tions and HRV indicators. Sample sizes, Gensini scores, and different aspects differ in each table (4, 5). However, we are yet unclear regarding the views of them expressed in the letter that the group distributions of HRV parameters in tables 3-5 in our study substantially overlap with each other and reduce the clinical applicability of the study results.

We sincerely hope that these responses can answer their questions.

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Obesity and coronary bypass

To the Editor,

We have read with great interest the article entitled "Obesity is still a risk factor in coronary artery bypass surgery" published in Anatol J Cardiol 2014; 14: 631-7 (1). The authors aimed to document the effects of obesity on surgical outcomes in patients undergoing coronary artery bypass surgery. They concluded that obesity was still a risk factor for occurrence of adverse events in cardiac surgery and the mortality rates were similar in obese and non-obese patients. We congratulate the authors for these valuable results.

There are several reports regarding the effect of obesity on postoperative mortality and morbidity after cardiac operations. Some of them concluded that obesity is a risk factor for both mortality and morbidity, and some concluded that obesity is a risk factor only for morbidity (2). This result is partially supported again with this article. However, the design of the article does not confirm the hypothesis of the manuscript because there is a statistical difference between the parameters which affect the operative mortality and morbidity, such as female gender, smoking, diabetes mellitus, and hypertension; even the result of the article is compatible with the literature (Table 3). We expect equality between the parameters which affect the prognosis. We assume that obesity is a risk factor not only for morbidity but also for mortality after coronary bypass surgery and after many other operations. There is a need to more detailed studies about the clarification of this difference between obese patients.

Furthermore, postoperative atrial fibrillation is a common complication after cardiac surgery and predicts increased morbidity and mortality. There are many studies in the literature which propose that atrial fibrillation is a risk factor for obese patients compared with that for non-obese patients (3). We believe that mortality resulting from obesity disagree with the results of postoperative atrial fibrillation which is more often seen in BMI <30 group, as shown Table 5. There is a need for further studies on this issue.

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Author`s Reply

To the Editor,

We would like to thank the authors for their contribution to our study entitled "Obesity is still a risk factor in coronary artery bypass surgery" published in Anatol J Cardiol 2014; 14: 631-7. (1) and their valuable comments. They have mentioned that, as outlined in Table 3, some preoperative demographic characteristics in obese and non-obese groups differed. They have also stated that these factors could play a role in postoperative morbidity and mortality. However, we know that female gender, diabetes, and hypertension are comorbidities of obesity. It is not easy to say that these factors played a direct role on adverse effects. It would be more reliable to state that the comorbidities of obesity increase these adverse outcomes. However, multivariate analysis could have been performed to increase reliability. We have re-analyzed the effect of each mentioned parameter on adverse effects

(female gender, hypertension, diabetes, and smoking) and did not find any statistically significant effects (p>0.05 each) on adverse outcomes except for increased rates of postoperative atrial fibrillation (POAF) in patients with diabetes (p=0.03).

The authors have also stated that they find the lower rates of POAF in obese group very conflicting. But, as mentioned in the original article, obese patients are more prone to insulin resistance which mandates increased use of perioperative insulin for strict blood glucose control. Insulin causes decrease in the occurrence rates of POAF (2). We find this explanation for the lower rates of POAF satisfactory.

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Noncompaction with dysmorphism, mental retardation, general wasting, and hypogonadism requires neurologic and sophisticated cytogenetic investigations

To the Editor,

We read with interest the article published entitled "Case of fatal heart failure with biventricular noncompaction, genital skeletal abnormalities and mental retardation." by Ataş et al. (1) regarding a 48-year-old female from consanguineous parents with dilated cardiomyopathy (dCMP), left ventricular hypertrabeculation/noncompaction (LVHT), primary amenorrhea, bilateral amazia, ovarian dysgenesis, uterine aplasia, hypergonadotropic hypogonadism, macrocephaly, facial acromegaly, arachnodactyly, pectus carinatum, and mental retardation who died from heart failure 4 months after being diagnosed with LVHT. We have the following comments and concerns.

We do not agree with the statement that LVHT is a genetic disorder. Although LVHT is associated with various monogenic disorders, in particular neuromuscular disorders (NMDs) and cardiomyopathies, and chromosomal defects (2), a causal relation between these genetic defects and LVHT has not yet been proven. The strongest argument against a causal relation is that only a small number of patients with NMDs, cardiomyopathies, and chromosomal defects present with LVHT (2). An argument in favor of a causal relation, however, is that LVHT also occurs familial (3).

The patient underwent cytogenetic investigation; however, it is not mentioned which technique was applied (1). Did the authors investigate complex chromosomal re-arrangements and micro-aberrations by means of fluorescence in-situ-hybridization (FISH) or microarray assays? In particular, did they apply multi-color FISH, telomere/subtelomer FISH, reverse painting, fiber FISH, quantitative FISH, or cobra-FISH?

According to Figure 1, the patient presented with generalized muscle wasting (1). Was this due to being bedridden prior to admission or was this due to involvement of the peripheral nerves or the skeletal muscles? Did the patient ever undergo a clinical neurologic investigation, nerve conduction studies, or needle electromyography? This is of particular importance because LVHT is associated with NMDs in more than half of the cases.

Concerning mental retardation and macrocephaly, it would be interesting to know cerebral imaging results. Was there cerebral atrophy, calcification, demyelination, or hydrocephalus? Did she ever develop seizures? Was an electroencephalogram ever recorded?

Because LVHT can be complicated by stroke embolism, it is important to understand whether the individual or family history was positive for stroke/embolism. Did cerebral imaging reveal previous embolic stroke? Furthermore, patients with LVHT and dCMP require oral anticoagulation with vitamin-K antagonists for primary prophylaxis of stroke/embolism (4). Did the patient receive phenprocoumon or warfarin in addition to heart failure therapy on dismissal?

Furthermore, because LVHT is complicated by arrhythmias, it would be worthwhile to know the results of long-term electrocardiography recordings. Did the two sisters and brother who deceased in childhood die suddenly? Was the family history positive for falls, syncope, fainting, or sudden cardiac death? Was an autopsy conducted in the three deceased children?

Because LVHT may be acquired in some cases (5), it would be interesting to know whether the patient had undergone previous echocardiographies and if these were revised for LVHT?

Overall, this interesting case merits further evaluation with regard to genetic background and possible neuromuscular or cerebral comorbidities. Only if LVHT patients are comprehensively investigated, the pathogenetic background of this enigmatic cardiac abnormality may be elucidated.

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