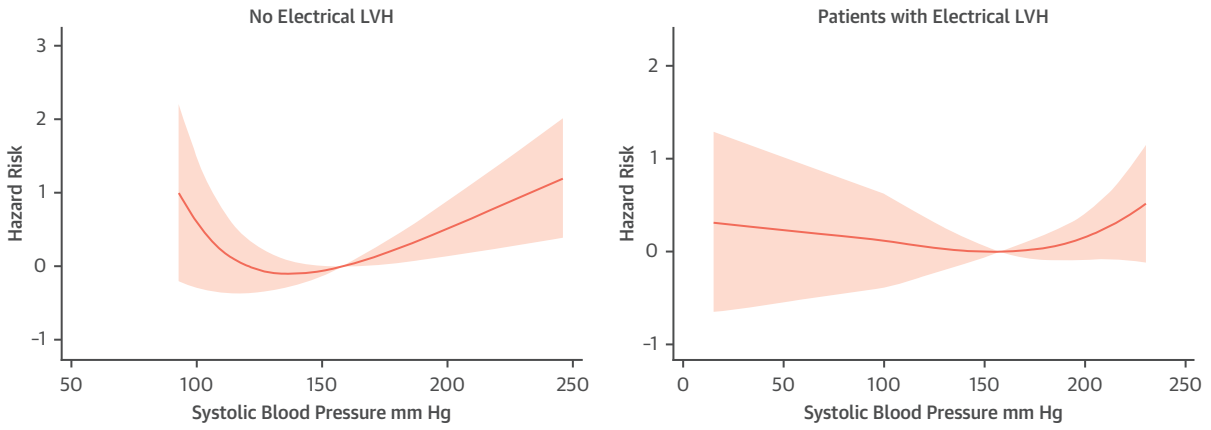
FIGURE 1 Systolic Blood Pressure and Early Death in Patients With Acute Stroke



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.

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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

unfavorable outcomes in stroke patients. Indeed, it is not surprising that initial studies reporting the prognostic usefulness of LVH in various populations frequently used ECG-based methods for LVH assessment. However, it is also not surprising that the sensitivity of ECG criteria for LVH is quite low compared with imaging methods, including echocardiography or cardiac magnetic resonance. Hence, a recent study has proposed a novel ECG criterion for LVH diagnosis to improve diagnostic performance over traditional criteria (2). Although this new criterion outperformed the classic Cornell criteria, its sensitivity was still 62% compared with echocardiography, the reference standard used in this study. Thus when discussing the predictive ability of ECG-derived LVH, it should be considered that a substantial portion of cases of true anatomic LVH may be misclassified as normal according to ECG criteria. Despite this limitation, we agree with Dr. Inamo and colleagues that ECG-defined LVH has prognostic relevance in stroke patients, as supported by data demonstrating that ECG-diagnosed LVH independently predicted outcomes even after adjusting for imaging-based left ventricular (LV) mass. It can be speculated that the development of ECG changes associated with increased LV mass per se may offer prognostic information by reflecting the complex alterations in electrical properties of myocardium.

With regard to the interaction between LV geometry and blood pressure (BP), we think that the findings by Inamo and colleagues, the U-shaped relationship of mortality with BP regardless of the presence of ECG-based LVH, should be cautiously interpreted, given the aforementioned limitation. In other words, the U-shaped association between mortality and BP in patients without LVH could stem from the misclassification of true LVH as normal. Furthermore, although their exclusion criteria included patients with significant aortic stenosis, there is a possibility that potential confounding diseases were not excluded, such as valvular heart disease (other than aortic stenosis), cardiomyopathy, or pericardial diseases. However, because our hypothesis also remains just an intriguing possibility, we definitely agree with Dr. Inamo and colleagues and the excellent accompanying editorial by Drs. Gillebert and Chirinos (3) that further studies are warranted to verify the interaction between LV remodeling and BP as prognosticators in stroke patients and to elucidate its underlying mechanisms.

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Coronary Artery Calcium Progression: Increasing CAC Is Associated With Increased Events

We read with interest the well written paper by Mori et al. (1) related to coronary artery calcium (CAC) progression. Despite the apparent emphasis on clinical utility implied from the title, “What Does It Really Mean” (1), the clinical importance of CAC progression is never addressed in the paper. The clinical perspectives section of this paper infers that better acquisition parameters using computed tomography are required to assess CAC progression, but Mori et al. (1) fail to cite or refer to even 1 study published related to CAC progression and subsequent cardiovascular (CV) events. The ultimate proof of a surrogate marker is that it can discern or predict CV endpoints, which Mori et al. (1) did not address. There are dozens of studies published demonstrating that CAC progression is associated with increased CV events (2), including such large prospective studies as MESA (Multi-Ethnic Study of Atherosclerosis) (3), as well as large observational studies in thousands of patients (4). The studies, encompassing more than 20,000 participants in total, consistently demonstrate that an increase in CAC is associated with a 4- to 7-fold increase in CV events, independent of the baseline CAC score, CV risk factors, and demographic variables.

Furthermore, studies have demonstrated that progression of CAC is significantly associated with an increase in both calcified and noncalcified plaque volume, paralleling an increase in atherosclerosis