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Pressure Wave Cardiography; Cardiac Diagnosis by Means of Noninvasive Continuous Blood Pressure Record

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Summary

The pulse pressure (PP) is proportional to the preceding interval (T) because of the restitution of contractility and the Starling mechanism, and is inversely proportional to the pre-preceding interval (T-1) because of potentiation of contractility. The aim of the present paper was to find if this relationship can be used for diagnostic purposes. Blood pressure was noninvasively and continuously recorded for 3 minutes (Peňáz method), in 26 healthy subjects, in 13 patients with congestive heart failure (NYHA I,II) and sinusoidal rhythms and in 21 patients with atrial fibrillations. By means of multidimensional regression analysis the coefficient D[T] and D[T-1] were calculated in each subject. D[T] expresses the relative role of the preceding, D[T-1] of the pre-preceding interval. The correlation between PP and T was small in subjects with sinusoidal rhythms. Subjects with particular correlation coefficients between PP and T-1 higher than 0.5 were used for further analysis (18 controls, 7 patients). The difference between D[T-1] in controls (0.30 ± 0.20) and in patients (0.48 ± 0.19) was significant (Wilcoxon $P<0.05$). In subjects with atrial fibrillations both D[T] and D[T-1] were higher in decompensated patients (Wilcoxon $P<0.05$). The ratio D[T]/D[T-1] was higher in patients with mitral stenosis than in patients with ischaemic heart disease (t-test, $P<0.05$). The test can be usefully employed as a screening test in medical practice.

Key words

Blood pressure monitoring – Interval-strength relationship – Congestive heart failure – Atrial fibrillations

Introduction

The role of inter-beat intervals in the regulation of myocardial contractility has been known since the classical description of Bowditsch's staircase (Bowditsch 1871). The force of contraction is increased by a longer preceding interval; this phenomenon is called restitution. By shorter pre-preceding and other foregoing intervals, this phenomenon is called potentiation (Bravený and Kruta 1959). Studying the potentiation and restitution of myocardial contractility on an isolated rabbit heart perfused according to Langendorf we suggested a mathematical model, which we used for a description of the relationship between intervals and isovolumic contraction magnitude by two simple equations:

$$C = f + g$$

where C corresponds to the contraction magnitude, f is a value, which is interval-independent and ischaemia sensitive, g is interval-dependent and ischaemia insensitive. f is decreased during ischaemia and nitrogen anoxia and increased during catecholamine, oubaine and Na-poor perfusion.

$$g = A * T + B * \exp(-k * T[-1]);$$

where T is the immediately preceding and T[-1] the pre-preceding cardiac interval; A,B and k are constants (Fišer 1980, Opitz and Fišer 1987). Neither the positive nor the negative inotropic factors influenced g and hence did not influence the relationship between contraction and intervals. As a consequence, decreased or increased contractility is connected with the

relative changes in the slope of the curve expressing the relationship between contraction magnitude and preceding respective pre-preceding intervals. This conclusion is supported by the former findings of the increase in the slope of the preceding interval-pulse pressure curve of decompensated patients with ischaemic heart disease and atrial fibrillations in comparison to patients with atrial fibrillations without signs of cardiac decompensation (Fišer and Semrád 1973).

The aim of the present paper was to test a non-invasive method which enables the evaluation of the myocardial function on the basis of the blood pressure record according to Peňáz. We tested patients with atrial fibrillations caused by ischaemic heart disease or mitral stenosis, patients with ischaemic heart disease with sinusoidal rhythm and healthy controls. The method is based on the study of the relative contribution in the changes of preceding and pre-preceding intervals to the magnitude of pulse pressure. The term Pressure Wave Cardiography for this method has been suggested.

Methods

Blood pressure was continuously recorded non-invasively in finger arteries by means of the volume-clamp method (Peňáz 1973) in 34 patients and 26 healthy subjects. All patients were treated by maintenance doses of digoxin. The patients were divided into 5 groups. We studied 6 patients with atrial fibrillations and ischaemic heart disease who had signs of left ventricular failure; 4 patients with atrial fibrillations and ischaemic heart disease without signs of cardiac decompensation; 6 patients with atrial fibrillation and mitral stenosis with signs of heart failure and finally 5 patients with atrial fibrillation and mitral stenosis without heart failure. Thirty-nine subjects had sinusoidal rhythms, of these, 26 were healthy controls and 13 patients with suspected ischaemic heart disease without signs of cardiac decompensation. They were selected according to anamnestic data of an exercise dyspnoea (NYHA I).

A one minute record of blood pressure and cardiac intervals was analyzed. Two coefficients D[T] and D[T-1] were calculated in each subject. Regression analysis was applied to find the regression coefficients B1 and B2 of the equation:

$$PP\{T,T[-1]\} = A + B1 * T + B2 * T[-1].$$

PP is the pulse pressure, T is the preceding interval and T[-1] pre-preceding interval. Using A, B1 and B2 both coefficients D[T] and D[T-1] were calculated (T and T[-1] are expressed in seconds):

$$D[T] = (PP\{0.9s,0.8s\} - PP\{0.7s,0.8s\}) / PP\{0.8s,0.8s\};$$

$$D[T-1] = (PP\{0.8s,0.7s\} - PP\{0.8s,0.9s\}) / PP\{0.8s,0.8s\}.$$

Equivalent relations are as follows:

$$D[T] = B1 * 0.2s / (A + B1 * 0.8s + B2 * 0.8s);$$

$$D[T-1] = B2 * 0.2s / (A + B1 * 0.8s + B2 * 0.8s).$$

D[T] expresses the relative contribution of restitution to the pulse pressure magnitude, D[T-1] the relative contribution of potentiation is seen on the scheme in Fig.1.

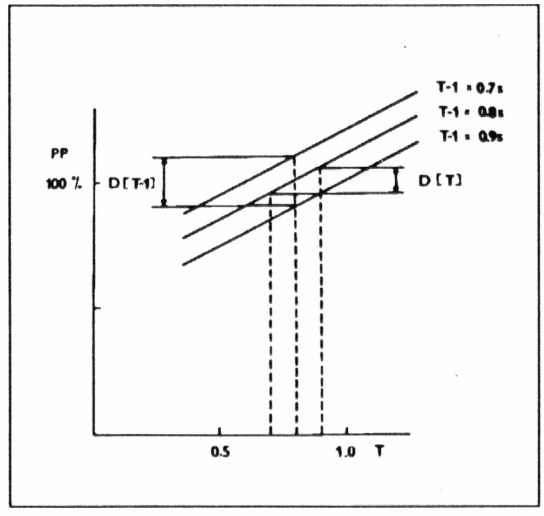

Fig. 1
Scheme of the calculation of D[T] and D[T-1]. The slopes and intercepts of the lines were calculated by means of multiple regression analysis.

The disadvantage of this approach is that the change of the average pulse pressure magnitude

during the measured period caused by the Starling mechanism increases the error of calculated values. For this reason we used an alternative method of calculation. We calculated the relative changes of pulse pressure caused by changes of preceding and pre-preceding intervals from beat to beat. Regression coefficients B1 and B2 were calculated from

(PP{T,T[-1]} - PP{T[-1],T[-2]}) / PP{T[-1],T[-2]} = A + B1 * (T - T[-1]) + B2 * (T[-1] - T[-2]).

T[-2] is an interval which precedes T[-1]. It follows that

D[T] = B1 * 0.2s * (1 - B1 * (mdt1 - 0.8s) - B2 * (mdt2 - 0.8s));

D([-1] = B2 8 0.2s * (1 - B1 * (mdt1 - 0.8s) - B2 * (mdt2 - 0.8s);

where mdt1 is the mean difference between T and T[-1]; mdt2 is the mean difference between T[-1] and T[-2]. We use the term "sliding method" for this approach.

Results

Both alternative approaches to calculations of D[T] and D[T-1] brought similar results in patients with atrial fibrillation. The sliding method in subjects with a sinusoidal rhythm is preferable because the partial correlation coefficients between preceding and pre-precedings intervals and pulse pressure magnitudes are higher in the majority of subjects. The results of D[T] and D[T-1] in patients with atrial fibrillation are seen in Fig 2. The difference between mean D[T] in patients with signs of heart failure and compensated patients (disregarding the cause of atrial fibrillation) is statistically significant (Wilcoxon, $P<0.05$), which corresponds to the theory. Mean D[T] was higher in decompensated patients.

Also D[T-1] was higher in decompensated patients (Wilcoxon, $P<0.05$). In patients with mitral stenosis D[T] was relatively increased in comparison to D[T-1]. It is not surprising because D[T] corresponds not only to the restitution of myocardial contractility but also to the speed of filling of the left ventricle during a diastole. And this speed is decreased in patients with mitral stenosis. We calculated the ratio D[T]/D[T-1] (± S.D.) in patients with ischaemic heart disease and in mitral stenosis (both with signs of heart failure) and found a significant difference (1.53 ± 0.29 versus 2.19 ± 0.76, Student t-test, $P<0.05$).

In subjects with a regular cardiac rhythm, no difference in D[T] between patients and controls was found. Mean D[T-1] was higher in patients when only subjects of both groups with partial correlation coefficients between pre-preceding intervals and pulse pressure magnitude higher than 0.5 were taken into account (18 controls, 7 patients, Wilcoxon, $P<0.05$). The histograms are seen in Fig. 3. The specificity of this method is low, there are relatively high values of D[T-1] in both groups, but sensitivity is relatively high, D[T-1] lower than 20 % was found in 45 % of the controls and not found in any patient.

Discussion

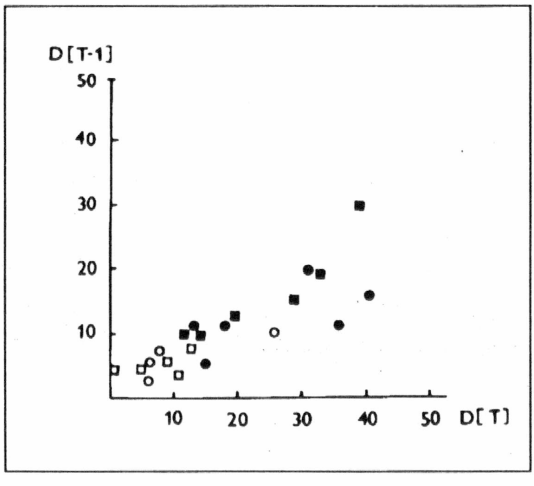

Fig. 2
D[T-1] plotted against D[T] in patients with atrial fibrillations (open symbols - compensated patients, closed symbols - decompensated patients, circles - ischaemic heart disease, squares - mitral stenosis).

The exact explanation of changes of D[T] and D[T-1] is difficult. The reason for this is that, despite the sophisticated methods used for studying the movement of Ca-ions in myocardial cells, a suitable model of Ca-

movement does not yet exist (Ikonomidis *et al.* 1990). On the other hand, the important role of Ca-ions in the interval-strength relationship is generally accepted. Despite the fact that the results correspond to the theory, the discriminating power important for using PWCG in clinical practice is relatively low. It is possible that longer records of blood pressure can increase the suitability of the method. On the other hand, lower D[T] or D[T-1] in decompensated patients need not be an error. The magnitude of D[T] and D[T-1] corresponds to the myocardial function and signs of heart failure such as oedema are influenced by other factors such as diuretics therapy. The error is probably increased by pulsus alternans, a phenomenon which occurred after an abrupt change of heart rate at higher mean heart rates. In a former study, we found that pulsus alternans occurred at lower average heart rates in decompensated patients (Fišer and Semrád 1972). Pulsus alternans increases the non-linearity of the system and so increases the error, because our calculation is based on a supposed linearity of the system.

The non-linearity is higher in patients with atrial fibrillation where the variation of intervals is higher than in subjects with a sinusoidal rhythm. The changes of intervals in the latter are so small that, in fact, the relationship between pre-preceding interval and pulse pressure is exponential and plays a negligible role.

Also the fact that the ratio D[T]/D[T-1] is not lower in all subjects with mitral stenosis in comparison to subjects with ischaemic heart disease need not be an error of the method only. The rheumatic fever, the usual cause of valvular disease, also attacks the working myocardium and the higher D[T] can be the result of decreased inotropy.

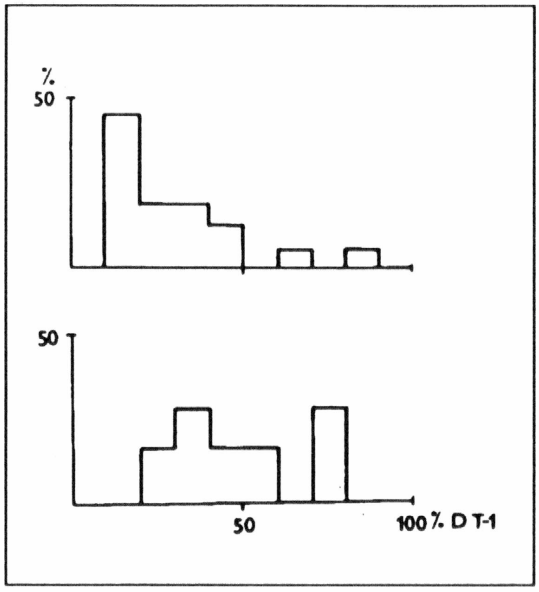

Fig. 3
Distribution histograms of D[T−1] in normal subjects (above) and in heart failure patients (below).

The most important advantage of this method is its versatility. Each blood pressure monitor based on the volume-clamp method with an A/D converter and PC can perform the calculation of D[T] and D[T-1] automatically. It is possible that longer continuous records of D[T] and D[T-1] could provide more valuable information. We can also not exclude the possibility that PCWG can be used for diagnosis of mitral stenosis in patients with a sinusoidal rhythm, a difficult task for an inexperienced physician.

Acknowledgement

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Reprint Requests

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