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SHORT COMMUNICATION

Atrial Contribution to Ventricular Ejection in Sequentially Paced Patients

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Summary

A new method for quantitative assessment of the atrial contribution to ventricular ejection in sequentially paced patients is described. The atrial contribution (AC) has been defined as the pulse pressure decrement (invasive arterial measurement by a canulla inserted into the brachial artery), expressed in percent of the control pulse pressure, induced by switching off the atrium activating impulse for one beat. In 17 patients, the AC was found to be atrioventricular (AV) interval dependent, the measurements were well reproducible (the mean difference between two measurements at different times was 9.3 %, S.D. 8.4). For the AV interval of 170 ms, it was found to be 29.3 % (+8.9) in patients with the sick sinus syndrome, 27.0 % (+3.2) in patients with complete AV block and only 10.8 % (+2.1) a in patient with complete AV block and heart dysfunction.

Key words

Atrial contribution – Pulse pressure – DDD pacing – AV optimization – AV interval

The physiological role of atrial contraction in circulation was studied in detail both experimentally (Mitchel *et al.* 1965, Skinner *et al.* 1963, Snyder *et al.* 1966) and clinically (Carleton *et al.* 1966a,b, Samet *et al.* 1968) in the sixties. It was already then that the importance of proper timing of the atrial contraction was emphasized (Carleton *et al.* 1966a,b, Mitchel *et al.* 1965). Later, this was confirmed by numerous clinical studies (Brecker *et al.* 1992, Iwase *et al.* 1986, Nishimura *et al.* 1995, Videen *et al.* 1986). In sequentially paced patients the atrioventricular (AV) interval has to be set. However, it is not easy to estimate its optimal value (Daubert *et al.* 1993). The optimal AV interval is important for coordination of the atria and ventricles. It is of particular importance in patients with left ventricular dysfunction, in whom the ventricular function might be even more dependent on the optimal atrial contraction (Rahimtoola *et al.* 1975, Samet *et al.* 1968, Videen *et al.* 1986). We describe here a method, by which the atrial contribution to ventricular filling can be estimated from the changes in the invasively measured arterial pulse pressure.

Seventeen patients, who were on the paced atrial and ventricular rhythm of 70/min on long-term medication, were included in this study. Seven were females and 10 males 67 years old on the average (min. 53, max. 79). Ten of them had a complete AV block, the rest were patients with a sick sinus syndrome. In all, clinical examination, ECG at rest, chest X-ray and standard nuclear ventriculography (gamma camera Helix, Elscint) were performed. Patients were divided into 3 groups: group 1 included 6 patients with a complete AV block but without signs of heart dysfunction. Heart dysfunction was defined as the left ventricular ejection fraction (EF) of 35 % or less (nuclear ventriculography) and the cardiothoracic ratio

(CTR) 0.52 or more. Group 2 included 4 patients with a complete AV block and signs of heart dysfunction. Group 3 included 7 patients with a sick sinus syndrome. None of the latter complained of symptoms of organic heart disease (apart from symptoms related to the sick sinus), all had EF more than 0.45 and CTR less than 0.48. In all patients, an arterial canulla was inserted into the brachial artery. The brachial artery pressure was measured by Siemens pressure transducer 746 and recorded by a Mingograf 803 recorder.

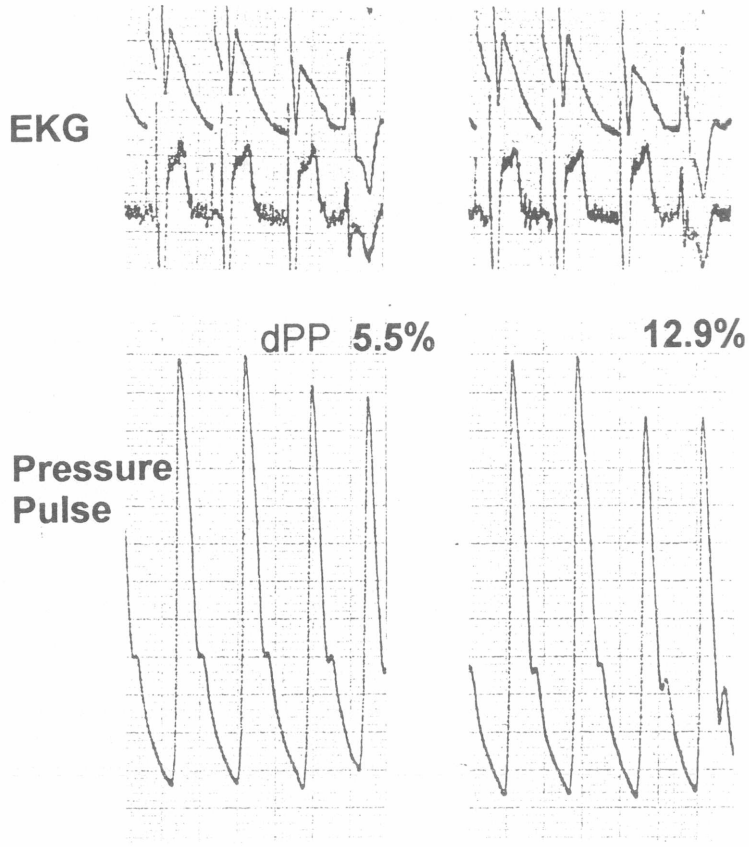

Fig. 1
Example of the pressure pulse record in one of the AVB III patients. In the first column, the AV interval of the sequential pacing was 120 ms, in the second 170 ms. Notice the missing atrial impulse and atrial activation in the ECG of the third beat. Decrement in the pulse pressure (dPP) is expressed in percent of the mean value of the two preceding beats.

The AV interval changes were induced at the absolutely constant ventricular rate of 70/min. Thus, any changes in PP, related to the rate-dependent changes in ventricular contractility, could be excluded. All the findings are related to changes in the sequence of mechanical contractions of atria and ventricles or to changes in contractility of the atrial myocardium. The patients were examined in the resting supine position, in apnoea following quiet expiration. All the induced changes in PP were expressed in % of the control pulse pressure (CPP). As the CPP, the mean of the two beats preceding the induced change was taken. Only beats the PP of which did not differ by more than 2 % from each other and the diastolic pressure of which did not shift for more than 1 % of the actual PP value were accepted for calculation. Only one beat (the one following the change in the AV interval) was used for calculations of the induced changes. Three runs of examinations were undertaken. At the end of each run, the pacing mode at which the effect of AV prolongation and shortening was studied, the pacer was switched to the VVI mode. As the intrinsic sinus activity was always suppressed (all patients had the intrinsic atrial rate lower than 70), on switching to the VVI mode, the CPP was followed by a beat which was not preceded by an atrial contraction. In other words, the beat in which the atrium contributed to the ventricular filling by a contraction at the defined AV interval was followed by a beat in which any atrial contribution was missing. In this way, it was possible to measure the atrial contribution (AC) for each tested AV interval from the changes in the arterial PP. It was possible to compare beats of different AV intervals (for instance by switching the interval from 120 ms to 170 ms) and to assess the atrial contribution at each of the AV intervals (by switching off the atrial impulse) (Fig. 1). The reproducibility of individual measurements in individual patients is demonstrated in Fig. 2. In all patients in whom at least two measurements of the AC at any interval examined were obtained, the individual measured values are plotted against their mean value. Altogether, 79 values of AC, calculated from at least 2 measurements at different AV intervals were obtained. The mean AC was 21.8 %

of the respective PCP, S.D. 9.35, min. 4.4, max. 51.7 %. If the mean of the AC values obtained from at least two measurements was taken as 100 %, and the differences between all the measurements for that particular AV interval in this patient were expressed in %, then the mean difference between measurements in all patients at all intervals (N=187) was 9.3 %, S.D. 8.4.

The measured values of atrial contributions in patients with complete AV block and with and without signs of heart dysfunction, and in patients with the sick sinus syndrome with otherwise "healthy hearts", are summarized in Fig. 3. As the number of patients, particularly the patients with heart dysfunction, was small, no attempt has been made to analyze the problem of the importance of AC in different types of heart dysfunction or even at different AV intervals. The apparently lower atrial contribution to the ventricular ejection in patients with the heart dysfunction was somewhat surprising. Some authors who compared the left atrial contribution to the ventricular filling in patients with ventricular dysfunction found greater atrial contribution to the ventricular filling in patients with a lower ejection fraction (Bristow *et al.* 1970, Videen *et al.* 1986). Clearly, this problem deserves further study with special methodological considerations.

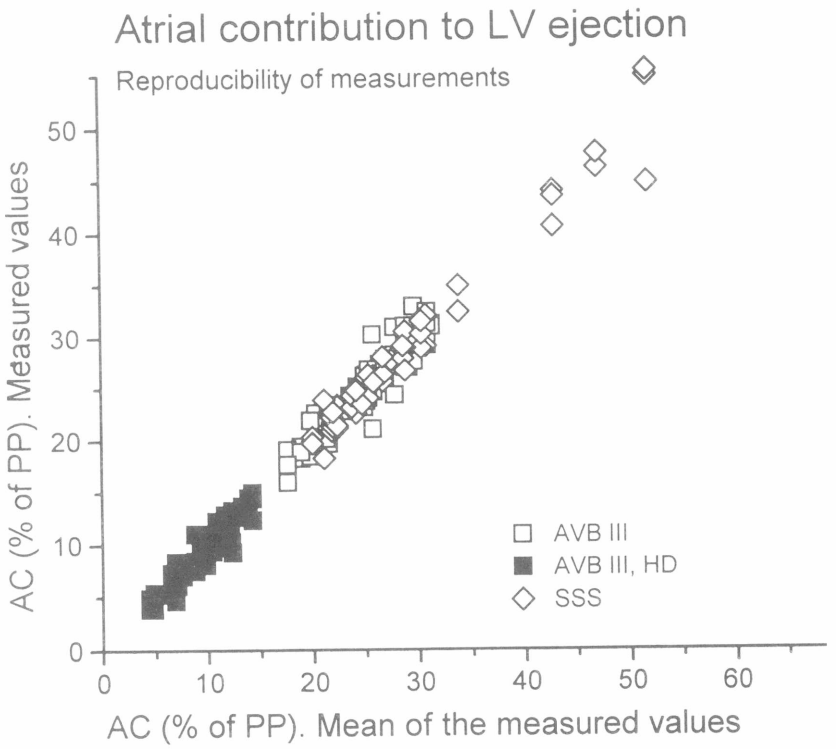

Fig. 2
Graphic illustration of reproducibility of the method. Individual measurements of the AC values are plotted against their means. Only values, where at least two valid measurements were obtained for the tested AV interval in the examined patients were used.

The demonstrated method makes it possible to find the optimal AV delay from invasively measured PP, and to quantify it. Its advantage is the clear definition of the atrial contribution: the testing shows how much of the pulse pressure (and presumably stroke volume) is lost when the atrium does not contribute to the ventricular filling. The arterial pulse pressure curve was used in the past even for calculating the stroke volume. What makes it particularly attractive in the sequentially paced patients is its precision in calculating the intraindividual beat-to-beat changes in stroke volume (Snyder *et al.* 1966, Warner and Rutishauser 1965, Warner *et al.* 1953). We evaluated only one beat following the intervention. After the sequential mode of pacing is switched to the VVI mode, only the first beat is not influenced by the atrial contraction: the atria contract later, at a time when the AV valve is already closed. After the first beat, the atrioventricular dissociation or even retrograde VA conduction makes the following beats unsuitable for further evaluation. The very first beat is also perhaps the only one which cannot be influenced by the atrial reflexes supposedly playing a role in the so-called pacemaker syndrome. Mitchell *et al.* (1965) employed a similar technique for quantifying the atrial contribution in an experimental setting in the dog. They produced complete heart block, excised the sino-atrial node and measured the aortic blood flow by an electromagnetic flow probe. The animal was kept on sequential pacing and an atrial asystole was produced by switching off the atrial pacing. They found the atrial contribution to be 20 %, S.D. 5 %.

Admittedly, changes in the brachial artery PP induced by AV changes may not provide the same information as the pulse pressure changes measured in the ascending aorta. On the other hand, the most relevant information that we are looking for at different AV intervals is the value at which the AC is maximal and the atrial contraction most efficient, whatever the "true" percentage of the atrial contribution to the ventricular stroke volume might be. To our best knowledge, there are no data suggesting that the maximal PP change found in the aorta could occur at a different AV interval than the maximal PP change found in the brachial artery.

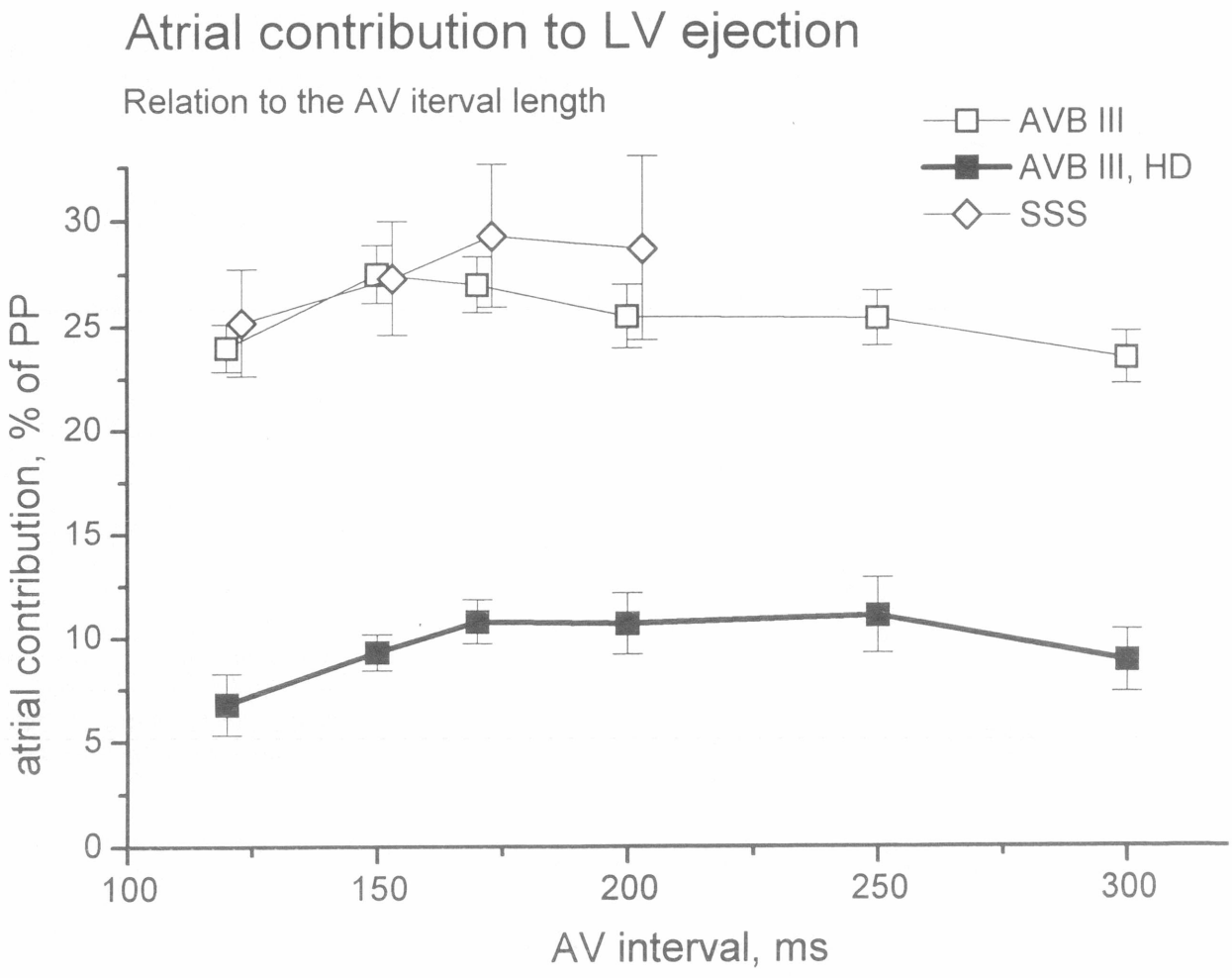

Fig. 3
Atrial contribution at different AV intervals in different groups of patients. AVB III: patients with complete AV block, AVB III, HD: patients with AVB III and heart dysfunction. SSS: patients with sick sinus syndrome. Means ± S.D. are given.

The greatest advantage of the demonstrated technique is the easy testing of the atrial contribution at different AV intervals and good reproducibility of the measured values (Fig. 2). The method believed to be the best for clinical assessment of the AV delay influence on ventricular filling, i.e. the Doppler measurement of the changes in the stroke volume (Daubert *et al.* 1993, Iwase *et al.* 1986), was found inaccurate in the study on patients with left ventricular dysfunction (Nishimura *et al.* 1995). Moreover, we were not able to find data on the reproducibility of this technique under clinical conditions when it is used for the AV delay optimization. Other techniques for AV delay optimization made use of indirect data on ventricular function, for instance an evaluation of the ventricular diastolic filling time (Bibra *et al.* 1986, Brecker *et al.* 1992). Notwithstanding the fact that the filling time is just one of the diastolic factors influencing the systolic ventricular function it was also found unreliable (Nishimura *et al.* 1995).

The disadvantage of the presented method is its invasivity. As there are experimental studies demonstrating a possible use of digital plethysmography (Fagrell and Lindvall 1979) or

photoplethysmography (Eliakim *et al.* 1973) for evaluating stroke volume changes, we believe a noninvasive technique for arterial pulse assessment could be employed to the same effect in the future. Our own preliminary data (not published) support this opinion.

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