the performance of the global residence time for LVT detection (receiver-operating characteristic area under the curve [AUC] = 0.79) outweighed that of the AWMS (receiver-operating characteristic AUC = 0.64; p = 0.05 for comparison of both AUCs). According to bivariate logistic regression analysis that included average residence time and AWMS, only the former was significantly associated with LVT (p = 0.001 and p = 0.20, respectively). LV volumes and ejection fraction were of no use to predict LVT detection in this cohort.

To the best of our knowledge, this is the first prospective study demonstrating that objective indices of LV stasis can be derived from bedside echocardiography. Our results suggest that stasis imaging is a more powerful predictor of thrombosis than AWMS (5) in the setting of AMI. Thus, stasis imaging is a promising tool to address the risk of cardioembolic stroke in the clinical setting.

Pablo Martinez-Legazpi, MEng, PhD*
Lorenzo Rossini, AEng
Candelas Pérez del Villar, MD, PhD
Yolanda Benito, DCS, DVM
Carolina Devesa-Cordero, MD
Raquel Yotti, MD, PhD
Antonia Delgado-Montero, MD
Ana Gonzalez-Mansilla, MD, PhD
Andrew M. Kahn, MD
Francisco Fernandez-Avilés, MD, PhD
Juan C. del Álamo, AEng, PhD
Javier Bermejo, MD, PhD

*Department of Cardiology
Hospital General Universitario Gregorio Marañón
Dr. Esquerdo 46
28007 Madrid, Spain
E-mail: pablom.legazpi@redcardiovascular.com

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Left Ventricular Hypertrophy Diagnosed on ECG Also Predicts Early Mortality After Stroke

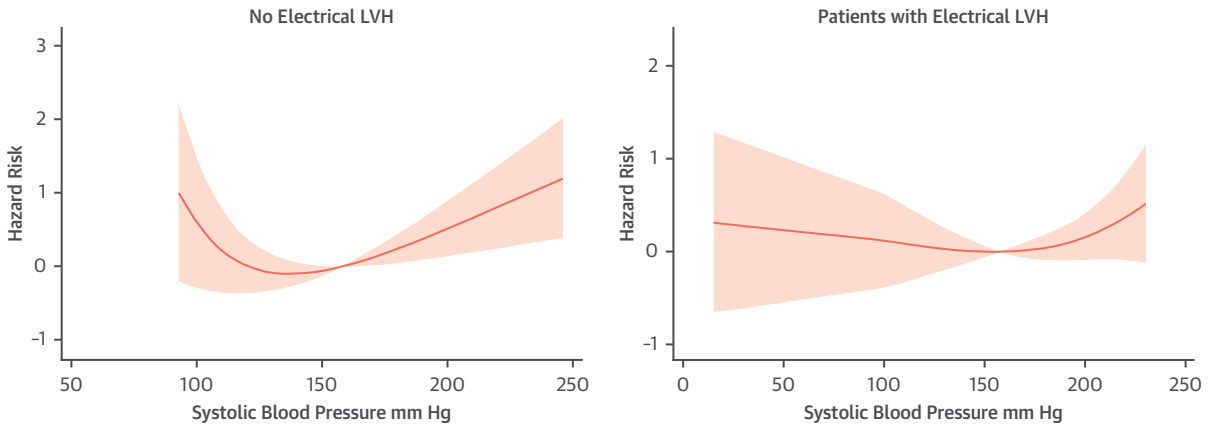
The paper by Park et al. (1) recently published in *iJACC* offers convincing evidence that patients with concentric left ventricular remodeling and eccentric and concentric left ventricular hypertrophy (LVH) have an increased risk of all-cause mortality after an ischemic stroke, as compared with patients with a normal left ventricle.

To ascertain whether electrocardiographic findings demonstrate a similar risk of death, we evaluated the risk of death at 1 and 12 months after an acute stroke, according to LVH defined by values of the Cornell voltage duration product ≥2,440 mV/ms. We analyzed data from the ERMANCIA II (Etude Réalisée en MArtinique et Centrée sur l'Incidence des Accidents vasculaires cérébraux) study, a prospective population-based cohort constituted in 2012 (2). Among the 544 patients with a first ever stroke who were initially enrolled, 32 patients with no electrocardiograms available, 17 with bundle branch block, 5 with pacemakers implanted, and 4 with at least moderate aortic stenosis were excluded from the study.

The final analysis included 486 patients (n = 262 men and 224 women; mean age 72.1 ± 15.5 years). LVH was found in 101 patients (20.8%). Patients with LVH had a higher prevalence of hypertension (84.2% vs. 61.0%; p < 0.001), and they were more likely to be receiving current antihypertensive treatment (70.9% vs. 49.8%; p < 0.001). Nonetheless, they had similar mean systolic and diastolic blood pressure levels at entry. They were also twice more likely to present with atrial fibrillation. Stroke subtype and the premorbid Rankin disability score were not different, but patients with LVH had a more severe initial clinical presentation, with National Institutes of Health Stroke Scale score of 11.4 ± 10.7 versus 8.3 ± 9.2 (p = 0.007).

Death occurred in 160 patients during the first 12 months. The crude cumulative 1-year mortality rate was 36.1% in patients with LVH versus 28.2% in controls. Kaplan-Meier survival analyses stratified by LVH showed significantly worse survival in the LVH group, with 1.86% and 1.67% hazard risks of death at 1 and 12 months, respectively (all p < 0.001). The excess risk remained significant after adjustment for age, sex, and stroke subtype and severity.

FIGURE 1 Systolic Blood Pressure and Early Death in Patients With Acute Stroke



Adjusted fractional polynomial Cox regression showing the relationship between systolic blood pressure and adjusted risk of all-cause death in the first month following stroke, according to the presence **(right)** or absence **(left)** of left ventricular hypertrophy (LVH) diagnosed by the voltage duration Cornell product on electrocardiogram. The **dark line** indicates the hazard ratio (log transformed), and the **shaded area** its 95% confidence interval. Adjustment variables included age, sex, waist circumference, history of hypertension or diabetes, stroke subtype, premorbid Rankin score, and National Institutes of Health Stroke Scale score on admission.

We also investigated the relationship between systolic blood pressure and rate of death in groups defined by the presence or absence of LVH. In both groups of patients with or without LVH we observed a U-shaped relationship, with a curve inflection around 150 mm Hg (Figure 1).

Thus these results further support the deleterious role of LVH in the occurrence of death after stroke, in accord with the paper presented by Park et al. (1). These results expand the initial findings observed using echocardiography to electrocardiographically defined LVH. These findings have immediate clinical implications, given the greater availability of electrocardiography over echocardiography in the early course of the disease in emergency departments and even in dedicated stroke units.

Some gray areas do exist, notably given the discrepancy between differences observed in patterns of systolic blood pressure and rates of death. This issue and the mechanisms involved need further investigations. Altogether, both studies diverge from a previous study suggesting a clinical benefit associated with higher left ventricular mass in patients with acute stroke who were treated with thrombolysis using intravenous tissue plasminogen activator (3). The studies also shed fresh light on a surprisingly forgotten issue and open a new field to improve the daily care of patients with stroke.

Jocelyn Inamo, MD, PhD*
Nathalie Ozier-Lafontaine, MD
Stephane Olindo, MD

*Département de Cardiologie
Centre Hospitalier Universitaire de Fort de France
BP 632
97200 Fort de France
Martinique
France
E-mail: Jocelyn.inamo@chu-martinique.fr

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THE AUTHORS REPLY:

We thank Dr. Inamo and colleagues for their interest in our paper (1) and congratulate them for performing a meaningful analysis using data from a previous cohort study to assess the prognostic role of electrocardiography (ECG)-determined left ventricular hypertrophy (LVH) in stroke patients. We would like to start by agreeing that LVH, whether measured by ECG or echocardiography, can be associated with