Quantifying Myocardial Function
by using 2D and 3D
Wall Motion Tracking Analysis

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Introduction
A 64-year-old male patient presented with moderate dyspnea but without angina for one year. For five years, the patient received diuretics due to arterial hypertension. Neither an anti-obstructive lung therapy nor a thyroid replacement therapy to treat hypothyroidism relieved symptoms. In December 2007, the patient presented with atrial fibrillation with a ventricular heart rate of 150/min. The GP prescribed anticoagulation medication, beta-blockers and AT2 receptor antagonists. The AT2 receptor antagonist had to be discontinued because of an angioedema. The diuretic therapy contributed to a weight loss of 10 kg and the heart rate was reduced to 91/min. Although the condition of the patient had significantly improved he was referred for a cardiac evaluation of a heart failure of unknown origin.

Clinically we find an obese patient weighing 102 kg, with a height of 182 cm, blood pressure of 120/72 mmHg and absolute arrhythmia. The general state is somewhat reduced. Splenomegaly is suspected and can be confirmed sonographically.

Findings
The ECG shows atrial fibrillation with a ventricular rate of 96/min with high amplitudes in the Wilson leads which suggests left ventricular hypertrophy (fig. 1).

An echocardiography was performed and the M-mode is presented in fig 2. Note the bright myocardium with asymmetrical hypertrophy and depressed ventricular function with akinetic septum. Figure 3 shows a diastolic frame in the 4-chamber view with the result of measurement.
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of LV function (modified Simpson). The ejection fraction is reduced to 40% and there seems to be some LV-hypertrophy. However, in the mid-ventricular short axis view there is normal LV-wall thickness (fig 8).

Doppler sonography shows restrictive filling (fig. 4) and pulmonary arterial hypertension (fig. 5).

Speckle tracking helps to quantify myocardial function and is helpful to quickly illustrate anatomy in clinical practice. Fig. 6 shows automatic measurement of systolic ejection fraction as well as regional quantification of myocardial function with longitudinal strain. Even though global longitudinal strain is reduced to 11%, transversal function is obtained with a transversal global strain of 23% at this early stage (fig. 7). Fig. 8 and 9 depict transversal 2D and 3D slices (midpapillary plane). Three-dimensional function is shown in fig. 10 with a global systolic ejection fraction of 41% with radial 3D strain and myocardial volume.

Amyloidosis is preliminarily diagnosed and verified by further work up which showed the lambda light chains to be highly elevated. A plasmocytoma was found with cardiac amyloidosis (AL type) and diffuse splenomegaly. A medical therapy was started.

Conclusions
Amyloidosis refers to abnormal protein deposits in tissue either as a primary cause or as the result of a chronic illness. Symptoms depend on the functioning of organs involved. Electrocardiography shows low amplitudes in late stages, but arterial hypertension can unmask this effect. Echocardiography depicts a bright speckled myocardial wall with myocardial hypertrophy and reduced systolic and diastolic function are suggestive of cardiac amyloidosis. Epicardial effusion is present in progressed state. Speckle tracking is a new feature of two-dimensional echocardiography to rapidly quantify global and regional myocardial function without angle dependency in clinical practice. The disease is confirmed histologically. Amyloidosis can be treated by correcting the underlying disease and the organ damage. Medical therapy depends on the type of amyloidosis, but in general prognosis is poor.