overall mortality (secondary endpoint). Previous meta-analysis failed to prove the effects of CRT alone on survival because trials that also included patients receiving a CRT with an added defibrillator function were also considered in their analysis. As it is highly unlikely that a trial comparing the effects of CRT alone with optimal medical therapy will ever be conducted, it was our purpose to attempt to give a definitive estimation of the effects of CRT on overall mortality in this specific patient population. The message of this meta-analysis is that there is enough evidence to strongly support CRT as a class I indication to improve survival in this selected group of patients with advanced systolic heart failure. Whether the level of evidence sustaining the recommendation to implant CRT devices should be A instead of B is probably more dependent on the definition used by the task force working group (some task forces only consider multiple randomized clinical trials and not meta-analyses as level of evidence A).

The second reason for conducting this meta-analysis was to evaluate in what way CRT affects the mode of death. Probably, no randomized clinical trial will ever be conducted for the purpose of answering this question. We think, as the CARE-HF investigators showed, that the positive effect of CRT on mode of death is probably time (remodelling)-dependent. Our meta-analysis showed that CRT did not affect the incidence of sudden cardiac death (SCD) during the follow-up covered by it (18.4 months). Nonetheless, CRT modified the mode of death, increasing the proportion of SCD relative to other modes of death. After long-term treatment, CRT probably also reduces the incidence of SCD; however, the proportion of patients dying suddenly remains high and the use of a combined CRT device with defibrillator function, when indicated, is warranted.

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The chronic heart failure is not so frequent in non-compaction

Isolated and associated non-compaction of the left ventricle is an uncommon cardiomyopathy, with an unclear natural history.1-3 Some authors described a bad prognosis for this disease.1,2 Instead, other published works, such as the Lilje’s1 work published in *European heart journal* in August, with a longer follow-up and with a larger cohort, reported a medium-long term good prognosis. Correlated with the prognosis is the ventricular dysfunction.

During this last year, we collected more than 230 patients in the Italian society of cardiovascular ultrasound registry. The incidence of chronic heart failure (EF < 45%) in our experience was around 50%. However, an important bias influences these data: all patients enrolled in a hospital division, where, in a large percentage, were admitted for symptoms. So the analysed population is a selected population. To demonstrate this hypothesis, we studied all first-degree relatives of 31 patients. In total, we observed 48 relatives affected by non-compaction. Of the total patients, 61% (19/31) presented an EF reduction, whereas of the relatives, only 2% (1/48) presented an EF reduction.

In our opinion, high incidence described by Lilje et al.1 of ventricular dysfunction is a consequence for a selected cohort.

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The chronic heart failure is not so frequent in non-compaction: reply

We appreciate the interest of Fazio et al. in our paper. We agree with their valuable comments in many regards.

1. Prognosis. Presumably by accident, Fazio et al. cited our data as describing both a bad and a good prognosis. In fact, we do consider the outcome concerning. At 12 months follow-up, the occurrence of congestive heart failure (CHF), arrhythmias, and thrombo-embolic events was 68.0, 20.0, and 13.9%, respectively. The mortality was 7.1%.1 These figures are even higher in several other studies.

2. CHF. The incidence of CHF in the population studied by Fazio et al. was 50–61%. These data are likewise concerning. They support our and previous findings of a high incidence of CHF in non-compaction of the left ventricular myocardium (NCVM), as do additional recent studies.2,3 We discussed several theories and findings explaining why NCVM may lead to CHF.1 Further data have since been published in support of this discussion.4

3. Selection bias. As mentioned within the paragraph ‘study limitations’ of our paper, our population—like the one studied by Fazio et al.—was undoubtedly prone to a referral bias of a tertiary care medical centre. However, not all patients were symptomatic. We evaluated all consecutive
patients sent for echocardiography during a specified period of time. Reasons for presentation were innocent heart murmurs, a family history of congenital heart disease, rule-out of cardiac side effects of non-cardiac medications, among others.

4. Patient populations. While comparing study populations, we would be interested to know the age of the patients and relatives studied by Fazio et al. Presumably most of them were adults. By which criteria did patients enter the registry; was it hospital admissions only? How many NCVM patients were entered with and without congenital heart disease? NCVM subpopulations may carry a different cardio-vascular risk.

5. Incidence. We do not know the total number of patients entered into the highly specialized Italian registry. But certainly, the number of NCVM patients entered in 1 year (>230) is remarkable; as is the number of first-degree relatives detected with NCVM (48/31). It has been repeatedly stated that NCVM appears to have been previously under-diagnosed all together.

Pretty much all existing data on NCVM has been prone to a selection bias. We do not know the prevalence and the natural history in a truly non-selected population. Based on existing data, including our own study and the one by Fazio et al., the occurrence of CHF in NCVM is concerning. It will be highly interesting to learn about details of the investigation by Fazio et al. addressing first-degree relatives and future similar data. The outlook may be more encouraging in incidental or familial discovery of NCVM.

Unfortunately, even such populations are prone to a selection bias.

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Septal alcohol ablation in hypertrophic obstructive cardiomyopathy: improving cardiac function by generating a myocardial scar

We read with great interest the article by van Dockum et al. on the improvement of systolic myocardial function of the left ventricular (LV) lateral (free) wall in patients with hypertrophic cardiomyopathy (HCM) after alcohol septal ablation (ASA). Using cardiac magnetic resonance (CMR) tissue tagging and three-dimensional strain analysis, the authors found that both maximum end-systolic strain index and systolic strain index rate improved significantly in remote myocardium.

This report shows for the first time that the reduction of the LV outflow tract gradient in symptomatic patients with obstructive HCM treated with ASA is associated with the improvement in intramural systolic function in the lateral wall remote from the ablated area. Although this is an interesting finding, there is a main point to be addressed in relation with the procedure. In Figure 1, there is a clear demonstration of a gross gadolinium late myocardial hyperenhancement in the interventricular septum attributable to the procedure, although there is no report of direct comparison with pre-procedural gadolinium myocardial enhancement in the same patient. It would be very interesting if myocardial hyperenhancement data derived by CMR before and after ASA could be provided by the authors. Such data would be very helpful to estimate the impact of ASA on the development of new fibrosis superimposed on an already existing one.

The most dramatic event in HCM is sudden death attributable to arrhythmogenic substrate owing to cardiac fibrosis. Cell death with subsequent healing and replacement fibrosis induced by ASA eventually leads to an increase in the already existing myocardial fibrosis, creating a substrate more prone to arrhythmic events. In other words, we are trying to improve patient’s symptoms by generating a scar tissue that may be deleterious long life, especially for young subjects. Data on sudden death after ASA are lacking. Therefore, as stated by Maron, avoidance of septal ablation in young patients is probably prudent, especially if the surgical option is feasible.

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