Coronary Artery Disease: Evaluation by the Multistage Treadmill Exercise Test and Right Atrial Pacing*

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Atrial pacing and treadmill exercise were performed by 28 patients to determine the value of each test in the clinical and functional evaluation of the patient with coronary artery disease (CAD). The functional aerobic impairment (FAI) in 20 patients was 48 percent (eight patients with unstable angina paced but not exercised) and the pacing ventricular function curve (VFC) was abnormal in 13 of these 20. Five patients with severe FAI (pain limited) had a normal pacing VFC. Although the FAI was greater the more extensive the CAD, no such correlation existed between the pacing VFC and the number of diseased coronary vessels. Exercise testing does not necessarily indicate the functional performance of the left ventricle since the VO₂ max is frequently pain limited. Atrial pacing, although less sensitive, permits a VFC determination, even in poorly motivated and non-ambulatory patients, and combined studies will indicate the patient's clinical, as well as myocardial status.

Since there may be no correlation between the control hemodynamic performance of the myocardium and the extent of coronary artery disease, some form of stress is usually required in evaluating this type of patient.¹ ² Most commonly, this is exercise stress either clinically or at cardiac catheterization,⁵ ⁶ but infusions of angiotensin,⁷ sustained hand grip,⁸ and atrial pacing¹¹ ¹⁴ are also utilized to determine the functional status of the left ventricle.

Although exercise may be the ideal method, the patient must be ambulatory and motivated, and the clinical situation should be stable. Therefore, unstable angina pectoris or a recent myocardial infarction is usually considered a reason to preclude this form of study. Many investigators have recently used atrial pacing in the evaluation of patients with coronary artery disease since it is a controlled reproducible stress, which lacks the systemic metabolic effects of exercise and requires no particular cooperation on the part of the patient other than that of routine cardiac catheterization.⁶ ¹⁶ We and others have also utilized the multistage treadmill exercise test developed by Bruce and his associates¹⁷ ²² to evaluate and follow up these patients, and this has been used in conjunction with atrial pacing performed at the time of cardiac catheterization.

This report describes our results in 28 consecutive patients with coronary artery disease, in whom both treadmill exercise and atrial pacing were performed. Although it is realized that each test is not measuring the same parameters, we have attempted to determine what relation exists between the two procedures. This was done to ascertain their individual and their combined potentials in determining the clinical and hemodynamic status of the patient with coronary artery disease. This knowledge is important since these techniques are so frequently used, especially in the preoperative and postoperative study of patients with coronary insufficiency.

METHODS

Twenty-eight consecutive patients with coronary artery disease undergoing hemodynamic and angiographic studies were evaluated by means of exercise and atrial pacing. There

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were 24 men and four women; the average age was 46 years (range 29 to 63). Informed written consent was obtained from all patients for both cardiac catheterization and exercise studies.

The exercise testing was carried out in only 20 patients, with the same procedures reported in some detail by Bruce and his associates, since eight patients were considered to have unstable angina pectoris. After a complete clinical examination (history, physical examination, 12-lead ECG, x-ray studies and appropriate laboratory studies), a multistage treadmill test of maximal exercise was performed. The patients were studied several hours postprandially; they had not smoked or taken nitroglycerin in the preceding two to three hours. The exercise began with the subject walking at 1.7 mph on a 10 percent grade for three minutes, followed by stepwise increases every three minutes to a higher level of exertion as tolerated by the patient (to 2.5 mph and 12 percent grade, 3.4 mph and 14 percent grade, 4.2 mph and 16 percent grade). Medically supervised testing was done under constant ECG lead V5 (telemetry) and blood pressure (clinical sphygmomanometer) monitoring. The patients were not permitted to hold on to a hand rail, but could rest one or two fingers of one hand, if necessary, on a flexible spring guide to maintain a fixed position on the treadmill. The end point for exercise was determined by the patient when severe dyspnea, fatigue, angina pectoris, dizziness, etc occurred. In addition, the medical supervisor could terminate the procedure if ataxic gait, arrhythmia, or hypotension was noted. The patients were also monitored closely after the exercise period. Expired air was collected every minute for the last three to four minutes of exertion or during early periods of the testing when clinical evaluation disclosed a significant decrease in exercise tolerance. If the last sample was obtained over less than one minute, the volume was extrapolated to one minute. Oxygen consumption was determined by standard techniques and the maximal oxygen consumption (VO2max) expressed as milliliters per kilogram per minute.

The exercise performance was also analyzed in terms of duration of walking and in relation to the patient’s functional aerobic impairment (FAI). The latter was derived from standard values, considering body weight, sex, age, level of ordinary activity, duration of treadmill exercise and oxygen intake. The FAI represents the percentage of deficit in a patient’s circulatory conductance of oxygen in relation to the functional aerobic capacity expected in health, and may be determined by the formula:

$$\text{FAI} = \frac{\text{predicted VO}_{2\text{max}} - \text{observed VO}_{2\text{max}}}{\text{predicted VO}_{2\text{max}}} \times 100$$

For purposes of further comparison, the patients were divided into three groups, according to their degree of FAI: Group 1, no to mild impairment (0-25 percent FAI); Group 2, moderate impairment (25-50 percent FAI); and Group 3, severe impairment (more than 50 percent FAI).

The next day, hemodynamic and angiographic studies were performed by previously described methods. With the patient under light pentobarbital sedation in the postabsorptive state and under local anesthesia with lidocaine, a needle was placed in the left femoral artery (LFA) and a cutdown was performed in the right antecubital area. By standard techniques, one catheter was placed in the pulmonary artery (PA) through an antecubital vein and another in the left ventricle (LV) retrograde from the brachial artery. A bipolar electrode catheter was positioned in the right atrium. All pressures were recorded through fluid-filled systems on an Electronics for Medicine DR8 recorder through Statham P23Db strain gauges. The midchest position served as the zero reference point, and mean pressures were determined electronically. Left ventricular end-diastolic pressure (LVEDP) was recorded on a high sensitivity scale and measured where the downslope of the left ventricular "A" wave coincided with the initial upstroke of the LV pressure. This usually occurred near the peak of the R wave of the electrocardiogram, or approximately 0.05 second after the Q wave. LVEDP was averaged over two respiratory cycles, or approximately ten beats. The time relation between the Q wave or R wave and the LVEDP at the control rate was used to ascertain the proper point on the LV pressure tracing for measuring the LVEDP at rapid rates.

After control arterial and intracardiac pressure measurements, the heart rate was gradually increased by right atrial pacing, beginning at a rate five to ten beats/minute above the control level. Subsequent rate increases were in increments of approximately ten beats/minute, with hemodynamic measurements recorded after a two-minute period at the particular heart rate. Cardiac output was determined by the dye-dilution method, during the control period, at an intermediate heart rate of 100-110/minute and at the maximum pacing rate. The highest pacing rate was determined individually for each patient and depended on his response. Pacing rates were limited in some patients because of chest pain or dyspnea, and further rate increases were discontinued if significant symptoms developed. Hemodynamic measurements were made at that particular level, and then pacing was discontinued. However, at least one, and occasionally two, sets of data were obtained after angina was induced in patients with coronary artery disease. In asymptomatic patients, attempts were made to reach heart rates of approximately 140-150/minute, but in two patients, the rate was limited by the development of Wenckebach-type second-degree atrioventricular block. In all patients, the induced changes in LVEDP were used to construct pacing ventricular function curves relating the pacing-induced changes in LVEDP to stroke work. The latter was calculated from the formula:

$$\text{Stroke work (SW)} = \frac{(\text{FAm}) - \text{LVEDP} \times SV \times 1.36}{100}$$

where (FAm) is mean femoral arterial pressure and SV is stroke volume. These function curves have been referred to as pacing ventricular function curves because, in the "classic" function curves, heart rate and other potential variables are held constant and alterations in stroke volume are produced by varying venous return. In these studies, stroke volume is being decreased by increasing heart rate. The "curves" should also be interpreted in relation to the control normal patient rather than to the classic description, since their shape appears to be more linear over a wide range of LVEDP’s. The slope of the pacing VFC may also be used to separate normal from abnormal responses, in accordance with the finding in a previous study that in the normal response the slope was greater than 2.0 gm-M SW/mm Hg LVEDP. On the basis of the configuration and slope of the pacing VFC, the patients with coronary artery disease could be divided into those with normal and those with abnormal myocardial function. Since data are available before and after the induction of angina pectoris, it is possible to evaluate myocardial function separately in each instance.
CORONARY ARTERY DISEASE EVALUATION BY MULTISTAGE TREADMILL EXERCISE TEST

Table 1—Exercise Tolerance of Patients with Coronary Artery Disease

<table>
<thead>
<tr>
<th></th>
<th>Heart Rate, Beats/Min</th>
<th>Sytolic Brachial Artery Pressure, mm Hg</th>
<th>Max. O₂ Intake, ml/kg/Min</th>
<th>Functional Aerobic Impairment, %</th>
<th>Tension Time Index</th>
<th>Tension Time Index, Pacing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>80 ± 3</td>
<td>129 ± 6</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercise</td>
<td>134 ± 5</td>
<td>154 ± 5</td>
<td>16.9 ± 5</td>
<td>48 ± 5</td>
<td>1982 ± 115</td>
<td>1879 ± 101</td>
</tr>
</tbody>
</table>

catheterization 12-lead electrocardiogram was used for comparison with the other parameters of myocardial function, with special emphasis on whether a diagnosis of a previous myocardial infarction could be made. The PR interval was also measured in each patient, since it increases with atrial pacing; this would tend to disturb the relation between atrial and ventricular contractions and might compromise myocardial function. True angina pectoris was considered present when the patient's chest discomfort had the usual characteristics and was similar to that occurring in the patient during exertion. This was distinguishable by careful questioning and familiarity with the patient's clinical syndrome, from the feeling of discomfort in the chest that patients sometimes experience during pacing.

After the physiologic studies were completed, left ventricular and coronary cineangiography were carried out. The techniques used, the evaluation of these films and the grading systems, which included separating generalized from individual vessels the right coronary artery, the left anterior descending, and the left circumflex arteries and any of their major branches.

RESULTS

Exercise Studies

On the basis of duration of exercise and/or their VO₂max, we determined that four patients with coronary artery disease had no or only mild FAI, five a moderate and 11 a severe reduction in their exercise tolerance (Table 1). The overall FAI for this group was 48 percent. Only four patients, three of whom had a mild and one a severe reduction in exercise tolerance, had a VO₂max above 20 ml/kg/min, with an average value for all patients of 16.9 ml/kg/min. Six had to discontinue exercise because of dyspnea, eight for pain, and two for "dizziness," and in four the main reason was fatigue. In no instance was it necessary for the medical supervisor to halt the test because of arrhythmia, ataxia, etc. However, one patient experienced ventricular tachycardia after he voluntarily stopped exercising because of dyspnea and fatigue. This was resolved spontaneously, with no aftereffects. Only five patients were able to reach stage three (3.4 mph-14 percent grade) in the exercise protocol. Significant ischemic ST segment changes appeared on the electrocardiograms of nine out of 20 patients. The modified tension-time index (heart rate x systolic arterial pressure) was 1982 ± 115 at angina during exercise. This did not differ significantly from the value of 1879 ± 101 at angina produced by atrial pacing (see below).

Atrial Pacing Studies and General Hemodynamic Considerations

Heart rate was increased by atrial pacing from a control value of 73 ± 6/min to a maximum of 124 ± 5/min. No significant change occurred in LVEDP, cardiac output or arterial pressure during pacing, and the usual decrease in stroke work and increase in PR interval was apparent (Table 2). Since these effects of atrial pacing in coronary artery disease have been commented on in detail elsewhere, no further evaluation has been made in this report. Similar hemodynamic responses to atrial pacing

<table>
<thead>
<tr>
<th></th>
<th>Heart Rate, Beats/Min</th>
<th>Mean Femoral Artery Pressure, mm Hg</th>
<th>Left Ventr. End-Diastolic Pressure</th>
<th>Cardiac Output, Liters/Min</th>
<th>Stroke Work, GmM/Min</th>
<th>PR Interval, Sec</th>
</tr>
</thead>
<tbody>
<tr>
<td>In 20 patients with stable angina</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>73 ± 6</td>
<td>103 ± 3</td>
<td>13 ± 2</td>
<td>4.5 ± 0.3</td>
<td>70 ± 7</td>
<td>0.16 ± 0.01</td>
</tr>
<tr>
<td>Atrial pacing</td>
<td>124 ± 5**</td>
<td>103 ± 3</td>
<td>13 ± 2</td>
<td>4.5 ± 0.4</td>
<td>44 ± 5**</td>
<td>0.23 ± 0.01**</td>
</tr>
<tr>
<td>In 8 patients with unstable or &quot;preinfarction&quot; angina</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>74 ± 5</td>
<td>101 ± 6</td>
<td>9 ± 1</td>
<td>4.7 ± 0.03</td>
<td>80 ± 9</td>
<td>0.15 ± 0.01</td>
</tr>
<tr>
<td>Atrial pacing</td>
<td>138 ± 6**</td>
<td>108 ± 7</td>
<td>8 ± 3</td>
<td>4.7 ± 0.03</td>
<td>55 ± 8**</td>
<td>0.22 ± 0.02**</td>
</tr>
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</table>

*Values are in standard error of the mean.
**Significant difference.
were noted in the eight patients with unstable or "preinfarction" angina (Table 2). Eleven patients experienced angina pectoris during atrial pacing, including seven of the eight having exercise-induced angina. Ischemic ST segment depression was noted in only six of 20 patients during atrial pacing. In patients with preinfarction angina, chest pain was induced in seven of eight, with significant ST changes in three (Table 2).

**Pacing Ventricular Function Curves (VFC)**

A pacing VFC could be constructed in all 20 patients with chronic angina prior to or in the absence of chest pain. In 13 (65 percent), this was abnormal. It became abnormal in three more, initially normal subjects, when angina was induced. In nine of 11 (83 percent) patients with angina, abnormalities in myocardial function were evident by the pacing VFC. In patients with preinfarction angina, abnormalities were noted in four of eight (50 percent) prior to pain and in six of seven (86 percent) when pain was present.

In Figure 1 we have compared the pacing VFC prior to angina with the same patients' exercise tolerance. All seven patients with normal pacing VFC had moderate or severe decreases in exercise tolerance. With abnormal pacing VFC, four patients had normal exercise responses and nine a significant decrease in exercise tolerance. These four patients with normal exercise tolerance exercised 8 min 48 sec, 6 min 52 sec, 10 min 10 sec, and 7 min, only the first patient stopping because of chest pain.

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**EXERCISE TOLERANCE**

![Graph showing exercise tolerance](image)

**NORMAL-MILD**

**NORMAL-ABNORMAL**

**PACING RESPONSE**

![Graph showing pacing response](image)

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**Angiographic Correlations**

Three patients had single-vessel, ten double-vessel and seven three-vessel coronary artery disease (average = 2.2/patient). Those with more severe coronary arterial disease tended to have the poorest exercise responses. Two of three patients with single-vessel disease had mild FAI, while eight of ten with double, and all seven with triple disease had moderate or severe FAI. However, ventricular func-
Severe exercise intolerance

**Figure 3.** Pacing ventricular function curves (VFC) prior to induction of angina pectoris in five patients whose exercise tolerance was severely limited only by development of chest pain. Results are normal when compared with those for accompanying normal pacing VFC.

As measured by the pacing VFC was not related to the number of vessels involved: one of three (33 percent) with single-vessel disease, three of 10 (30 percent) with two-vessel and three of seven (43 percent) with three-vessel disease had normal pacing responses in the absence of angina.

Left ventricular cineangiography disclosed localized ventricular abnormalities in 13 patients, generalized disease in six and only one with a grossly normal ventricle in the absence of chest pain. This last patient's FAI was severe, due to chest pain, and he stopped exercising in only 2 min 40 sec. His pacing VFC was normal before induced angina, but abnormal afterwards. There was generally no correlation between the angiographic appearance of the left ventricle and the FAI: three patients with localized defects in contractility had no or mild FAI, three had moderate FAI and seven had severe FAI.

Pacing VFCs were normal in seven patients with no or localized abnormalities, and were abnormal in seven patients with local changes. All six patients with generalized abnormalities in contraction had an abnormal pacing VFC.

**Electrocardiographic Correlations**

Ten patients had normal resting electrocardiograms or only nonspecific ST-T abnormalities; in ten, findings were consistent with old myocardial infarctions. No correlation was found between the resting electrocardiogram and the patient's response to pacing or exercise.

**Discussion**

The objective of any exercise testing is to determine the performance characteristics of the patient, which cannot be adequately determined by taking a clinical history and performing a physical examination. This is especially important in the patient with ischemic heart disease whose subjective complaint may be influenced by various emotional factors. The disease itself is not always predictable, and the physical and laboratory findings may be normal, despite severe disease. The recent tendency in exercise testing has been to determine maximal exercise capacity with the measurement of maximum oxygen intake (VO_{max}), since this value is now considered the international standard for physical fitness. This latter measurement depends on age, lean body mass, the sex of the patient, and usual degree of activity. Normal standards have been developed that allow comparisons among patients based on the above factors. Since a linear relation exists between the duration of exercise and the oxygen intake, the former can also be used to clinically estimate the latter and, hence, the FAI of the patient. FAI, as determined in this manner, represents the patient's overall impairment but does not necessarily define the various disease components, which may require further investigation (eg, electrocardiograms, cardiac catheterization).

Recently developed reliable atrial pacing methods and the utilization of these techniques in patients with angina provided a new dimension to the evaluation of the patient with ischemic heart disease. Since then, numerous investigators have used various atrial pacing methods to increase heart rate and myocardial work in a controlled fashion, thereby enhancing myocardial oxygen consumption and stressing the coronary reserve.
capacity.\textsuperscript{39,40} Through atrial pacing it has been possible to induce angina pectoris as well as electrocardiographic and hemodynamic changes.\textsuperscript{9-10} In addition, recent studies show that these techniques may also be of value in determining myocardial function through the production of ventricular function curves by means of pacing-induced changes in LVEDP and stroke work.\textsuperscript{10,12} Some studies have shown, however, that atrial pacing is inferior to exercise in illustrating ventricular dysfunction, probably because the ventricle is subjected to greater stroke work during exercise.\textsuperscript{41,42} These studies question the value of atrial pacing in the absence of induced angina. In our experience, atrial pacing did disclose left ventricular abnormalities in the absence of angina or prior to the induction of angina in approximately 60 percent of the patients with coronary artery disease.\textsuperscript{10,11} These abnormalities were noted in patients with chronic angina and preinfarction angina, and were shown in postoperative studies. The results of our investigation are similar. The pacing ventricular function curve was abnormal in 13 of 20 patients (65 percent) with chronic angina prior to or in the absence of chest pain. Similar findings were noted in patients who had had preinfarction angina.

The maximal exercise capacity is reduced in subjects with coronary artery disease, whether or not angina is induced.\textsuperscript{22,17} However, in many of these patients the VO\textsubscript{2max} is a pain-limited value and probably does not have the same physiologic meaning as a true VO\textsubscript{2max}.\textsuperscript{18} In other words, the patient discontinues exercising because of pain; thus, it is impossible to determine a valid maximal exercise performance. Since a properly performed exercise test is reproducible, the graded treadmill exercise test is valuable for following the patient's clinical course, especially when drug or surgical interventions are utilized;\textsuperscript{17,22} however, it does not necessarily indicate the functional status of the left ventricular myocardium, one of the important factors that ultimately determine morbidity and mortality.\textsuperscript{43} This is illustrated by the fact that some patients in this study showed a severe decrease in exercise capacity. They all experienced chest pain and stopped walking at low exercise levels, but they had normal pacing ventricular function curves during cardiac catheterization in the absence of angina (Fig 3). When angina pectoris was subsequently induced by atrial pacing, the pacing VFC became abnormal, a typical response. In essence, then, their myocardial function was abnormal during angina, and, hence, the exercise test performance is impaired during this ischemia. However, since the myocardium functions normally in the absence of ischemia, such patients may be better candidates for a surgical procedure that could eliminate the ischemia and leave them with an essentially normal myocardial response. The other patients in that group having a severe decrease in exercise tolerance had abnormal myocardia, as disclosed by the pacing VFC, even without the induction of overt ischemia. Surgical intervention in these patients who may have chronic, irreversible myocardial changes such as fibrosis, might not produce the anticipated improvement in their myocardial performance, as determined hemodynamically, although pain relief may be expected.

At the other end of the spectrum are those patients having little exercise impairment but whose pacing VFC disclosed abnormal myocardial function (Fig 2). Only one of these patients experienced chest pain, so they were generally limited only by dyspnea and fatigue. Apparently their cardiac reserve mechanisms permitted them an adequate external treadmill exercise response, but since their pacing VFCs were abnormal, hemodynamic studies may have indicated large increases in left ventricular volume and end-diastolic pressure in order to maintain stroke volume during exertion.\textsuperscript{5,6} This type of response has been termed “abnormal left ventricular dynamics” by Ross et al.\textsuperscript{44} and also represents an abnormality of myocardial function.

Hemodynamic studies during exercise in patients with coronary artery disease almost invariably indicate abnormalities of myocardial function, many of which occur prior to the actual manifestation of angina pectoris. These abnormalities include elevations of left ventricular end-diastolic pressure, pulmonary wedge and arterial pressure and systemic arterial pressures, with increases in mean systolic ejection rate, stroke volume and work and cardiac index less than those occurring during the normal exercise response.\textsuperscript{5,6} Obviously, the determination of intracardiac hemodynamics during exercise provides information unavailable from the treadmill test, and, in addition, is more sensitive than the pacing VFC. Unfortunately, being an invasive technique, it cannot be used for routine serial patient follow-up, and one must rely on noninvasive techniques, which may indicate the clinical status more precisely than the myocardial status.

Although atrial pacing is not always as sensitive in determining the abnormal myocardium as exercise stress, it has several important advantages that may offset some of its limitations. The pacing test does not require that a patient be either ambulatory or motivated. It is easier to perform, lacks the systemic, metabolic and hemodynamic changes associated with exercise, and permits precisely con-
trolled and reproducible heart rate changes, which can be useful in comparing the myocardial status before and after intervention. Besides permitting the construction of pacing ventricular function curves, atrial pacing provides information on the level at which angina pectoris occurs, and permits other hemodynamic and electrocardiographic changes to be noted at reproducible heart rates that can be compared before and after various interventions. In our experience it has been possible to construct pacing ventricular function curves in 95 percent of patients with coronary artery disease in the absence of angina pectoris; in approximately 60 percent this function has been abnormal.\textsuperscript{10,11} We have therefore inferred that the pacing ventricular function curve, although less sensitive than hemodynamic studies during exercise, can disclose ventricular abnormality in the absence of overt ischemia. Therefore, this important state of the myocardium can be determined in most patients by a simple reproducible technique. Those patients with significant myocardial abnormalities have the greatest morbidity and mortality from coronary artery surgery.\textsuperscript{43} Therefore, if further studies disclose a good correlation between the pacing VFC and operative risk, we will have a method of distinguishing those patients with the greatest likelihood of doing well from those with the greatest risk.\textsuperscript{43} Another important advantage of the pacing test was shown by the patients included in this study. Although we wished to exercise a consecutive series of patients, it was impossible because eight subjects were considered to have preinfarction or unstable angina, and exertion was believed hazardous. Atrial pacing was carried out without incident, however, and four of eight patients had normal pacing ventricular function curves in the absence of angina pectoris. The ability to evaluate myocardial function in these patients may have important prognostic implications as noted above.

Because of the importance of the myocardial factor in coronary artery disease, the variable progression of the disease, and the introduction of new surgical approaches, as much clinical information as possible is needed to evaluate these patients. Graded exercise studies provide important information that enables us to determine the patient's clinical status, although, as shown in this study, not necessarily disclosing the functional status of the myocardium. When these studies are combined with a hemodynamic evaluation using exercise and/or atrial pacing, information on the patient's myocardial status as well as clinical status can be obtained. If these techniques are utilized before and after various interventions, then the value of the intervention and the fate of the patient may be determined.

References

Dora Angela Duncan (1878-1927) was born in San Francisco. Isadora read everything she could find about dancing. Paris was the scene of her first genuine acceptance as an innovator. Her triumphs in Hungary were duplicated in Germany. Money poured in and she moved as an equal in artistic circles of painters, sculptors and poets. Isadora reached the first crest of her life and of her career in 1904-1905. Her first journey in 1905 was a history-making event. Into the stronghold of classical ballet she went and started an artistic revolution. The Russian appearance successfully completed, Isadora returned to Germany. Walter Damrosch invited her to dance at the Metropolitan Opera and Isadora triumphed. Back in Nice in 1907, she was interested in buying a Bugatti sports car. She ignored advice on what she should wear in an open car and looped a red shawl with its long fringe around her neck. The car jumped forward, the shawl had caught in the spokes of a wheel. In one instance of violent strangulation, Isadora was dead.

Terry, W.: Isadora Duncan—Her Life, Her Art, Her Legacy, New York, Dodd, Meade, 1964