The influence of fluid and diuretic administration on the index of atrial contribution in sequentially paced patients

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Aims To examine whether acute changes in patient hydration can change atrial contribution (AC) to circulatory function. Methods and results Atrial contribution was quantified by beat-to-beat changes in the amplitude of pulse oximetry signal in 24 paced outpatients. Changes in body weight were used for assessment of changes in total body water. The first measurement was performed at steady state. The second measurement was made after infusion of saline (5 mL/kg) and the third measurement was obtained 2 h after a bolus of furosemide (1 mg/kg). Changes found after furosemide administration (compared with steady state): a substantial decrease in body weight from median 78.6 (interquartile range 65.7–86.5) to 77.1 (64.4 –85.6) kg (P < 0.001), accompanied by an increase in AC from 30.4 (20.2–47.1) up to 43.3 (30.6 –80.9)% (P < 0.001). An increase in heart rate and shortening of the atrio-ventricular conduction time occurred during acute hypohydration in some of the subjects. Conclusion Administration of furosemide was followed by a decrease in body weight and an increase in AC to stroke volume. This suggests that in conditions where pre-load is reduced cardiac output is preserved by an increase in AC enforced by sympathetic activation.

KEYWORDS
Atrial contribution; Acute effect of diuretic; Pulse oximetry; Sequential cardiac pacing; Sympathetic activation

Introduction

The left atrium acts as a reservoir, which converts the continuous inflow from the pulmonary veins into the pulsatile aortic blood flow produced by the ventricle. The atrium with its thin muscular wall contains about three times as much blood as the ventricle at the end of diastole. When properly timed, its contraction helps to optimize the performance of the ventricle, i.e. ‘atrial contribution’ (AC).1–4

Previous work has focused primarily on the importance of AC in pathological conditions. It has been shown that forceful contraction of the hypertrophied atrium is essential for maintaining appropriate stroke volume when the ventricular function is impaired by a previous myocardial infarction5–10 or hypertrophic cardiomyopathy (HCM).11 However, very little is known about atrial function under conditions when diastolic ventricular function is normal or mildly abnormal. Researchers in the past analysed the short-lasting effects of hydrostatic pressure changes11,12 and effects of intravenous fluids13,14 on atrial function. However, studies analysing the impact of mild dehydration (a rather frequent clinical situation) on AC have not been performed.

The aim of the study was to measure the AC index derived from the pulse oximetry signal in patients 2 h after administration of a standardized dose of intravenous furosemide. All the patients included in the study were paced for different clinical reasons by an implanted atrioventricular (AV) sequential pacemaker.

Methods

Patients

Twenty-four outpatients were enrolled in the study. They all gave informed consent to the study, which was approved by the local Ethics Committee. The mean age was 60 ± 12 years (range 20–75 years). Ejection fraction (EF) of the left ventricle varied widely from 24 to 77% (mean 53 ± 12%). A detailed description of subjects including indications for cardiac pacing and long-term medication is given in Table 1.

We performed echocardiography in all patients. In 18 patients, echocardiographic examination in 2D mode was performed 1 week...
before the measurement. In six patients, 2D-mode echocardiographic examination was performed within 3 months of the measurement. Additionally in these six patients, we estimated EF using nuclear ventriculography 5 days before the actual measurement. In all patients, echocardiography excluded moderate or severe congenital or acquired valvular disease, shunts in the heart or the great vessels. All showed no evidence of atrial dysfunction. All patients were paced for different types of symptomatic bradycardia (Table 1). Two were additionally treated for diabetes mellitus, five for coronary heart disease, and eight for arterial hypertension. During the study, long-term medications (Table 1) were not changed and the activity sensor of the pulse generator was switched off.

Methods

A measure of the AC index was made using the arterial waveform trace obtained from a pulse oximetry signal.15,16 In short, after quiet expiration, the pacing mode was changed from DDI (sequential) to VVI (ventricular) pacing mode for a few beats. This change temporarily stopped atrial contractions. In the first beat after loss of atrial contraction, the left ventricular filling, its stroke volume, pressure-pulse amplitude, and the pulse oximetry signal amplitude decreased. The AC index (Figure 1) was then calculated from an equation: 
\[
\frac{\text{Amplitude of Beat0}}{\text{Amplitude of Beat1}} \times 100
\]
A constant ventricular rate was maintained throughout measurement. The index of AC estimated from pulse oximetry signal has previously been validated against the index of AC assessed from the aortic pulse pressure.16 The beat-to-beat changes in the amplitude of the pulse oximetry signal correlate closely with the simultaneous pulse-pressure amplitude changes measured in the ascending aorta with a catheter-tip manometer.15,17 We have previously demonstrated using our unpublished paired data that this method for measuring AC index is highly reproducible when analysed by the method of Bland and Altman.18 The mean difference of paired data (n = 153) obtained at AV interval of 150 ms was only 0.3 (standard deviation 5.2)%.

In the present study, the AC index was estimated at the end of three periods: (i) steady-state period (refer to index steady), (ii) post-infusion state (refer to index infusion), and (iii) post-diuretic state (refer to index diuretic). The post-infusion state was achieved by intravenous infusion of saline at 5 mL/kg of body weight over 30 min. The dose of 5 mL/kg of body weight was chosen to minimize the risk of inducing acute pulmonary oedema in patients with systolic dysfunction of the left ventricle (Table 1). Another reason was because the two heaviest patients weighed 100 and 102 kg. The volume of infusion was 500 mL. This required at least 25 min to be delivered into a peripheral vein. At the end of each period, the patients passed urine, and the body weight (W), systolic blood pressure (BP), diastolic blood pressure (BPd) were measured concurrently with the AC index. After the second series of measurements (end of post-infusion state), furosemide (1 mg/kg body

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Pacing</th>
<th>EF (%)</th>
<th>AH</th>
<th>L VH</th>
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</table>

M, male; F, female; SSS, sick sinus syndrome; AV II, second-degree AV block; AV III, complete AV block; HCS, carotid sinus syndrome; AH, presence of arterial hypertension in personal history. Long-term medication is indicated by: Diur, diuretic; BB, beta-blocker; CaB, calcium channel blocker; ACE-I, angiotensin-converting enzyme-inhibitor.
weight) was administered as an intravenous bolus. A loop diuretic was chosen for its natriuretic and negligible direct arteriolar dilating effect. Effects of loop diuretics are similar to various situations in real life where loss of sodium and water occurs, e.g. sweating.

The AC index was estimated at AV intervals of 125, 150, 175, 200, 225, and 250 ms in all patients with intrinsically AV conduction longer than 250 ms. In all patients, the AC index was measured for AV intervals from 125 to 200 ms. For further evaluation, optimal AV delay was used, i.e. the delay for which the AC index was highest at the end of steady state.

Ejection fraction of <50% was considered as systolic dysfunction of the left ventricle. Left ventricular hypertrophy (LVH) was considered to be present if the interventricular septum or the posterior wall of the left ventricle exceeded 11 mm.

A change of at least 30% was chosen as a criterion of significant change in AC after diuretic administration. This value correlates with studies dealing with similar issues of acute changes in the wall of the left ventricle.


Statistical analyses

Because the distribution of the source data was not normal in the whole group of 24 patients, we used medians and interquartile ranges (first quartile–third quartile) as well as non-parametric statistical tests for the description of data. The changes in variables in the first group of 16 patients were evaluated using Wilcoxon’s test with Bonferroni’s correction for multiple comparisons to avoid false results. The changes in variables in the whole group of 24 subjects were validated by Wilcoxon’s test.

Because of the heterogeneity of the observed group (including patients with LVH, low EF, and combinations of these; Table 1), further testing had to be performed in order to assess whether variations in results were due specifically to these disorders. Using Fisher’s exact test, the AC changes in all participants were examined with respect to present combinations of depressed function and/or hypertrophy of the left ventricle.

Fluid infusion was omitted in the last eight patients. Statistical analysis by Mann-Whitney U test was performed to find any impact of fluid infusion on changes in AC induced by administration of diuretic in groups with and without infusion.

Results

The changes in body weight, BPs, BPd, and AC for the first 16 patients are shown in Table 2. When comparing findings at the end of the post-infusion state with the steady state, there were significant increases in body weight and BPs (for both \( P < 0.010 \)). The changes in BPd were small (\( P = 0.080 \)). There were only negligible changes in AC (\( P = 0.897 \)). As the infusion of fluid did not lead to any significant changes in AC in the first 16 subjects, fluid administration was omitted in the last eight patients. These eight patients received diuretic immediately after the measurements of \( W_{\text{steady}}, \text{BPS}_{\text{steady}}, \text{BPd}_{\text{steady}}, \text{and AC}_{\text{steady}} \).

After the diuretic, body weight (\( W_{\text{diuretic}} \)) was significantly lower when compared with \( W_{\text{steady}} \) and \( W_{\text{infusion}} \) (for both \( P < 0.001 \)). The \( \text{BPS}_{\text{diuretic}} \) was decreased when compared with \( \text{BPS}_{\text{infusion}} \) (\( P < 0.005 \)), but there were no significant changes when compared with \( \text{BPS}_{\text{steady}} \) (\( P = 0.186 \)). The \( \text{BPd}_{\text{diuretic}} \) showed an insignificant fall when compared with \( \text{BPd}_{\text{steady}} \) (\( P = 0.085 \)) and \( \text{BPd}_{\text{diuretic}} \) (\( P = 0.021 \)). These changes were accompanied by a rise in AC. The median of \( AC_{\text{diuretic}} \) was higher than \( AC_{\text{steady}} \) and \( AC_{\text{infusion}} \) (for both \( P < 0.001 \); e.g., Figure 2).

Medians (interquartile ranges) of measured parameters in all 24 patients are given in Table 3. Compared with the steady state, administration of the diuretic was followed by a decrease in \( W \) (\( P < 0.001 \)) and in BPd (\( P = 0.041 \)). The AC increased significantly (\( P < 0.001 \), Figure 3). There was an insignificant fall in the BPs (\( P = 0.134 \)).

Possible relationships of AC reactions to administration of diuretic with the combination of EF and LVH were explored by Fisher’s exact test. The null hypothesis of no relation was not rejected (\( P = 0.395 \)).

Differences in body weight, BPs, BPd, and index of AC between post-diuretic state and steady state are shown in Table 4. As \( P \)-values indicate, there were no deviations caused by infusion of fluid.

Discussion

The results show that administration of a diuretic decreases body weight. The fall in weight is accompanied by an increase in the index of AC (and most likely also the AC itself) and a less apparent decrease in blood pressure. The explanation seems to be an increase in the active participation of the atria (more forceful contraction) in the physiological response to the short-duration dehydration and, presumably, to a consequent fall in the cardiac output. In six of our patients, the magnitude of AC reached values of >80% (i.e. after administration of furosemide, >80% of the blood that filled the ventricle was expelled into the ventricle by atrial contraction). Similar values have been reported by Ruskin et al., who described an increase in the systolic volume due to a properly timed atrial contraction up to 145% of the systolic volume of beats in which the AC was lost.

Increase in AC index was likely to be caused by increased contractility of the atrial myocardium, mediated by sympathetic activity. Previous work by Sigwart et al. supports the suggestion that sympathetic activity plays an

\[
\text{Table 2 Evaluation of the first 16 subjects}
\]

<table>
<thead>
<tr>
<th>( n = 16 )</th>
<th>( W ) (kg)</th>
<th>( \text{BPS} ) (mmHg)</th>
<th>( \text{BPd} ) (mmHg)</th>
<th>AC (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Steady state</td>
<td>79 (73.3–86.5)</td>
<td>135 (125–148)</td>
<td>80 (70–89)</td>
<td>27 (19.8–42.8)</td>
</tr>
<tr>
<td>Post-infusion</td>
<td>79.4 (73.3–86.9)</td>
<td>140 (130–160)</td>
<td>80 (70–90)</td>
<td>25.9 (20.2–42.4)</td>
</tr>
<tr>
<td>Post-diuretic</td>
<td>77.8 (72.4–85.6)</td>
<td>130 (120–139)</td>
<td>70 (66–80)</td>
<td>38.1 (26.9–62.4)</td>
</tr>
</tbody>
</table>

Medians and interquartile ranges of body weight (\( W \)), BPs, BPd, and AC index obtained in the steady state, after administration of fluid and after injection of furosemide in the first 16 subjects.

\( ^*P < 0.010 \), post-infusion state compared with steady state.

\( ^*^*P < 0.001 \), post-diuretic state compared with steady state.

\( ^*^*^*P < 0.001 \), post-diuretic state compared with post-infusion state.

\( ^*^*^*^*P < 0.005 \), post-diuretic state compared with post-infusion state.
important role in regulation of atrial contractile function. Myocardial ischaemia and a blood pressure fall induced by a short-lasting inflation of a balloon in the left anterior descending artery was followed by a highly significant increase in the AC to the ventricular filling within 30 s. Most of our patients with high degree AV block had no spontaneous AV conduction. As a result, it was impossible to measure PV interval as a criterion of intrinsic sympathetic activity. During the course of the study, no index of sympathetic activity was measured.

Patient 14 suffered from neurally mediated syncope with vasodepressive and cardioinhibitory components. The patient was on long-term treatment with fludrocortisone and midodrine to prevent further pre-syncopal episodes. Despite a decrease in body weight of 1 kg, the AC decreased from 21.1% in steady state to 17.7% in post-diuretic state. This is compatible with the hypothesis that sympathetic activity plays an important role in the force of atrial contraction, as there was parasympathetic dominance at the time of the measurement.

We have demonstrated that AC seems to increase during dehydration (due to diuretics or perhaps due to a dehydration of any type) to maintain cardiac output regardless of underlying systolic function. This implies that the physiological response to decreased pre-load appears to be an increase in AC to ventricular performance. This is consistent with previous work, in patients with impaired ventricular compliance (HCM, fibrotic changes of the ventricular wall due to an old myocardial infarction, etc.), where cardiac output is maintained by a more forceful atrial contraction.11,14,22–25

The increase in AC during dehydration was noted in all our patients with two exceptions (Patients 14 and 24) regardless of presence of systolic dysfunction or hypertrophy of the left ventricle. In other words, it might be assumed that the atria help to keep the cardiac output within normal limits when ventricular filling is hampered due to decreased inflow of blood and pathology of the ventricular wall.

Other studies have used Doppler echocardiography to assess the effect of changes in pre-load induced either by infusion of nitroglycerine or by application of tourniquets. It was shown that changes in pre-load induced by infusion of nitroglycerine26,27 or application of tourniquets on all four extremities28 was followed by a significant decrease in

### Table 3  Evaluation of all 24 subjects

<table>
<thead>
<tr>
<th></th>
<th>n = 24</th>
<th>W (kg)</th>
<th>BPs (mmHg)</th>
<th>BPd (mmHg)</th>
<th>AC (%)</th>
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<tr>
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<td>130(116–140)</td>
<td>80(70–89)</td>
<td>30.4 (20.2–47.1)</td>
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<tr>
<td>Post-diuretic</td>
<td>77.1</td>
<td>120(120–134)</td>
<td>73(66–80)</td>
<td>43.3 (30.6–80.9)</td>
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<tr>
<td>Wilcoxon’s test</td>
<td>P &lt; 0.001</td>
<td>P = 0.134</td>
<td>P = 0.041</td>
<td>P &lt; 0.001</td>
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</table>

Medians and interquartile ranges are tabulated. Results of Wilcoxon’s test for body weight (W), BPs, BPd, and AC index in steady state and after administration of diuretic are in the last row.

**Figure 2** Example of an extreme decrease in pulse amplitude of the pulse oximetry signal (black arrow) in Patient 4, induced by changing the pacing mode from DDI to VVI. Grey arrow marks the loss of atrial activation on ECG.
amplitude of the E-wave and shortening of the deceleration time, whereas the A-wave remained almost unchanged. The ratio of the velocity-time integral of the A-wave and of the velocity-time integral of the whole diastolic inflow increased. Thus, echocardiographic studies also imply that the pre-load reduction may enhance the AC to ventricular filling. Notwithstanding the inaccuracies of the Doppler method, nitroglycerine induces a purely pharmacological effect. On the contrary, loop diuretics cause loss of sodium and water without remarkable dilation of arteries, a condition that mimics real life.

Dernellis et al. compared patients with HCM with and without outflow obstruction of the left ventricle with a control group. The active contribution of the left atrium to the load of the left ventricle, estimated from the Doppler signal obtained from the mitral ostium, was higher in patients with or without obstruction than in controls. The AC to filling was enhanced on rising from a supine to upright position.

No statistically significant changes in AC were found after intravenous administration of fluids. This may be due to the small volume of fluid load given. None of our patients exhibited signs of left ventricular failure during the study, indicating that their atria probably operated on the flat portion of the pressure-volume curve. In this case, a much larger volume load would have been necessary to increase pressure inside the atria substantially to produce some decrease in AC. Our results also showed that exclusion of the infusion from the study protocol did not affect the response to a diuretic.

The study has several limitations. Only an indirect index of the AC to the systolic ventricular output was measured. In contrast, the data obtained were entirely observer independent. For ethical reasons, we could not measure parameters that could quantify the mean pressure in the left atrium (pulmonary capillary wedge pressure) and the cardiac output invasively. However, changes in blood pressure and cardiac output induced by diuretics have been well described in the past. This study showed that administering furosemide at a dose of 1 mg/kg leads to an increase in AC. It would be interesting to establish whether a standard dose of furosemide (40 mg) has a similar effect.

The schedule of the study and the study population with chronotropic incompetence and with conduction disturbances did not allow us to measure indices of intrinsic sympathomimetic activity. Further studies are required in order, more precisely, to uncover the role of the sympathetic system on AC. A study design including the administration of beta-blockers might distinguish between variations in AC due to pressure changes within the heart chambers and due to increased contractility of the atrial myocardium.

We did not use Doppler echocardiography for assessment of the left ventricular diastolic inflow and left atrial size measurements.

Conclusion

A short duration infusion of saline increased the body weight and BPs, but did not influence the AC index; whereas furosemide administration caused a marked reduction in body weight, a slight decrease in arterial pressure, and a substantial increase in the AC index.

We believe that sympathetic activation enhances atrial contraction, which may maintain cardiac output near normal limits in conditions where the ventricular filling is reduced.

Acknowledgements

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