

Left ventricular hypertrophy and duration of systemic sclerosis

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Table 2. Age, disease duration, severity of disease, and parameters of left ventricular (LV) hypertrophy in patients with and without septal or/and posterior LV wall thicknesses > 13 mm

	Wall thickness		P-value
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Disease duration (years)	5.5 ± 5.9	5.9 ± 6.5	NS
Scleroderma score	24.7 ± 8.4	26.9 ± 9.7	NS
Interventricular septum thickness (mm)	10.4 ± 1.3	15.3 ± 2.0	< 0.001
LV posterior wall thickness (mm)	9.7 ± 1.9	11.0 ± 1.9	NS
LV mass index (g m ⁻²)	100.0 ± 28.7	143.7 ± 24.8	< 0.005

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Disease duration (years)	5.5 ± 5.9	5.9 ± 6.5	NS
Scleroderma score	24.7 ± 8.4	26.9 ± 9.7	NS
Interventricular septum thickness (mm)	10.4 ± 1.3	15.3 ± 2.0	< 0.001
LV posterior wall thickness (mm)	9.7 ± 1.9	11.0 ± 1.9	NS
LV mass index (g m ⁻²)	100.0 ± 28.7	143.7 ± 24.8	< 0.005

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Left ventricular hypertrophy and duration of systemic sclerosis

DEAR SIR, We have described in this journal a consecutive series of patients with systemic sclerosis, and claimed that these subjects often show increased left ventricular (LV) wall thickness and abnormal diastolic function [1]. Hegedüs & Czirjak [2] have now studied 71 patients with systemic sclerosis and found similar mean septal thickness as in an age- but not sex-matched, fairly small ($n = 16$) healthy control group. Recently, Marshall *et al.* also found normal wall thicknesses and LV dimensions in 24 patients with systemic sclerosis, despite signs of reduced LV compliance [3], as in our patients [1, 4]. However, Hegedüs & Czirjak [2] found that the septal thickness or posterior wall thickness exceeded 13 mm in 14 (20%) cases. Moreover, there were 22 (31%) patients with a 'wall thickness', probably meaning either septal or posterior wall, of < 8 mm. Thus 20% of patients had increased LV wall thickness, although due to thin walls in some patients the group mean values did not indicate LV hypertrophy. We cannot determine whether the same is true for the study by Marshall *et al.* [3]. However, it seems likely that a heterogeneous differently selected study population contributed to the failure to reveal LV hypertrophy in these two investigations. Furthermore, it is important to determine both the wall-thickness-to-volume ratio, and LV mass and mass index, in order to draw conclusions on the prevalence of LV hypertrophy. Our study, as well as several previous investigations, indicates the presence of LV hypertrophy in systemic sclerosis. Autopsy studies have described either cardiac hypertrophy or LV hypertrophy in systemic sclerosis [5-7] and in an early echocardiographic study that mentioned LV walls, hypertrophy was found in 70% of patients in a small group [8] (Table 1).

The low number of patients with a thin wall in our study contributes to the increased group means indicating LV hypertrophy in our patients [1]. There

Table 1. Left ventricular hypertrophy (LVH) in autopsy or echocardiographic studies

Author	Year	Type of study	Finding
Sackner <i>et al.</i> [5]	1966	Autopsy	LVH in 11/25 patients
D'Angelo <i>et al.</i> [6]	1969	Autopsy	Increased relative heart weight in 58 patients vs. 58 controls
Bulkley <i>et al.</i> [7]	1976	Autopsy	LVH in 22/52 patients
Gottdiener <i>et al.</i> [8]	1979	Echocardiography	Increased LV wall thickness in 7/10 patients
Kazzam <i>et al.</i> [1]	1990	Echocardiography	Increased LV wall thickness and mass index in 30 patients vs. 48 controls
Marshall <i>et al.</i> [4]	1990	Echocardiography	No LVH in 24 patients vs. 24 controls
Hegedüs & Czirják [2]	1990	Echocardiography	LVH in 14/71 patients

were only five (17%) patients with a wall thickness (septum or posterior wall) of < 8 mm, and of these, one patient had a septal thickness of 16.9 mm despite a posterior wall thickness of 7.2 mm. Hegedüs & Czirják [2] advocate a reciprocal relationship between disease duration and wall thickness in patients with systemic sclerosis, which they suggest may be secondary to the fibrotic process. In contrast to the findings of Hegedüs & Czirják, our five patients with thin walls had a disease duration of only 2.7 ± 1.7 years. Moreover, our 10 patients with a wall thickness of > 13 mm had a disease duration similar to that of the remaining 20 patients (Table 2). Like the patients in the study of Hegedüs & Czirják (mean disease duration 9.6 years), the patients in the study of Marshall *et al.* had a longer disease duration (mean 13.8 years) than our patients (5.6 years). It may very well be that our patients with thin LV walls are closest to normal, that our patients and those of Hegedüs & Czirják with hypertrophy represent an intermediate group with myocardial fibrosis, and their patients with thin walls and long disease duration might represent the most severe cases, with fibrosis and myocardial degeneration, i.e. more or less dilated cardiomyopathy. However, the sparse information on their patients provided by Hegedüs & Czirják does not allow any conclusions to be drawn. The matter is intriguing and should stimulate further research, in which myocardial biopsy in patients with various disease durations would be an interesting adjunct to echocardiography.

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Table 2. Age, disease duration, severity of disease, and parameters of left ventricular (LV) hypertrophy in patients with and without septal or/and posterior LV wall thicknesses > 13 mm

	Wall thickness		P-value
	≤ 13 mm (n = 20)	> 13 mm (n = 10)	
Age (years)	53.0 ± 12.4	57.5 ± 15.2	NS
Disease duration (years)	5.5 ± 5.9	5.9 ± 6.5	NS
Scleroderma score	24.7 ± 8.4	26.9 ± 9.7	NS
Interventricular septum thickness (mm)	10.4 ± 1.3	15.3 ± 2.0	< 0.001
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