**TABLE 1 Patient Details Specific to History of SCAD and Subsequent Chest Pain Symptoms**

| Patient # | Age*, yrs | SCAD Risk Factors | Initial SCAD Management | Timing of Symptoms | % of Cycles Affected | Evaluation of Recurrent CP | Treatment |
|---|---|---|---|---|---|---|---|
| 1 | 35 | 5 days PP | PCI | 5 days pre-menstrual | 100 | PCI to residual dissection; persistent pain without ischemia on subsequent stress test | Short-acting nitrate prn |
| 2 | 42 | Prior SCAD | Unsuccessful PCI; CABG | 1–2 days pre-menstrual | 5–10 | No ischemia on stress test | Short-acting nitrate prn, CCB |
| 3 | 39 | 13 days PP, FMD | Unsuccessful conservative;† PCI | 1–2 days pre-menstrual | 90 | Patent coronary arteries on CA | Short-acting nitrate prn, CCB, beta-blocker |
| 4 | 46 | None | Conservative | 1–2 days pre-menstrual | 100 | Improved coronary artery caliber on CA | Long-acting nitrate, ranolazine, endometrial ablation |
| 5 | 44 | None | Conservative | 1–2 days pre-menstrual through menses | 100 for 2 yrs, now 5–10 | No ischemia on stress test; stable CCTA | Short-acting nitrate prn |
| 6 | 34 | FMD | Unsuccessful conservative; PCI | 1–2 days pre-menstrual through menses | 50 | Complex PCI for in-stent stenosis; intracoronary thrombus requiring emergent CABG; persistent pain without ischemia on stress test; patent stents/grafts on repeat CA | Ranolazine, CCB, beta-blockers, long-acting nitrate |
| 7 | 45 | EDS type IV | Unsuccessful conservative; unsuccessful PCI; interval CABG | 1–2 days pre-menstrual during first 2 days of menses | 100 | Patent coronary arteries on CCTA | Short-acting nitrate prn, CCB, long-acting nitrate |
| 8 | 43 | Emotional stress | Conservative | 3 days pre-menstrual during first 2 days of menses | 100 | No ischemia on stress test; patent coronaries on CA | Short-acting nitrate prn |
| 9 | 37 | 10 weeks PP | PCI | 1–2 days pre-menstrual | Only heavy cycles | No ischemia on stress test | Short-acting nitrate prn |
| 10 | 37 | 15 days PP | CABG | 1–2 days pre-menstrual | 50 | No ischemia on stress test | Short-acting nitrate prn, long-acting nitrate, ranolazine |
| 11 | 41 | None | Conservative | 1–2 days pre-menstrual | 20 | No ischemia on stress test | Short-acting nitrates prn, CCB |

*Age in years at the time of SCAD. †Conservative = conservative management during which the patient received medications but no other invasive treatment strategies at time of SCAD.

CA = coronary angiography; CABG = coronary artery bypass grafting; CCB = calcium-channel blocker; CCTA = coronary computed tomography angiography; CP = chest pain; EDS = Ehlers-Danlos syndrome; FMD = fibromuscular dysplasia; PCI = percutaneous coronary intervention; PP = postpartum; prn = pro re nata (when necessary); SCAD = spontaneous coronary artery dissection.

The novel observation of catamenial chest pain among SCAD patients emphasizes the potential vascular significance of ovarian hormones among these patients. If catamenial chest pain is recognized, and after excluding ongoing ischemia/infarction, anti-ischemic therapies can be titrated to achieve optimal symptom control. The role of pregnancy, endogenous/exogenous hormones, endometrial ablation, and menopause in SCAD needs to be further explored to better discern management of post-SCAD contraception, pregnancy considerations, and recurrent symptoms.

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# Recovery of Left Atrial Contractile Function After Maze Surgery in Persistent Longstanding Atrial Fibrillation

Atrial fibrillation (AF), the commonest dysrhythmia, affects nearly 4.5 million people in Europe and

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2.2 million in the United States. Prevalence increases with age (4% at age >60 years and 9% at age >80 years), and its effect on quality of life and health resources is considerable. Fibrillating atria have no contractile function. The most serious complication is thromboembolic stroke (12,500 strokes are attributed to AF annually in the United Kingdom). To reduce thromboembolism, patients are anticoagulated, which increases bleeding risk. AF also exacerbates heart failure and is a rising public health burden as the population ages.

The maze procedure creates lesions in the atria to obstruct the macro-re-entry circuits essential to sustain AF. Maze is most commonly performed as an adjunct to major cardiac surgery and significantly increases 12-month freedom from AF, restoring sinus rhythm (SR) in 44% to 94% of treated patients compared with 5% to 33% of control subjects (1). Restoring SR does not guarantee return of atrial contractile function, and both thromboembolism and heart failure exacerbation are related to the loss of contractile function (2).

Studies of atrial transport after maze are limited by small samples, selection bias, and lack of matched control subjects. The HESTER (Has Electrical Sinus Translated into Effective Remodelling?) matched cohort study compared atrial transport in patients whose SR was restored by maze with those in SR before and after cardiac surgery. Maze patients were in SR at least 1 year after receiving maze for persistent longstanding AF as an adjunct to cardiac surgery. Control patients were in SR before and at least 1 year after cardiac surgery, matched 1-to-1 for time since procedure (±6 months), age (±5 years), sex, type of surgery, left ventricular function, and risk profile (logistic EuroSCORE). The aim was to assess whether the 2 cohorts had equivalent left atrial (LA) function, primarily active left atrial ejection fraction (ALAEF):

$$ALAEF = 100 \times \frac{LAV_{preA} - LAV_{min}}{LAV_{preA}}$$

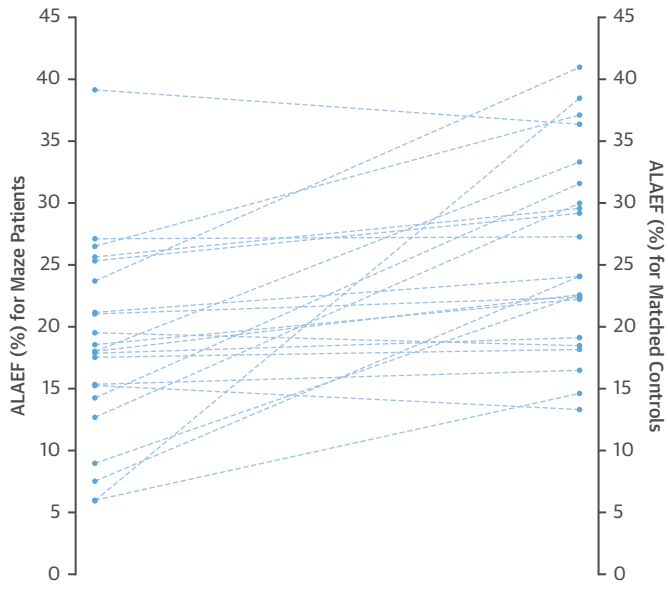
where LAVpreA = pre-A wave left atrial volume and LAVmin = minimum left atrial volume. Secondary outcomes were LA volume measurements, active and passive stroke volume, and LA ejection fraction.

LA function was evaluated by transthoracic echocardiography and multislice cardiac magnetic resonance imaging. For each modality, a single operator blinded to patient identity performed the test and interpreted the findings. A mixed effects linear regression model was fitted, including treatment and matching variables (fixed effects) and matched pairs (random effects). An unconstrained covariance model was assumed. The estimated treatment coefficient was taken as the mean ALAEF difference. In normal subjects in SR, mean ALAEF was 43 ± 18.2% (3), which could be taken as the minimum clinically important difference in ALAEF.

Between 2013 and 2015, 22 pairs were recruited and had LA functional measurements. Maze patients had lower mean ALAEF (18.4%) than control subjects (26%). One-to-one comparison shows that control subjects had higher ALAEF in all but 3 pairs (**Figure 1**). After adjusting for the paired design and matching variables, mean ALAEF was 8.03% lower in maze than control subjects (95% confidence interval: −12.43% to −3.62%; p = 0.0015).

Patients with chronic AF may have persistent LA dysfunction even after restoration of SR by ablation. Buber et al. (2) reported that absence of LA contraction, despite SR restoration, is associated with a significant increase in the risk of thromboembolic stroke after maze. Global and regional atrial dysfunction may be the result of a combination of injury from the ablation process and pre-existing disease (2). The 2 adverse features of asymptomatic AF, thromboembolism and effect on cardiac function, are both directly related to atrial function. Restoring SR without restoring function is unlikely to be of clinical benefit. The HESTER study provides evidence that function is indeed restored after adjunct maze, with potential clinical benefits in reducing

**FIGURE 1 ALAEF Measurements for Individual Patients Undergoing the Maze Procedure and Their Matched Control Subjects**




ALAEF = active left atrial ejection fraction.

thromboembolic and heart failure risk. Determining whether patients can safely stop taking anticoagulants after SR is restored by a maze procedure requires long-term follow-up and stroke surveillance beyond the HESTER study. The varying rates of LA functional recovery after maze means that it would be prudent to measure atrial function before considering anticoagulation withdrawal.

In summary, a return to SR after adjunct maze is associated with recovery of LA function but with a mean ALAEF smaller in maze patients than in control subjects. This functional recovery and the variability observed within it may have important implications for survival, heart function, and clinical decisions on long-term anticoagulation.

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# Deferred Stenting in STEMI

## Still an Interest in Selected Patients?

Deferred stenting (DS) in the setting of ST-segment elevation myocardial infarction (STEMI) has been the subject of numerous controversies, but the current view is that DS should be restricted to very selected cases, as demonstrated once again by Lønborg et al. (1). Notwithstanding, it is widely acknowledged that thrombus plays a key role in the pathophysiology of STEMI, and the size and composition of thrombus may affect the results of primary percutaneous coronary intervention, given the risk of distal embolization. The ratio of plaque to thrombus is highly variable among culprit lesions; it is also highly variable with time, and this represents the conceptual basis of the DS strategy. Despite the overall negative results of this study in terms of microvascular obstruction (1), we think that there is still a place for a "tailored DS strategy," provided that thrombus-laden lesions can be selected. In this respect, Lønborg et al. (1) showed a highly significant interaction between DS and lesion length that may represent an interesting parameter for selection of patients who are potentially eligible for DS.

This finding is perfectly in line with our observation that, in patients with STEMI who are treated with DS, the longer the culprit lesion, the greater is its shortening with time (2). In particular, for lesions longer than 23.7 mm (very close to the 24-mm cutoff reported by Lønborg et al. [1]), this shortening could reach more than 7 mm (2). Within such a brief time frame, this result clearly implies that the length of the lesion is associated with thrombus content. The paper by Loøborg et al. (1) strongly fuels the hypothesis of a prognostic advantage of DS in the presence of a long lesion, probably through thrombus regression. Although it is a post hoc analysis with all the inherent limitations of such a study, we find this result extremely promising for tailoring the best strategy during primary percutaneous coronary intervention. However, we acknowledge that routine DS in all patients with STEMI is probably not an option. We believe that further adequately designed clinical trials that have an evaluation of thrombotic load and lesion length as their basis should be encouraged.

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