Studies Utilizing the Portable Electromagnetic Ballistocardiograph

II. The Ballistocardiogram as a Means of Determining Nicotine Sensitivity

By Harry Mandelbaum, M.D., and Robert A. Mandelbaum, M.D.

Persons who have failed to habituate to the cardiovascular effects of nicotine will demonstrate changes in their ballistocardiogram after smoking. These ballistocardiographic changes produced by smoking are of more abnormal quality and are found more frequently in patients with coronary artery and hypertensive heart disease than in normal subjects. The ballistocardiograph provides an accurate objective means of determining whether nicotine has a deleterious effect upon the myocardial function of a patient. In a significant proportion of patients with heart disease who demonstrated an abnormal ballistocardiographic response to smoking, clinical improvement was obtained after a period of tobacco abstinence.

The deleterious effects which tobacco may have on patients with heart disease have been given considerable study by various investigators. The peripheral vasospastic action of nicotine has been proved conclusively by Roth.1 Graybiel and his co-workers2 and Roth1 have shown the great frequency with which nicotine, either by inhalation or by intravenous administration, can cause electrocardiographic changes consisting of tachycardia and decrease of T-wave amplitude. The T-wave changes are similar to those found after atropinization. It is likely that the action of nicotine on the vagus and sympathetic ganglions is the cause of the electrocardiographic, blood pressure and pulse rate changes. These observations have been recorded in normal subjects after smoking as well as in patients with heart disease. Cases in which the use of tobacco caused angina pectoris associated with electrocardiographic changes have been described3-5 but are considered of infrequent occurrence. Ralli and Oppenheimer6 consider that tobacco angina results from vasoconstriction of the coronary arteries. Graybiel and his co-workers2 believe that it is due to a sudden increase in the work of the heart, as shown by the increase in heart rate and blood pressure.

In other reports,6-8 abstinence from smoking for extended periods of time has resulted in a decrease in the severity or frequency of the attacks of angina pectoris, as well as an occasional reversion to normal of the electrocardiogram. While the fact that nicotine can affect the cardiovascular dynamics in certain susceptible persons has been well established, a great deal of doubt9,10 has been cast upon the importance of absolute abstinence from smoking in the routine care of patients with heart disease. On the other hand, many clinicians agree with Moschcowitz7 that, because of the angiospastic11 action of nicotine, along with the clinical experience that many patients with angina pectoris do well after the cessation of smoking, more attention should be paid to nicotine sensitivity in the study of patients with heart disease.

A satisfactory test to determine which patients are sensitive to tobacco is needed. That there is individual susceptibility1, 9, 10 to nicotine has been established. Blood pressure rises of 22 mm. Hg systolic and 20 mm. Hg diastolic as well as increases in heart rate of 50 beats per minute have been described following smoking.4 No significant difference in the degree or relative frequency of these blood pressure or heart rate changes has been found in persons with heart disease compared with normal subjects. Retinal spasm was noted in hyper-
tensive patients as an effect of nicotine. Mathers and his associates believe that an increase in the heart rate of more than 25 beats per minute may be regarded as the most reliable index of hypersensitivity to the immediate effects of nicotine.

It is an accepted fact that nicotine affects the cardiodynamics of particular individuals by causing tachycardia, an increase in the blood pressure and occasionally changes in the electrocardiogram. A more impressive test, however, would be one that demonstrates a direct effect upon the subject's myocardial efficiency. In doing ballistocardiographic studies upon subjects with neurocirculatory asthenia, essential hypertension and coronary artery disease, we have observed that a number of patients showed definite changes in their ballistocardiograms following smoking. These changes in the ballistocardiographic pattern demonstrated in almost every case that the ventricular ejection wave was of abnormal quality. Ballistocardiographic changes after smoking were found also in several persons who were apparently without heart disease. All of the ballistocardiograms were made with the direct electromagnetic ballistocardiograph previously described upon subjects who had rested for 15 minutes, who had not eaten for three or more hours, and who had not smoked for 24 hours. After the completion of a ballistocardiogram taken under resting conditions, the subjects were asked to smoke their particular brand of cigarette or cigar. Many subjects did not care to finish more than half of the cigarette or an inch of the cigar. Positive tests were noted as frequently after abbreviated periods of smoking as when the entire cigarette or cigar was smoked. Positive tests were also occasionally found when the particular cigarette used was of the "denicotinized" variety. No note was made as to whether or not the subject inhaled. The postsmoking ballistocardiogram was taken two minutes after the completion of smoking and was followed immediately by a ballistocardiogram taken after the performance of a modified two-step exercise test.

Levy and his co-workers used the Nickerson ballistocardiograph to estimate changes in cardiac output in response to smoking. Their findings were a generalized fall in cardiac output in normal subjects, averaging 1.8 per cent of the presmoking output, and a rise in cardiac output in persons with heart disease, averaging 6.4 per cent. No follow-up studies were done in these patients with heart disease who were sensitive to nicotine to determine whether the cessation of smoking would result in an improvement of the presmoking resting ballistocardiogram or in any subjective relief of angina. Furthermore, it is of interest to note that in their study no significant changes were found in the electrocardiograms after smoking of the subjects who were sensitive to nicotine, as determined by the ballistocardiogram and the pulse rate changes. In a recent study, Caccese and Schrager have described abnormal changes in the ballistocardiographic records of persons with and without heart disease following cigarette smoking. The Dock electromagnetic ballistocardiograph was the recording instrument in the latter report.

In our studies on subjects suspected of sensitivity to tobacco, three characteristic patterns could be recognized in the ballistocardiograms as a positive reaction to smoking: (1) the hyperkemic* response; (2) abnormal variation in the inspiratory-expiratory complexes; and (3) the diminished HIJ and prominent K pattern.

A group of subjects, consisting primarily of normal subjects, of persons with complaints of palpitation, and of subjects previously diagnosed as having neurocirculatory asthenia, demonstrated the hyperkemic response in which the amplitude of the HIJK complexes became increased; an associated tachycardia was the rule (fig. 1). However, we did not consider these hyperkemic responses as evidence of a harmful nicotine effect unless some abnormal configuration was noted in the ballistic pattern as well. The hyperkemic nicotine response, however, contributed to or was the cause of symptoms such as palpitation, chest pain, dizziness or throbbing headache, in many of these persons.

The second type of abnormal smoking test

* Hyperkemia indicates increased velocity of ejection of blood from the ventricles, causing larger amplitude of the ballistic record.
was the development of increased respiratory variation in the ballistocardiogram, with complexes of poor amplitude in the expiratory phase (figs. 4, 6). Occasionally, all the complexes in the ballistocardiogram became bizarre and of low amplitude, resulting in a Brown classification of grade 3 or 4. These findings were more common in subjects with angina pectoris. No such findings were recorded in normal controls.

Particularly in hypertensive subjects, the abnormal configuration in response to smoking was the appearance of the diminished HIJ deep K pattern (figs. 3, 6). In many, this change occurred only in the expiratory phases of the ballistocardiogram.

Changes in the ballistocardiogram following exercise, similar to these specific nicotine effects of increased respiratory variation with complexes of poor amplitude in the expiratory phase and/or the appearance of the low HIJ deep K pattern, have been previously described in subjects with hypertension and coronary artery disease whose basal tracings had been normal or only slightly abnormal. In subjects suspected of sensitivity to smoking, where the immediate postsmoking ballistocardiogram showed no variation from the basal ballistocardiogram, exercising immediately after smoking demonstrated an abnormality where a previous exercise test was not abnormal. These instances, however, were not common.

It is not our purpose, in this report, to determine the statistical frequency of nicotine sensitivity in any particular group of subjects, but rather to emphasize that the ballistocardiograph appears to be of great clinical importance in the recognition of those persons who have never adapted themselves to the toxic action of nicotine. The more serious types of ballistocardiographic abnormalities resulting from smoking occurred most frequently in subjects who had coronary artery disease. There is no question, however, that people who have no heart disease may exhibit abnormal ballistocardiograms in response to smoking (fig. 1). In a series of 50 subjects, from 16 to 60 years of age, who by all standards were believed to be without heart disease, 28 per cent demonstrated an abnormality in their ballistocardiogram following smoking. All of these positive ballistocardiographic smoking tests were obtained from persons less than 40 years of age. No sex difference in smoking response was noted. In persons with coronary artery or hypertensive heart disease, most of whom demonstrated an abnormal presmoking ballistocardiogram, we have found the incidence of positive smoking tests to be more than twice that obtained in persons without heart disease.

Since the ballistocardiogram represents primarily the initial cardiac output velocity of blood ejected from the ventricles, which depends entirely upon myocardial vigor, the development of ballistocardiographic abnormalities following smoking in the cardiac patient is of clinical importance. The physician responsible for the guidance of a patient with coronary artery disease or hypertension, upon recognizing a change suggestive of impaired myocardial efficiency in the ballistocardiogram following the smoking test, should be insistent that this patient stop smoking.

As a concomitant finding with the ballistocardiographic changes, we have also observed the rises in blood pressure and the tachycardia noted by other observers in response to smoking. However, we have found positive ballistocardiographic smoking tests in the absence of a rise in the heart rate or blood pressure (fig. 5; upper two traces). Not infrequently, nicotine tachycardia was associated with a ballistocardiogram which did not demonstrate any abnormality in configuration and which was considered normal. Many of the patients with positive smoking test ballistocardiograms had electrocardiographic studies done following smoking. Only two of 35 patients with angina pectoris so tested demonstrated significant electrocardiographic changes after smoking. Several patients, following a short period of smoking, complained of nausea and dizziness. The fact that all tests were done on fasting subjects may account for the frequency of these symptoms. In all subjects with these complaints, ballistocardiographic abnormalities were found. In 10 subjects, who were observed for several hours, ballistocardiographic ab-
normalities could be recognized in tracings taken from five minutes to as long as 45 minutes after smoking had been completed. The average time required for the postsmoking ballistocardiogram to return to the resting state was 10 minutes.

The fact that tobacco can cause added distress in subjects with coronary artery-hypertensive heart disease, in whom the ballistocardiogram shows an increase in abnormality with smoking, was confirmed clinically by the subjective improvement in 30 out of 35 patients with the anginal syndrome after abstention from nicotine. Eight of these 35 patients showed improvement in either their resting or their postexercise ballistocardiogram after abstaining from nicotine for two months. Four young subjects with hypertension (fig. 5) and early, deep K-stroke patterns in the resting ballistocardiogram, who had positive smoking tests, responded to a period of nicotine abstention by the improvement to normal of the basal ballistocardiogram and appreciable decrease in their blood pressure.

White believes that there is a condition justifiably called "tobacco heart" but that it is primarily a functional derangement and not an organic heart disease. We have observed two nondiabetic normotensive women in the fifth decade of life (fig. 2) who had complaints characteristic of coronary artery disease. Both had normal electrocardiograms and abnormal resting ballistocardiograms; after the smoking test, the ballistocardiograms showed a progression in the degree of abnormality. These
women became asymptomatic and were found to have almost normal ballistocardiograms at rest and after exercise after two months of abstention from nicotine. Whether the continued use of tobacco by these two patients who had failed to habituate to nicotine would have played a role in the development of organic heart disease is a matter of conjecture.

ILLUSTRATIVE CASE REPORTS

*Case 1 (fig. 1)*; Hyperkemic Response. A. W., male, 32 years of age, weighing 155 pounds. This patient, a heavy smoker, suffered from attacks of dizziness accompanied by tinnitus and nausea. Occasional attacks of palpitation often accompanied or preceded the dizziness. His blood pressure was 140/80. Physical examination, roentgenogram of the chest, electrocardiogram and basal metabolism studies were normal. 1. The basal ballistocardiogram was normal. 2. After smoking half a cigarette, tachycardia occurred; the blood pressure rose to 150/90 and a ballistocardiogram with abnormal respiratory variation was obtained. The patient felt completely well during the entire test period. The increased amplitude of the K and L waves puts this nicotine response in the hyperkemic category.

*Case 2 (fig. 2)*; Hyperkemic Response and Tobacco "Heart Disease." S. S., female, 46 years of age, weighing 114 pounds. This patient was a heavy smoker and complained of headaches, palpitation and angina on effort. The attacks of angina had been increasing in frequency and degree, forcing her to give up her work. Her blood pressure showed lability, varying from 140/85 to 185/98. Physical examination, roentgenogram of the chest, electrocardiogram and basal metabolism were normal.

On March 25, 1951, 1, the basal ballistocardiogram shows grade 2 respiratory variation with low HIJ waves in the expiratory phase. 2. After smoking, note the tachycardia and the large amplitude of the complexes which characterize the hyperkemic response; the slurred and short HIJ strokes are evident in the expiratory phase.

The patient has not smoked since March 25, 1951, and has been free of complaints since May 15, 1951. On May 29, 1951, 3, the basal ballistocardiogram shows marked improvement; notched J waves are present in the expiratory phases. 4. After performance of the exercise test, short HIJ waves are present. Note the diminished IJ amplitude in the fourth and seventh complexes. This patient has been without complaints since giving up the use of tobacco.

*Note:* The upper channel in each figure represents the ballistocardiogram; the lower channel, the concomitant tracing taken from the left radial pulse. The J wave is recorded at the apex of the radial pulse.
waves can be noted in the second and fifth complexes.

Case 3 (fig. 3); Essential Hypertension; Characteristic Low HIJ Deep K Response. S. G., male, 52 years of age, weighing 186 pounds. This patient has been a heavy smoker for many years and complained of recurring headaches. His blood pressure over a period of years showed marked lability, 170/90 to 210/105. Physical examination and roentgenogram of the chest were normal. The electrocardiogram showed left ventricular hypertrophy. 1. The basal ballistocardiogram shows normal respiratory variation with tall L waves. 2. After smoking half a cigarette; note the short HIJ strokes especially in the expiratory phases, which gives prominence to the K wave. 3. The next morning; no smoking for 24 hours. A ballistocardiogram taken after performance of a two-step exercise test. (The basal ballistocardiogram preceding the two-step exercise was the same as the original.) The HIJ deflections are decreased in amplitude but not to the extent produced by nicotine. This patient improved clinically after cessation of smoking; his blood pressure has not risen above 165/90.

Case 4 (fig. 4); Angina Pectoris; Increase in Inspiratory-Expiratory Variation. L. H., male, 41 years of age, weighing 181 pounds. Since his discharge from the Army, he has been aware of angina on effort. Of late, the attacks required nitroglycerin for relief. He had reduced his smoking and used denicotinized cigarettes. Many examinations and repeated electrocardiograms were normal. His blood pressure was 128/84. Physical examination, roentgenogram of the chest, electrocardiogram and basal metabolic rate were normal. 1. Basal ballistocardiogram normal. 2. After smoking (a denicotinized cigarette was used in this instance), the inspiratory-expiratory variations permit classification of grade 2 after Brown. 3. After abstinence from smoking for 24 hours, the exercise test showed the inspiratory-expiratory variation to be grade 1 according to Brown. This patient has had diminished frequency of angina since losing weight and giving up smoking.

Case 5 (fig. 5); Angina Pectoris; Essential Hypertension; Improvement after Abstinence. I. L., female, 41 years of age, weighing 188 pounds. Her complaints were of angina of effort, which in the last few months had become of more frequent occurrence and of greater severity. Hypertension was of at least five years' duration.

On January 4, 1951, the blood pressure was 190/105. Physical examination, roentgenogram of the chest and basal metabolism were normal. The electrocardiogram showed left axis deviation. 1. Basal ballistocardiogram was classified grade 1 after Brown. The fourth complex shows a notched J wave; the short HIJ deep K pattern is in evidence. 2. After smoking, the ballistocardiogram may be classified as grade 3; note absence of tachycardia.

The patient gave up smoking January 4, 1951, and after a few months improvement was striking; the attacks of angina occurring only after extended effort. On July 28, 1951, the blood pressure was 145/90. The electrocardiogram showed no change. 3. The basal ballistocardiogram is now normal. 4. After the exercise test, the ballistocardiogram is classified grade 1.

Case 6 (fig. 6); Myocardial Infarction; Smoking Sensitivity. B. K., male, 30 years of age, weighing 155 pounds. He had been complaining of angina pectoris since October, 1950. He was a heavy smoker. In May, 1951, the attacks of angina became more frequent and on June 30, 1951, he suffered an anterior myocardial infarction. After four weeks in bed, he has gradually increased his activities.

August 2, 1951. The blood pressure was 128/82. Physical examination and roentgenogram were normal; electrocardiogram shows evidence of a healed anterior myocardial infarction. 1. Basal ballistocardiogram shows excellent myocardial recovery. 2. After smoking a cigarette, tachycardia decreases in the amplitude of all complexes, grade 1 respiratory variation and low HIJ waves are in evidence. 3. Exercise test (eight trips) after the effects of the nicotine were permitted to wear off (30 minutes later); the ballistocardiogram is grade 1. This patient has been forbidden to smoke.
HARRY MANDELBAUM AND ROBERT A. MANDELBAUM

Summary

In detecting those subjects who have never adapted themselves to the toxic action of nicotine, the ballistocardiograph has proved to be a valuable instrument. The characteristic ballistocardiographic patterns that are obtained after smoking have been described. These patterns have been found in some smokers who were apparently without heart disease, as well as in cardiac subjects. However, the more marked ballistocardiographic abnormalities were recorded in patients with hypertension, coronary artery disease and neurocirculatory asthenia.

The clinical importance of determining nicotine sensitivity has been proved by the high incidence of subjective improvement in our patients with coronary artery and hypertensive heart disease, with positive smoking tests, after a period of tobacco abstinence. Not infrequently, progressive improvement in the ballistocardiograms of those nicotine sensitive patients kept pace with their symptomatic relief.

It is our opinion that no patient with cardiac complaints, who shows a positive ballistocardiographic smoking test, should be permitted the use of tobacco.

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References

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HARRY MANDELBAUM and ROBERT A. MANDELBAUM

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