

FLUCTUATION OF ENERGY OF SPECTRAL COMPONENTS OF THE ECG SIGNAL DURING
AND AFTER CLINICAL DEATH AND IN THE EARLY RESUSCITATION PERIOD

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UDC 616-036.882+616-036.882-008.66]-07:
616.12-008.3-073.97-073.584

KEY WORDS: spectral analysis of the ECG; fluctuations of energy of the power spectrum; diene; clinical death; early resuscitation period

Data of spectral analysis of repetitive physiological processes can provide a highly accurate criterion for evaluating the quality of self-regulating processes and their time course during life [5]. It has been shown [3] that fluctuations of the power spectrum of signals reflecting the cardiac rhythm and blood pressure (BP) provide a description of changes in control of the cardiovascular system associated with various diseases and, in particular, essential hypertension, diabetes, etc. Estimation of an autonomic parameter of the cardiac rhythm in patients with ischemic heart disease (IHD) during the bicycle ergometry test has revealed an inappropriately high level of strain on the regulatory systems with excessive sympathetic provision for physical activity [2]. The attention of research workers also has been drawn to the possibility of spectral analysis of the ECG. For instance, changes in frequency characteristics of the spectrum of the ECG signal have been found in patients with acute respiratory failure [1].

The aim of this investigation was to study fluctuations of the power spectrum of the ECG signal by the fast Fourier transform method during rapid dying, in the period of clinical death, and in the early resuscitation period, in order to find new quantitative methods of evaluating the ECG in these states.

EXPERIMENTAL METHOD

Experiments were carried out on 17 anesthetized (trimeperidine 10 mg/kg, pentobarbital 8 mg/kg) mongrel dogs of both sexes weighing 10-20 kg. In the experiments of series 1 (nine animals) clinical death was caused by acute blood loss from the femoral artery through a catheter. The average duration of the dying process was 9 ± 3 min and of clinical death 7 ± 2 min. The animals were revived by rapid intraarterial injection of the lost blood with 0.2-0.7 ml of 1:10,000 adrenalin solution, artificial ventilation of the lungs (AVL) and, should ventricular fibrillation develop, external cardiac massage and defibrillation. The duration of observation in the postresuscitation period was 2 h. To obtain a more accurate picture of the relationships discovered, the experiments of series 2 were conducted (on eight animals) in the early resuscitation period, in which the circulation was arrested by passage of an alternating current from the main supply system (127 V) through the region of the heart, inducing ventricular fibrillation. Cardiac activity was restored 9 min after circulatory arrest by cardiac massage, AVL, intravenous injection of 0.5-1.0 ml of 1:10,000 adrenalin solution in 20-30 ml polyglucin (dextran), followed by defibrillation.

The ECG was recorded in three orthogonal (Frank's) leads (standard amplification 1 mV) on a "6-NTK" electrocardiograph and "SDR-41" four-channel tape recorder ("Nihon Kohden," Japan). The SB-1-Ts-02 spectrobiograph was used for spectral analysis.

In each derivative 5-7 cardiac cycles were analyzed and two regions of the ECG were processed: the QRS complex and T wave. The amplitude of the frequency peaks was estimated within frequency bands 0.5-5, 5.5-10, 10.5-15 Hz, and over 21 Hz (bands 1, 2, 3, and 4), respectively. To estimate the total power of the spectrum, the amplitude values obtained were averaged and represented by the index Am. Fluctuations of the index Am were estimated as the ratio of the maximal to minimal values (A_{\max}/A_{\min}) obtained by analysis of a chosen time cut of the cardiac cycle (the coefficient of fluctuation at CF).

Institute of General Resuscitation, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR, V. A. Negovskii.) Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 108, No. 11, pp. 531-533, November, 1989. Original article submitted March 24, 1989.

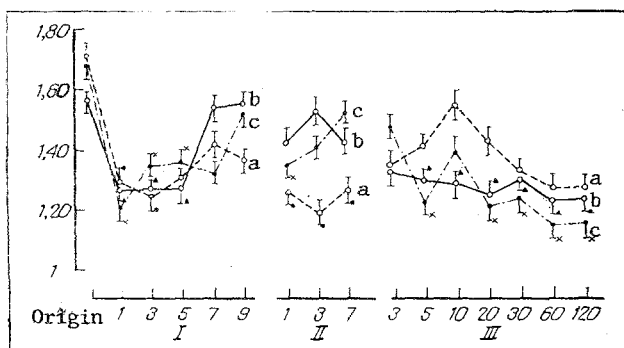


Fig. 1. Time course of CF during the dying process and clinical death and in the early resuscitation period in X, Y, and Z derivations. Abscissa, periods of investigation (in min); ordinate, CF (in min); ordinate, CF (in conventional units); a) X derivation; b) Y; c) Z. $p < 0.05$; I) period of diene; II) of clinical death; III) recovery period.

EXPERIMENTAL RESULTS

Analysis of the observations in the early resuscitation period in the two series of experiments revealed similar changes in the value of CF for the T wave, so that it was possible to combine the results. The character of changes in CF for the QRS complex and T wave in the early recovery period was similar.

Changes in CF in correlated orthogonal derivatives during the dying process and clinical death and in the early recovery period are shown in Fig. 1. The greatest decrease in the value of CF in all derivations analyzed occurred between the 1st and 3rd minutes of dying. By the 7th-9th minutes the value of CF had fallen, although differences from the initial values were not significant.

The period of clinical death was characterized by a significant decrease in CF in the X derivation between the 1st and 7th minutes and in the Z derivation in the 1st minute. By the 3rd minute of clinical death, CF in the Y derivation was higher than in the X derivation.

In the early recovery period CF in the X derivation rose until the 10th minute, but thereafter it fell steadily until 60 min. Meanwhile in the Y and Z derivations no increase in CF was found in the first minutes. Moreover, their values were significantly lower than initially, and remained within these limits until the 120th minute of observation.

Fluctuations of the power characteristic of the spectrum of the ECG signal in corrected orthogonal X, Y, and Z derivations during the dying process and clinical death and in the early recovery period thus revealed quite distinct phases. The first few minutes of dying were characterized by a significant decrease in the Am_{max}/Am_{min} ratio. In the late stages of dying and in the period of clinical death this coefficient increased and came close to its initial value. During resuscitation there was a second fall (like that at the 1st-5th minutes of dying) in the value of CF. Consequently, changes in CF do not coincide with the well-known characteristic periods of hyper- and hypodynamia, which are observed in the early recovery period after clinical death of relatively short duration from acute blood loss.

It can be tentatively suggested that a low CF reflects a process of compensation (in the 1st-5th minutes of dying) and of normalization of functional activity (toward the 60th-120th minute of the recovery period), whereas the increase in CF which was observed toward the end of the dying period is evidence of a breakdown of competition. This is in agreement with the currently expressed view that many pathological processes are characterized by an increase in the degree of orderliness, and not in the degree of chaos, as was hitherto considered [4]. Consequently, assessment of the degree of fluctuation of the energy spectrum of the ECG signal can serve as a sensitive indicator of changes in the mechanisms of regulation of cardiac activity during the dying process and clinical death and in the early recovery period.

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