between the 2 tests: 122 ± 15 beats/min versus 121 ± 16 beats/min.

Patients with SPEPP+ latent obstruction more frequently reported PP symptoms: 92% versus 29% ($p = 0.007$). At rest, they had longer posterior mitral leaflets: 1.7 ± 0.3 cm versus 1.2 ± 0.3 cm ($p = 0.002$). Initial post-exercise gradients were mildly higher in patients who ultimately were SPEPP+: median 34 mm Hg versus 16 mm Hg ($p = 0.008$). Overall, in 9 of 13 SPEPP+ patients, there was a change in therapy to relieve obstruction. Patients with SPEPP+ latent obstruction were more frequently treated with beta-blockade: 100% versus 43%; 5 patients received disopyramide and 3 underwent septal myectomy. In patients with gradient-lowering therapy because of SPEPP+, Minnesota quality of life decreased from 29 ± 19 to 12 ± 19 ($p < 0.02$), but there was no change in SPEPP− patients.

We selected patients with HCM whom we suspected had LVOT obstruction as the cause of their symptoms, despite no significant obstruction with standard provocations and after symptom-limited conventional stress exercise. In these patients, 13 of 20 (65%) were found to have substantial LVOT gradients after SPEPP, which led to an alteration in treatment for 9 of 13 patients.

Treatment choices for relief of limiting symptoms in HCM depend heavily on the presence of LVOT obstruction. Patients' severe symptoms and outflow obstruction may be treated successfully by addition of medications or septal reduction. In contrast, there are limited options for patients who are nonobstructed. Exercise echocardiography is an excellent modality for provocation in patients who might otherwise be categorized as nonobstructed (2).

However, even after a stress exercise echocardiogram that does not provoke gradient, the clinician may still suspect obstruction in certain patients. It is our current clinical practice to employ SPEPP after conventional stress echocardiography for patients with putative nonobstructive HCM who have: 1) New York Heart Association 3 symptoms, especially PP worsening, who otherwise would have limited treatment options; 2) long leaflets or mild systolic anterior motion but no demonstrable gradients; and 3) syncope but where arrhythmias cannot be detected, even after prolonged monitoring.

Valsalva and standing increase gradient due to a reduction in venous return, a decrease in LV volume, and an increase in the overlap between LV inflow and outflow (2). Consequent decrease in stroke volume increases sympathetic activity, augmenting obstruction. In healthy individuals after a meal, peripheral vascular resistance decreases because of mesenteric vasodilation, with a secondary increase in stroke volume. In HCM, deleterious hemodynamic effects of a meal include limited rise of stroke volume, greater elevation in right heart pressures, and provocation of LVOT gradients (1). During exercise alone, augmented contractility increases obstruction. We have shown that selected patients who have no obstruction after conventional stress echocardiography performed fasting and imaged supine may develop large gradients (median 83 mm Hg) when exercised after eating and when the gradient is acquired upright. We posit that SPEPP more profoundly alters load and contractility than conventional stress echocardiography.

The diagnosis of latent obstruction altered therapy in 9 of 13 (69%) of our patients. Five received disopyramide and 3 had surgical septal myectomy. Patients who received new therapy because of SPEPP+ latent obstruction experienced a dramatic reduction in symptoms. A limitation of the study is that LVOT gradients after stress were not interpreted blindly. Because study patients were selected by clinical suspicion, detection rates may not be generalizable.

Not infrequently, findings in a symptomatic patient with HCM will raise the clinical suspicion that the patient is suffering from LVOT obstruction, even if routine stress echocardiography is negative. Performing stress echocardiography in the PP state with gradient acquired in the upright posture (SPEPP) correctly diagnoses latent obstruction and thus focuses management on gradient reduction.

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Letters to the Editor

Do Work Accidents Play Any Role in the Increased Risk of Death Observed in 25- to 44-Year-Old Patients After Syncope?

We read with great interest the paper by Ruwald et al. (1) dealing with the prognosis of healthy individuals discharged from the emergency department or hospital after a syncope episode.

Surprisingly, fainters 25 to 44 years of age had a risk of death higher than that of subjects older than 75 years of age, differing from what was previously reported (2). Moreover, in individuals 25 to 44 years of age with syncope, the long-term all-cause mortality

rate was twice that observed in the syncope group older than 75 years. This is unexpected too because the long-term (1 year) prognosis of syncope was found to be related to aging and comorbidities (3) that are likely to be more frequent in elderly individuals.

The authors hypothesized that there could have been an underdiagnosis of unrecognized cardiovascular diseases in syncope patients 25 to 44 years of age (1).

Here we propose an additional potential explanation. A preliminary analysis of our STePS (Short-Term Prognosis of Syncope) (3) database indicated that 1-year syncope recurrence in working-age (18 to 65 years) patients was as high as 14%. Significant hazard ratio values of recurrent syncope were also reported by Ruwald et al. (1) in the 2 groups of syncope subjects 25 to 44 years of age and 45 to 74 years of age. In a setting of hazardous occupations, syncope recurrence might lead to fatal work accidents in the group of syncope patients 25 to 44 years in the present study who, potentially, are active workers. According to the EUROSTAT Health and Safety at Work in Europe report (4), most of work fatal accidents are classified as occurring after "loss of control," "slipping," "stumbling," and "falling." All these conditions might be the consequence of an occult syncope, producing a sudden loss of consciousness and postural tone.

We wonder whether work accidents, produced by a hidden syncope recurrence, might play any role in the increased risk of death found by Ruwald et al. (1) in patients 25 to 44 years of age. A potential answer might be found by matching the present study data with occupational accidents recordings. Should this hypothesis be confirmed, an unexplored scenario characterized by remarkable social implications could be opened.

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Does the Absence of Comorbidities Really Identify Low-Risk Syncope Patients?

We read with great interest the paper by Ruwald et al. (1) on the prognosis among healthy individuals discharged with syncope as the primary diagnosis. However, we wonder whether the subjects enrolled in the study can be effectively considered low-risk patients. Syncope is, most of the time, the first manifestation of several diseases, some of which are life-threatening, such as pulmonary embolism (2). In our opinion, the absence of comorbidities cannot by itself identify low-risk patients. Indeed, none of the risk stratification tools for syncope in the emergency department (ED), derived in both the short or long term, consider comorbidities as risk factors, except for cardiac diseases (3). On the contrary, patient admission to the hospital might be due to the clinical perception that the patient is not at low risk of adverse outcomes. During hospital admission, a specific cause of the syncopal episode is identified in only 30% to 50% of the cases (4,5). Thus, syncope as primary discharge diagnosis is not necessarily consistent with the benignity of the clinical presentation.

Moreover, Ruwald et al. (1) observed an increased mortality rate from syncope after some years, whereas in the first year, data were inconclusive. If syncope itself were responsible for the increased mortality, should we not observe such increase in the short term rather than in the long term when comorbidities (3) seem to play a more relevant role?

Finally, from our point of view, low-risk patients should be those without comorbidities but who were also discharged from the ED. It would be of great interest to re-analyze the data from this point of view. We think that this analysis could strengthen the study results.

There might be a misprint in Table 3 because the 95% confidence interval of the point estimate does not include the point estimate itself.

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