Hypertrophic cardiomyopathy (HCM) is an inherited cardiac disorder characterised by abnormal left ventricular hypertrophy, with an early systolic peak (S1), followed by a sudden systolic velocity curve (S2) (fig 1). Paired data were analysed with Wilcoxon signed rank test. Unpaired data were analysed with Mann-Whitney U test.

### RESULTS

All patients showed normal systolic LV size and function (mean (SD) LV end diastolic diameter 48 (7) mm, LV end systolic diameter 22 (10) mm). LV systolic function was normal (mean (SD) LV fractional shortening 46 (10)%, LV ejection fraction 58 (9%) and LV end systolic wall thickness 14 (5) mm). The average LVOT size (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm, fractional shortening 46 (10)%), was associated with significant LVOT obstruction. The MSSD notch was present in 15/26 patients (58%) and occurred in all patients simultaneously. All patients showed normal systolic LV size and function (mean (SD) LV end diastolic diameter 48 (7) mm, LV end systolic diameter 22 (10) mm) and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 48 (7) mm, LV end systolic diameter 22 (6) mm). The average LVOT area by planimetry (0.5 (0.3) mm²) was 25 mm² greater than the normal LVOT area (0.3 (0.2) mm²) in 14 patients (54%) and in 13 patients (46%) in the control group. An MSSD pattern was present in 15/26 patients (58%) and occurred in all patients simultaneously. All patients showed normal systolic LV size and function (mean (SD) LV end diastolic diameter 48 (7) mm, LV end systolic diameter 22 (10) mm) and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 48 (7) mm, LV end systolic diameter 22 (6) mm). The average LVOT area by planimetry (0.5 (0.3) mm²) was 25 mm² greater than the normal LVOT area (0.3 (0.2) mm²) in 14 patients (54%) and in 13 patients (46%) in the control group.

### PATIENTS AND METHODS

Twenty-six HCM patients (15 male, mean (SD) age 48 (17) years) were included in the study. All patients showed normal systolic LV size and function (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm, fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 48 (7) mm, LV end systolic diameter 22 (6) mm). The average LVOT area by planimetry (0.5 (0.3) mm²) was 25 mm² greater than the normal LVOT area (0.3 (0.2) mm²) in 14 patients (54%) and in 13 patients (46%) in the control group. An MSSD pattern was present in 15/26 patients (58%) and occurred in all patients simultaneously. All patients showed normal systolic LV size and function (mean (SD) LV end diastolic diameter 48 (7) mm, LV end systolic diameter 22 (6) mm) and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 48 (7) mm, LV end systolic diameter 22 (6) mm). The average LVOT area by planimetry (0.5 (0.3) mm²) was 25 mm² greater than the normal LVOT area (0.3 (0.2) mm²) in 14 patients (54%) and in 13 patients (46%) in the control group. An MSSD pattern was present in 15/26 patients (58%) and occurred in all patients simultaneously. All patients showed normal systolic LV size and function (mean (SD) LV end diastolic diameter 48 (7) mm, LV end systolic diameter 22 (6) mm) and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 40 (5) mm, LV end systolic diameter 22 (6) mm) fractional shortening 46 (10)% and significant LVOT (mean (SD) LV end diastolic diameter 48 (7) mm, LV end systolic diameter 22 (6) mm). The average LVOT area by planimetry (0.5 (0.3) mm²) was 25 mm² greater than the normal LVOT area (0.3 (0.2) mm²) in 14 patients (54%) and in 13 patients (46%) in the control group.
rest does not exclude the presence of LVOT obstruction during exercise.

In conclusion, the presence of an MSSD pattern in the septal TDI velocity trace, defined and identified by the presence of two systolic velocity peaks and a sudden interpolated deceleration notch, identifies HCM patients with clinically important LVOT obstruction. TDI analysis of septal longitudinal motion patterns may constitute a new diagnostic tool additional to the conventional continuous wave Doppler examination for gradient measurement. It may help to verify the presence of an LVOT gradient, particularly in difficult imaging conditions such as exercise testing and in the presence of mitral regurgitation. This additional information is likely to reduce the number of false negative studies in patients where conventional Doppler methods fail to identify the site of gradient development, and it will identify false positive cases where the continuous wave Doppler beam has been misaligned and records mitral regurgitation instead of the LVOT velocity. Whether the presence or absence of an MSSD notch will improve risk stratification in HCM patients, or whether it may help to validate the response to treatment, remains to be studied prospectively.

Authors’ affiliations
O-A Breithardt, A Franke P Hanrath, Medizinische Klinik I, Universitäts-Klinikum, RWTH, Aachen, Germany

G Beer, B Stolle, F Lieder, T Lawrenz, H Kuhn, Klinik für Kardiologie und Internistische Intensivmedizin, Klinikum Bielefeld-Mitte, Akademisches Lehrkrankenhaus der Westfälischen-Wilhelms Universität Münster, Bielefeld, Germany

Correspondence to: Ole-A BreithardtMD, Medizinische Klinik I, Univ.-Klinikum Aachen, Pauwelsstrasse 30, D-52057 Aachen, Germany; olebreithardt@gmx.de

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