Assessment of vasomotor oscillations with Fourier analysis of biological tissue impedance

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Abstract. Fourier analysis revealed a number of periodicities in small variations of bioimpedance of human finger including the major spectrum peaks at the frequencies of heart beats, respiration, and Mayer wave (0.1 Hz). These periodic variations of bioimpedance were detected under the normal conditions and during blood flow arrest in the hand by a pneumatic cuff placed on the arm. They are explained by periodic variations in systemic blood pressure and by oscillations of regional vascular tone resulted from neural vasomotor control. During normal blood flow, the greatest variations in bioimpedance were observed at the heart rate, and their amplitude surpassed by an order of magnitude the amplitudes of respiratory oscillations and Mayer wave. In contrast, during blood arrest, the largest amplitude of rhythmical changes of the impedance characterized the oscillations at respiration rate, while the amplitude of oscillations at the heart rate was the smallest. During normal respiration and circulation, two side cardiac peaks were revealed in bioimpedance amplitude spectrum which disappeared during respiration arrest and thought to reflect the amplitude respiratory modulation of the cardiac output via sympathetic influences. During normal breathing, the second and the third harmonics of the cardiac spectrum peak were split reflecting frequency respiratory modulation of the heart rate by parasympathetic influences. The results favour applicability of Fourier analysis of bioimpedance variations in assessment of regional neural influences and neurogenic modulation of cardiac activity.

1. Introduction
A spectacular progress in microelectronics resulted in appearance of low-noise chips and powerful computers which made it possible to measure and analyze small bioimpedance variations with a laboratory-made high-resolution impedance converter and original software. We analyzed bioimpedance variations in human finger with Fourier transform in the frequency band of 0.08–15.0 Hz under the normal conditions and during circulation arrest in the arm optionally combined with expiratory delay for 40 sec. The aim was to assess the neurogenic contribution to bioimpedance variations, which probably result from vasomotor activity of sympathetic nerve system.

2. Methods
Bioimpedance was measured with original impedance converter based on synchronous detection principle resulting in total (“basic”) real part of impedance in the frequency band of 0–15 Hz (R_e) and the variable component of this real part in the frequency band of 0.08–15 Hz (R_v). The measuring
frequency was 100 kHz. The resolving power of R. channel was 250 microOhm in the range of ±4 Ohm. To obtain the amplitude Fourier spectrum, R. record was cut into 25.6-sec fragments containing 4096 points. FFT was calculated with Hanning window. The amplitude spectra of 2 to 4 such fragments were averaged. The impedance converter was connected to the proximal phalanx of the middle human finger (n=14) with two output current wires and two input voltage wires coupled to two Ag/AgCl wire electrodes made of silver wire (0.4 mm in diameter) covered with narrow gauze bandages soaked in physiological saline. The distance between these electrodes was about 2 cm. To damp the mechanical vibrations caused by heart and lungs, the arm was fixed to a massive support. Circulation in the arm was temporarily arrested with a pneumatic cuff inflated to at least 2-fold systolic pressure to prevent the spread of hydraulic wave in the blood vessels of the arm.

3. Results

Figure 1 show basic impedance \( R_\infty \) (b) and variable impedance component \( R_\sim \) measured under normal conditions (a) and during circulation arrest in the left arm (c). While the large oscillations of bioimpedance in (a, b) resulted mostly from pumping action of the heart, the smaller ones (c) observed during circulation arrest were not cardiac reflecting hypothetically the neural vasomotor activity.

The nature of these oscillations can be elucidated by frequency-domain analysis. Figure 2 shows amplitude Fourier spectrum during intact circulation. Peak 1 was observed at the frequencies of 0.1–0.2 Hz. Oscillations of arterial pressure at this rate are known as “Mayer wave” [1] so the corresponding peak is termed here as Mayer’s. The frequencies of peaks 2 and 3 coincided with respiration and heart rate, correspondingly. The large cardiac peak 3 and its harmonics 3”, 3’”, 3’’” spaced equidistantly along the frequency axis represented the large-scale oscillations clearly seen in the raw graphic data (figures 1, a, b). Other bioimpedance oscillations revealed by Fourier analysis cannot be directly observed. The cardiac peak and its harmonics were accompanied by two smaller left (L) and right (R) peaks spaced symmetrically from the central peak at the distance equal to the respiration rate. These side peaks and the respiratory peak 2 simultaneously disappeared during respiration arrest for 40 sec (figure 3). Thus, the cardiac side peaks 3L and 3R depend on respiration.

The trident spectrum structure with a central and two symmetrical side peaks is well known in physics as reflecting the amplitude modulation of one periodic process (here the heart beats) with another (and slower) periodic process (respiration). The amplitude of the side peaks is proportional to the “depth” of such modulation. Thus, the amplitude spectrum of bioimpedance variations makes it possible to assess the intensity of respiratory control over the force of the heart beats [2, 3].
Figure 2. Amplitude spectrum of bioimpedance variations in human finger over broad frequency range with Mayer's peak (1), respiratory peak (2), four cardiac harmonics (3 to 3'''), and the side peaks (3L, 3R, etc). Note the break in ordinate.

Another important observation is splitting of higher harmonics of the cardiac peak observed under normal conditions in some cases (figure 3, a). Such splitting was far less pronounced under respiration delay (figure 3, b). Splitting of a peak can be explained by frequency modulation of basic oscillatory process with another process going on at a smaller rate; in this case, splitting can result from vagal modulation of the heart rate at the respiration frequency. In this experiment, the oscillations were cut off below 0.3 Hz, so no Mayer peak is seen in figure 3.

Figure 3. The respiratory (2) and side cardiac peaks (L and R) are observed under normal conditions (a) but disappeared during expiratory delay (b).

In order to assess the changes in bioimpedance of non-pulsatile (non-cardiac) origin, we compared the bioimpedance spectra during normal circulation (figure 4, a) and during blood arrest in the arms (figure 4, b). Circulation arrest did not eliminate oscillations of bioimpedance although diminished their amplitude (figure 4, b). Under these conditions, the periodic bioimpedance oscillations could be produced either by neurogenic vasomotor influences or by spontaneous vasomotions. The latter are characterized with a broad range at the low frequencies of 0.008 – 0.11 Hz [4]. The narrow shape of all peaks in Fig. 4 does not favour the hypothesis of spontaneous nature of the corresponding bioimpedance oscillations. Thus, all the peaks in figure 4, b are probably neurogenic and vasomotor in nature.

Arrest of circulation dramatically reduced the cardiac peak, which is a trivial consequence of the fact that during normal circulation the major variations in bioimpedance are produced by pumping action of the heart. Surprisingly, the respiratory peak 2 and Mayer’s peak 1 decreased by no more than 2-fold. It means that during normal circulation, the heart and systemic blood pressure are not the major players who control these rhythmic variations of bioimpedance. During circulation arrest, the
respiratory peak surpassed the cardiac peak in amplitude; moreover, in contrast to the cardiac peak, it was accompanied by the second and third harmonics indicating that neurogenic respiratory oscillations are far more powerful than the cardiac ones.

![Amplitude spectra of bioimpedance variations under normal conditions (a) and during circulation arrest (b). Note different vertical scales.](image)

Combined respiration and circulation arrest resulted in elimination of all peaks except the Mayer’s one, which however decreased 3-fold. This is another surprising observation indicating the major role of respiratory process in sympathetic vasomotor supply, since respiration delay can eliminate neurogenic influences normally observed at the cardiac rate.

4. Discussion
Fourier analysis of bioimpedance variations reflecting blood filling and fluid redistribution in biological tissue can assess activity of periodic neurogenic vasomotor influences both during normal and arrested circulation. During normal circulation, the neurogenic vasomotor supply to human finger is predominantly modulated at Mayer’s and respiratory rate, while the overwhelming part of the heart-rate bioimpedance variations reflects the pumping action of the heart. During arrested circulation, the bioimpedance variations seem to be exclusively neurogenic in nature with the largest contribution of oscillations at the respiration rate. The maximum frequency of bioimpedance variations under these conditions (1.4 Hz, the third respiratory harmonic) can assess the upper limit of frequency range of neurogenic vasomotor control.

The trident structure of cardiac peak and the amplitude of the side peaks can assess the depth of respiratory modulation of the stroke volume. The width of splitting of the higher cardiac harmonics reflects the depth of frequency modulation of the heartbeats by respiratory system. Thus, Fourier analysis of bioimpedance variations seems to be a new powerful tool to examine the regional neurogenic vasomotor influences and central interaction between cardiovascular and respiratory systems.

5. References
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