surgery (Table 3). Moreover, if the absence of effective ligation as assessed by echocardiography was included in the model, OR increased up to 11.9 (95% CI 1.5 to 93.6), $p = 0.02$, and the presence of LA or LAA thrombus also remained independently associated with the occurrence of an embolic event (Table 3).

If only patients without LAA ligation were included in the analysis, events were more frequent in patients with larger LAA size (8.6 ± 1.0 mm vs. 7.7 ± 1.1 mm in patients with and without embolic events respectively; $p = 0.001$) and in those with greater LASEC score (64% of patients with embolic event had a score 3 or 4 compared with 21.3% of patients without event; $p = 0.0005$). The presence of LA or LAA thrombus was close to reaching statistical significance (12% vs. 3.3% in patients with and without embolic events respectively; $p = 0.06$). After multivariate analysis, the only independent variable associated with the occurrence of embolisms in these group of patients was the LASEC score (OR 3.0 [95% CI 1.5 to 6.0]; $p = 0.002$) (Table 4).

DISCUSSION

LAA ligation during surgical procedures. Several studies have confirmed that the LAA plays a very important role in

Table 3. Factors Independently Associated With Embolic Events

Logistic Regression Procedure Evaluating the Absence of LAA Ligation Performed at Surgery			
	B	OR (95% CI)	p Value
Absence of LAA ligation at MVR	1.9	6.7 (1.5–31.0)	0.02
LA or LAA thrombus	1.8	5.8 (1.2–27.3)	0.03
Age (per yr)	0.02	1.0 (0.9–1.1)	0.5
Female gender	0.07	1.1 (0.4–2.8)	0.9
Time from MVR to TEE (per month)	0.01	1.0 (0.9–1.0)	0.2
LVEF	0.01	1.0 (0.9–1.1)	0.7
Mitral regurgitation	−0.2	0.8 (0.5–1.3)	0.3
LA size	0.03	1.0 (0.9–1.1)	0.2
Sinus rhythm	−0.03	1.0 (0.3–3.5)	0.9
Logistic Regression Procedure Evaluating the Absence of Effective LAA Ligation in TEE			
Absence of LAA ligation in TEE	2.5	11.9 (1.5–93.6)	0.02
LA or LAA thrombus	1.6	5.1 (1.1–23.2)	0.04
Age (per yr)	0.02	1.0 (0.9–1.1)	0.5
Female gender	0.09	1.1 (0.4–2.9)	0.9
Time from MVR to TEE (per month)	0.005	1.0 (0.9–1.0)	0.2
LVEF	0.01	1.0 (0.9–1.1)	0.5
Mitral regurgitation	−0.3	0.8 (0.5–1.2)	0.3
LA size	0.03	1.0 (0.9–1.1)	0.3
Sinus rhythm	−0.07	0.9 (0.3–3.4)	0.9

In both models the same factors were evaluated, excepting absence of LAA ligation (at MVR in the first model or at TEE in the second model).

CI = confidence interval; OR = odds ratio. Other abbreviations as in Table 1.

Table 4. Factors Independently Associated With Embolic Events Evaluated Only in Patients Without LAA Ligation Performed at Surgery (n = 147)

	B	OR (95% CI)	p Value
LASEC*	1.09	3.0 (1.5-6.0)	0.002
Age (per yr)	0.01	1.0 (0.9-1.17)	0.7
Female gender	0.37	1.5 (0.5-4.4)	0.5
Mechanical mitral prosthesis	0.004	1.0 (0.9-1.2)	0.9
Mitral regurgitation	0.37	1.4 (0.7-3.0)	0.3
LAA size	0.29	1.3 (0.8-2.2)	0.3
LA size	-0.03	1.0 (0.9-1.1)	0.2
Sinus rhythm	0.18	1.2 (0.2-6.9)	0.8
LA or LAA thrombus	1.13	3.1 (0.6-17.6)	0.2

*OR related to each grade of increase of severity. Logistic regression procedure including the following variables: age, gender, mechanical mitral prosthesis, LA size, LAA size, sinus rhythm, LA thrombus, mitral prosthetic regurgitation. Abbreviations as in Tables 1, 2, and 3.

the development of thromboembolic strokes. In patients with rheumatic and nonrheumatic AF, at least 60% and 90% of LA thrombi, respectively, are located in the LAA (1-3). Therefore, it is likely that ligation of the LAA would greatly reduce the risk of stroke.

Since the early rheumatic mitral stenosis surgical procedures (17,18), LAA ligation has been performed attempting to reduce the risk of embolization. Ligation of the LAA during mitral valve replacement surgery is still controversial. Some authors propose that prophylactic appendage removal should be performed whenever the chest is open as a method of preventing future strokes (26). Surprisingly, the benefit of carrying out such a commonly performed procedure is poorly documented in scientific literature, and its use has been sporadic and governed by intuition alone (3). The utilization of this technique depends on the methodology and insight of the different surgical teams despite that it is a recommended procedure in the ACC guidelines (27).

Orszulak et al. (28) evaluated the risk of stroke in elderly patients during the early postoperative period after a mitral valve replacement with a Carpentier-Edwards biological prosthesis and found a strong correlation between late stroke in patients having the LAA ligated when patients underwent mitral valve replacement and coronary bypass grafts. However, in the group of patients with isolated mitral valve replacement and in the overall group, the only independent variable associated with a greater risk of late stroke was an advanced heart class.

Juratli et al. (29) analyzed the use of LAA ligation during mitral valve surgery (50% underwent mitral valve repair and 23% received a mechanical prosthesis) as an alternative to anticoagulant treatment with warfarin. Left atrial appendage ligation did not provide an adequate protection from thromboembolic events in the absence of effective anticoagulation treatment with warfarin. Our study population, unlike the patient population studied by Juratli et al. (29) and Orszulak et al. (28), had received acenocumarin treatment in 82% of cases, and in all the cases when a patient received a mechanical prosthesis. In addition, the majority of subjects

were diagnosed with chronic rheumatic mitral valve disease, and a greater enlargement of the LA was observed.

LAA incomplete ligation. A special group of patients are those with incomplete surgical ligation of the LAA. Katz et al. (25) recently reported that 36% of patients had incomplete ligation of the LAA after a mitral valve surgery, and 22% of them suffered an embolic event. In our study the frequency of incomplete ligation of the LAA was only 10.3%, of which only one case suffered an embolic event. There is no doubt about the influence of the surgical technique on these findings. In the series of Katz et al. (25), a running suture was performed, whereas our surgeons carried out a double suture (a purse string suture as well as a running suture). This double suture probably provides a more stable LAA ligation. The high incidence of incomplete ligation of the LAA should be noted, and further investigation is required to establish if this can result in an increased incidence of postoperative embolic events.

Selection of patients for LAA ligation. The physiological role of the LAA is minimal, and consequently, its removal has been advocated over the years to reduce stroke risk. At this point, an interesting application of TEE may be to identify patients for LAA ligation; TEE can define a high-risk population if a low or absent LAA flow velocity profile or the presence of LASEC is identified (8). It could be argued that ligation of the LAA is unnecessary in those high-risk patients because they will receive anticoagulation therapy for decades. However, it is well known that, despite clear guidelines, warfarin is either not used or used improperly on a large scale (30-32). In addition, warfarin levels are affected by a large number of dietary and drug factors (3). Thus, it is clear that variations in the therapeutic effectiveness of long-term treatments are almost the norm, and it is in these changes where the appearance of embolic events can take place. Thus, it would appear sensible to eliminate the fundamental origin of the embolic events.

Our study shows for the first time that LAA ligation during surgery of mitral valve replacement, performed in a high-risk population, is consistent with a reduction of the risk of late embolism (6.7-fold reduction in embolic risk). Moreover, if a complete ligation is achieved and confirmed with TEE, a further reduction in embolism risk is observed (11.9-fold reduction in embolic risk). In addition, the TEE identification of LAA or LA thrombus, higher LA size, or higher degrees of LASEC is associated with a higher risk of late embolisms. These findings represent a strong negative association between an embolic event and ligation of the LAA during surgical intervention, thus providing clear evidence for the benefit of ligation of the LAA during surgical procedure as a method of preventing a postoperative embolic event. Because we studied mainly patients with rheumatic valvular disease, our results may be applicable only to this population. However, an ongoing randomized trial of LAA occlusion during routine coronary artery bypass graft surgery (Left Atrial Appendage Occlusion Study [LAAOS] study) (33) will be able to provide definitive

evidence about LAA ligation in patients without rheumatic valvular disease.

Study limitations. The study population consisted of patients referred to the echocardiography laboratory for mitral valve prosthesis evaluation using TEE. This pretest referral bias can result in an amplification of the beneficial effect of LAA ligation if fewer patients who had mitral valve replacement and LAA ligation would be referred for echocardiographic study. Another limitation is the lack of information about anticoagulation levels in these patients. Although all patients with a mechanical prosthesis were anticoagulated, it could be possible that lower international normalized ratio levels influenced the development of an embolic event. Despite its evident clinical relevance, there is no reason to think that patients with LA appendectomy have a different chronic anticoagulation profile than patients without it. Thus, the degree of chronic anticoagulation should not be a confounding factor in this setting.

Our study supports the surgical LAA ligation together with the mitral valve replacement. It provides new information about potential impact of LAA ligation until new data of a randomized study with blinded event verification are available (33).

Reprint requests and correspondence: Dr. Miguel Ángel García-Fernández, Laboratorio de Ecocardiografía, Servicio de Cardiología, Hospital General Universitario Gregorio Marañón, Doctor Esquerdo 46, 28007, Madrid, Spain. E-mail: magfeco@primustel.es.

REFERENCES

1. Al Saady NM, Obel OA, Camm AJ. Left atrial appendage: structure, function, and role in thromboembolism. *Heart* 1999;82:547-54.
2. Ischemic stroke associated with atrial fibrillation: the demographic and clinical characteristics and 30-day mortality in a hospital stroke registry: the European Community Stroke Project, Florence unit. *Ann Ital Med Int* 1996;11:20-6.
3. Blackshear JL, Odell JA. Appendage obliteration to reduce stroke in cardiac surgical patients with atrial fibrillation. *Ann Thorac Surg* 1996;61:755-9.
4. Vemmos KN, Bots ML, Tsibouris PK, et al. Stroke incidence and case fatality in southern Greece: the Arcadia Stroke registry. *Stroke* 1999;30:363-70.
5. Petersen P, Godtfredsen J. Risk factors for stroke in chronic atrial fibrillation. *Eur Heart J* 1988;9:291-4.
6. Agmon Y, Khandheria BK, Gentile F, Seward JB. Clinical and echocardiographic characteristics of patients with left atrial thrombus and sinus rhythm: experience in 20,643 consecutive transesophageal echocardiographic examinations. *Circulation* 2002;105:27-31.
7. Pollick C, Taylor D. Assessment of left atrial appendage function by transesophageal echocardiography: implications for the development of thrombus. *Circulation* 1991;84:223-31.
8. Garcia-Fernandez MA, Torrecilla EG, San Roman D, et al. Left atrial appendage Doppler flow patterns: implications on thrombus formation. *Am Heart J* 1992;124:955-61.
9. Mugge A, Kuhn H, Nikutta P, Grote J, Lopez JA, Daniel WG. Assessment of left atrial appendage function by biplane transesophageal echocardiography in patients with nonrheumatic atrial fibrillation: identification of a subgroup of patients at increased embolic risk. *J Am Coll Cardiol* 1994;23:599-607.
10. Kamp O, Verhorst PM, Welling RC, Visser CA. Importance of left atrial appendage flow as a predictor of thromboembolic events in patients with atrial fibrillation. *Eur Heart J* 1999;20:979-85.
11. Kato H, Nakanishi M, Maekawa N, Ohnishi T, Yamamoto M. Evaluation of left atrial appendage stasis in patients with atrial fibrillation using transesophageal echocardiography with an intravenous albumin-contrast agent. *Am J Cardiol* 1996;78:365-9.
12. Daniel WG, Nellesen U, Schroder E, et al. Left atrial spontaneous echo contrast in mitral valve disease: an indicator for an increased thromboembolic risk. *J Am Coll Cardiol* 1988;11:1204-11.
13. Black IW, Hopkins AP, Lee LC, Walsh WF. Left atrial spontaneous echo contrast: a clinical and echocardiographic analysis. *J Am Coll Cardiol* 1991;18:398-404.
14. Tsai LM, Chen JH, Fang CJ, Lin LJ, Kwan CM. Clinical implications of left atrial spontaneous echo contrast in nonrheumatic atrial fibrillation. *Am J Cardiol* 1992;70:327-31.
15. Gonzalez-Torrecilla E, Garcia-Fernandez MA, Perez-David E, Bermejo J, Moreno M, Delcan JL. Predictors of left atrial spontaneous echo contrast and thrombi in patients with mitral stenosis and atrial fibrillation. *Am J Cardiol* 2000;86:529-34.
16. Hwang JJ, Kuan P, Chen JJ, et al. Significance of left atrial spontaneous echo contrast in rheumatic mitral valve disease as a predictor of systemic arterial embolization: a transesophageal echocardiographic study. *Am Heart J* 1994;127:880-5.
17. Bailey C, Olsen A, Keown K, et al. Commisurotomy for mitral stenosis technique for prevention cerebral complications. *JAMA* 1952;149:1085-91.
18. Madden J. Resection of the left auricular appendix. *JAMA* 1948;140:769-72.
19. Odell JA, Blackshear JL, Davies E, et al. Thoracoscopic obliteration of the left atrial appendage: potential for stroke reduction? *Ann Thorac Surg* 1996;61:565-9.
20. Nakai T, Lesh MD, Gerstenfeld EP, Virmani R, Jones R, Lee RJ. Percutaneous left atrial appendage occlusion (PLAATO) for preventing cardioembolism: first experience in canine model. *Circulation* 2002;105:2217-22.
21. Sievert H, Lesh MD, Trepels T, et al. Percutaneous left atrial appendage transcatheter occlusion to prevent stroke in high-risk patients with atrial fibrillation: early clinical experience. *Circulation* 2002;105:1887-9.
22. Hatle L, Angelsen BA, Tromsdal A. Non-invasive assessment of aortic stenosis by Doppler ultrasound. *Br Heart J* 1980;43:284-92.
23. Cooper JW, Nanda NC, Philpot EF, Fan P. Evaluation of valvular regurgitation by color Doppler. *J Am Soc Echocardiogr* 1989;2:56-66.
24. Fatkin D, Kelly RP, Feneley MP. Relations between left atrial appendage blood flow velocity, spontaneous echocardiographic contrast and thromboembolic risk in vivo. *J Am Coll Cardiol* 1994;23:961-9.
25. Katz ES, Tsiamtsiouris T, Applebaum RM, Schwartzbard A, Tunick PA, Kronzon I. Surgical left atrial appendage ligation is frequently incomplete: a transesophageal echocardiographic study. *J Am Coll Cardiol* 2000;36:468-71.
26. Johnson WD, Ganjoo AK, Stone CD, Srivvas RC, Howard M. The left atrial appendage: our most lethal human attachment! Surgical implications. *Eur J Cardiothorac Surg* 2000;17:718-22.
27. Bonow RO, Carabello B, de Leon AC, et al. ACC/AHA Guidelines for the Management of Patients With Valvular Heart Disease: Executive Summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Patients with Valvular Heart Disease). *J Heart Valve Dis* 1998;7:672-707.
28. Orszulak TA, Schaff HV, Pluth JR, et al. The risk of stroke in the early postoperative period following mitral valve replacement. *Eur J Cardiothorac Surg* 1995;9:615-9.
29. Jurati N, Wilkoff B, Tchou P, et al. Left atrial appendage ligation during mitral valve surgery may increase the risk of late thromboembolic event (abstr). *J Am Coll Cardiol* 2002;39:85A.
30. Munschauer FE, Priore RL, Hens M, Castilone A. Thromboembolism prophylaxis in chronic atrial fibrillation: practice patterns in community and tertiary-care hospitals. *Stroke* 1997;28:72-6.
31. Schlicht JR, Davis RC, Naqi K, Cooper W, Rao BV. Physician practices regarding anticoagulation and cardioversion of atrial fibrillation. *Arch Intern Med* 1996;156:290-4.
32. Stafford RS, Singer DE. National patterns of warfarin use in atrial fibrillation. *Arch Intern Med* 1996;156:2537-41.
33. Crystal E, Lamy A, Connolly SJ, et al. Left Atrial Appendage Occlusion Study (LAAOS): a randomized clinical trial of left atrial appendage occlusion during routine coronary artery bypass graft surgery for long-term stroke prevention. *Am Heart J* 2003;145:174-8.

Role of left atrial appendage obliteration in stroke reduction in patients with mitral valve prosthesis: A transesophageal echocardiographic study

Miguel Ángel García-Fernández, Esther Pérez-David, Juan Quiles, Juan Peralta, Ismael García-Rojas, Javier Bermejo, Mar Moreno, and Jacobo Silva

J. Am. Coll. Cardiol. 2003;42;1253-1258

doi:10.1016/S0735-1097(03)00954-9

This information is current as of February 9, 2012

Updated Information & Services	including high-resolution figures, can be found at: http://content.onlinejacc.org/cgi/content/full/42/7/1253
References	This article cites 32 articles, 22 of which you can access for free at: http://content.onlinejacc.org/cgi/content/full/42/7/1253#BIBL
Citations	This article has been cited by 32 HighWire-hosted articles: http://content.onlinejacc.org/cgi/content/full/42/7/1253#otherarticles
Rights & Permissions	Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: http://content.onlinejacc.org/misc/permissions.dtl
Reprints	Information about ordering reprints can be found online: http://content.onlinejacc.org/misc/reprints.dtl