BALLISTOCARDIOGRAPHIC OBSERVATIONS

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Although it has long been known that body movement occurs with each heart beat (Gordon, 1877), and in spite of great progress during recent years, the value of ballistocardiography as a diagnostic aid remains uncertain. The object of the present investigation was to assess its clinical value utilizing a direct body-type displacement ballistocardiograph. In addition, the effects of the inhalation of amyl nitrite upon the pattern IJ stroke of the ballistocardiogram was studied in normal subjects and in a group suffering from coronary artery disease.

METHOD AND MATERIAL

All the ballistic tracings were recorded by means of the Sanborn photoelectric ballistocardiograph, a commercially developed instrument, consisting essentially of a cross-bar placed across the patient's shins, into which has been built an optical system. This mobile portion of the apparatus emits a rectangular field of light, which is received by a stationary photoelectric cell, resting on a fixed table. Fluctuations in the field of light are registered by the photosensitive element of the cell, the voltage generated being proportionate to the amount of light impinging on the cell. The output of the cell is connected to an amplifier type of direct writing electrocardiograph. A pure displacement ballistocardiogram is therefore recorded. The only physical connection between the crossbar and cell is the beam of light which does not introduce compliance effects (Rappaport *et al.*, 1953). A filter system is attached to the photoelectric cell.

Three hundred ballistocardiograms were recorded on 75 persons. This series is composed of three groups each of 25 subjects, comprising healthy adults under the age of 30 years, healthy adults between the ages of 40 and 65, and patients with proven coronary artery disease between the ages of 38 and 70. Of the healthy controls a few were patients referred to the hospital and found to have normal cardiovascular systems, the remainder being members of the hospital staff. The subjects judged to have coronary artery disease all had a complete study by the Department of Cardiology of the Royal Infirmary. All patients with evidence of congestive heart failure, left ventricular failure, or somatic tremor, and any who had suffered myocardial infarction within the previous two months were excluded from this series. There were 15 women among the young controls, 5 in the older normals, and 6 in the coronary artery group. Eight of the 25 subjects with coronary artery disease had suffered myocardial infarction from ten weeks to five years previously and three had a raised blood pressure.

With few exceptions the subjects came to the laboratory two hours after the last meal. Ballistocardiograms were recorded as soon as the patient was connected up to the apparatus, this varying from 2 to 7 minutes after lying down. All had, however, been sitting in the department for at least 15 minutes before the first record. Records were taken during quiet respiration followed immediately by graphs with respiration suspended in expiration and then inspiration. Records

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were taken with the filter *in* and *out*. Six leads were therefore available for analysis after each tracing. The blood pressure in the right arm was then recorded. This procedure was repeated after the subject had been lying down for 15 minutes and again at 30 minutes. A fourth ballistocardiogram was then recorded during and immediately after the inhalation of a 3-minim capsule of amyl nitrite. None of the patients had experienced the effects of amyl nitrite before and all recordings were made with the subjects lying in the horizontal position. As a further control 6 normal young persons were subjected to the identical procedure two months after their first record.

Although all the subjects had had the effects of amyl nitrite explained to them before the first record the original method was modified for 6 healthy young persons. After their records had become "basal" the effects of amyl nitrite were re-explained and a series of tracings, including blood pressure estimations, were immediately recorded. Five minutes after this emotional stimulus a second ballistocardiogram was recorded. As a further control this procedure was followed 5 minutes later by the inhalation from an empty capsule, the subjects believing it to contain amyl nitrite. Five minutes later a further resting ballistocardiogram was taken and this was followed finally by a record taken during and immediately after the inhalation of a capsule containing amyl nitrite.

Standard lead II of the electrocardiogram was simultaneously recorded to identify and time the ballistic waves. To allow for possible Valsalva and Mueller effects the expiratory and inspiratory results were always compared with the records taken during quiet respiration. The beam of light generated from the optical system was kept constant, the electrocardiogram standardization was fixed at 1 cm.=1 MV., paper speed was 25 mm. per second and all measurements were expressed in either millimetres or seconds. The nomenclature adopted was that suggested in the first report of the Committee on Ballistocardiographic Terminology (Starr *et al.*, 1953), and the analysis of each ballistocardiogram followed a modification of the scheme suggested by Brown *et al.* (1952).

In order to measure the IJ stroke the H wave is first identified. This is a positive deflection coinciding with, or immediately following, the electrocardiographic QRS complex. The negative or footward deflection following the H is the I wave and the subsequent positive deflection is the J wave. The distance between the depth of I and the summit of J represents the IJ stroke. This is illustrated diagrammatically in Fig. 1a.

FIG. 1.—Diagrammatic representation of a typical ballistocardiogram occurring in (a) normal subjects and (b) patients with coronary artery disease. Both diagrams are twice the natural size and have been drawn from actual records to illustrate the striking ballistic difference that exists between the two groups.

RESULTS

Group A. Twenty-five Normal Subjects under 30 years of age. In this group the inhalation of amyl nitrite produced a striking increase in the height of the IJ stroke in tracings taken with respiration suspended in expiration, and an even more highly significant increase when respiration was suspended in inspiration. A marked increase in heart rate and a slight but significant rise in both the systolic and diastolic blood pressures also occurred (Table I). The IJ values and heart rate increased in every instance, the maximum

TABLE I
BALLISTOCARDIOGRAPHIC CHANGES DUE TO INHALATION OF AMYL NITRITE

	Group A				Group B				Group C			
	Before amyl nitrite		After amyl nitrite		Before amyl nitrite		After amyl nitrite		Before amyl nitrite		After amyl nitrite	
	Mean	S.D. of mean	Mean	S.D. of mean	Mean	S.D. of mean	Mean	S.D. of mean	Mean	S.D. of mean	Mean	S.D. of mean
Heart rate	72.6	± 8.6	126	± 14.9	70.6	± 9.6	106.7	± 17.2	70.4	± 10.8	93.2	± 14.0
Systolic B.P. ..	118.8	± 11.1	125.6	± 13.7	127.7	± 15.4	131.5	± 18.6	137.5	± 26.1	137.4	± 29.7
Diastolic B.P. ..	69.6	± 7.3	75.9	± 10.3	77.8	± 11.7	81.8	± 11.8	82.9	± 15.7	84.4	± 17.5
Expiratory IJ stroke in mm. ..	4.02	± 1.68	6.56	± 2.83	4.19	± 2.84	5.52	± 2.87	2.36	± 1.01	2.62	± 1.46
Inspiratory IJ stroke in mm. ..	3.92	± 1.75	7.68	± 3.43	5.04	± 3.67	6.69	± 3.20	3.3	± 1.76	4.12	± 2.47

Group A consists of healthy subjects aged thirty or less, Group B consists of normal persons aged 40–65 years, and Group C patients with proven coronary artery disease. Each group consists of 25 subjects. One set of IJ readings was omitted from Group B because of somatic tremor.

risers being from 6 to 11 mm. (expiratory IJ), 6 to 13.5 mm. (inspiratory IJ) and heart rate from 73 to 158 a minute. The systolic blood pressure increased in 19 subjects, remained unchanged in two and fell in four. The range was +38 to –6 mm. Hg. The diastolic pressure rose in 19 instances, did not change in three and fell in three, the range being +30 to –10 mm. Hg. In all tracings the K wave and subsequent diastolic waves showed a similar increase in depth after the administration of amyl nitrite, illustrated in Fig. 2.

FIG. 2.—The effect of amyl nitrite on the ballistocardiogram in a young normal subject. (a) Basal expiratory record: heart rate 63 a minute, B.P. 94/60. (b) Basal inspiratory record: heart rate 60 a minute. (c) Expiratory record after amyl nitrite: heart rate 112 a minute, B.P. 110/60. (d) Inspiratory record after amyl nitrite: heart rate 100 a minute. In both (c) and (d) all the visible ballistic waves have increased in amplitude. The ST/T wave changes on the electrocardiograms are unusual.

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The filter *out* results are not published in detail for they were considered too variable to be of scientific value. The main differences between filter *in* and *out* tracings were increased amplitude of all component waves, a greater degree of weaving of the base line and a tendency to an increased HK time with the filter *out*. In a hundred consecutive normal records the HK time was greater with the filter *out* in 75 instances, equal in 20 and greater with the filter *in* in 5 instances. The HK time ranged from 0.18 to 0.38 sec. with the filter *out* and 0.16–0.32 sec. with the filter *in*. The filter *out* values were greater by 0.02–0.16 sec. (mean 0.035 sec.). When the filter *in* values were greater the corresponding figures were 0.02–0.06 sec. (mean 0.036 sec.).

In none of the young normals was any qualitative abnormality detected, although in many instances, both before and after amyl nitrite, the IJ stroke varied slightly from beat to beat in the same lead, irrespective of the phase of respiration and with a constant heart rate and P–R interval.

In the 6 normal subjects who had the identical procedure repeated two months later the results showed no increase in mean blood pressures, either systolic or diastolic, a lesser IJ and a similar pulse rate response to amyl nitrite as compared with the first series (Table II).

In the 6 subjects who were tested successively under emotional strain, with a placebo, and with amyl nitrite, the emotional strain caused no change of note, the placebo caused a slight increase in heart rate, and the amyl nitrite caused a highly significant increase in heart rate and some IJ increase (Table III).

TABLE II
BALLISTOCARDIOGRAMS AFTER A SECOND INHALATION OF AMYL NITRITE IN SIX NORMAL SUBJECTS

	First Inhalation				Second Inhalation			
	Before amyl nitrite		After amyl nitrite		Before amyl nitrite		After amyl nitrite	
	Mean	S.D. of mean	Mean	S.D. of mean	Mean	S.D. of mean	Mean	S.D. of mean
Heart rate	67.3	±5.96	131.3	±11.7	66.3	± 6.86	127.0	±17.6
Systolic B.P. .. .	122.0	±8.67	134.0	±14.0	115.0	±13.5	119.6	±14.4
Diastolic B.P. .. .	72.3	±7.09	77.6	± 5.4	66.6	± 5.46	69.0	± 6.3
Expiratory IJ stroke in mm. .. .	3.8	±1.63	6.5	± 2.5	4.3	± 1.84	7.3	± 2.4
Inspiratory IJ stroke in mm. .. .	4.0	±1.14	8.0	± 2.9	4.4	± 1.91	7.7	± 3.2

The identical procedure was repeated on six healthy young persons two months later.

TABLE III
BALLISTOCARDIOGRAPHIC CHANGES DUE TO EMOTION, PLACEBO, AND AMYL NITRITE

	Emotion				Placebo				Amyl nitrite			
	Before emotion		After emotion		Before placebo		After placebo		Before amyl nitrite		After amyl nitrite	
	Mean	S.D. of mean	Mean	S.D. of mean	Mean	S.D. of mean	Mean	S.D. of mean	Mean	S.D. of mean	Mean	S.D. of mean
Heart rate	72	±14.6	77	±13.7	72	±12.2	85	±11.8	74	±12.0	119	±18.6
Systolic B.P. .. .	120	±12.9	126	±12.6	124	±12.5	120	± 7.2	120	±10.1	123	±12.3
Diastolic B.P. .. .	71	± 5.6	74	± 3.5	71	± 5.02	70	± 4.8	71	± 5.2	75	± 8.9
Expiratory IJ stroke in mm. .. .	3.1	± 1.9	3.5	± 2.09	3.5	± 2.09	3.6	± 2.16	3.5	± 2.21	5.6	± 3.4
Inspiratory IJ stroke in mm. .. .	4.6	± 1.8	4.6	± 2.22	4.7	± 2.39	4.1	± 2.44	4.1	± 1.98	7.4	± 4.0

Six healthy young subjects had the effects of amyl nitrite reiterated to them, were given an empty capsule and then amyl nitrite to inhale. The IJ stroke was measured in only five owing to a technical error.

Group B. Twenty-five Normal Subjects between 40 and 65 years of age. In 5 subjects the initial ballistocardiograms were abnormal in form and resembled tracings obtained from the coronary artery group. In one person there was a history of alcoholism, one was diabetic, one was very obese, and another was found to have albuminuria. The fifth appeared to be otherwise healthy.

The changes in IJ values, heart rate and blood pressure after amyl nitrite were less significant than in the younger group (Table I). Heart rate increased in 24 of 25 subjects, remaining unchanged in one case. The maximum increase was from 68 to 125 a minute. Systolic blood pressure increased in 15, fell in 6, and remained unchanged in 4, the range being +26 to -12 mm. Hg. Diastolic pressure increased in 17, fell in 4, and remained unchanged in 4 with a range of +20 to -6 mm. Hg.

In one instance somatic tremor after amyl nitrite prevented an accurate assessment of the IJ stroke and this case was omitted. After amyl nitrite the expiratory IJ increased in 16 instances, remained unchanged in 3, and fell in 5, the maximum being from 7.5 to 13.5 mm. Inspiratory values increased in 18, remained unaltered in 3, and fell in 3 instances, the maximum increase being from 5 to 14 mm. The mean IJ values correlated well with those of Group A. One case in this group had pulsus bigeminus, and this record showed a definite quantitative difference in all the major ballistic waves with each extrasystole (Fig. 3).

FIG. 3

FIG. 4

FIG. 3.—Normal subject over 40 years of age with coupled rhythm. Resting expiratory record: heart rate approximately 60 a minute, B.P. 114/64. With the ectopic beats the IJ stroke, K, L and M waves are of smaller amplitude.

FIG. 4.—Patient with coronary artery disease showing the typical appearance of low variable and largely unidentifiable ballistic complexes. Heart rate 68 a minute, B.P. 126/88. The standardization is the same as in Fig. 2, 3, and 5.

Group C. Twenty-five Subjects with proven Coronary Artery Disease between 38 and 70 years of age. A feature of this group was that normal ballistocardiograms were obtained in 2 of 25 cases and in another 3 patients the graphs differed from normal only in the amyl nitrite response. One of the two patients with normal ballistocardiograms had experienced myocardial infarction two years previously. All five were ambulant and liable only to minor attacks of angina at this time.

Mean heart rate, blood pressure and IJ changes after amyl nitrite were less than in Groups A and B (Table I). Maximum changes were heart rate from 68 to 120, systolic pressure from 152 to 170 mm. Hg, diastolic 70-86 mm. Hg, expiratory IJ 5-7 mm., and inspiratory IJ 4-10 mm. Expiratory IJ values increased in 12, fell in 8, and remained unchanged in 5, and inspiratory values increased in 15, fell in 5, and remained unchanged in 5. Systolic blood pressure increased in 10, fell in 12, and remained unchanged in 3, while diastolic blood pressure increased in 11, fell in 8, and remained unchanged in 6 instances. The systolic range was +18 to -10 mm. Hg, and the diastolic +16 to -10 mm. Hg.

In the three hypertensive subjects systolic pressure increased in two and remained unchanged in one, the diastolic rose in two and fell in one instance. The systolic range was +10 to zero mm. Hg, and the diastolic +8 to -14 mm. Hg.

In all but the five subjects with normal or near normal records the ballistocardiograms revealed impairment or loss of regularity of onset and definition of complexes. In many the ballistic waves were completely unrecognizable and in most, the IJ values were of low and variable amplitude. The wave form was very variable and no definite pattern could be established, nor was there any significant qualitative or quantitative improvement after the administration of amyl nitrite (Fig. 1b, 4, 5, and Table I).

FIG. 5.—The effect of amyl nitrite on the ballistocardiogram in patients with coronary artery disease. (a) Basal expiratory record: heart rate 75 a minute, B.P. 176/90. (b) Basal inspiratory record: heart rate 70 a minute. The ballistic complexes in both leads are of low variable amplitude and are largely unidentifiable. (c) Expiratory record after amyl nitrite: heart rate 100 a minute, B.P. 176/98. (d) Inspiratory record after amyl nitrite: heart rate 100 a minute. The ballistic complexes remain of low and variable amplitude and are largely unidentifiable. Further flattening of the electrocardiographic T wave has occurred.

DISCUSSION

In recent years particular interest has been shown in the ballistic IJ stroke which was believed to be closely related to cardiac output (Starr *et al.*, 1939; Molomut and Allen, 1946; Nickerson, 1945, 1947). Apart from the theoretical implications of variable body tissue damping effects and an inability to determine accurately the spatial vector from records taken with the patient recumbent, the findings in this series suggest that factors other than cardiac output are important in the production of the IJ stroke.

Expressed in millimetres, basal IJ stroke values were found in some instances to be as much as ten times greater in normal subjects of the same age than in patients suffering from coronary artery disease. After the inhalation of amyl nitrite, a powerful vasodilator, both IJ values and heart rate increased markedly in normal subjects; in isolated cases these both doubled. These changes were independent of alterations in the blood pressure, although for individual cases this more often increased than decreased. In patients suffering from coronary artery disease, and to a lesser extent in normal subjects, the IJ amplitude often varied from beat to beat, independently of heart rate, P-R interval, phase of respiration or somatic tremor (Fig. 2, 3, 4, 5). IJ values measured after the patient had been lying down for 5 minutes and again after 30 minutes often revealed that the former was two to five times greater than the latter, there being no significant changes in blood pressure or heart rate. In bigeminy the extrasystoles showed smaller ballistocardiographic complexes. The smaller IJ was compatible with a diminution in stroke volume, but there were also smaller K, L, and M waves with each ectopic beat (Fig. 3).

It was also observed that the depth of the K wave increased after the inhalation of amyl nitrite (Fig. 2), and as this drug diminishes peripheral resistance it would suggest that other factors are important in the production of this wave.

These findings suggest that the elasticity of arterial and arteriolar vessel walls are important in determining the amplitude of the IJ stroke and other ballistocardiographic waves. The marked variability that may occur from beat to beat cannot, however, be explained on this hypothesis alone. It is possible that general body tissue damping effects are important in this respect. These findings all indicate, however, that attempts to estimate cardiac output by means of the ballistocardiogram are open to criticism. It would seem justifiable to express IJ results in terms of millimetres, providing all standardization criteria are described and the type of instrument employed is stated.

The blood pressure changes due to the inhalation of amyl nitrite proved interesting. In the young normals the mean systolic and diastolic pressures increased slightly, although individual blood pressures often fell, after breathing amyl nitrite. Neither an emotional stimulus nor the inhalation of a placebo caused any statistical alteration in blood pressure, although with emotion the trend was to increase. Yet when six subjects had the test repeated two months later the heart rate and IJ stroke responded as before, but the mean systolic and diastolic pressures remained normal whereas with the first investigation both had increased. This would suggest an emotional factor as the cause of the rise in blood pressure, an observation previously reported (Evans *et al.*, 1939). It is well known that the effects of the nitrite ion vary with body posture. When the subject is horizontal there occurs little or no change in blood pressure, but when erect both the systolic and diastolic pressures fall steeply (Wilkins *et al.*, 1938). As all records in the present series were taken with the patient recumbent, these blood pressure findings are therefore not surprising.

Normal ballistocardiograms were found in 2, and patterns that were almost normal in 3 of the 25 patients suffering from coronary artery disease. All these subjects were ambulant and liable to only slight effort pain at the time of their ballistocardiograms. A normal ballistocardiogram therefore does not exclude the presence of coronary artery disease.

It is interesting to speculate on the possible prognostic implications of a normal ballistocardiogram in the presence of angina pectoris. If it be true that such patients have a more adequate coronary artery circulation then it would be reasonable to anticipate a better prognosis. The exact cause of the abnormal graphs found in coronary artery disease is unknown, but the observations made in the normal groups suggest that peripheral arteriosclerosis may be as important as coronary sclerosis. Whatever the explanation, the difference is so striking that ballistocardiography may prove to be a useful procedure in the diagnosis of latent coronary artery disease. A long-term follow-up of such cases is essential for the reason that the pattern now described may well prove not to be specific for uncomplicated coronary artery disease. In the presence of any other organic cardiac disease the ballistic results should be treated with caution.

Apart from tachycardia the only other electrocardiographic response to amyl nitrite was the development of depressed S-T segments and T wave inversion that occurred in two ischaemic patients and two normal young women aged 19 and 22 years respectively. This would suggest increased heart work due to amyl nitrite and probably implies a relative and temporary coronary insufficiency, which in the young normal patients indicates that it may be physiological.

SUMMARY AND CONCLUSIONS

An abnormal ballistocardiographic pattern is common in patients handicapped by coronary artery sclerosis. Its recognition may prove of value in the diagnosis of latent coronary disease.

A normal ballistocardiogram does not exclude the presence of coronary artery disease.

The inhalation of amyl nitrite produces a transient increase in amplitude of the major ballistic waves in normal people. In subjects with coronary artery disease these alterations are minimal.

Evidence is presented which suggests that the ballistic IJ stroke cannot be accurately correlated with cardiac output.

Emphasis is placed on the state of the arterial and arteriolar vessel walls in the production of ballistic complexes.

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