temporal resolution and reduced radiation doses, these develop-
ments include the introduction of complementary explorations
for the detection of ischemia (perfusion, noninvasive determina-
tion of functional repercussion of stenosis, etc.). These advances
have made MDCT one of the most sensitive and specific methods
for ruling out significant coronary artery disease, second only to
invasive coronary angiography. The noninvasive nature of MDCT
moreover brings added benefits, including the detection of subclinical coronary artery disease, the potential to characterize
high-risk plaques, and prognostic value.

2. Technical considerations. The diagnostic performance of MDCT
could have been improved with an optimized spatial resolution of
the reconstructions, achievable by modifying the slice thickness,
the between-slice increase and filters as described
by other authors working with exactly the same type of
scanner. Additionally, given the mean body surface area
observed in the study population (although the benchmark parameter in cardiac CT is body-mass index), a tube potential of
100 kV would have improved luminal contrast in the coronary
arteries, thereby facilitating image interpretation and exponen-
tially reduces the radiation dose. Such dose reductions are line
with Society of Cardiovascular Computed Tomography guide-
lines, which recommend the establishment of quality assurance
procedures to meet the following objectives: sufficient diagnost-
ic quality in ≥ 95% of scans, a demonstrable diagnostic accuracy
at least 75% that of invasive coronary angiography, and a mean
radiation dose at the reference level (12 mSv according to the
most recent guidelines). Today, with a careful acquisition
protocol and the latest scanners, doses are normally in the
region of 1-2 mSv or even lower, well below the 7-10 mSv in
invasive coronary angiography and the 8-10 mSv in isotope studies
with gamma radiation, demonstrated to be more
harmful than X rays.

3. Methodological considerations. An Agatston score > 400 is not
equivalent to the detection of significant coronary artery disease
by MDCT because this threshold drags down the specificity of
the method, with 20% of patients with this score having no
disease. The authors’ statement in the Discussion that “MDCT
has low diagnostic specificity” seems to me to be inappropriate.
What limits specificity is setting the significance threshold at
≥ 50% when the “reference pattern” is ≥ 70% for invasive
coronary angiography (luminogram) and MDCT is based on this
same “luminogram”, with the advantage of assessing the
coronary wall. The ≥ 50% significance threshold was established
in the cited study by Hoffmann, in which final cost-effectiveness
did not reach statistical significance. In contrast, the Goldstein
study, using a significance threshold of ≥ 70%, showed a
significantly positive cost-effectiveness for MDCT ($2137 for
MDCT compared with $3458 for standard; P < .0001).

The major scientific societies now accept the diagnostic value of
both techniques and their complementary nature, especially in
non-diagnostic MDCT studies and studies that indirectly evaluate
the functional repercussion of intermediate or limiting stenosis, an
evaluation achieved directly with pressure guides in invasive
coronary angiography.

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Comentarios a la evaluación del dolor torácico agudo mediante
eccardiografía de ejercicio y tomografía computarizada
multidetectores. Respuesta

To the Editor,

We would like to thank Dr Catalán for her comments and to clarify certain points.

Although major technological progress has been made in cardiac multidetector computed tomography (MDCT) since
2008 when the above-mentioned study was started, it is important to recognize that both the myocardial perfusion study and the recent evaluation of functional repercussion using MDCT discussed
by Dr Catalán are emerging techniques that are not included in
clinical practice guidelines. Noninvasive estimation of the coronary reserve flow using MDCT, whose analysis is still not
widely available, could be promising in the future, but its
diagnostic value in addition to MDCT angiography is still to be
determined for acute chest pain.

Dr Catalán states that the results could have been improved by a
different image reconstruction according to the study by
Rixe et al.2 The device used in our study provides a rotation time of
370 ms, inferior to the 330 ms used by Rixe et al. To compensate
for the loss of sharpness of the coronary lumen, we used 0.7 mm
slices and 0.4 mm increments instead of the 0.6 × 0.3 mm
suggested by Rixe et al, resulting from the tests performed and
consensus among 3 observers. For the same reasons, a tube current
of 120 kV was maintained, similar to that used by Rixe et al, instead
of the suggested 100 kV.

Our article acknowledges the specificity of MDCT was affected
by the 50% stenosis cut-off value, which is why we conducted
another analysis at 70%, producing a considerable improvement in
specificity. However, we did not think that an Agatston Ca score > 400 significantly impaired the specificity of MDCT, as only 1 in 5 patients with a Ca score > 400 did not show acute coronary syndrome. With similar devices, in the presence of a Ca score > 400, the proportion of nonconclusive studies increases, luminal stenosis is overestimated and the specificity of the technique is severely limited; moreover, a Ca score of > 400 has been shown to be an excellent predictor for significant coronary disease. Along the same lines, Goldstein et al. recommended performing single-photon emission computed tomography (SPECT) when Ca scores were > 100, markedly lower than the 400 score used in our study.

Finally, in our opinion, the cost-effectiveness differences between the studies by Hoffman et al. and Goldstein et al. were not exclusively due to the differences in the cut-off values chosen for stenosis (50% vs 70%). Moreover, there were differences in the prevalence of acute coronary syndrome in the MDCT group (9% vs 4.4%), as well as large differences in the percentage of additional tests conducted in the control groups of the 2 studies (45% vs 100%, respectively), which contributed to the discrepancies observed.

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The Genetic Background of Left Ventricular Hypertrabeculation / Noncompaction Remains Vague

El trasfondo genético de la hipertrabeculación/miocardiopatía no compactada ventricular izquierda sigue sin estar claro

To the Editor,

We read with interest the article by Rodríguez-Serrano et al. about familial left ventricular hypertrabeculation/noncompaction (LVHT) associated with a novel alpha-cardiac actin gene (ACTC1)-mutation in 4 family members (II:4, III:4, III:6, IV:1), of whom 3 (II:4, III:4, III:6) presented with noncompaction and 1 with hypertrabeculation of the explanted heart. We have the following comments and concerns.

We do not agree with the statement that the described ACTC1-mutation “caused” LVHT. LVHT is associated with mutations in a large number of different genes but no proof has ever been provided for any of these associations that a particular mutation is truly causative of this myocardial abnormality. Reservations against a causal relation comes from the following arguments: first, in most cases of hereditary disease in which LVHT has been described, only a limited number of mutation carriers also had LVHT. Second, LVHT may be a dynamic abnormality that may not be present at birth in single patients (acquired LVHT) and may more rarely even disappear during life. Third, most of the few patients with acquired LVHT did not carry a mutated gene and did not have LVHT on previous echocardiographic or other cardiac imaging studies. Fourth, according to the authors themselves, the pathogenicity of the detected ACTC1 variant was neither confirmed nor excluded by in silico analysis. Fifth, the mutated genes so far associated with LVHT are responsible for a variety of hereditary disorders, ranging from cardiac to neuromuscular disease, including hereditary neuropathies and cobalamin-C deficiency. Sixth, LVHT frequently occurs in patients with chromosomal defects (eg, p1.36 syndrome).

Given these arguments, we consider LVHT to be a secondary myocardiabnormality in compensation for other cardiac disease, possibly induced by upregulation of regulatory genes.

Concerning the index patient, some confusion derives from the description of the explanted heart as having shown LVHT but this is not mentioned in the pedigree. Instead, the authors describe the patient as having “left ventricular hypertrabeculation”. What is the difference between noncompaction and left ventricular hypertrabeculation? In our understanding, noncompaction and hypertrabeculation are 2 different terms for the same entity. The term hypertrabeculation, however, appears to be the more favorable one since it is descriptive and does not imply a causal relation.

Since there is no general agreement on the definition of LVHT, it would be interesting to know if LVHT in the 4 individuals presented would meet Chin’s or Stöllberger’s diagnostic criteria.

The echocardiographic image of patient IV:1 is not convincing. Why was LVHT absent on echocardiography? Were the cine loops of this investigation revised? Was LVHT truly absent? If truly absent, what was the reason for the discrepancy with the histologic finding in the explanted heart? Since it is mentioned that this patient had undergone heart transplantation, a picture of the explanted heart would be helpful.

Although involvement of the skeletal muscle in ACTC1-mutations has not been reported, it is advisable to investigate all individuals with LVHT neurologically. This is because neuromuscular disorders