THE ROLE OF CARDIAC MOVEMENTS IN THE FORMATION OF THE SYSTOLIC BALLISTOCARDIOGRAPHIC COMPLEX

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At the foundation of contemporary concepts pertaining to the origin of the ballistocardiogram (BCG) and the principles of its alterations lie the classic experiments of the American physiologist, Starr, using artificial reproduction of systole in cadavers [10]. Changing the volume and rate of blood expulsion in the aorta and pulmonary artery by means of syringes, the author obtained both the normal ballistocardiographic complexes and the various alterations of their form that are encountered in clinical practice. This proof of the important role played by the blood flow in the genesis of the BCG is so convincing that interpretation of the results of a ballistocardiographic examination is constructed exclusively upon analysis of the forces relative to this flow.

Along with this, experimental facts have accumulated showing that the ballistic waves are retained even after blockading the blood circulation, and their amplitude may be even greater than the original [5, 6, 7, 9]. On this basis, certain investigators do not attribute the main generating role to blood flow, but rather the movements of the heart itself [5, 6]. Others only regard these results as proof of the participation of both factors in the formation of the ballistocardiogram [7, 9]. Developing this point of view, Frederick and Eddleman [7], in experiments on dogs, showed that the ballistic effect of the displaced myocardial mass is proportional to the force of the cardiac contraction, and doubtlessly is summated with the ballistic effect of the displaced blood.

The use of these data in interpreting ballistocardiographic changes in humans is complicated by the fact that neither the effect of the myocardial factor on the formation of the complex, nor the normal relationship of the two origins, have yet been studied. In addition, in analysing the BCG of patients with arteriosclerosis associated with coronary insufficiency, we were convinced that not every form of the curve could be explained exclusively by changes in the forces related to blood flow [2].

EXPERIMENTAL METHODS AND RESULTS

We demonstrated two basic types of changes in the form of the systolic complex. One of these (Fig. 1b) was essentially observed in patients with predominant involvement of the coronary arteries, an unaltered aorta, normal or slightly increased cardiac dimensions, and a normal arterial pressure level. The principal decrease in the initial systolic waves ("fused H–J" or "late negative deflection") and the high level of the respiratory waves are easily explained by the reduced initial rate of blood expulsion by the ventricles.

The other type (Fig 1c) was noted in patients in whom the coronary insufficiency was coupled to hypertension, or where there were manifest atherosclerotic changes in the aorta. The cardiac dimensions in these patients were increased, mainly due to hypertrophy of the left ventricle. However, with significant development of myocardiosclerosis we also observed, as a rule, changes of the first type in these patients.

The form of the K wave, and its shortening, can be explained by shifts in the aortic hemodynamics, and the normal level of the respiratory waves, present in the curve of this type, by the relative increase in the role of the left ventricle. The form of the initial complex, however, cannot be related only to changes in the rate of expulsion.

Its reduction is equivalent for both the stroke force and the force of recoil. However, in curves of this type the corresponding elements change to a varying degree : the J wave is diminished with considerably greater intensity than the I wave, the latter often being the deepest wave of the entire complex.

Considering the hemodynamic characteristics of the patients studied, and the previously indicated data in the literature, we postulated that this group of alterations in the systolic complex is caused by an increased effect from the forces related to movements of the heart, and that the factor determining these interrelationships appears to be hyperfunction of the hypertrophied myocardium.

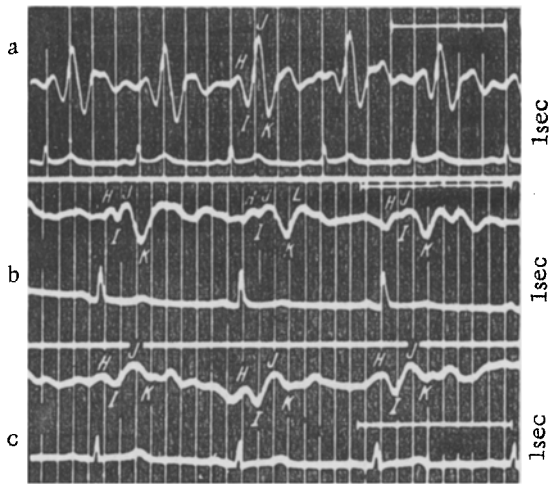

Fig. 1. Changes in the form of the systolic complex. a) Normal BCG; b) first type of change; c) second type of change. In Figs. b and c the EKG lags by 0.04 seconds for technical reasons.

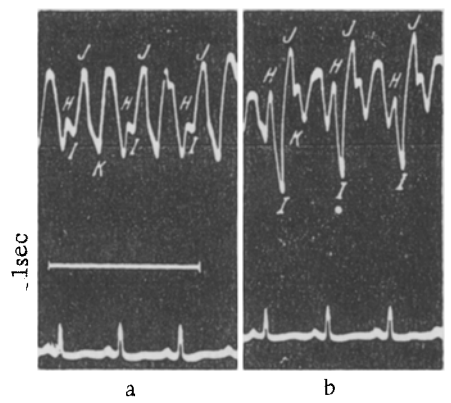

Fig. 2. BCG and EKG following compression of the descending aorta. a) Original tracings; b) after compression. For technical reasons, there is a lag in the EKG of 0.04 seconds.

Significant development of the myocardiosclerosis, and the subsequent reduction of contracting capacity which it causes, apparently removes this factor, thus involving a transition to the ballistocardiographic changes of the first type.

We tested this hypothesis in short term experiments on dogs. The experiments were principally carried out under ether narcosis. In the majority of the animals this was accompanied by incision into the chest cavity and catheterization of the heart and the great vessels. The BCG was recorded by means of an electromagnetic pickup, which was securely fastened to the frontal articulation [3]; during the recording artificial respiration was cut off in the position of expiration. The EKG was simultaneously recorded, and in the majority of the experiments, we also recorded curves for the pressure in the auricles, the aorta and the pulmonary artery in different combinations.

In order to elucidate the effect of cardiac movements on the formation of the systolic complex under conditions of an uninterrupted blood circulation, we increased the individual significance of the forces associated with these movements by repeated mechanical compression of the descending aorta. Marked elevation of the peripheral resistance in this case sets up conditions which lower both the rate of expulsion and the stroke volume; on the other hand, it brings with it an increase in the presystolic dimensions of the heart, and an increase in the force of its contractions [4]. By this means, the magnitude of the systolic volume and the rate of expulsion do not have to be lost, and can be sustained at the original level or decrease, but the forces associated with displacements of the heart are augmented.

As can be seen in Fig. 2, the corresponding changes in BCG are reflected by a marked deepening of the I wave. The J wave remains almost unchanged. Shortening and clefting of the K wave does not appear as a result of the increased role of the cardiac movements, but is related to changes in the properties of the aortic compression chamber, and the speed and volume of the blood flow in the aorta [1, 8].

We attained an approximation of the hemodynamic conditions found in patients with BCG changes of the first type by reducing the force of the cardiac contractions; this was done by ligating the anterior descending coronary artery. It is known that this procedure results in a reduction in the volume and speed of blood expulsion; it also decreases the rapidity of movements of the heart itself. Compensation for the latter disturbance in the coronary blood circulation is accomplished by an increase in the activity of the uninjured myocardium [4]. One of the vital links in this mechanism appears to be an increase in the presystolic dimensions of the heart. Thus, the forces related to

cardiac movements are obviously reduced less intensively than the forces generated by blood expulsion.

Changes in the BCG following ligation of the coronary artery followed this relationship (Fig.3) : waves I and J were significantly reduced, but shortening of the I wave was less intense. In addition, reduction in the original speed of systolic blood expulsion caused a relative deepening of the K wave [1, 10]. The general form of the complex (fused H-J) corresponded to the first type of tracings obtained from investigations on patients (see Fig. 1b).

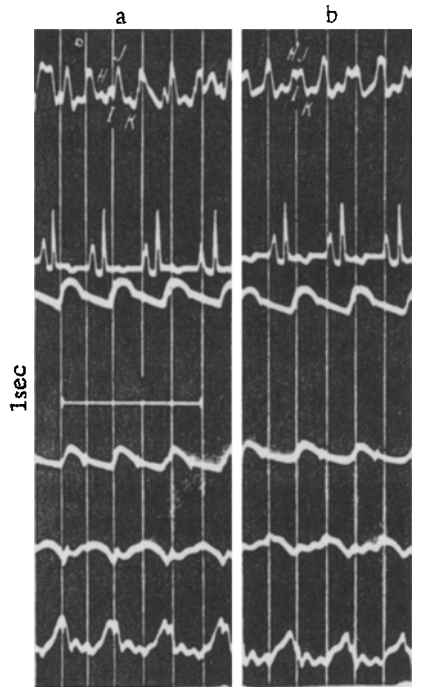

Fig. 3. BCG and EKG following ligation of the coronary artery. a) original curves; b) after ligation. Meaning of the curves (from above downward): BCG; 1st lead of the EKG; pressure in the aorta; pressure in the pulmonary artery; pressure in the left auricle; pressure in the right auricle.

In order to approximate the hemodynamic conditions of patients with associated hypertension, or with manifest atherosclerosis of the aorta, we performed a third series of experiments in which we ligated the coronary artery after placing a clamp on the descending aorta. Elevation of the peripheral resistance in this case played a dual role. On the one hand, it gave impetus for the compensatory mechanisms, providing a relative increase in the forces associated with the cardiac movements, and on the other, it caused an increased load, which, after ligation of the coronary artery, led to accelerated exhaustion of the energy reserves and the compensatory potentials of the myocardium. By thus providing a progressive reduction in the contracting capacity of the cardiac muscle under conditions of compensatory hyperfunction, elevation of the cardiac load played the same role as development of myocardiosclerosis in patients with the second type of BCG changes. In distinction to natural conditions, an important factor was absent in the experiment which would serve to elevate the myocardium's potential for hyperfunction— its hypertrophy. This would seem to be one of the reasons that processes occupying many years developed in the course of several hours in this short term experiment.

In Figure 4 we present the changes in the systolic complex of the BCG, corresponding to the basic stages of the experiment. Compression of the aorta caused a typical change in the amplitude relationships and a rise in the aortic pressure. Subsequent ligation of the coronary artery brought about a lowering of the systolic and pulse pressures in the aorta and a reduction in the amplitude of all waves with preservation of the amplitude relationships present in the BCG with changes of the second type (see Fig. 1c). Progressive lowering of the amplitude of the waves was replaced, after a while, by a predominant reduction in the I and J waves, while the systolic complexes acquired the "fused H-J" form. In two trials, in order to attain this effect, we utilized supplementary reduction of the contracting capacity of the myocardium, achieved by thermal injury of the ventricular walls. This transition to changes of the first type was accompanied by a lowering of the aortic pressure.

Thus, in the last two series of experiments we reproduced the essential hemodynamic conditions of the patients under study, and obtained analogous changes in the form and amplitude of the ballistocardiographic complexes. Also analogous were the conditions for the transition from the second type of changes to the first: in our records the diagnosis of "remission of hypertension in association with progression of atherosclerosis" is encountered only in those patients in which there are manifest sclerotic changes in the hypertrophied myocardium, and where the ballistocardiographic complexes correspond to the first type of changes and are distinguished by low amplitude.

The results of these experiments, in our opinion, suggest that the mean amplitude of the systolic ballistic waves is proportional to the absolute magnitude of the forces arising from cardiac contractions. The form of the systolic complex depends, to a significant degree, on the forces related to blood flow and to movements of the heart itself. Reduction of the forces of both origins, with a small relative increase in the ballistic effect of the cardiac movements, leads to changes of the first type.

A significant increase in the ballistic contribution of the cardiac movements and a reduction in the specific importance of the forces connected with blood flow, leads to changes of the second type, regardless of the absolute magnitude of the acting forces. These relationships depend upon the compensatory hyperfunction, the effectiveness of which may differ, depending upon what state the myocardium is in as regards its contracting function. However, just as with sufficiently effective strengthening of cardiac activity normal levels are attained for the stroke volume and, mainly, the rate of expulsion, so with lowering of these levels (primarily – the initial rate of expulsion) the speed of the cardiac movements is relatively elevated, and the cardiac mass is absolutely increased. Inasmuch as the basis of these force interrelationships for the second type of changes in the systolic complex is compensatory hyperfunction, the transition to the first type is apparently caused by exhaustion of the compensatory potentials within the cardiac muscle, and by a decrease in the speed of the cardiac movements. This conclusion is in agreement with the fact that cardiac insufficiency is most frequently encountered in patients with manifest myocardiosclerosis. Regardless of the clinical characteristics associated, as a rule, with the changes in the BCG of the second type, the systolic complexes of these patients are altered according to the first type, and are distinguished by the lowest amplitude.

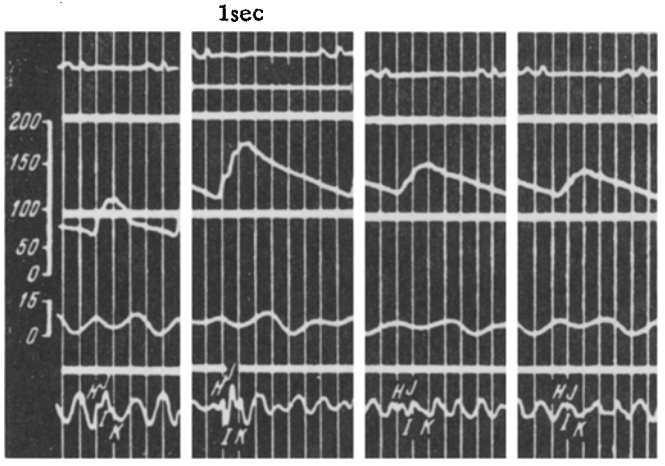

Fig. 4. EKG and BCG following ligation of the coronary artery and elevation of the peripheral resistance. a) Original tracings; b) following compression of the descending aorta; c) following ligation of the coronary artery; d) 1 hour, 20 minutes after the first, and 30 minutes after the second, procedure. Meaning of the tracings (from above downward) : EKG lead; pressure in the aorta; pressure in the left auricle; BCG.

Thus, a change in the systolic complex of the first type may either be the result of lowering the contracting force of a myocardium which is still capable of increasing its mechanical function, or may be due to exhaustion of this capacity. Changes of the second type may be related to a varied degree of strain on the compensatory mechanism, and, with its exhaustion, they change over to the first type. Thus, the question of the significance of these types, especially in a comparative analysis, must be resolved by considering the mean amplitude of the systolic waves and, certainly, must be judged in light of the data from the clinical examination.

The material from these investigations, and the data in the literature, all bear evidence that the form of the systolic complex is always modified according to one of these two types. When there is asynchrony in the cardiac function a serrated or indented form for one or another element is superimposed on the basic contour of the tracing, creating the impression of polymorphism.

The limited ways in which the form of the systolic complex changes is a result of the nonspecific nature of the conditions under which the described types form. Thus, the principles noted are obviously acceptable in analyzing the results of a ballistocardiographic investigation for all conditions.

Interpreting the relative deepening of the I wave as an expression of a relative increase in the ballistic contribution of the cardiac movements is completely in accord with the hemodynamic aspects of the conditions in which this change is encountered (coarctation or thrombosis of the aorta, acute massive hemorrhage, compensated mitral insufficiency, etc.). Depending on the conditions of blood flow in the aorta, in some cases the K wave is simultaneously contracted, while in others it is unchanged (mitral insufficiency). With equal changes in the forces associated with the blood flow and the cardiac movements, the form of the systolic complex remains normal (compensated aortic insufficiency, myxedema).

Supplementing the existing concepts on the genesis and principles of changes in the systolic complex of the BCG, the data in this report gives rise to increased possibilities for appraising the functional state of the heart by using data from the ballistocardiograph.

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All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. *Some or all of this periodical literature may well be available in English translation.* A complete list of the cover-to-cover English translations appears at the back of this issue.
