Hemodynamics of the Master Two-Step Test in Hypertension and Healed Myocardial Infarction

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Although the two-step exercise test of Master1 has been used extensively to produce electrocardiographic changes of acute coronary artery insufficiency, results of directly measured hemodynamic changes during the recovery period have not been reported. Indirectly measured hemodynamic changes following this two-step test have been reported infrequently since the tables of standardized stress were derived in 1929 from the recovery heart rates and indirect blood pressures in healthy subjects.2-4 Measurements of the cardiac output by Fick or indicator-dilution principles have not been reported, although the total body oxygen consumption and the energy cost have been determined.5 Hemodynamic data obtained during the change from the upright during exertion to the supine position during recovery might help explain the delay in development of maximal chest pain and of maximal ST-segment changes until the second minute of recovery, as is frequently observed in subjects with coronary artery insufficiency.6

The electrocardiographic two-step test is usually restricted to individuals with normal electrocardiograms but not necessarily to those with normal cardiovascular systems. Therefore, subjects with diastolic hypertension or healed myocardial infarction were studied. None of the subjects had angina pectoris. To determine whether there is a difference in their hemodynamic responses to recovery from exertion a comparison was made between hypertensive subjects with labile diastolic hypertension and those with persistent diastolic hypertension.

From brachial artery pressure and dye-dilution curves alternately recorded in pairs at 1- to 2-minute intervals for 10 minutes with the subject recumbent following a single two-step test, seven hemodynamic events were measured. From these, nine other indices of function were calculated. It was observed that a marked increase in total peripheral resistance during systolic ejection developed during the second and third minutes of recovery in the subjects with persistent diastolic hypertension. The findings suggest an explanation for the delay in the appearance of the maximal chest pain and electrocardiographic changes of coronary artery insufficiency.

Methods

Subjects

A total of 16 subjects were studied. All subjects had a functional capacity of I or II (New York Heart Association). No subject had angina pectoris or an organic heart murmur, and none had been receiving medication. The subjects were divided into three categories by clinical criteria prior to analysis of the data.

The first group, consisting of five Negro subjects, had labile diastolic hypertension with indirect blood pressures that fluctuated over several months of observation from below 140/90 to above 150/100 mm. Hg. Two of the five were men. The mean age was 43 (31 to 52) years. Their mean body surface area was 1.8 M.2. All had normal electrocardiograms and none had cardiomegaly.

The second group, consisting of seven Negro subjects, had persistent diastolic hypertension with indirect diastolic blood pressures that had exceeded 100 mm. Hg persistently during at least 2 months of observation. Four of the seven were men. The mean age was 43 (33 to 53) years. Their mean body surface area was 1.9 M.2. Six subjects had normal electrocardiograms. One had an abnormal tracing. This showed the ST and T changes of left ventricular hypertrophy. Three of the seven subjects had radiographic evidence of cardiomegaly.

The third group, consisting of four Caucasian men without hypertension, had coronary artery...
disease. Each had a myocardial infarction more than 18 months prior to the study. Their mean age was 54 (47 to 70) years, and their mean body surface area was 1.8 M.2. One had a normal electrocardiogram. The other three had abnormal tracings with QRS patterns of old myocardial infarction. One had cardiomegaly by radiography.

**Procedure**

The single two-step test was performed according to the standard technic of Master.1 A practice test was performed by each subject after an 18-gage thin-wall Courmand needle had been percutaneously placed in the right brachial artery and another in a vein of the basilic system at the right antecubital space. Fifteen of the subjects were able to complete the scheduled number of two-step trips within 10 seconds of the standard 90 seconds. Each of the three groups accomplished a mean of 20 two-step trips.

At least 20 minutes after the practice test two successive dye-dilution curves and an intra-arterial pressure curve were recorded with the subject remaining at rest and recumbent. The two-step test was then repeated. Fifteen to 20 seconds following the end of exertion, with the subject again recumbent, the first arterial pressure curve was recorded, followed by a dye-dilution curve the inscription of which began a mean of 30 seconds after exertion. These curves were recorded in successive pairs at minute intervals the first 5 minutes, and at 1- to 2-minute intervals the second 5 minutes.

**Equipment**

The recording equipment consisted of an Electronics-for-Medicine photographic recorder, a Statham P23Db strain gage, a Waters XC 250A densitometer cuvette, Harvard Apparatus Company withdrawal pump (19.4 mL/min.) and a Sanborn 151 direct-writing recorder with which arterial pressures were recorded at a paper speed of 25 mm./sec.

**Dye-Dilution Curves**

Brachial artery dilution curves were obtained after the rapid intravenous delivery of 4 to 6 mg. of indocyanine green from 1-ml. polyethylene cartridges connected in tandem with flushing 10-ml. syringes filled with normal saline. The injection of 10 ml. of normal saline alone fails under these conditions to affect the densitometer significantly. The withdrawn blood was returned immediately. The volume of the withdrawal system between artery and densitometer was 1.2 ml. Calibration of the dye curves was obtained through the continuous successive withdrawal through the densitometer of five blood samples containing known amounts of dye.

Because of the ease of measurement, areas of the dilution curves used in the calculations were determined with a planimeter rather than by the method of Hamilton7 from curves replotted on semilogarithmic paper. The ascending limb of decreasing concentration of the planimetrically measured curves was extended to the baseline by inspection. The cardiac outputs calculated from planimetrically measured areas were compared to the cardiac outputs calculated from replotted curves. The cardiac outputs from the planimetric method exceeded the cardiac outputs from the replotting method by a mean of 182 ± 332 mL/min. (p < 0.001) in 35 curves from the 16 subjects of this study and from 19 other subjects. This difference was greater when determined from curves of large amplitude with a relatively narrow time base (cardiac output < 7.0 L/min.) than when determined from curves of small amplitude with a relatively wider time base (cardiac output > 7.0 L/min.) by 204 ± 162 mL/min. (p < 0.001) and by 163 ± 425 mL/min. (p > 0.05), respectively.

Successive peripherally determined cardiac outputs do not vary significantly with the time between injections in this laboratory. The first of two cardiac outputs obtained by peripheral injection and sampling within 1 to 2 minutes of each other showed a mean that was 56 ± 407 mL/min. larger than the second output in 36 subjects. Cardiac outputs measured 10 minutes after completion of the standard two-step exercise showed a mean of only 5 ± 570 mL/min. greater than the second of the pre-exercise pair of resting cardiac outputs in 19 subjects.

**Measurements and Calculations**

The following were determined for all 16 subjects: heart rate, minute output in L./min., stroke volume in ml., “central blood volume” in ml., and maximal systolic and diastolic pressures in mm. Hg.

Only in the last nine subjects studied were records made at a paper speed that permitted the determination of ejection times and systolic mean pressures. Of these nine subjects three were from the group with labile diastolic hypertension and six were from the group with persistent diastolic hypertension. All nine had normal electrocardiograms.

With these additional measurements the following indices were calculated: the tension time8 or pressure time per stroke and per minute (pressure time per stroke = ejection time in seconds × systolic mean pressure in mm. Hg; pressure time per minute = pressure time per stroke × heart rate); left ventricular systolic ejection rate (ejection rate = stroke volume in ml. per

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ejection time in seconds) systolic total peripheral resistance (systolic peripheral resistance = systolic mean pressure in mm. Hg × 1332 per ejection rate); left ventricular external work per stroke and per minute (left ventricular external work per stroke = 0.0136 × systolic mean pressure in mm. Hg × stroke volume in ml.; left ventricular external work per minute = left ventricular external work per stroke × heart rate) and left ventricular external power per stroke and per minute (left ventricular external power per stroke = left ventricular external work per stroke per ejection time in seconds; left ventricular external power per minute = left ventricular external power per stroke × heart rate).

Sixteen Subjects

All of the mean determinations of heart rate, cardiac output, stroke volume, "central blood volume," and maximal systolic pressure during the first minute of recovery were significantly elevated (p < 0.05) above the resting level for the 16 subjects.* The maximal diastolic pressure was unchanged. Thereafter the rate of return to the resting level varied with the hemodynamic function.

The heart rate returned to the resting level rapidly after exercise (fig. 1A). In 15 of the 16 subjects it had returned to within 10 beats per minute of the resting level by the end of the third minute. It took over 10 minutes to return to this level in one subject with coronary artery disease.

The mean minute-by-minute elevation in the recovery cardiac output and stroke volume can be seen in figure 1B. The cardiac output in 13 of the 16 subjects remained 200 ml. or more above the resting level for at least 7 minutes and four were still above this level at the end of 10 minutes. The mean cardiac output remained significantly different from the resting level through the sixth minute (p < 0.02). The stroke volume had returned

* A table of each value including the maximal diastolic pressure, systolic mean pressure, and ejection time for each of the subjects during each of the 10 minutes of recovery can be obtained from the authors on request.

Figure 1A

Mean change from the resting level in heart rate following a single two-step test in four subjects with coronary artery disease, five with labile diastolic hypertension, and seven with persistent diastolic hypertension.

Figure 1B

Mean change from the resting level in cardiac output (above) and stroke volume (below), as in figure 1A.
Mean Hemodynamic Values at Rest

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DH, entire group with diastolic hypertension; LDH, subgroup with labile diastolic hypertension; PDH, subgroup with persistent diastolic hypertension; CAD, group with coronary artery disease; SD, standard deviation; No., number of subjects per group; BSA, body surface area in square meters; HR, heart rate per minute; SV, stroke volume in ml.; CO, cardiac output in L./min.; CV, central volume in ml.; SP, systolic blood pressure in mm. Hg; DP, diastolic blood pressure in mm. Hg; SM, mean systolic blood pressure in mm. Hg; ET, systolic ejection time in sec.; SER, systolic ejection rate in ml./sec. per stroke; SPR, systolic peripheral resistance in dynes/sec./cm.²; PT, pressure time in mm. Hg sec. per ST (stroke) and per MIN (minute); LVW, left ventricular work per ST and per MIN in Gm. M. and Gm. M./100, respectively; LVP, left ventricular power per ST and per MIN in Gm. M./sec. and Gm. M./sec./100, respectively.

to within 10 ml. of resting by the end of the third minute in all seven with persistent diastolic hypertension but fell to this level within 3 minutes in only two of five subjects with labile diastolic hypertension and in only two of four subjects with coronary disease. The central blood volume returned to within 200 ml. of resting by the end of the third minute in 12 of 16 subjects.

The systolic pressure did not return to the resting level rapidly (fig. 1C). It returned to less than 10 mm. Hg above the resting level within 2 minutes in only two subjects, within 5 minutes in 10 subjects, and in more than 10 minutes in four subjects.

Hypertension

In three of the five subjects with labile diastolic hypertension and in six of the seven subjects with persistent diastolic hypertension additional functions dependent upon ejection time and systolic mean pressure could be calculated. Their mean resting values are summarized in table 1. In the nine subjects the mean heart rate, systolic pressure, cardiac output, stroke volume, systolic mean pressure, pressure time per minute, total peripheral resistance during systole, ejection rate, left ventricular external work, and left ventricular external power indices were significantly different (p < 0.05) from the resting level during the first minute of recovery. All but the systolic peripheral resistance were significantly different during the second min-

**Figure 1C**

Mean change from the resting level in maximal systolic pressure (above) and systolic mean pressure (below) following a single two-step test, as in figure 1A. Note during the first two minutes the significant mean rise in mean systolic pressure compared to the falling maximal systolic pressure of the group with persistent diastolic hypertension.
The diastolic pressure was not significantly different either minute.

The mean ejection, which was significantly shortened \((p < 0.01)\) from 0.279 (0.24 to 0.32) second at rest to 0.252 (0.20 to 0.30) second during the first minute in nine subjects, had returned to the resting level by the end of 2 minutes in seven of the nine subjects. This required nearly 3 minutes for two subjects who had persistent diastolic hypertension.

The systolic mean pressure (fig. 1C) of the nine subjects continued to rise during the second minute of recovery but fell thereafter.

It reached the resting level within 9 minutes in three of the six subjects with persistent hypertension and within 2 to 6 minutes in all three with labile hypertension.

The mean pressure time per stroke (fig. 2A) during the first minute of recovery was approximately the same as at rest in both hypertensive groups. During the second minute there was a significant rise \((p < 0.01)\) above resting in the mean pressure time per stroke in all nine hypertensive subjects. The resting level was reached between 5 and 6 minutes by two subjects with labile hypertension and between 7 and 8 minutes by two with persistent hypertension. In the remaining six subjects the resting level was reached after the ninth minute. The mean pressure time per minute for the nine subjects during the first minute of recovery was nearly double that at rest. The resting level was reached between 2 and 6 minutes by two subjects with labile hypertension and in the seventh minute by

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**Figure 2A**

Mean change from the resting level in pressure time per minute (above) and pressure time per stroke (below) following a single two-step test in three subjects with labile diastolic hypertension and in six with persistent diastolic hypertension. Note the absence of significant change from resting in pressure time per stroke during the first minute of recovery and the significant rise during the second minute in contrast to the elevated but falling pressure time per minute at these same times. The pressure time indices are the same as the tension time indices of Sarnoff.8

**Figure 2B**

Mean change from the resting level in total peripheral resistance during systolic ejection, as in figure 2A. Note during the second and third minutes the significant overshoot above the resting level in the group with persistent diastolic hypertension, as compared to the group with labile diastolic hypertension.
two with persistent hypertension. In the remaining five subjects the resting level was reached after the ninth minute.

The mean systolic peripheral resistance (fig. 2B) was approximately two thirds the resting level during the first minute of recovery. By the end of the third minute the resistance had increased above the resting level in all six subjects with persistent hypertension \((p < 0.02)\). It took 4, 8, and over 10 minutes for the reduced systolic peripheral resistance of the three subjects with labile hypertension to return to the resting level.

The mean ejection rate (fig. 2C) remained significantly elevated above the resting level through the fifth minute \((p < 0.01)\). The resting level was reached by the sixth to seventh minute by one subject with labile hypertension and by three subjects with persistent hypertension. In the remaining five subjects this level was reached after the ninth minute.

The mean left ventricular work and the left ventricular power (fig. 3) elevated 169 to 303 per cent 30 seconds after exercise, fell toward the resting level rapidly during the next 90 seconds, and fell more gradually in the following 8 minutes.

**Group Comparisons**

The four subjects with coronary artery disease differed significantly \((p < 0.05)\) from the 12 with hypertension only in their systolic and diastolic pressure at rest and in the systolic pressure the first minute after exercise.

The three subjects with labile diastolic hypertension differed from the six with persistent diastolic hypertension in six of the 16 functions. At rest the difference was significant \((p < 0.05)\) for systolic and diastolic pressures and the mean systolic pressure. During the first minute following exercise the difference was significant \((p < 0.05)\) for systolic pressure, left ventricular external power per stroke and per minute; during the second minute for systolic pressure and mean systolic pressure; and during the third minute for systolic pressure and systolic peripheral resistance.

**Discussion**

**Indocyanine Dilution Curves**

In the resting state measurements of cardiac output by indicator-dilution technics are reasonably accurate.9 During more dynamic states such as that which immediately follows exertion, the validity of the indicator-dilution method is questionable. The basic assumption of a constant distribution of transit times for the indicator particles traveling through the vascular system10 may not be valid under these circumstances. If it is valid, then a mean cardiac output is determined for the period of the indicator's transit, despite the lack of a "steady state." Should the assumption not be valid and should the hemodynamic changes alter the distribution of the transit times, we believe it is unlikely that the changes occur rapidly enough during the usual mean transit time of 18 seconds to distort the measurement of cardiac output seriously. If it is assumed that these changes occur no more rapidly than the changes in heart rate, which is the most rapidly changing function, then the maximum error in cardiac output determinations would be less than 18 per cent. We therefore believe that this method provides useful and reasonably accurate compara-
tive cardiac output estimates under the conditions of this study.

Rapid repetitive determinations of dye curves might be modified by the hemodynamic changes mentioned above, by dye recirculation, by total circulatory mixing rate of the dye, or by the dye clearance rate. We have no data concerning dye recirculation. The dye mixing rate and clearance rate, however, do not significantly modify subsequent curves, for we have found in 36 subjects that the difference in cardiac outputs from two successive curves recorded peripherally at rest at 1- to 2-minute intervals is not significant.

While the determination of cardiac outputs by indicator-dilution methods under the conditions of this study probably is valid, determination of the "central blood volume," especially early in recovery, is not valid. A "central blood volume" can be determined satisfactorily from the peripheral site in a resting state. But measurements made from the upper extremities in conjunction with lower extremity exercise are subject to large error.11

Two-step Test

The exertion recommended by Master re-

![Figure 3](http://circ.ahajournals.org/)

*Mean change from the resting level in cardiac work (above) and cardiac power (below), as in figure 2A. The stroke indices are on the left and the minute indices are on the right.*

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results in the return of heart rate and blood pressure to within 10 beats per minute and 10 mm. Hg above the resting level by the end of 2 minutes in healthy subjects who recover in the sitting position.\textsuperscript{2} Recovery in the supine position corresponding to the usual technic of the electrocardiographic two-step test was used in this study. While the heart rates generally returned to the resting level within 2 minutes’ time, the blood pressures returned more slowly, as did the other hemodynamic functions. Although this may have been partly due to the reduced functional capacity of this group of subjects, it may also have been due to the differences in position during recovery. This discrepancy between heart rate and other indices of cardiac function further demonstrates that the heart rate taken alone is a very poor index of cardiovascular recovery from submaximal exertion of short duration in subjects with good cardiac reserve.

Clinical observations of several types suggest that the recovery posture after exertion is important. Lying down occasionally precipitates angina pectoris. Also cardiac pain appearing during the two-step test frequently does not become maximal until the second or third minute after lying down, at which time the electrocardiographic ST-T changes are usually maximal.\textsuperscript{6} This pain accentuation during recovery in the supine position occurs at a time when the pain is ordinarily subsiding during recovery in the upright position, in our experience.

An explanation for the supine precipitation or accentuation of cardiac pain and, conversely, its relief by sitting or standing is suggested by the hemodynamic changes noted in the first 3 minutes of recovery after exertion observed in this study. The systolic peripheral resistance and pressure time per stroke were the only functions that increased in amplitude during the second minute of recovery instead of returning toward the resting level, as did the other functions. The pressure time per stroke increased due to the rapid lengthening of ejection time without reduction in the mean systolic pressure. Concurrently, the pressure time per minute decreased owing to the rapid slowing of the heart rate. The pressure time per stroke, which is the same as the tension time index of Sarnoff, and the pressure time per minute are both determinants of myocardial oxygen need \textsuperscript{8} in the absence of a change in heart size. Interestingly, as seen in figure 2A, these two indices have become dissociated. Based upon the pressure time per stroke index the myocardial oxygen consumption per stroke should be increasing, but based upon the pressure time per minute index, the net myocardial oxygen consumption per minute should be decreasing as a result of the decrease in heart rate. Besides the mean systolic pressure, the ejection time, and the heart rate, a determinant of myocardial oxygen consumption not involved in the calculation of the above pressure time indices is the mean ventricular size.\textsuperscript{12} If the heart is enlarging as the pressure time per stroke is increasing, there would be a further increase in myocardial oxygen consumption per minute that could more than compensate for the decrease that would occur as a result of cardiac slowing.

Transient enlargement of the heart on lying down after exertion might possibly occur through three mechanisms. First, a rapid increase of the peripheral resistance with its after loading effects, as during recovery in this study, is followed by a decrease in stroke volume. This could result in an increased residual cardiac volume and mean ventricular size.\textsuperscript{13} Second, in the upright position the heart size is smaller than in the supine position and becomes still smaller during exercise.\textsuperscript{14} Conversely, the heart increases in size, at least returns to normal, following the cessation of exercise, presumably due to a reversal of exercise reflexes. It may further increase in size on the assumption of the supine position due to a shift of blood to the thorax. Third, if angina pectoris results, an additional increase in heart size probably occurs.\textsuperscript{15}

Although total body oxygen demand and external cardiac work are decreasing during the recovery period, internal cardiac work and myocardial oxygen need could increase through the mechanisms described above. This would accentuate pre-existing coronary artery insufficiency and myocardial oxygen deficit.
Consequently, angina pectoris and the associated electrocardiographic changes might first appear or become accentuated several minutes after lying down following exercise.

**Hypertension**

The most interesting difference between the labile and persistent diastolic hypertension groups is in the recovery reactivity of the total peripheral resistance determined during systole. At rest and during the first minute of recovery the peripheral resistance was the same in both groups. Thereafter, the persistent diastolic hypertensive subjects responded with a marked increase of the resistance to a value significantly above the resting level in contrast to the gradual return of the peripheral resistance to the resting level in the group with labile hypertension. The origin of this apparently abnormal reflex response is not readily apparent. Whether these two small groups of subjects represent different stages of the same disease or different conditions cannot be determined from the data.

**Summary and Conclusions**

Peripheral arterial pressure and indicator-dilution curves were recorded alternately in pairs at 1- to 2-minute intervals following the single Master two-step test in five subjects with labile diastolic hypertension, in nine with persistent diastolic hypertension, and in four with healed myocardial infarction. From these curves seven hemodynamic functions were determined. In nine of the 12 hypertensive subjects nine other indices of function were calculated.

In the hypertensive subjects during the first to the third minutes of recovery there was complete dissociation of the pressure time per stroke which was increasing, and the pressure time per minute, which was decreasing. Concurrently the total peripheral resistance during systolic ejection had significantly increased above the resting level after its reduction at the termination of exercise. This rebound in peripheral resistance was limited to the subjects with persistent diastolic hypertension. The significance of these hemodynamics relative to the delayed accentuation of angina pectoris and the associated electrocardiographic ST-T changes 2 to 3 minutes after the termination of two-step exertion is discussed.

**References**

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