Measurement of Haemodynamic Parameters during Dobutamine Echocardiography by Automated Impedance Cardiography

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Background: Dobutamine stress echocardiography (DSE) has become a widely used method for the evaluation of patients (PTS) with suspected coronary artery disease (CAD). However, little is known about the haemodynamic effects of such short-term, high-dose dobutamine infusions. Methods: We investigated 50 PTS who were referred for DSE. Heart rate (HR), stroke volume (SV), cardiac output (CO) and index of contractility (IC) measured by automated impedance cardiography. Baseline values were compared to the corresponding values at peak stress with dobutamine. All PTS underwent coronary angiography within the next 1-7 days. For interpretation of the data, PTS were subdivided into 3 groups: PTS without CAD (A), PTS with single vessel disease (B) and PTS with multivessel disease (C). Results: At baseline, there was no significant difference between the three groups. The infusion of dobutamine resulted in a significant increase of HR, SV, CO and IC in all PTS. At peak stress, there was no significant difference between PTS of group A and B, but PTS of group C showed a significantly smaller increase of SV, CO and IC (p < 0.05). Conclusions: Automated impedance cardiography allows continuous haemodynamic monitoring during DSE. In PTS with multivessel disease, the impaired systolic performance of the left ventricle during pharmacological stress could be clearly demonstrated.

INTRODUCTION:
Physical exercise by bicycle- or treadmill stress testing are the classical methods for functional evaluation of the cardiovascular system. During the recent years, stress echocardiography has emerged as a new and exciting diagnostic method for the evaluation of patients with suspected coronary artery disease (CAD). Stress-induced left ventricular wall-motion abnormalities have been shown to be a more sensitive marker of functional relevant myocardial ischaemia than stress-induced ECG abnormalities [1,2]. In patients who cannot adequately exercise, pharmacologic stress echocardiography with dobutamine is an accepted and useful method to induce cardiovascular stress [3,4].

However, little is known about the haemodynamic effects of dobutamine stress echocardiography (DSE) on stroke volume (SV) and cardiac output (CO) in patients with suspected CAD, although it is likely that stress-induced myocardial ischaemia should significantly impair systolic pump function. Because so far, stress echocardiography only concentrated on evaluating the presence, absence or worsening of stress induced wall-motion abnormalities we wanted to add complementary haemodynamic information to echocardiographic stress testing.

Issues of our study were therefore (a) to investigate the haemodynamic responses of patients with or without CAD to the standard protocol for DSE and (b) to investigate potential differences in these haemodynamic responses in different patient groups. Because of its feasibility for non-invasive monitoring of the cardiovascular system, we decided to use automated impedance cardiography for the haemodynamical measurements.

PATIENTS AND METHODS
We investigated 50 patients (37 m, 13 f, mean age 60.8 ± 8.5 years) by the standard protocol for DSE-testing. All patients were referred for coronary angiography to our institution and gave informed consent for participation in the study.

Dobutamine was administrated intravenously by an infusion pump at increasing doses from 5 up to 40 μg/kg/min at 3 min intervals. Criteria for the termination of the DSE-test were the detection of stress-induced wall-motion abnormalities, severe angina pectoris, significant arrhythmias, hypertension (blood pressure ≥ 220 mmHg) or achievement of the age-predicted target heart rate (85 % of 220 minus age). Echocardiographic images were digitally stored at each stage of the dobutamine infusion. In the event of adverse effects, significant ST shifts or severe angina, the dobutamine infusion was stopped by the injection of a short-acting betablocker. During both protocols, blood pressure and the ECG were recorded every three minutes. A DSE-test was defined as pathologic, if dobutamine-induced new left ventricular wall-motion abnormalities occurred during the test or a worsening of pre-existing wall-motion abnormalities could be observed compared to baseline recordings at rest.

Haemodynamic monitoring was performed by an automated impedance cardiography (AIC) system (cardioscreen professional, Medis GmbH, Ilmenau, Germany). The AIC system consisted of a standard PC with the data processing software and a transmitting unit. The four pair electrode method was used for analysis of the thoracic impedance field. Correct identification of the points B (opening of the aortic valve), P (maximum systolic flow) and X (closure of the aortic valve) on the impedance cardiography curve were confirmed by marker channels. The modified Bernstein formula was used for calculation of SV, the index of contractility was calculated as follows: IC = (dz/dt)max/Z0. Respiratory and other artefacts of the impedance signal or the ECG were automatically eliminated by the AIC system from further analysis. For calculation of the hemodynamic parameters, the software was programmed to average HR, SV, CO and IC automatically every 5 consecutive beats.
Coronary angiography was performed in all patients within 1-7 days after the DSE test. Depending on the results of the cardiac catheterization (significant CAD was defined as ≥ 50 % lumen narrowing in at least one coronary vessel), patients were subdivided into three groups: patients without relevant CAD (group A), patients with single vessel disease (group B) and patients with multivessel disease (group C).

The Mann-Whitney rank sum test (a-test) was used for statistical analysis of independent variables. The Wilcoxon test was used for comparison within the groups. A p value of ≤ 0.05 was regarded as significant. All values are expressed as mean values +/- standard deviation (SD).

RESULTS

The results of measurements at baseline and peak stress with dobutamine were compared between the three groups and are given in table 1. Three patients had to be excluded because of inappropriate quality of the thoracic impedance signal. The mean dose of dobutamine until termination of the test was 31.8 ± 4.5 μg/kg/min. The sensitivity of the DSE-test for the detection of patients with functional relevant CAD was 79 % (27/34), the specificity 88 % (14/16), respectively.

Baseline values of HR, SV, CO and IC did not significantly differ between patients with or without CAD. The infusion of dobutamine resulted in a significant increase of these parameters in all three groups, but there was no significant difference in peak values of SV, CO and IC between patients without CAD (group A) and patients with single vessel disease (group B). However, significant differences in SV, CO and IC were found between the patients of group B if they were compared to the patients of group C: patients with multivessel disease (group C) showed a significantly smaller increase of all three haemodynamic parameters than patients with single vessel disease (p ≤ 0.05).

DISCUSSION

Although DSE has become a well-established and broadly accepted diagnostic method for the evaluation of patients with suspected or proven CAD, little is known about the haemodynamic effects of the short-term, high-dose dobutamine infusions used for pharmacologic stress testing. Besides an invasive, study of Pierard and co-workers [5] no studies have been reported in which the haemodynamical alterations of SV and CO during DSE have been investigated. The results of the present study support Pierard's findings and preliminary results of our group in a smaller population [6,7]. In contrast to our previously reported findings were haemodynamic data only correlated with echocardiographic results, all patients of the present study underwent angiography so that a direct comparison of the haemodynamic results to the extent and severity of CAD was possible. Interestingly, no significant differences between patients with single vessel disease and patients without significant CAD could be found. The pathophysiological explanation for these findings is, that SV in patients with single vessel disease can obviously be maintained for a relatively long time by hypercontractility of the non-ischaemic myocardial areas or collateralisation, before haemodynamically relevant depression of left ventricular function occurs.

The results of our study show that AIC can be used for haemodynamic measurements during DSE and can provide clinically useful information concerning left ventricular functional performance during cardiovascular stress.

REFERENCES