Table 1
Characteristics of the study population according to LVH patterns.

Variable	CLVH	ELVH	p
Age* (years)	59.2 ± 10.7	59.7 ± 11.3	0.58
BS** (m^2)	1.6	1.7	0.017
BMI**	2.6	2.7	0.18
LVMI** (g/m^2)	123.6	108.6	<0.0001
RWT**	0.49	0.37	<0.0001

*Mean; **Median; LVH = left ventricular hypertrophy; CLVH = concentric left ventricular hypertrophy; ELVH = eccentric left ventricular hypertrophy; BS = body surface; BMI = body mass index; LVMI = left ventricular mass index; RWT = relative wall thickness.

Table 2
Sensitivity and *p* values of the electrocardiographic criteria according to LVH patterns.

Variable	CLVH (*n* = 326)	ELVH (*n* = 437)	p
	Sensitivity% (CI)	Sensitivity% (CI)	
[(S + R) X QRS]	40.8 (35.4–46.3)	25.8 (21.8–30.2)	<0.0001
Sokolow-Lyon	16.5 (12.7–21.0)	11.4 (8.6–14.8)	0.0433
Cornell voltage	22.0 (17.7–26.9)	15.1 (11.8–18.8)	0.0172
Cornell duration	11.6 (8.3–15.6)	8.7 (6.2–11.7)	0.1812
Romhilt-Estes	15.6 (11.8–20.0)	10.3 (7.6–13.5)	0.0356
R_aV_L	12.5 (9.1–16.6)	8.9 (6.4–12.0)	0.1203
Perugia	34.0 (28.9–39.4)	23.8 (19.8–28.0)	0.0020
Strain	20.2 (16.0–25.0)	13.7 (10.6–17.3)	0.0181

LVH = left ventricular hypertrophy; CLVH = concentric left ventricular hypertrophy; ELVH = eccentric left ventricular hypertrophy; CI = confidence interval.

The level of agreement between the three observers ranged from 0.82 (QRS) to 0.98 (R and S amplitude).

Few studies have explored the power of ECG to discriminate the geometric patterns of LVH, maybe because of its known limitations [3].

Of the eight criteria analyzed, only R_aV_L and Cornell duration showed no significant differences, and are the two least capable criteria to discriminate the two patterns of LVH. Our findings show that sensitivity was always higher for the diagnosis of concentric LVH, certainly a reflex of more severe disease and myocardial deformation associated with this pattern, which is known to be related to a worse prognosis.

Aktoz et al. [4] analyzed 125 hypertensive patients using the Sokolow–Lyon, Cornell voltage, Cornell product, Gubner and $R_aV_L > 11$ mm criteria and concluded that the diagnostic value of ECG is acceptable, i.e., it has positive specificity and predictive value to differentiate normal and abnormal ventricular geometry, with modest accuracy in hypertensive patients. Nonetheless, its diagnostic value for the prediction and differentiation of the specific geometric patterns of LVH is poor.

The most of the electrocardiographic criteria analyzed showed statistically significant differences in the identification of the concentric and eccentric types of hypertrophic hearts, and this corroborates their usefulness in the diagnosis of the two established patterns of LVH.

The authors of this manuscript have certified that they comply with the Principles of Ethical Publishing in the International Journal of Cardiology (Shewan and Coats 2010;144:1–2).

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Troponin rise in healthy subjects during exercise test

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Cardiomyocyte necrosis is accompanied by a rise and fall of circulating troponin levels. The increased precision in the low analytical end of the hs-cTnT assay contributes to earlier clinical decisions [1]. The universal definition of myocardial infarction in patients presenting with symptoms of cardiac ischemia and/or changes in EKG requires a rise and fall of troponin, where at least one measurement is above the 99th percentile limit [2]. No consensus is obtained how large such a change has to be in order to be classified as significant. Such calculations must consider both biological variation and the analytical variation at the actual level. Participants in extreme sports leak cTn to the circulation at levels high above the accepted 99-percentile limit [3,4]. With the addition of suspect clinical symptoms like chest pain or typical ischemic ECG-signs, they would have fulfilled the international criteria for myocardial infarction. The physiological background of such a leakage is largely unknown. The intention of the present work was primarily to see if physical activity prior to blood sampling might contribute to the biological variation of cTn. Our second aim was to see if the fitness level of the participants might be of relevance.

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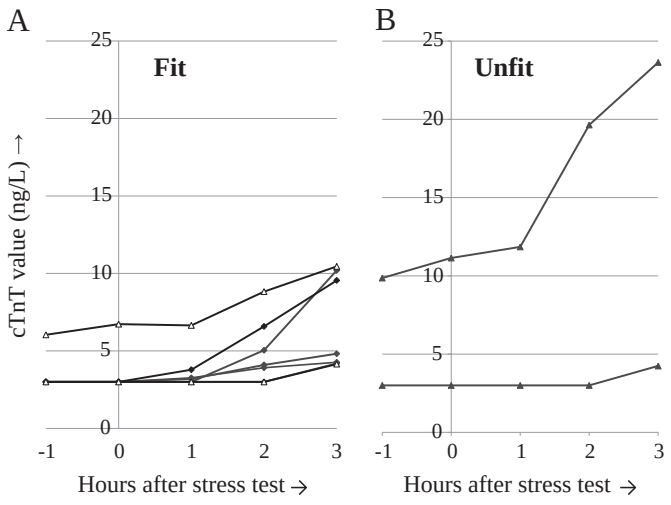

Fig. 1. A and B: The developing level of troponin is illustrated in the group of trained (fit) and untrained (unfit). Female participants are illustrated by closed and males by open symbols.

19 volunteers (55–67 years) were recruited from Oslo University Hospital staff. The study protocol was approved by the regional ethics committee. The participants filled out a questionnaire, which was used to select them into two physical fitness groups due to reported monthly physical activity. 16 of the 19 included volunteers were female. 7 participants in the female group reported little physical activity (<2 times per month, maximal 30 min of duration); the other 9 subjects reported better physical fitness (regular physical activity, 4–8 times per month, 45–120 min of duration). Of the three males, 1 reported little physical activity and 2 reported better physical fitness. All 19 participants were healthy, with no history of cardiovascular disease or use of any regular pharmacological treatment. Venous Li-heparin blood samples were drawn: before, immediately after completion of the bicycle stress test, and at 1, 2 and 3 h post-exercise. Hs-cTnT was measured in each sample (Roche Diagnostic Modular analyzer E170). The bicycle stress test started with a 50 W output for 1 min., thereafter increasing the strain by 10 W each minute, until the participant claimed being exhausted. Continuous and categorical data were compared using independent *t*-test and chi-square test. P-values <0.05 were considered statistically significant (SPSS for Windows, version 17.0). All participants except 2 started with a cTnT level ≤ 3 ng/L. After the stress test 9 of 19 participants (47%) had a rise in circulating hs-cTnT (Fig. 1A and B). A higher frequency of cTnT release was observed in the better trained group, 7 out of 9 (78%), compared to only 2 of 10 (20%) in the less trained group (p-value 0.012). Total physical performance (females) was better in the well trained group 1083 ± 161 (Watt min) versus 823 ± 232 in the other group (p-value 0.02). This was true also in the male group, but they were too few to draw any statistical conclusions. Comparing the two fitness groups (females, mean values) for age (58.6 years), weight (66.1 kg), BMI (24.2 kg/m^2), blood pressure (128/81 mm Hg), resting and maximum heart rate (66 and 160 bpm), glucose (6.0 mmol/L) and cholesterol (6.2 mmol/L) did not give any significant statistical differences. One participant (male) who had a cTnT release during the first sampling hours performed additional blood sampling at 4, 5 and 18 h after the stress test. The hs-cTnT levels increased continuously to a sharp plateau at 4 h; from where it declined slowly without normalizing at 18 h.

Our data clearly indicate that daily life physical strain in healthy individuals might be followed by a rise in hs-cTnT. Obviously this phenomenon is not restricted to extreme sports. This questions the relevance of calculating biological variation of hs-cTnT without considering the influence of modulating factors. Contrary to our pretest expectation the better trained individuals had the greatest cTnT increase after physical stress. We do not believe the elevations found in our study are coming from necrosis or ischemia, if so, training should be hazardous. Recently such elevations were described among young, well trained individuals after stress test [5], where no correlation to measured $O_{2\ peak}$ uptake was found. If necrosis is the mechanism of cTn release, the heart must recruit new cardiomyocytes in order to maintain its contractile capacity. However, it has been shown that such regenerative capacity only is in the order of approximately 1% per year in an adult over 25 years of age, and less than 0.45% per year at the age of 70 years [6]. This reduces the plausibility of brisk regenerating heart cells. We do all have minor levels of cTn in our circulation. Given the claimed cardio specificity, it must indicate daily minor leakages from the heart. Our belief is that the observed leakage of cTnT, more pronounced among the well trained, is coming from the cytosolic compartment of the cells and not from the thin filaments, and that this is a universal phenomenon probably related to myocardial strain due to two observations: 1) kinetics of release: Shave [5] and we (one observation) observed that the peak appeared 4 h after the strain. In cardionecrosis due to ablation the peak values appears at 8 h [7]. 2) The half-life ($T_{1/2}$) of cTn (T,I,C) is reported to approximately 3–5 days [8]. Our hypothesis is that cytosolic cTn's are scavenger products from the thin filaments and that well trained individuals with a larger contractile apparatus also might have a greater cytosolic reservoir. Recently such an increased membrane permeability mechanism has also been proposed [9]. If so, leakage during strain might result in a higher leakage of cTnT in the well trained individuals. This study does have the following limitations: The groups were small and the male participants were few.

In summary, the present study showed significant elevations of cTn T in healthy subjects after a standardized exercise test. This observation may have major impact on clinical practice in the emergency room.

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