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HISTORY AND CLINICAL VALUE OF THE BALLISTOCARDIOGRAM - A REVIEW OF CLINICAL AND EXPERIMENTAL RESEARCHES

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SVMMARIVM — Ex aliquot annorum investigationibus perspectum est multo maioris esse ponderis in ballistogrammate efficiendo ea quae cum myocardica contractione connectuntur, quam haemodynamica illa, quae multa sunt, ab Auctoribus permultis considerata.

Ceterum ex clinico hominum haud paucorum examine constat ballisticos complexus non solere secundum certas normas ac rationes in singulis cardiacorum morborum generibus variare, prout apud Auctores scriptum legimus: ea igitur quae ballistocardiographica methodus vocatur parum sane valet ad morbi species coniciendas et agnoscendas.

The origin of ballistocardiography may be traced back to GORDON's [28] studies in 1887, when, for the first time in history, he recorded body oscillations of subjects lying on a bed suspended from the ceiling by cables. Though no attempts at recording had been made so far in the field, the existence of a « motus corporis » consequent to the « motus cordis » described by HARVEY had long been known: in 1786, for instance, PARRY noticed it in a patient whose heart worked at an excited pace. Later on this phenomenon was studied by an increasingly large number of Authors, but only after 1939 BCG studies resulted in a systematic theoretical approach and practical ap-

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plication, after STARR's [63-75] clinical and experimental research.

Even though most of the time we do not agree with this Author's conclusions, we must give full credit to Starr and co-workers — especially in such a work as this, where his ideas are amply criticized — for conducting the first researches on the value and significance of ballistocardiography in normal and pathological cases on the basis of a sound and later universally accepted technique.

Since 1940 BCG research has been increasing remarkably all over the world, and we can hardly give a full account of its developments in this short paper; we shall therefore only mention some monographs, such as those by BROWN et al. (1952) [4], SIBILIA (1953) [61], DOCK et al. (1953) [9], ALVAREZ MENA (1955) [1], MERLEN and DESRUELLES (1956) [46], RAGER (1957) [52], NOORDERGRAF (1957) [50], SCARBOROUGH (1960) [59], JONNART (1960) [35, 36] and the « Seminar of Ballistocardiography » [60] published in 1959 in the « American Journal of Cardiology ».

Concomitant with the development of research on the significance of the ballistocardiogram, to which scientists from different countries brought their contribution, there came a number of studies on the related recording techniques. What was quite unfortunate at the time was the imprudent spreading of often enthusiastic news on the interest found in BCG for diagnostic and prognostic purposes.

The technical equipment most commonly used in BCG research falls into a few basic categories, namely: STARR's non-damping high frequency table; NICKERSON's [48, 49] low-frequency table, with critical damping; the different models of ultra-low-frequency table permitting acceleration, as devised by JONNART, TALBOTT [76], BURGER [5], etc. Finally, we should mention the direct recording techniques, which employ photo-electric, piezo-electric and electromagnetic apparatuses.

The availability of such a wide range of technical aids in the field of research explains why the findings of the different Authors are not always comparable and why the reader is unable to get a clear picture of the situation from the relevant literature.

* * *

The first problem confronting the different Authors when assessing the importance of BCG research, was that of determining the significance attributable to the deflections of the BCG tracing. To this end a common approach has been adopted, especially under the influence of Starr's researches, in order to establish a cause-and-effect relationship between the different stages of the hemodynamic phenomena and the appearance of the ballistic curve.

A first assumption was that the BCG waves are to be related to the amount of blood released by the ventricles at each systole; STARR initially stated that each abnormality of the cardiac discharge curve should match with an abnormal shape of the ballistocardiographic tracing.

At a later date, on the basis of further experimental research, STARR and HAMILTON concluded that BCG shape and width are determined not so much by cardiac output as by the discharge rate and by the acceleration of blood flow due to ventricular systole.

On these grounds STARR explains the genesis of ballistic waves by Newton's action-reaction principle, in the sense that acceleration causes backflow in the sense of movement. A typical example usually mentioned by way of analogy is that of a running vehicle, whose occupants are thrown back at the time of acceleration and forward at braking. The oriign of the individual waves would therefore be as follows:

— the H wave would represent the backflow towards the heart base during isometric systole (HAMILTON [30] or

should be related to the deceleration caused by the contracting atrium in the bloodstream at the level of the venous arches in the neck; in LUISADA's [39] opinion, it is determined by the acceleration of venous flow due to distention of the atrium floor;

— the I wave would be due to the acceleration of the ascending bloodstream in the two arterial trunks (ascending and pulmonary aorta);

— the J wave would represent the reaction to two combined forces, i.e. blood flow deceleration at the level of the aortic arch and of the pulmonary branches plus the acceleration in the ascending aorta;

— the K wave would express deceleration of flow in the abdominal aorta and its branches, or the impact of peripheral resistance as a whole;

— lastly, the so-called diastolic waves L, M, N, O — which some Authors consider as artificial and actually as simple harmonic vibrations, presumably due to interference effects — should be related to the rapid filling of the ventricles and to the hemodynamic phenomena of return circulation.

The first objections to this interpretation were raised around 1954-55, eventually resulting in a reasoned revision of STARR's statements, which prove all the time less adequate to explain the clinical and experimental findings.

In 1954 COSSIO [6] and co-workers investigated BCG modifications due to experimental myocardic lesions in dogs, and came to highly interesting conclusions, particularly as refers to experiments on complete stoppage of blood flow by ligating the superior and inferior vena cava: in these conditions, they actually obtained a well-defined BCG tracing, showing the same number of deflections as a normal BCG. Quite clearly, then, the single deflections cannot reasonably be attributed to hemodynamic phenomena due to blood flow, i.e. to other than

cardiac factors (peripheral resistance, impact of the aortic arch, etc.), for, should we accept this assumption, then we should not see them at work in the absence of cardiac output, as in the case of the dogs with either vena cava ligated.

THOMAS and co-workers (1955) [77] reported similar findings after identical experiments on dogs: they remarked no significant modification in the BCG, but for reduced width of the I-J and J-K intervals in some of the animals and an increase in systolic waves in others; the typical sequence of the ballistic waves was not even disturbed by arterial occlusion (preceding or following venous occlusion).

These experiments evidence that BCG waves are produced as long as the myocardium contracts (independently of blood ejection), while the other factors, about which they had so subtly discussed, have little significance, if at all. These conclusions, strongly adverse to STARR's traditional interpretation, are opposed by HONIG and TANNEY [33]: COSSIO's findings — they claim — are obtained by the direct recording technique, and the phenomena are obviously disturbed by the typical « resonance » of the tissues, particularly of fatty ones. These Authors, however, admit that at least the first of the major BCG waves — which is large in the case of a hypertrophic heart, small in the case of hyposystole, and is still recorded in the absence of venous input, arterial inflow, or even of the circulating liquid — is exclusively due to the contracting heart.

A study by MC-GREGOR and co-workers (1957) [45] quashes the objection to the direct technique: using an extra-low-frequency table (0.2 c.p.s.) they repeated the experiments first conducted by COSSIO and THOMAS with identical results: total occlusion of either vena cava leads to decrease and eventual disappearance of diastolic waves; on the other hand, systolic waves suffer no change, or at times appear even wider.

The data available to us from systematic clinical and experimental observations made by us in the same period,

according to different techniques, also vouchsafe a critical evaluation of the assumptions advanced to explain the genesis of BCG waves.

We used an oscillating table working at a natural high frequency and simultaneously recorded the ECG tracing from a peripheral lead; in several cases the phonocardiogram, humeral and carotid sphygmograms and digital plethysmograms were also taken. Morphological alterations were classed as follows:

1) BCG with notched systolic waves or excessive voltage of the H wave;

2) BCG with gradual decrease of the I wave down to the appearance of Starr's « early M wave »;

3) BCG with decreased voltage in respect of all systolic waves and prevalence of so-called diastolic waves;

4) completely altered BCG.

It is our intention, in this short paper, to submit the results of our recent researches and our conclusions as to the value and limits of ballistocardiography, on the basis of our personal experience [11-25; 40-42; 57-58].

* * *

BCG analysis on 300 clinically healthy subjects and on over 600 cardiopathic patients, presenting different etiological pictures, allowed us to collect the following evidence:

a) individuals aged below 40 almost invariably showed the typical polyphase morphology of a normal BCG, its width varying with the subjects; beyond this age limit, morphological alterations of varying significance are found more or less frequently; only 25% of the subjects aged 60 and over showed a BCG whose morphological appearance was within the accepted limits;

b) morphological alterations of the BCG are frequent in the presence of cardiopathies (valvular insufficiency, myocarditis, coronaropathies, pericarditis, hypertensive conditions in systemic and pulmonary circulation, congenital heart diseases, and particularly in the case of coronaropathies and infarction these alterations, however, do not show a definite and constant relationship with the different types of heart diseases considered; they should rather be related to the functional conditions of cardiac activity, whose deterioration is the cause of their appearance or aggravation.

In healthy subjects and in cardiopathic subjects with complete arhythmia due to atrium fibrillation (30 cases), the duration of ventricular ejection (calculated on the basis of the interval between the I and II tone of the phonocardiogram) was related to the amount and morphology of the ballistic impulse, the latter being expressed as velocity of body displacement.

The results obtained did not point to a correlation between ventricular ejection time and intensity of ballistic impulse.

A systematic BCG investigation carried out on 30 subjects with complete atrioventricular heart-block evidenced the typical polyphase oscillations due to atrial activity, morphologically similar to the ventricular BCG. The deflections recorded amount to 20-40% of ventricular deflections for half the time taken by these. Because of the different timing, the apex of the I wave in the atrial BCG maintains a constant distance from the origin of the P wave in the ECG, and the time interval between the electrical and the mechanical phenomenon varies from 0.12″ to 0.15″. The existence of an atrial BCG, morphologically similar to the ventricular one, contrasts with the hypothesis that the conditions of ventricular ejection and the deceleration of blood flow in the larger vessels have any bearing on the genesis of BCG waves; we can actually exclude any relationship between atrial BCG and blood inflow into the arterial vessels; nor could we explain otherwise how movem-

ents in a direction apposite to blood flow (during atrial and ventricular systole) could determine such a morphological pattern; it also appears unlikely that venous backflow from the two auricular cavities may produce an impulse of such magnitude as to cause body movement, as some Authors (DE LALLA [8], JONNART) uphold.

The systematic study of ballistocardiograms in connection with the *various types of extrasystole* was found particularly interesting to understand the genesis of ballistic waves; our observations on 100 subjects allowed us to conclude that there is no constant relationship intensity of peripheral pulse and amplitude of extrasystolic ballistic oscillations; in some cases only the sphygmogram and the BCG show an identical amplitude, while quite frequently an ample ballistic impulse corresponds to early extrasystole (with non-recordable peripheral pulse); on the other hand, a series of very low ballistic waves contrasts with a wide sphygmogram. This clearly evidences the important influence of peripheral hemodynamic factors on the amplitude of ballistic oscillations and the variability of the ballistic impulse, wherears the mechanical phases of ventricular systole vary during extrasystole to a more or less significant but uniform extent.

The BCG tracings of 15 healthy subjects taken before, during and after partial exclusion of the vascular bed by compressing the vessels of the lower extremities, were found useful to determine whether a definite relationship exists between ballistic waves and blood flow in the vascular system.

We were able to note that the ballistic curve shows scarce or no alterations during the modifications of the vascular bed and of peripheral resistance (due to compression at the groins) as well as during modification of blood flow back to the right auricle occurring in the hyperemic phase following decompression.

The alteration of the ballistic tracing in drug-injected men and animals. Nicotinic acid administered intravenously prod-

uces general dilatation of arterial and capillary vessels as well as faster circulation, but does not modify BCG morphology and voltage. On the contrary, during peak action of positively tonotropic and inotropic drugs (e.g. adrenalin, veritol) an overall BCG increase, not matched by a corresponding increase of the K wave, was noted in most cases, in spite of more intense compression. Hypotensive drugs (acetylcholine, hexamethone, tetramethylammonium, prostigmin) do not induce significant or constant morphological modifications in the BCG of both experimental animals and normal subjects.

By temporarily occluding the pulmonary artery in man by means of CONDORELLI's catheter ending in a small inflatable balloon, we can study the effects of right ventricle activity on BCG morphology under these conditions. No alteration of the ballistic waves is remarked during insufflation and stenosis as well as in subsequent relaxation. Nor is any result obtained in dogs by directly causing stenosis of the pulmonary aorta or by ligating the abdominal aorta: the BCG remains actually morphologically unchanged or presents negligible voltage variations.

As a last point, *researches* were also conducted on *Amphibians* (Bufo vulgaris): the recordings of the movements of the animal and of its isolated heart on an appropriate high-frequency micro-table showed that also in this case the polyphase ballistic tracing obtained in connection with ventricular systole does not differ substantially from the pattern observed in upper class animals, despite the fact that the circulatory system in Bufo is quite other than in Mammals (cor triloculare monoventricolosum, symmetrical aortas, etc.). If, then, we open the thoracic cage and cut the vascular peduncle, we find that cardiac activity without any release of circulating blood does not cause any change in BCG morphology and voltage as long as heart contractions persist. Also the *isolated heart* (which, when enclosed in a rubber envelope containing Ringer's

amphibian liquid, goes on pulsating for a long time) produces ballistic oscillations similar to those emanating from the whole animal.

* * *

All these clinical and experimental observations substantiate our statement that, more than to the other hemodinamic factors considered so far, prevailing importance should be attributed to the ballistic effects connected to heart contraction in the genesis of BCG waves.

In view of the foregoing one can easily understand the difficulty of interpretation, the momentary hopes and constant disappointments involved in the different attempts at a practical application of BCG techniques: the inadequacy of the method to supply reliable data for diagnostic and prognostic use is fully apparent in the clinical study of the patients.

It is by now an accepted fact that no heart disease has a characteristic BCG pattern of its own. This is the truly disheartening conclusion reached after so many studies, although a few Authors today still refuse to accept it as final.

It is significant that the ballistocardiogram can have a normal shape or show no pathognomic alterations even in cardiopathic patients with circulatory troubles (shunts, obstacles to circulation, valvular insufficiency, etc.) which — in the opinion of Starr and others should produce the most marked irregularities. Also the decreased amplitude and, in case, actual flattening of the K wave are not constantly (PRATI et al. [51]) associated with occlusion at any level of the aorta (BROWN et al.; NICKERSON; HEIM DE BALZAC [31]; RAGER): this occurrence is, in fact, observed also in connection with other phenomena (DONOSO et al.) [10].

The warm hopes initially laid in BCG as a valuable tool for an early detection of coronary diseases have been proved unfounded by clinical experience. It has been stated that BCG alterations can be observed when the ECG offers not the

slightest indication of myocardial injury — a very important fact for establishing a diagnosis, as everyone will readily appreciate. The problem is, however, that this does not always come to pass, that these alterations do not manifest themselves according to a unique pattern, and can in any case be detected even in healthy subjects.

To conclude, ballistocardiography, at the present state of things, has hardly any clinical significance: it is in fact our view that no definite interpretation can be given of any abnormal shape or amplitude of BCG waves: constant alterations are found only in connection with gross damage to myocardial contracting ability, resulting in phenomena of circulatory insufficiency, irrespective of the specific heart disease observed in the patient.

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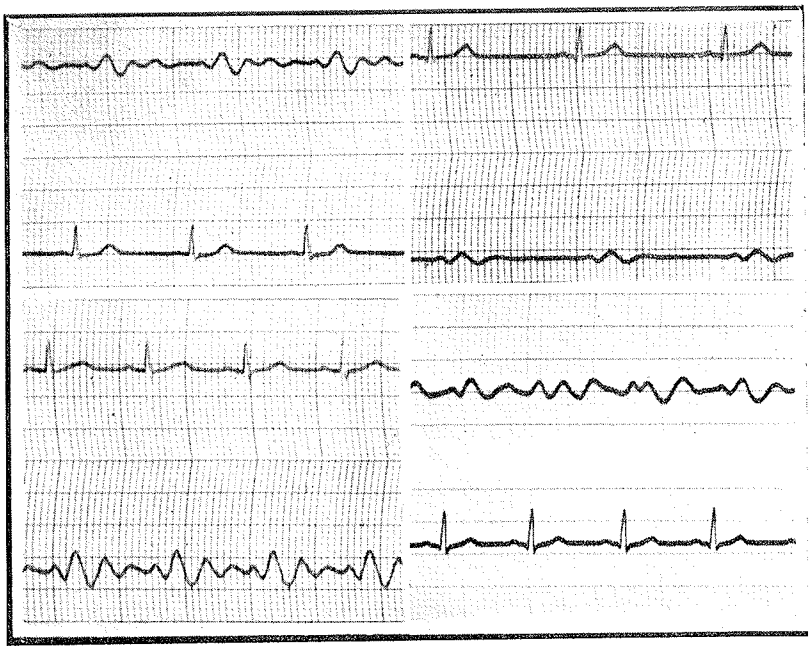

FIG. 1 — Bcg of a healthy subject. Left to right and up to down: Normotype 30 y.o., normal bcg. Brachytype 30 y.o., normal bcg. Athlete 32 y.o., normal bcg. Normotype 27 y.o. suffering from respiratory changes.

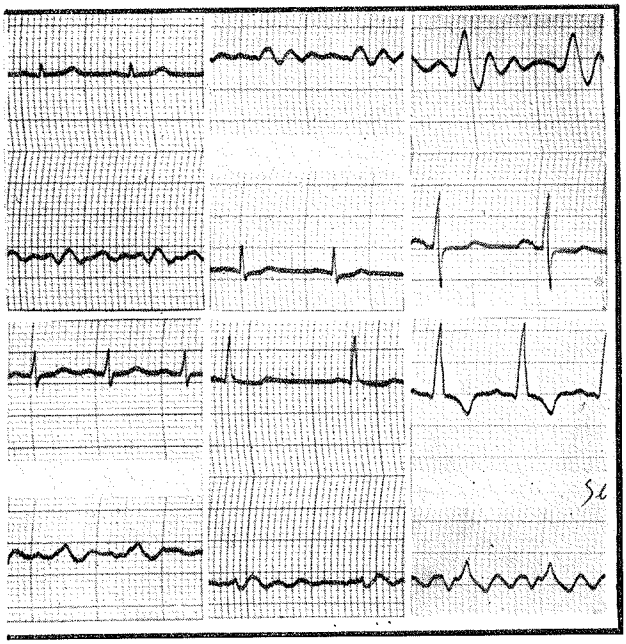

FIG. 2 — Bcg of cardiac patients. Left to right and up to down. - S.C., 24 y.o., mitralic stenosis, bcg within the limits of normality. - G.R. 39 y.o., mitro-aortic vice, bcg slightly changed. - P.A. 54 y.o., aortic insufficiency, normal bcg, but high voltage. - S.R. 32 y.o., mitralic stenosis with signs of circulatory insufficiency, bcg altered. - R.I. 44 y.o., aortic insufficiency together with signs of circulatory insufficiency and bcg changes.

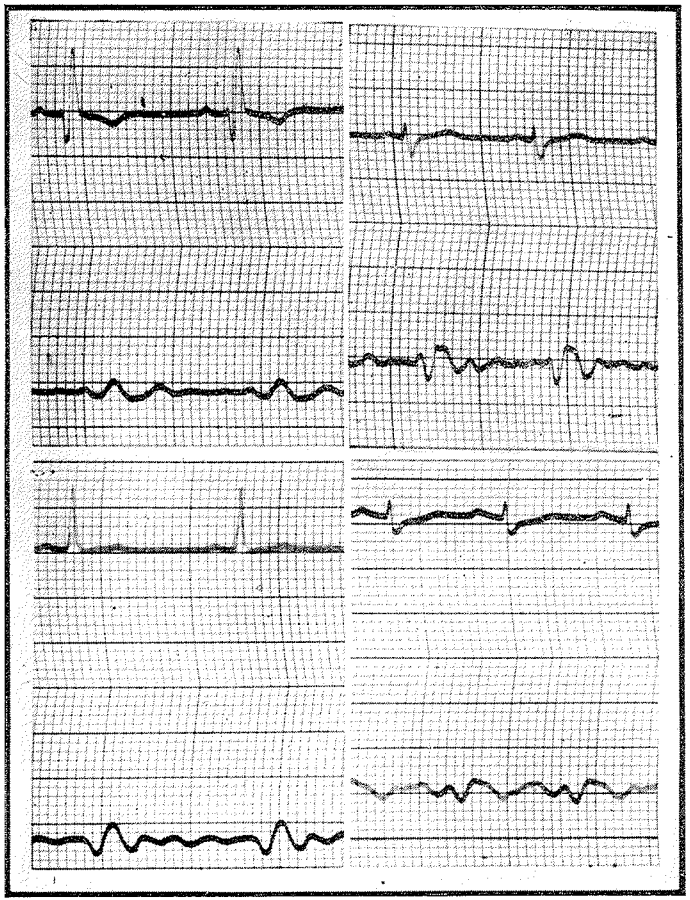

FIG. 3 — Bcg of cardiac patients. Left to right and up to down. C.R. 27 y.o. Fallot's tetralogy, bcg at the limits of normality. - S.B. 9 y.o. Fallot's tetralogy with signs of circulatory insufficiency and changes of bcg. - C.B. 22 y.o. stenosis of aortic isthmus, bcg at the limits of the normality. - V.L. 18 y.o., stenosis of the tricuspid valve with signs of circulatory insufficiency and changes of bcg.

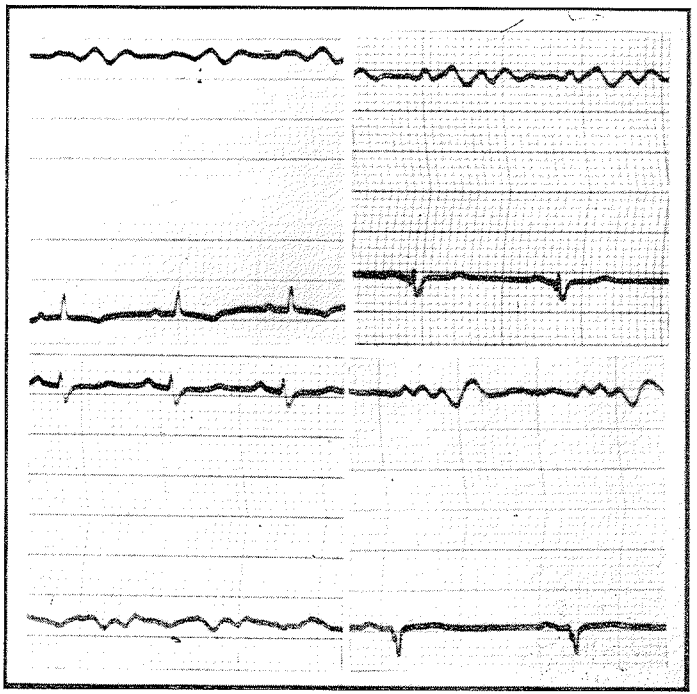

FIG. 4 — Bcg of cardiac patients. Left to right and up to down. L.M. 38 y.o., chronic pericarditis, bcg slightly changed. - R.T. 48 y.o., antero-lateral infarction; light changes in bcg. - P.S. 15 y.o., chronic pericarditis with signs of circulatory insufficiency changes of the bcg. - M.S. 45 y.o., anterior infarction, bcg changed.

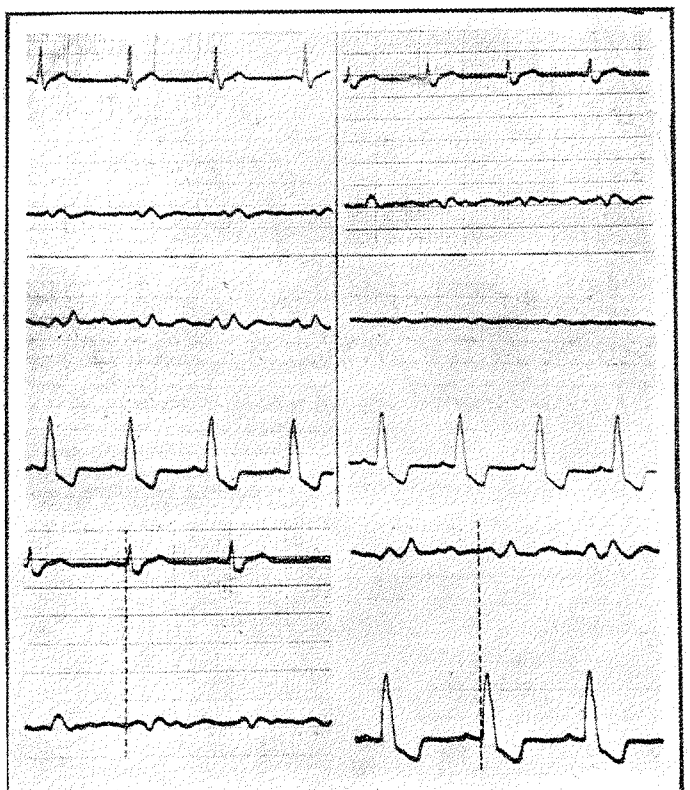

FIG. 5 — Bcg in troubles of intraventricular right and left conduction. Absence of parallelism between ECG and BCG changes; the last has been founded altered only in patients suffering from circulatory insufficiency.

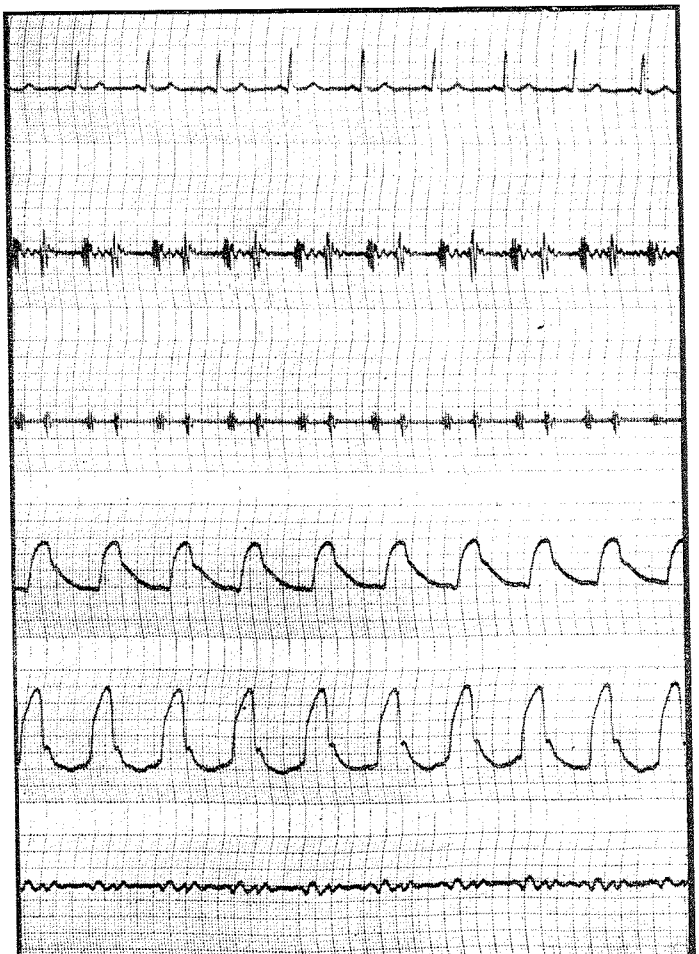

FIG. 6 — Chronological relations among ECG, phonocardiogram, arterial pulse, BCG.

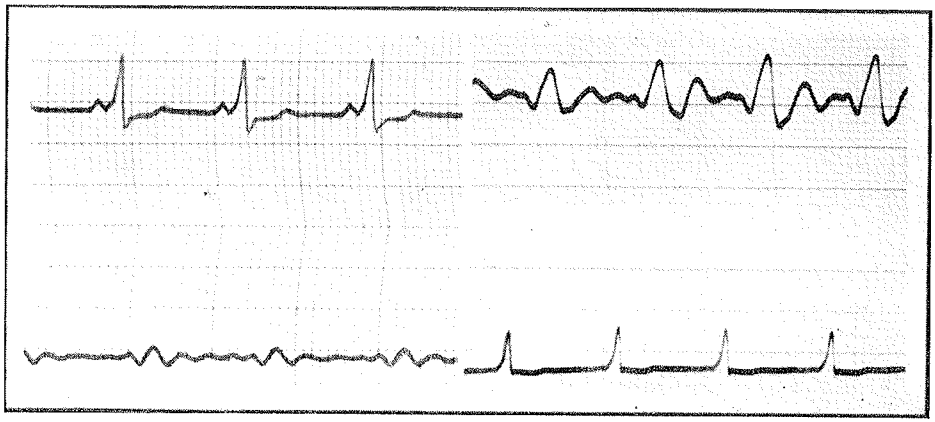

FIG. 7 — Wolf-Parkinson-White syndrome. No morfological changes of the bcg.

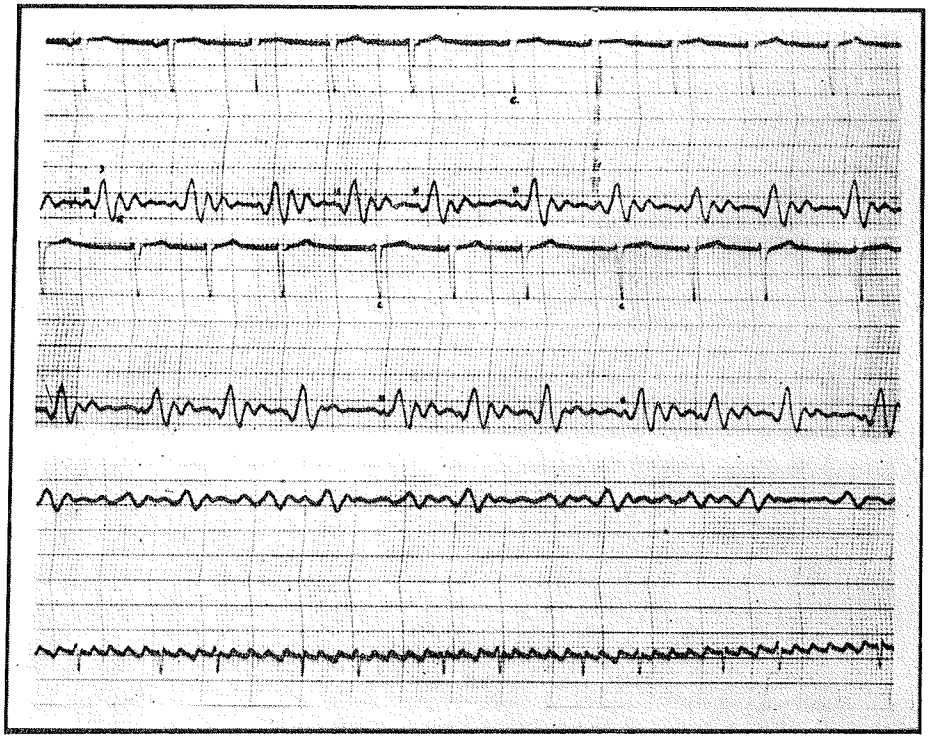

FIG. 8 — Atrial flutter. BCG within the limits of normal, showing changeable amplitude according with the duration of the heart cycle.

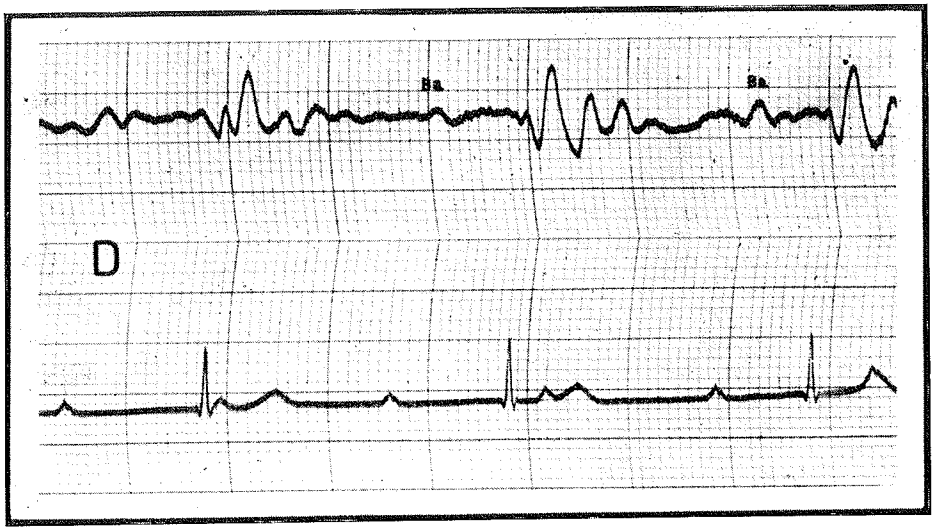

FIG. 9 — Atrial bcg in atrio-ventricular blocks.

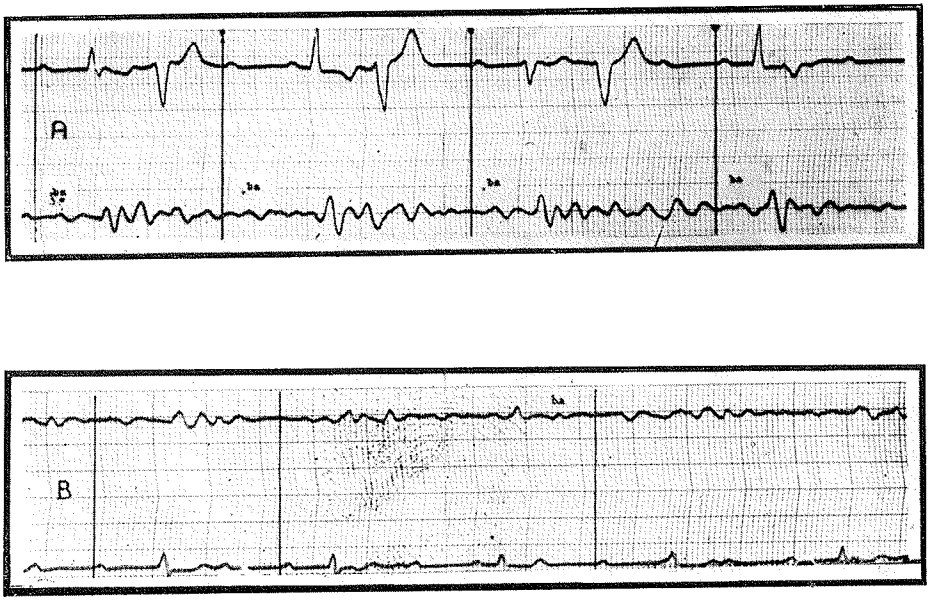

FIG. 10 — Atrial bcg in atrio-ventricular blocks.

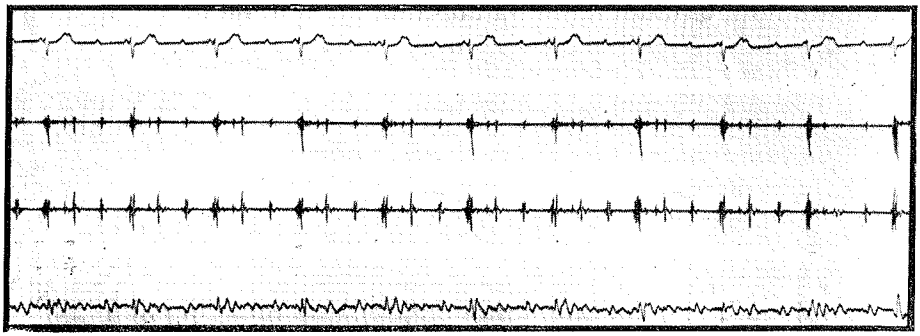

FIG. 11 — Atrio-ventricular dissociation. Incostant increase of bcg corresponding to the stroke of gun.

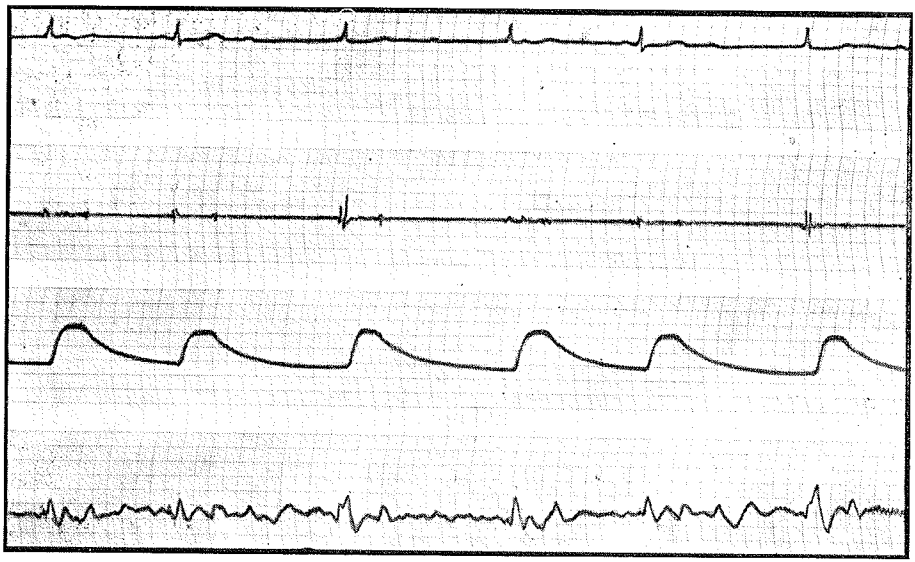

FIG. 12 — Atrio-ventricular dissociation. Incostant increase of bcg corresponding to the stroke of gun.

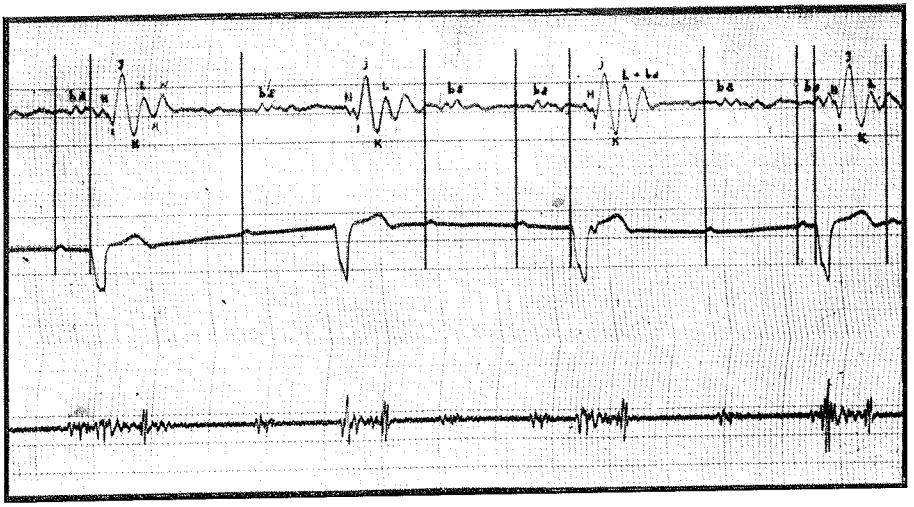

FIG. 13 — Whole atrio-ventricular dissociation; atrial bcg.

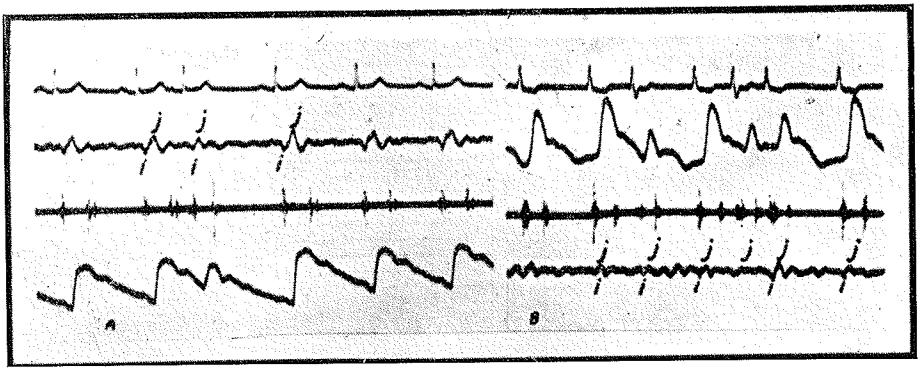

FIG. 14 — A) Supra-ventricular extra-systoles with sphygmogram and balistic pulse reduced in width. B) Ventricular extra-systoles in a patient suffering from atrial fibrillation. No connections between the sphygmogram and the balistic pulse.

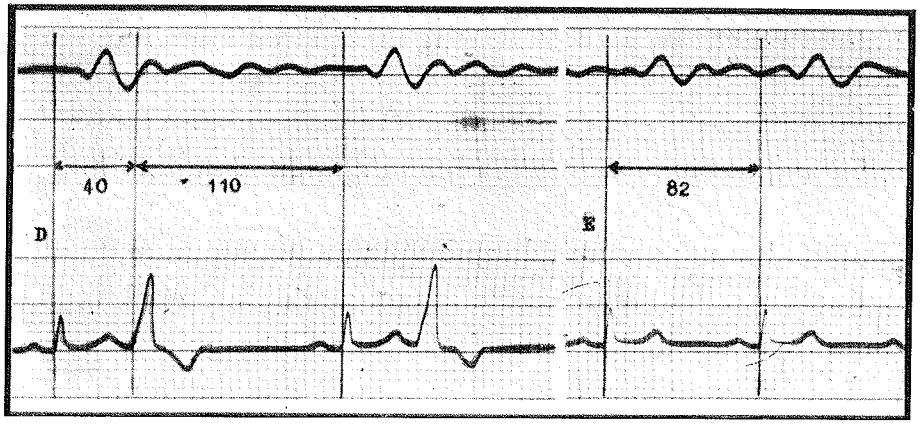

FIG. 15 — On the left: bigeminism with an extra-systolic unapparent bcg. On the right: same cases following a spontaneous vanishing of the bigeminism.

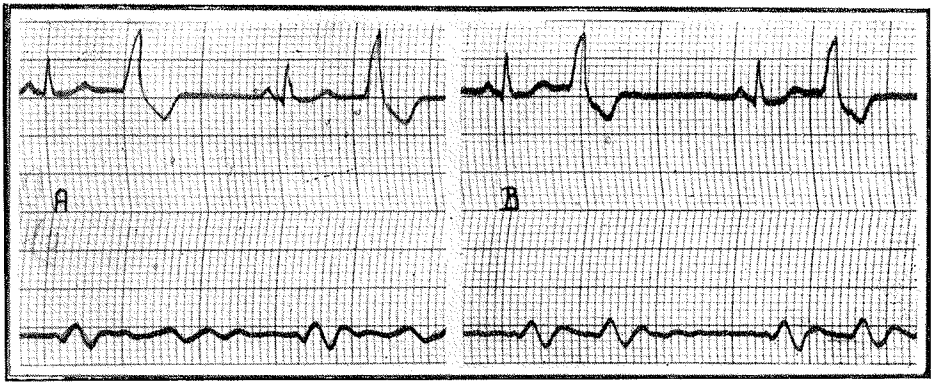

FIG. 16 — On the left: extra-systolic bigeminism with bcg reduced in width. On the right: same case following administration of epinephryne; large increase of amplitude of extra-systolic bcg.

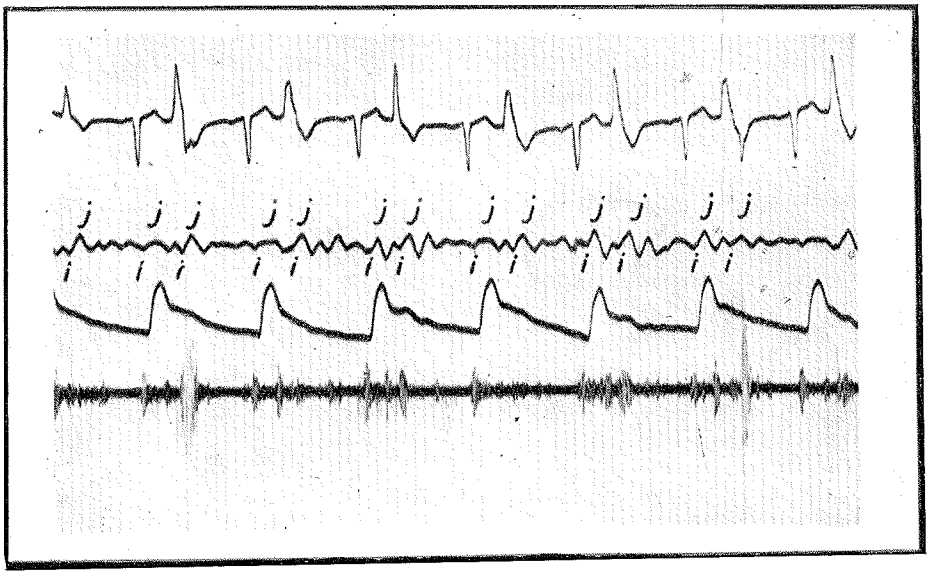

FIG. 17 — Connections between sphygmogram and balistic pulse in extra-systoles; ventricular extra-systoles with invisible sphygmogram and wide balistic pulse.

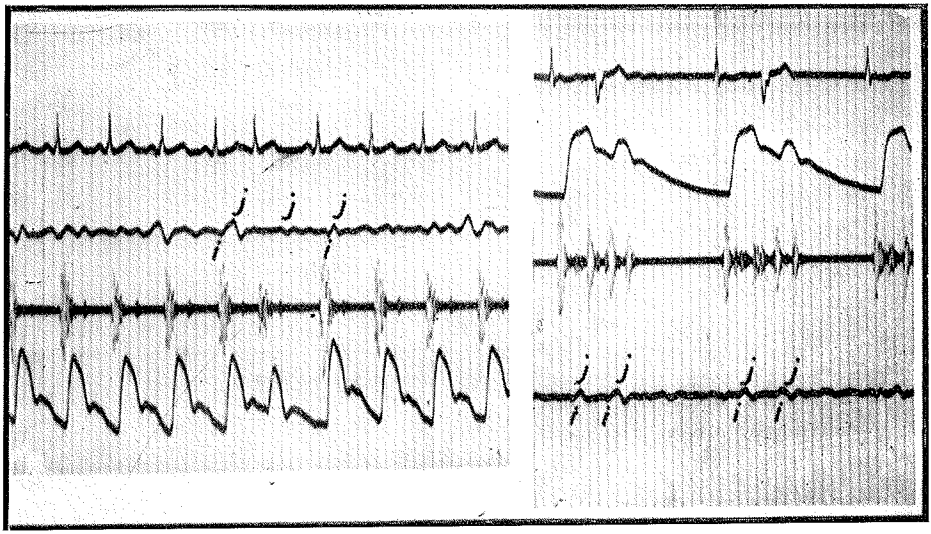

FIG. 18 — Left: supraventricular extra-systoles with small bcg and wide humeral sphygmogram. Right: ventricular extra-systoles with bcg wider than that of the former systole connected with a smaller humeral sphygmogram.

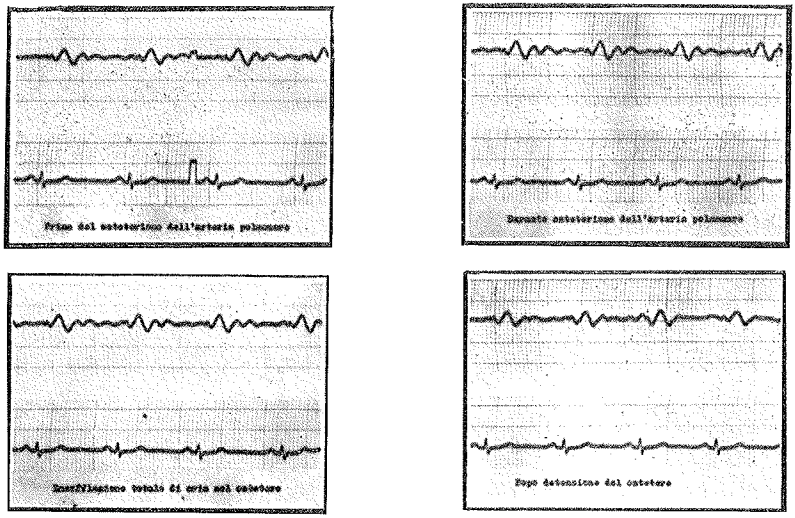

FIG. 19 — Lack of changes in bcg during and after temporary stenosis of pulmonary artery in human subjects.

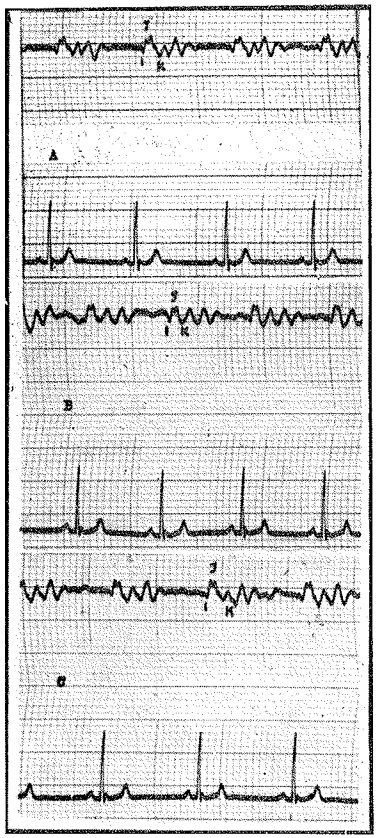

FIG. 20 — Lack of changes of bcg during stenosis of abdominal aorta in dog.

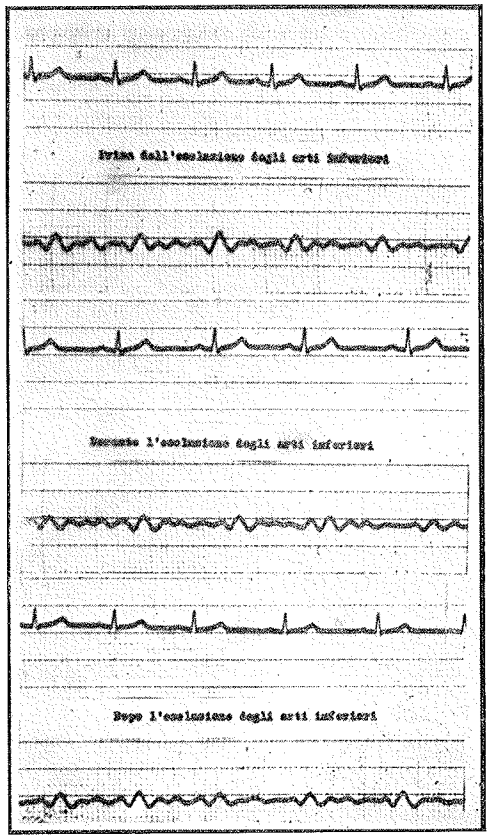

FIG. 21 — Lack of changes of bcg during and after the exclusion of lower limbs circulation in man.

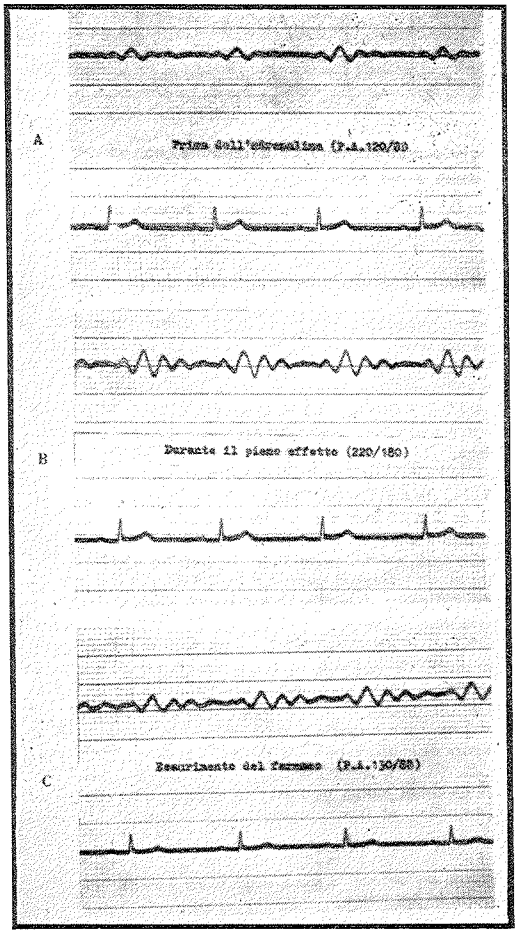

FIG. 22 — Bcg during epinephryne administration in man. Increasing voltage of bcg during the apical effect of the drug.

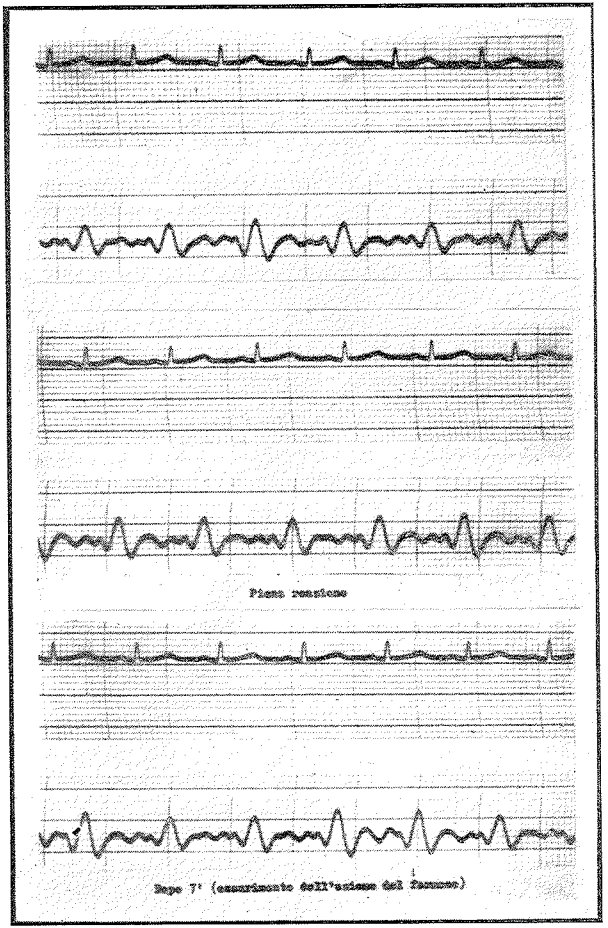

FIG. 23 — Bcg during administration of nicotinic acid in man; lack of morphological changes even during the apical effect of the drug.

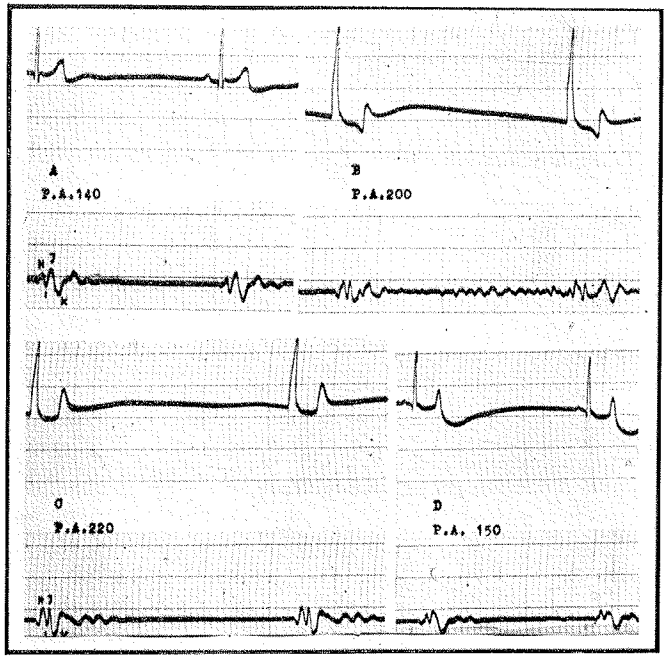

FIG. 24 — Bcg during nor-epinephryine administration in dog; lack of changes during the apical pharmacological effect.

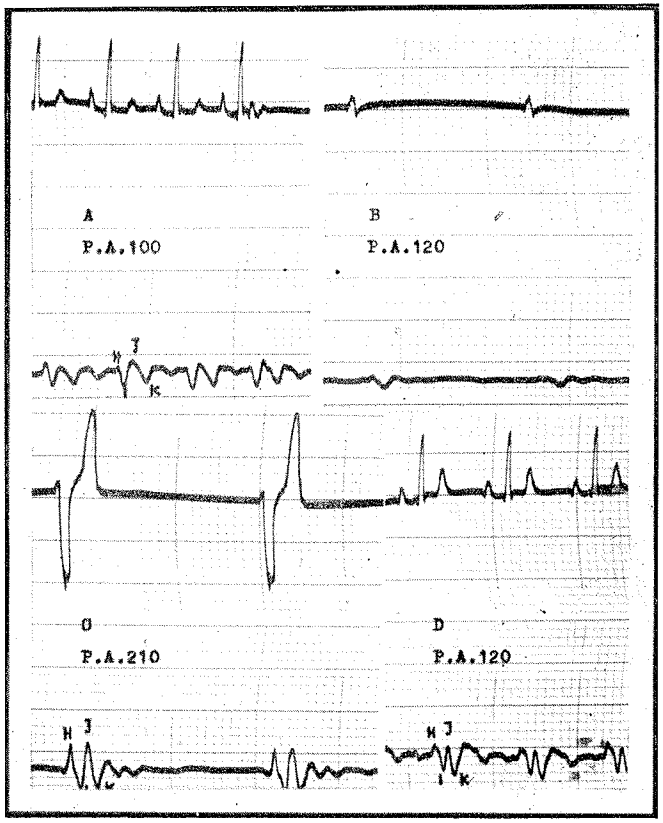

FIG. 25 — Bcg during veritol administration in dog; consisting voltage increase during the apical pharmacological effect.

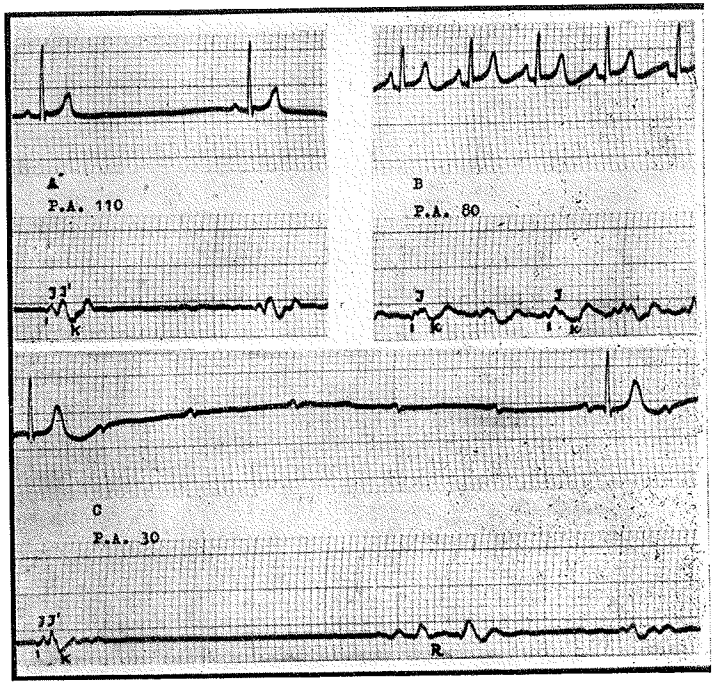

FIG. 26 — Bcg during acetilcoline administration in dog; lack of changes even during the apical pharmacological effect.

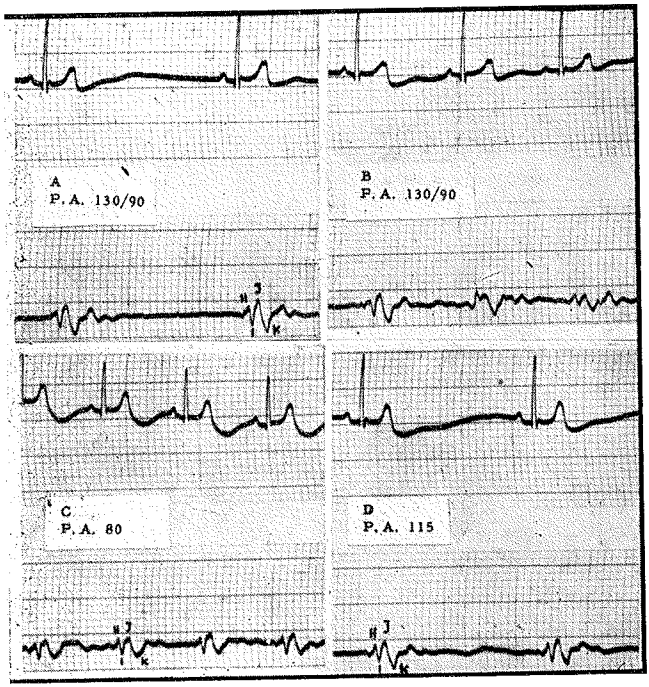

FIG. 27 — Bcg during prostgmine administration in dog; lack of changes even during the apical pharmacological effect.

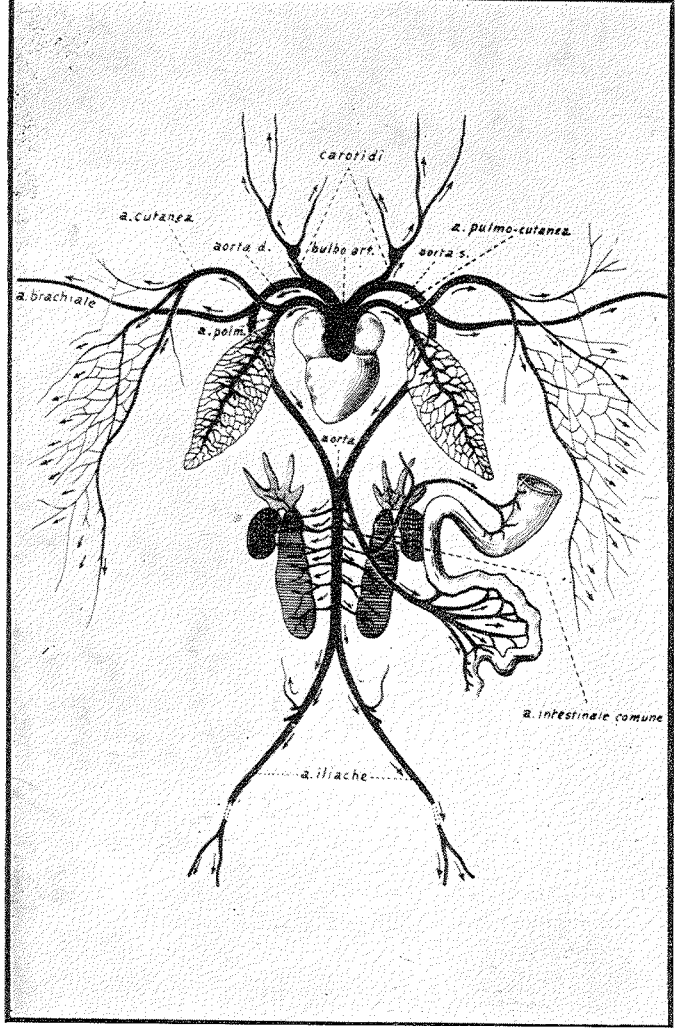

FIG. 28 — Circulatory system in amphibians.

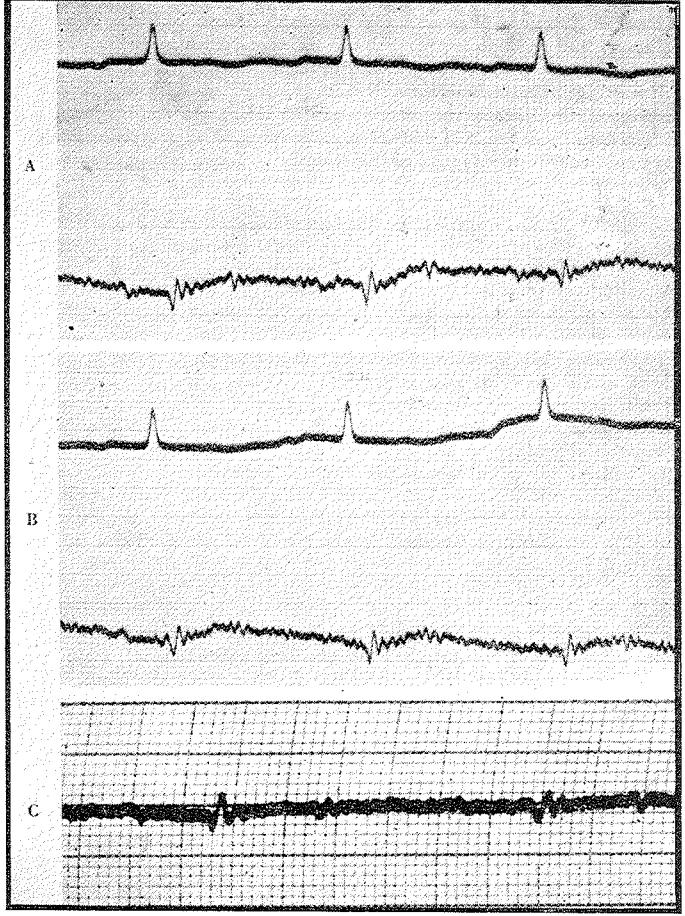

FIG. 29 — Bcg in amphibians. A) Bcg of the whole animal. B) Lack of changes of bcg after cutting of vascular peduncle. C) Bcg of isolated heart, the morphology of which is analogous to that of the whole animal.