

Scientific letters

Heart Failure, Nonvalvular Atrial Fibrillation and Anticoagulation Control With Vitamin K Antagonists***Insuficiencia cardiaca, fibrilación auricular no valvular y control de la anticoagulación con antagonistas de la vitamina K*****To the Editor,**

Classic anticoagulation therapy with vitamin K antagonists (VKA) reduces the risk of thromboembolic events very significantly in patients with nonvalvular atrial fibrillation (NVAF). Recent studies in Spain have shown that the quality of VKA anticoagulation is poor and that about half of these patients have a time-in-therapeutic range (TTR) of < 65%.^{1,2} It would be useful to identify which variables predict or are associated with worse VKA anticoagulation control. Heart failure (HF) or ventricular systolic dysfunction has been associated with poor control in several studies.^{3–5} The hepatic congestion caused by HF appears to lower VKA metabolism in the liver, increasing the anticoagulant effect, and leading to an INR (International Normalized Ratio) of > 3, and a higher risk of bleeding. Heart failure is common in patients with NVAF and is one of the thromboembolic risk factors in the CHADS₂ and CHA₂DS₂-VASc indices. Therefore, VKA anticoagulation is also common in these patients. Some authors have suggested that new direct anticoagulants could be first-line therapy for patients with heart failure, because the mechanism of action in liver metabolism is independent of HF in these novel drugs. However, one study has found no relation between HF and poor control with VKA anticoagulation,⁶ and therefore we believe it would be useful to have more data to help clarify this issue.

To provide more data, we analyzed the results of the recent CALIFA study.¹ This registry enrolled 1056 patients with NVAF receiving VKA at 120 cardiology clinics in Spain between November 2013 and March 2014. Data were included for all INR test results in the 6 months before the baseline visit. The patients' general characteristics are described in the published article.¹ Half of the patients (50.1%) had associated heart disease, and almost a quarter (22.2%) had HF. Of the 235 patients with HF, 88 (8.3% of the total and 37.5% of patients with HF) had systolic dysfunction, with left ventricular ejection fraction < 45%. In the entire sample, the TTR was < 65% in 47.3% of patients according to the Rosendaal method, and the mean TTR was 63.8% ± 25.9%. There were no differences in the TTR between patients with or without a history of HF or left ventricular systolic dysfunction. The percentage of patients with poor anticoagulation control (TTR < 65%) was actually lower among those with a history of HF, although the differences were not statistically significant (47.4% vs 52.6%; *P* = .189, NS), with an odds ratio of 0.786 (95% confidence interval, 0.557–1.109; *P* = .176, NS) in the bivariate analysis. The results were similar regardless of whether the ejection fraction was more or less than 45%. As already reported,¹ in the multivariate analysis, the independent variables associated with poor anticoagulation quality (TTR < 65%) were moderate or severe kidney disease (glomerular filtration rate < 60 mL/minute), routine nonsteroidal anti-inflammatory drugs, antiplatelet therapy,

and absence of angiotensin receptor blockers. The results remained unchanged when HF was added as a variable to the multivariate analysis by forced inclusion. The mean TTR was similar in patients with or without a history of HF (64.1% ± 26.7% vs 62.1% ± 25.5%, NS).

Our results suggest that the quality of VKA anticoagulation in patients with NVAF is not affected by the presence or absence of HF (or by preserved or reduced ejection fraction), which contrasts with the results of several studies published to date,^{3–5} but coincides with those of another study reporting the same finding.⁶ The mechanisms by which HF could alter the effect of VKA are related to the reduced VKA metabolism in the liver by the cytochrome P450 system due to decreased oxygen, particularly in the liver cells in acinar zone 3, where there is greatest cytochrome enzyme activity.⁴ However, this tissue hypoxia occurs mostly in decompensated HF, and as some studies have shown,³ the risk of worse VKA anticoagulation can be stratified by certain HF characteristics (liver enzyme changes, serum sodium, diuretic requirements, and decompensation frequency). In short, HF severity and liver involvement, but not a history of HF alone, are predictors of poor anticoagulation control. Therefore, our results show that, in general, patients with NVAF and a history of HF respond to VKA similarly to those without a history of HF. However, patients with frequent decompensation or more severe HF should be monitored more closely.

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Refractory Electrical Storm: A Role for Transient Sympathetic Blockade



Tormenta arrítmica refractaria: papel del bloqueo simpático transitorio

To the Editor,

Electrical storm (ES) is defined as the appearance of 3 or more episodes of ventricular tachycardia or fibrillation within 24 hours requiring antitachycardia therapy or cardioversion/defibrillation.¹ In practice, the number of episodes is usually much higher, producing an extremely serious situation. The role of the sympathetic nervous system in ES is well established,² and sympathetic blockade has been shown to effectively control these situations.³ The first studies with sympathetic blockade centered on its use to treat congenital long-QT syndrome in patients unresponsive to beta-blocker therapy, and it has recently been applied in the treatment of ES.⁴

The goal of this study was to present our accumulated experience with transient sympathetic blockade (TSB) in the treatment of a series of patients with refractory ES, defined as ES persisting after conventional therapy for the control of ES and its immediate causes.

Refractory ES was diagnosed according to the judgment of the responsible physician, without prior specification of criteria for episode duration, number, or toleration. In all patients, TSB was first attempted by left stellate ganglion block (LSGB); patients with recurrent ES or poor initial control after LSGB were scheduled for

bilateral block by thoracic epidural anesthesia. LSGB was performed by ultrasound-guided bolus injection of local anesthetic via the paratracheal route. The procedure was carried out at the patient's bedside in the coronary care unit by anesthesia unit staff. Ultrasound guidance was also used to place a soft catheter for continuous infusion of 0.2% ropivacaine. The mean infusion rate was 7 mL/h, with an initial rate of 6 mL/h and a maximum of 12 mL/h. The efficacy of TSB was evaluated by clinical observation: the efficacy indicators were clinical signs of Horner syndrome for LSGB and relief of anterior chest pain in patients given thoracic epidural anesthesia. In all patients, TSB was used as an addition to conventional antiarrhythmic therapy and in no instances replaced or required withdrawal of baseline pharmacologic treatment. In 2 patients, LSGB did not achieve sufficient electrical control, and these patients were given thoracic epidural anesthesia with 0.2% ropivacaine. Sympathetic blockade was withdrawn according to the medical team's criteria after the patient had been free of arrhythmias for 48 hours.

The effectiveness of the technique was estimated by comparing the number of episodes of sustained ventricular arrhythmia before and after TSB. The statistical significance of the reduction in the number of episodes after LSGB was assessed by the Wilcoxon test for paired data.

Between March 2012 and December 2014, our team performed TSB on 8 patients diagnosed with refractory ES. The mean patient age was 58 years, and 75% had severe left-ventricular systolic dysfunction. Baseline patient characteristics, the trigger for ES, and the initial treatment are summarized in the [Table](#).

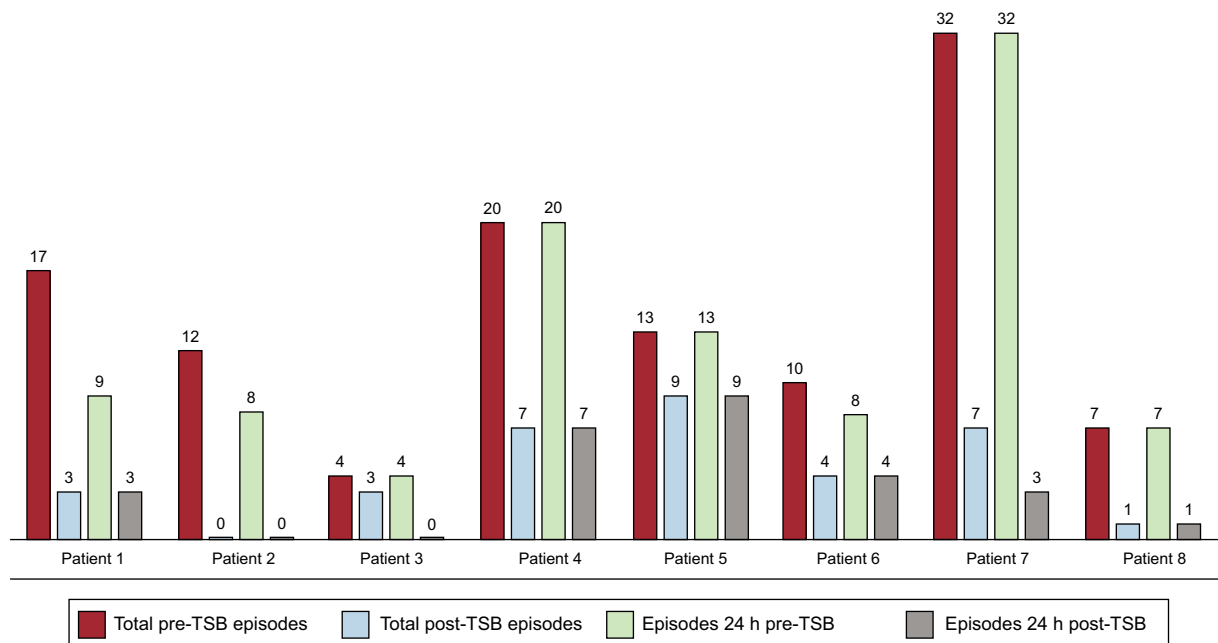


Figure. Number of electrical storm episodes, defined as the presence of sustained ventricular arrhythmias. The graph shows total episodes and episodes in the 24 hours before and after transient sympathetic blockade TSB: Transient sympathetic blockade.