Beat-to-beat blood-pressure fluctuations and heart-rate variability in man: physiological relationships, analysis techniques and a simple model

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Chapter 7

Relationships between short-term blood-pressure fluctuations and heart-rate variability:

II. A simple model

In this chapter we give a tentative interpretation of the phase spectra of RR-intervals against pressure variables (chapter 6) by means of the beat-to-beat model that was presented in chapter 5.

7.1 Abstract

A simple model of the beat-to-beat properties of the cardiovascular system is used to interpret the results of spectral analysis of blood-pressure and interval data as presented in chapter 6. The model consists of two equations, one representing the fast regulation of interval by the systolic pressure (baroreflex), the other one representing a Windkessel approximation of the systemic arterial system.

The model, when applied to interval and blood-pressure data from resting subjects, explains the lack of respiratory variability in the diastolic pressure values. The baroreflex equation seems to describe the data only in the region of respiratory frequencies. The shape of the phase spectrum of systolic pressures against intervals is modelled by difference equations, but no physiological interpretation of these equations is given.

7.2 Introduction

In chapter 6 we presented power spectra of beat-to-beat RR-interval (RRI) and blood pressure (BP) fluctuations. These spectra showed that both RRI- and BP-variability can be attributed quantitatively to its various causes, particularly to respiratory influences and the 10-second-rhythm. It was remarkable that almost no respiratory influence was seen in the spectrum of diastolic pressures.

We also presented cross-spectra of blood pressure values against intervals, which showed that systolic pressure variations lead interval variations by about two beats (2 s) in the 10-second-region; however, in the respiratory region, the intervals seemed to vary together with the systolic pressures.
In the present paper we try to explain some of the results and indicate how the cross-spectra may be interpreted. Our point of departure is a simple beat-to-beat model of the cardiovascular system (chapter 5, or DeBoer et al., 1983).

7.3 Methods
Blood pressure and ECG from resting subjects were recorded. Beat-to-beat pressure values and intervals were derived from these recordings and were used to calculate power and cross-spectra. Successive interval and pressure values were considered to be equidistantly spaced at distances equal to the mean interval length. Details of the data acquisition and the estimation of the spectra are given in chapter 6.

The cross-spectrum between a pressure variable (e.g., systolic pressure $S$ or diastolic pressure $D$) and the interval $I$ consists of two parts. The (squared) coherence spectrum $k^2(f)$ is a measure of the correlation between pressure and interval variability in a certain frequency band, and has values between 0 (no correlation) and 1 (complete correlation). The phase spectrum $\phi(f)$ indicates the phase difference between the signals; a negative value of $\phi(f)$ implies the pressure variation to lead the interval variation. If the coherence is low, the phase cannot be estimated reliably (Jenkins and Watts, 1968, ch.9).

In some cases, the data were band-pass filtered by applying a Digital Fourier Transform to the values, setting the unwanted contributions equal to zero, and applying an inverse Fourier Transform.

7.4 Model
7.4.1 Relations between beat-to-beat blood-pressure and R-R interval values.
To interpret the power and cross-spectra, we start with part of the simple beat-to-beat model as presented in a previous chapter (chapter 5). The following, physiologically plausible relationships between successive pressure and interval values were used to describe experimental BP- and RRI-data:

a) $I_n = a_0 S_n + c_1$ (baroreflex)
b) $D_n = c_2 S_{n-1} \cdot \exp(-I_{n-1}/T)$ (Windkessel)

In these equations $S_n$ and $D_n$ are the systolic and diastolic pressure, respectively, occurring during RR-interval $I_n$. 

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Eqn.a) states that an increase in systolic pressure $S_n$ implies an immediate increase in interval-length $I_n$. This is due to the fast, vagally mediated baroreflex-loop, aimed at keeping the blood pressure constant (Sleight, 1980). When a systolic pressure wave arrives at the baroreceptors in the aorta and the carotid sinuses, it causes a burst of afferent nerve spikes, travelling towards the Central Nervous System (CNS). The amount of nerve output depends on the pressure felt by the baroreceptors (Arndt et al., 1977). The pressure information is processed in the CNS, which then sends an efferent spike burst along the vagal nerve towards the cardiac pacemaker, thus leading to an adjustable amount of delay of subsequent heart beats. (The sympathetic effects on heart rate are neglected here, as they are much slower.) The most simple description of this process is eqn.a). As we consider data from resting subjects with small variations around the mean values, a linear approximation for the relationship between $S_n$ and $I_n$ is acceptable.

The constant $a_0$ is the so-called baroreflex-sensitivity coefficient and has values in the range 5-30 ms/mmHg (Smyth et al., 1969; Pickering et al., 1972). These values were found in experiments in which the blood pressure was increased or decreased by drugs, and the subsequent change in interval was compared with the change in blood pressure.

Eqn.b) is a formulation of the Windkessel model for the systemic circulation during diastolic run-off. The value of the diastolic pressure $D_n$ depends on the previous systolic pressure $S_{n-1}$ and on the length of the previous interval $I_{n-1}$. $\tau$ is the time-constant of the Windkessel. The equation can be linearized by rearranging and taking logarithms:

\[
\log(S_{n-1}/D_n) = I_{n-1}/\tau - \log(c_2),
\]

with $\Delta$ the logarithmic decrement.

In chapter 5 eqns.a) and b) were found to be in reasonable agreement with experimental BP- and RRI-data. In the next sections the phase spectra as implied by these equations are compared with spectra as calculated from the experimental data of fig.2 of chapter 6. The mean interval length $I$ of the 960 considered beats was 0.93 s. The power spectra of intervals (fig.1a), of systolic pressures (fig.1b, solid line) and of diastolic pressures (fig.1b, dashed) are again
Fig. 7-1a Power spectrum $P(f)$ of RR-intervals from a resting subject, showing peaks at 0.1 Hz (10-second-rhythm) and around the respiratory frequency.

Fig. 7-1b Power spectrum $P(f)$ of systolic pressure $S$ (solid line) and of diastolic pressures $D$ (dashed line). In the latter spectrum the respiratory peak is absent.

Fig. 7-1c Squared coherence spectrum $k^2(f)$ (dashed line, between 0 and 1), and phase spectrum $\phi(f)$ (solid line, between $-180^\circ$ and $180^\circ$) of systolic pressure $S$ against interval $I$. A high coherence implies a strong link between pressure and interval variations, as is the case around 0.1 Hz and in the respiratory region. When the coherence is high ($>0.5$), the phase is reliably estimated (heavy line). The phase is negative when pressure variations lead interval variations (e.g., at 0.1 Hz). In the region of respiratory frequencies, the phase difference between pressure and interval variations is small.

Fig. 7-1d Squared coherence spectrum $k^2(f)$ (dashed line) and phase spectrum $\phi(f)$ (solid line) of diastolic pressure $D$ against interval $I$. The phase spectrum shows no trend.
7.4.2 The baroreflex equation

The baroreflex equation a) implies that no phase difference exists between variations in $S$ and in $I$. Fig.1c shows that this phase difference is only approximately zero in the region of respiratory frequency (0.20-0.35 Hz). Around 0.1 Hz a definite lead of pressure exists of around $60^\circ = 1/6$ th period, corresponding with $10/6 = 1.7$ s or approximately 2 beats. Hence eqn.a) does not describe the data over the whole frequency range from 0 to 0.5 Hz. The scatter plot of $I_n$ against $S_n$ (fig.2a), which shows no clear linear relationship (correlation coefficient $r = 0.25$), does not corroborate equation a) either.

Thus, the phase spectrum between $I$ and $S$ (Fig.1c) suggests that the baroreflex-equation a) is valid only in the region of normal respiratory frequency. An estimate of the baroreflex sensitivity coefficient $a_0$ should therefore be based on this frequency-band only. In order to study only respiratory variations, the following simple method was used. The 0.1 Hz waves and other low-frequency fluctuations were removed from the BP- and RRI-variations by differencing of successive pressure or interval values, i.e., $X_n^{d} = X_n - X_{n-1}$. This is equivalent to filtering the data with a high-pass filter with gain-function $4 \sin^2(\pi f)$ (fig.2b; Jenkins and Watts, 1968, Ch.7.3).

![Fig.7-2a Scatter plot of R-R intervals $I_n$ against systolic pressures $S_n$ occurring during this interval. The baroreflex equation implies a linear relationship between $I_n$ and $S_n$, which is not seen from the data.](image-url)
Fig. 7-2b Gain-function of the difference filter as used to remove the low-frequency variability from I and S. As the mean interval length $\bar{T}$ is 0.93 s, the filter is defined from 0 to $1/2\bar{T} = 0.54$ Hz.

Fig. 7-2c Power spectrum of RR-intervals after application of the difference filter to remove the variability due to the 10-second-rhythm and to slower fluctuations. Almost only respiratory variability is left.

Fig. 7-2d Power spectrum of systolic pressures after application of the difference filter.

Fig. 7-2e Squared coherence spectrum $k^2(f)$ (dashed) and phase spectrum $\varphi(f)$ (solid) of differenced intervals against differenced systolic pressures. No appreciable difference with fig. 1c. See text.
For the data of fig. 1a, b this technique produces power spectra of $I^d$ and of $S^d$ as shown in Figs. 2c, d. The 0.1 Hz peaks are now minimized. The cross-spectrum of $S^d$ against $I^d$ is not changed (fig. 2e), because the phase shift due to the difference filter is identical for $I^d$ and for $S^d$. The existing minor differences between fig. 1c and fig. 2e are due to the averaging of the spectral values, preceding the calculation of the cross-spectrum. Fig. 2f is the scatter plot of $I^d_n$ against $S^d_n$. As expected, the correlation coefficient is higher than in fig. 2a ($r = 0.76$), permitting an estimation of the coefficient $a_0 = 9.7 \text{ ms/mmHg}$.

A different estimate of the baroreflex sensitivity coefficient was obtained by applying a band-pass filter between 0.2 Hz and 0.35 Hz to the data (see section 7.2); linear regression of the filtered data of intervals on systolic pressures now lead to a value $a_0 = 13.2 \text{ ms/mmHg}$ ($r = 0.94$). This value, which lies within the wide range of values obtained by other authors (section 7.4.1), will be used later on.

7.4.3 The Windkessel equation

According to the linearized equation by a fixed delay of one beat exists between the logarithmic decrement $L$ and $I$; this amounts to a phase difference between $L$ and $I$ increasing linearly with frequency from $0^\circ$ at $f = 0$ to $180^\circ$ for $f = 1/2T = 0.54$ Hz. This is actually observed in the calculated cross-spectrum of $L$ against $I$ (fig. 3a).
1. We estimated the timeconstant $\tau$ by linear regression of $L_n$ on $L_{n-1}$, after removing frequency components under 0.05 Hz from $I$ and $L$. This was done because for these frequencies the power in the spectra of $I$ and $L$ is high, but the coherence is low. Hence the correlation between $L_n$ and $L_{n-1}$ improves if the low-frequency component is removed. After filtering the time constant was estimated as $\tau = 1.85 \text{ s} (r = 0.87)$, which is in agreement with published values for the time constant of a Windkessel approximation to the systemic circulation (Simon et al., 1979; Watt and Burrus, 1976).

The evidence presented above suggests that the baroreflex equation a) holds only in the region of the normal respiratory frequency (0.20-0.35 Hz), but that the Windkessel equation b) is approximately valid for all frequencies above 0.05 Hz. In the following, eqns.a) and b) are used to explain why a respiratory peak should be absent in the spectrum of diastolic pressures.

We assume small variations of $D_n$, $S_n$ and $I_n$ around their mean values $D$, $S$ and $I$, and write $D_n = D + d_n$, $S_n = S + s_n$ and $I_n = I + i_n$. Then eqn.b) becomes:

$$b) \quad d_n = \frac{D}{S} \cdot s_{n-1}/S - i_{n-1}/\tau$$

Around the respiratory frequency, the baroreflex-equation a) may be used in the form:

$$a) \quad i_n = a_0 \cdot s_n.$$ 

So eqn.b) can be written as:

$$b) \quad d_n = \left( \frac{D}{S} \right) s_{n-1}/S - a_0/\tau.$$ 

Hence, no diastolic variability exists ($d_n = 0$) if $a_0 = \tau/S$. We consider here the case of $D = 75 \text{ mmHg}$, $S = 143 \text{ mmHg}$, $a_o = 13.2 \text{ ms/mmHg}$ (section 7.3.2) and $\tau = 1850 \text{ ms}$, and we find: $d_n = -0.01 \cdot s_n$. The respiratory variations in $D$ are only one per cent of the variations in $S$ and no respiratory peak is expected in the power spectrum of $D$.

Fig.3b illustrates qualitatively the argument presented above. A few consecutive heart beats are depicted. The systolic pressure is higher during beat $n$ by
If the systolic value $S_n$ is higher than the previous value $S_{n-1}$ and if the interval length $I_n$ does not change, the diastolic pressure $D_{n+1}$ will be higher than the previous one $D_n$ (broken lines). However, the baroreflex transforms the increased systolic pressure into a longer interval and so the diastolic pressure does not vary (solid line). This mechanism explains the lack of respiratory variability in the diastolic pressures. Consequently, it can be argued that the lack of respiratory variability in the diastolic pressures is a token of a functioning baroreflex (DeBoer et al., 1985f,g). The argument presented above is only valid for the variability in the respiratory frequency-band, because only there the systolic pressures and the intervals vary together. Therefore the 10-s variability in the diastolic pressures is not suppressed (cf. fig.1b).

7.4.4 Relations between the spectra
As the pulse pressure $P_n$ is defined as the difference between systolic pressure $S_n$ and diastolic pressure $D_n$, it is to be expected that the power spectrum of $P$ and the phase spectrum of $P$ against $I$ can be derived from the power and phase spectra of $S$ and $D$. Figs.4a,b show the directly calculated power spectrum of $P$ and phase spectrum of $P$ against $I$ (solid lines), and the spectra as computed from the spectra of $S$ and $D$ (dotted lines; only between 0.05 Hz and 0.15 Hz). For frequencies above 0.15 Hz, little variability in diastolic
pressure exists (fig. 1b) and hence for these frequencies the spectra of P and of P against I (figs. 4a, b) resemble the spectra of S and of S against I (figs. 1b, c), respectively.

In the range 0.05-0.15 Hz the spectrum of P was calculated from the spectra of S and D as indicated in fig. 4c. The Fourier transform of P is a complex quantity, having amplitude and phase. The components of \( \vec{P} \) can be obtained by vector-subtraction of the Fourier components of \( \vec{S} \) and \( \vec{D} \). The example in fig. 4c shows the calculation for \( f = 0.1 \) Hz. The phase of \( \vec{I} \) was defined as 0°.

The phase differences between \( \vec{S} \) and \( \vec{I}(-62°) \) and between \( \vec{D} \) and \( \vec{I}(-79°) \) were read from fig. 1c and fig. 1d, respectively. The lengths of the vectors \( \vec{S} \) and \( \vec{D} \) were taken from fig. 1b. The vector \( \vec{P} \) was found as the difference between \( \vec{S} \) and \( \vec{D} \).

![Fig. 7-4a](image1)

**Fig. 7-4a** Power spectrum \( P(f) \) of pulse pressure \( P \), calculated directly (solid line), and from the power and cross-spectra of \( S \) and \( D \) (dotted line; only between 0.05 Hz and 0.15 Hz).

**Fig. 7-4b** Squared coherence spectrum \( k^2(f) \) (dashed) and directly calculated phase spectrum \( \Phi(f) \) (solid line) of \( P \) against \( I \). The dotted line is the phase spectrum as calculated from the power and cross-spectra of \( S \) and \( D \).

**Fig. 7-4c** Diagram of the calculation of the Fourier components of \( P \) for \( f = 0.1 \) Hz by vector-subtraction of the Fourier components of \( S \) and \( D \). Phase angles and lengths of the vectors \( S \) and \( D \) are read from figs. 1b-d.
and $\sqrt{1}$ its (squared) length and phase angle ($-21^\circ$) are plotted in figs.4a,b (dotted lines). The correspondence between the dotted line and the directly calculated values is good, which was to be expected. This shows that the spectra of $D$, $S$ and $P$ are not independent, and that each of them may be computed from the other two.

7.5 Discussion

We presented a beat-to-beat model of part of the cardiovascular system. We checked whether this model could be confirmed by the power and cross-spectra of blood pressure and RR-intervals (chapter 6, or DeBoer et al., 1985). The baroreflex equation a), relating interval length to systolic pressure, held approximately only for respiratory frequencies (0.20-0.35 Hz). The Windkessel equation b), describing the diastolic pressure as a function of previous systolic pressure and interval, was confirmed by the shape of the phase spectrum of $L$ against $I$, with the logarithmic decrement $L$ defined as $L_n = \log(S_{n-1}/D_n)$. Using the model equations, it becomes clear why the respiratory variations in the diastolic pressure values are small: a high value of the systolic pressure would be followed by a high diastolic value; but variations in systolic pressure and in interval go together at the respiratory frequency, possibly due to the fast baroreflex, and so the high systolic value implies a lengthened interval, which tends to lower the diastolic pressure. As these two influences are counteracting, the diastolic pressure will remain more or less unaffected under respiratory variations of systolic pressure and interval. Our model states that the absence of respiratory influence in the diastolic pressure is consequential to a functioning baroreflex, and so a diminished vagal regulation of heart rate should become manifest in the appearance of a respiratory peak in the spectrum of diastolic pressures.

Scher and Young (1970) altered the blood pressure in unanesthetized dogs by sinusoidal inflation of implanted aortic cuffs and found a good correlation between interval duration and systolic pressure during the interval. They considered this "no-lag" response as vagal and found values of the baroreflex sensitivity coefficient between 10 and 45 ms/mmHg (cf. section 7.3.1). When the pressure was considerably decreased, the interval-response was slower, due to sympathetic effects. Using stimulation frequencies of 0.05-0.20 Hz, they found
little or no phase difference between variation in systolic pressure and interval. This is not in line with our results, as we find 60°-90° phase difference around 0.1 Hz. Possibly the spontaneous fluctuations in heart rate and blood pressure as studied by us are not directly comparable with the imposed pressure variations in the study of Scher and Young. The difference in species may also be important; dogs usually show a much larger respiratory sinus arrhythmia than is seen in man, which suggests a difference in the cardiovascular control system. In addition, the posture of dogs is often different from the posture of men.

One is tempted to approximate the phase spectrum of systolic pressure against interval (fig.1c) by a line from -90° at $f = 0$ Hz to +90° at $f = 1/2f = 0.54$ Hz. This line corresponds with the phase spectra belonging to the difference equations $I_n = \alpha(S_n - S_{n+2})$ or $S_n = \beta(I_n - I_{n-2})$, as can be seen using Z-transforms (Jury, 1973; Jenkins and Watts, 1968, ch.8.4); the first of these equations is then expressed by:

$$I(Z) = \alpha \cdot (S(Z) - Z^2 S(Z)) = \alpha \cdot S(Z) \cdot (1-Z^2),$$

and, if we put $Z = \exp(2\pi j f)$:

$$I(Z) = \alpha \cdot S(Z) \cdot (1-\exp(4\pi j f)).$$

This amounts to a phase difference between $S$ and $I$ that goes linearly from -90° for $f = 0$ to +90° for $f = 1/2f$, i.e. similar to the phase spectrum of fig.1c. The second difference equation, when expressed as $S(Z) = \beta \cdot I(Z) \cdot (1-Z^2) = \beta \cdot I(Z) \cdot (1-\exp(-4\pi j f))$, leads to the same phase spectrum. However, both difference equations are non-causal, as they predict an interval from a subsequent systolic pressure, and a systolic pressure from a not yet ended interval, respectively. Therefore they do not seem to be attractive for modelling the physiological relationship between $S$ and $I$. The difference equations are more fully discussed in appendix 7.A1.

Hence, we have no explanation for the shape of the phase spectrum of systolic pressures $S$ against intervals $I$. This spectrum is not in conformity with the simple baroreflex influence as modelled by eqn.a). Both difference equations which we present describe the shape of the phase spectrum, but a physiological interpretation is not available. The lead of around 2 s of systolic pressure variability on interval variability in the 10-second region may be related to sympathetic regulation, which has latencies in this range (Levy and Martin, 1979).
It appears to be necessary to model the influence of blood pressure on RR-interval length in a more elaborate way than was done in this paper. On physiological grounds the model should at least include some sympathetic regulation of heart-rate, in addition to the vagal regulation of equation a).

We showed that the spectrum of pulse pressures can be derived from the spectra of systolic and diastolic pressures, due to the identity \( P_n = S_n - D_n \) (section 7.3.4). In a similar way other -- more interesting -- relationships between pressures and/or intervals should be reflected in the various spectra.

Two final remarks should be made. First, the direct relationship in variability of interval and of systolic pressure in the respiratory region need not be caused solely by baroreflex influence, but may also be due to a simultaneous influence of respiration-coupled efferent nervous activity on RR-interval and on blood pressure, since correlation does not imply causality. The fact that for higher respiratory frequencies the interval seems to lead the systolic pressure may also be an indication for this. Second, it need not be that a single regulatory mechanism is commanding both the 20-second-variability and the respiration-linked variability, and so different explanations may be needed for both phenomena.
Appendix 1: Phase spectra of the difference equations

In section 7.5 we argued that the phase spectra between interval and systolic-pressure variability can be modelled by any of two different difference equations, but we were not able to give a physiological interpretation of these equations. In this appendix the power and phase spectra belonging to these difference equations are shown for simulated data. We also give two difference equations that model the shape of the phase spectra of intervals and diastolic pressures and again show calculated spectra of simulated data.

The phase spectra between intervals and systolic pressures resemble a straight line between \(-90^\circ\) at 0 Hz and \(+90^\circ\) at \(1/\bar{I}\) Hz, with \(\bar{I}\) the mean interval length (fig.1c, 2c, 3c). The following difference equations produce identical phase spectra:

\[ l_n = \alpha \cdot (s_{n-1} - s_{n+2}) \quad (A1), \]

and:

\[ s_n = \beta \cdot (l_{n-1} - l_{n-2}) \quad (A2). \]

Using Z-transforms, we found in section 7.5 that these equations can be written as:

\[ I(Z) = \alpha \cdot S(Z) \cdot (1 - \exp(4 \pi i \tilde{f})) = -2 \cdot \alpha \cdot S(Z) \cdot \sin(2 \pi \tilde{f}) \cdot \exp(2 \pi i \tilde{f}) \quad (A1), \]

and:

\[ S(Z) = \beta \cdot I(Z) \cdot (1 - \exp(-4 \pi i \tilde{f})) = 2 \beta \cdot I(Z) \cdot \sin(2 \pi \tilde{f}) \cdot \exp(-2 \pi i \tilde{f}) \quad (A2), \]

respectively.

Both equations describe an identical phase relation between systolic pressures and intervals, but the amplitude relations between the interval spectrum and the spectrum of systolic pressures as implied by equations A1 and A2 are different. For eqn. A1 the ratio between the power spectra of intervals \(P_I(f)\) and of systolic pressures \(P_S(f)\) is \(P_I(f)/P_S(f) = (I(Z)/S(Z))^2 = 4 \alpha^2 \sin^2(2 \pi \tilde{f})\); for eqn. A2 this ratio is \(1/(4\beta^2 \sin^2(2 \pi \tilde{f}))\). Hence, for \(f \rightarrow 0\) Hz \(P_I(f) \gg P_S(f)\) (eqn. A1), or \(P_I(f) \ll P_S(f)\) (eqn. A2). The same holds for \(f \rightarrow 1/\bar{I}\).

Similarly, difference equations can be constructed that model the phase spectrum of diastolic pressure against intervals; this spectrum has approximately the value \(-90^\circ\) over the whole frequency range (fig.1d). Difference equations that produce a constant phase difference \(-90^\circ\) are:

\[ l_n = D_{n-1} - D_{n+1} \quad (A3), \]
as well as:

\[ D_n = I_{n+1} - I_{n-1} \quad (A4). \]

The phase spectra for these two difference equations are again found by means of the Z-transform:

\[ D(Z) = D(Z(Z-1) - (Z-1)) = D(Z)(\exp(-2\pi ifl) - \exp(2\pi ifl)) = -2D(Z)\sin(2\pi f1) \quad (A3), \]

and:

\[ D(Z) = D(Z(Z-1)) = D(Z)(\exp(2\pi ifl) - \exp(-2\pi ifl)) = 2D(Z)\sin(2\pi f1) \quad (A4). \]

Eqns. A3 and A4 lead to similar amplitude relations between the spectra of intervals (I) and diastolic pressures (D) as was the case for intervals and systolic pressures (S) as shown above.

Hence, both for the spectrum of I against S and of I against D, two difference equations are found to agree with the phase spectra. This dual set of difference equations is to be compared with the similar situation if differential equations are sought that may explain a phase-difference of 90° between two signals x(t) and y(t): the two differential equations x(t) = y'(t) and y(t) = -x(t) imply as a relation between the Fourier-transforms \( X(\omega) = i\omega Y(\omega) \) and \( Y(\omega) = -i\omega X(\omega) \), respectively, and both relations lead to a phase-difference of 90° (but different ratios of the power spectra).

The two difference equations mimicking the phase spectrum of I against D are not causally interpretable.

To illustrate the above considerations, we show in figs.A1,2 power and cross-spectra of simulated "systolic pressures", "diastolic pressures" and "intervals", using the difference equations A1-A4. For Fig.A1, the systolic values \( S_n \), diastolic values \( D_n \) and intervals \( I_n \) were computed for \( n = 1 \) to 975 as:

\[ S_n = 100 + \delta_n, \]
\[ I_n = 1 + 0.05 (S_n - S_{n+2}) + \varepsilon_n, \] (cf. eqn.A1),
\[ D_n = 50 + 20 (I_{n+1} - I_{n-1}) + \eta_n, \] (cf. eqn.A4),
\[ M_n = 75 + \gamma_n. \]

with \( \delta_n, \varepsilon_n, \eta_n \) and \( \gamma_n \) independent Gaussian noise with mean zero and standard deviation 5, 0, 0.5, 1 and 4, respectively. Note that the simulated "mean values" \( M_n \) consist only of noise.
Fig. 7-A1 Power spectra of I (a), S and M (b) and D (c), and cross-spectra of S against I (d), M against I (e) and D against I (f). The data are simulated by difference equations (see text). Most noteworthy are the shape of the phase spectrum of S against I, going from $-90^\circ$ at $f = 0$ Hz to $+90^\circ$ at $f = 0.5$ Hz, and of the phase spectrum of D against I, at a constant value of $-90^\circ$. These phase spectra resemble the ones computed from experimental data. The spectra are fully discussed in the text. Horizontal: frequency (Hz), vertical: power (figs. A1a-c), coherence (figs. A1d-f, dashed) and phase (figs. A1d-f, solid).
Fig. 7-A2 As Fig. A1, but a different set of difference equations is used for the simulation.
For Fig. A2, $S_n$, $D_n$, $I_n$ and $M_n$ were computed as:

$$D_n = 50 + \delta_n,$$
$$I_n = 1 + 0.05(D_{n-1} - D_{n+1}) + \epsilon_n,$$  \hspace{1em} \text{(cf. eqn. A3)},
$$S_n = 100 + 20(I_n - I_{n-2}) + \eta_n,$$  \hspace{1em} \text{(cf. eqn. A2)},
$$M_n = 75 + \zeta_n,$$

with $\delta_n$, $\epsilon_n$, $\eta_n$ and $\zeta_n$ as above.

The phase spectra between $I$ and $S$ (figs. A1d and A2d) are seen to be identical and in correspondence with theory; likewise the phase spectra between $I$ and $D$ (figs. A1f and A2f). The power spectra are "white" for $S$ in fig. A1b, and for $D$ in fig. A2c. The power spectra of $I$ in fig. A1a and fig. A2a show the expected $\sin^2(2\pi f)$-shape: low values for $f = 0$ Hz and $f = 1/2T = 0.5$ Hz, and a maximum for $f = 1/4T = 0.25$ Hz.

The power spectra of $D$ in fig. A1c and of $S$ in fig. A2b have a $\sin^4(2\pi f)$-shape.

The power spectra of $M$ are white (figs. A1b, A2b), and the coherence spectra of $I$ against $M$ have low values throughout (figs. A1e, A2e).

In summary: this appendix shows that the stated difference equations lead to results resembling the experimentally obtained phase spectra of $I$ against $S$ and of $I$ against $D$; however, the non-causal character of the equations hinders a physiological interpretation of the equations.