The Effects of Training on the Time Components of the Left Ventricle, and Cardiac Time Components: Sedentary versus Active Individuals.

A review of previous research was completed to determine (a) the response of the cardiac time components of the left ventricle to varying types and intensities of training programs, (b) the probable physiological explanations for these responses, and (c) the significance of the changes which did or did not occur. It was found that, at rest, training caused a lengthening in diastole (13.8 percent), ejection period (4.5 percent), and isovolumetric contraction period (9.2 percent). The electromechanical lag shortened by 3.7 percent. Following a submaximal exercise bout diastole was 23.7 percent longer, ejection period 12.8 percent longer, isovolumetric contraction period 14.7 percent longer, and electromechanical lag unchanged as a result of training. These changes are generally thought to reflect adaptation to stress and shifting autonomic nervous control. (A second article, "Cardiac Time Components: Sedentary versus Active Individuals," is attached. This article discusses research which shows that the differences between active and sedentary males parallel the changes that training programs produce in the left ventricular time components. (Author/RC)
An important question concerning the cardiac intervals is whether or not they reflect changes in the human organism as a result of physical training or conditioning. In an attempt to answer this question, a review of the research has been conducted. Specifically, three factors were investigated:

1) The response of each interval to varying types and intensities of training programs;
2) The probable physiological explanations for these responses; and
3) The significance of the changes which did or did not occur.

The number of studies which have investigated training responses of the cardiac time components is small (1-8, 9-17). The training programs were largely concentrated in the walk-jog-run, continuous aerobic-type activities. Only one study (3) dealt with weight training and three (4, 6, 13) with specific sports—these being soccer, a badminton-handball combination, and cycling, respectively. The subjects were generally college or middle-aged men, although two (11, 12) dealt with females. Training lasted anywhere from 6-24 weeks with the subjects working out 1-1/2 to 5 days per week. The exercise stress situations where included were generally bicycle ergometer rides or step tests both submaximal and "all-out"; and, although techniques have now been developed for recording the cardiac intervals during work, the majority of these studies reported post exercise values.

Because of the small total number of subjects and the wide variations in the studies, it was decided to analyze the results in terms of percent change of the mean values pre and post training, both at rest and 1/2 to 1-1/2 minutes after a given submaximal exercise bout. This analysis was done using the factor groupings suggested by Franks and Cureton (7). No information was available for either the isovolumetric relaxation or rapid filling phases.
Mean data are presented in Table 1. These figures vary somewhat from those reported by Franks, et al (9). Reasons for these differences undoubtedly include: 1) all authors in the present review did not analyze all of the cardiac intervals; therefore, different subjects are included in each variable; 2) small variations in the equipment, recordings and measuring techniques existed between studies; 3) data from both males and females of all ages have been treated together; and 4) the time of recording the post-exercise measures varied, as well as the submaximal exercise that was used.

FACTOR I: LEFT VENTRICULAR DIASTOLE

The intervals measuring or showing the highest relationship with diastole showed the greatest response to training (Figure 1). Diastole (dia) lengthened 13.6% at rest (1, 2, 4, 5, 6, 8, 12, 16, 17) and was 23.7% more resistant to shortening from submaximal exercise following training (5, 6, 17). Cycle time (CT) and its inverse, heart rate (HR), both changed approximately 10% at rest (1-8, 10, 12-17), but 16.1% and -13.7% following the standard exercise bout (5, 6, 10, 17). The seeming discrepancy in these latter two figures can probably best be explained as being due to the fact that the heart rate was analyzed using the nearest whole number of beats per minute, whereas the cycle time figures are in the far more exact units of milliseconds.

The slowing down of the heart rate and concurrent lengthening of the cycle time and diastole undoubtedly reflect an increase in the level of parasympathetic influence in the nervous regulation of the heart (7).

This slowing and lengthening are deemed important, since it is the diastole phase in which ventricular rest, ventricular filling and coronary circulation occur. Thus, the fewer number of contractions, longer rest, and more complete
TABLE 1
CARDIAC TIME COMPONENTS - MEAN VALUES

<table>
<thead>
<tr>
<th>Factor</th>
<th>Variable</th>
<th>Pre-Training</th>
<th>Post Training</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Rest</td>
<td>Post Exercise</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Milliseconds</td>
<td>Milliseconds</td>
</tr>
<tr>
<td>1</td>
<td>Diastole</td>
<td>538</td>
<td>337</td>
</tr>
<tr>
<td></td>
<td>Cycle Time</td>
<td>869</td>
<td>583</td>
</tr>
<tr>
<td></td>
<td>Heart Rate</td>
<td>70</td>
<td>105</td>
</tr>
<tr>
<td></td>
<td>(b/min)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Ejection Period</td>
<td>287</td>
<td>203</td>
</tr>
<tr>
<td></td>
<td>Total Systole</td>
<td>391</td>
<td>332</td>
</tr>
<tr>
<td></td>
<td>Mechanical Systole</td>
<td>326</td>
<td>276</td>
</tr>
<tr>
<td>3</td>
<td>Isovolumetric</td>
<td>66</td>
<td>34</td>
</tr>
<tr>
<td></td>
<td>Contraction</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Period</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tension Period</td>
<td>107</td>
<td>105</td>
</tr>
<tr>
<td>4</td>
<td>Electromechanical</td>
<td>54</td>
<td>48</td>
</tr>
<tr>
<td></td>
<td>Lag</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
TRAINING CHANGES

REST

POST SUBMAXIMAL EXERCISE

FACTOR 1
LEFT VENTRICULAR DIASTOLE

PRE TRAINING
POST TRAINING

MILLISECONDS
(1000: 1 SEC.)

DIASTOLE
CYCLE TIME
HEART RATE

S2-Q
R-R'
60/CT

+10.9%
+13.8%
-10%

+16.1%
+23.7%
-13.7%

HEART RATE

HR: B/MIN.
filling represent a more efficiently functioning heart.

FACTOR 2 LEFT VENTRICULAR SYSTOLE

Those intervals associated with left ventricular systole or contraction all showed a tendency to lengthen at rest and to be more resistant to shortening following a submaximal exercise session at the end of training (Figure 2). At rest, total systole (TS) increased 6.1% (2,5,8), mechanical systole (MS) 7.7% (2,5,6,8), and ejection period (EP) 4.5% (2,4,12,14,16,17). The post exercise values increased by 4.2% (5), 8.3% (5,6) and 12.8% (17), respectively. Of the three systolic intervals, the ejection period is the most important and it tends roughly to parallel stroke volume (5,7).

Due to the relationship with stroke volume, a lengthened ejection period would seem to indicate a more complete emptying of the chamber and a larger stroke volume, at rest and in recovery from work. This may, at least in part, be a result of the longer diastole and more complete diastolic filling. As with the lengthening of diastole, this change would be considered a positive cardiovascular response.

FACTORS 3 AND 4 PRE-EJECTION INTERVALS

Tension period (TP), isovolumetric contraction period (ICP), and electromechanical lag (EML) are also part of systole—most specifically, the pre-ejection phases (Figure 3). Tension period, the sum of ICP and EML, exhibited a 9.4% increase at rest (2,3,4,5,11,13,14,17) and a 1.9% greater length following submaximal activity (4,5,17). Its two component parts, however, moved in opposite directions or not at all. At rest, the isovolumetric contraction period lengthened by 9.2% (-4,6,10-12,16,17), while the electromechanical lag shortened by 3.7% (-8,10-12,15-17).
TRAINING CHANGES

FACTORS 3 & 4
SYMPATHETIC TONE, SPREAD OF ELECTRICAL STIMULATION

PRE TRAINING
POST TRAINING

POST SUBMAXIMAL EXERCISE

EEG
EEG

CONTRACTION
CONTRACTION

MECHANICAL PERIOD
MECHANICAL PERIOD

LAG
LAG

Q-S1
TS-EP
MS-EP

ISOVOLUME-TENSION
ISOVOLUME-TENSION

ELECTRIC PERIOD
ELECTRIC PERIOD

PERIOD
PERIOD

+9.2%
+9.4%

+14.7%
+1.6%

0%
-3.7%

MILLISECONDS
(1000 = 1 SEC.)
Following submaximal exercise the ICP increased by 14.7% post training (4,6,17), while the EML showed no variation. The difference in response would tend to reinforce the importance of measuring ICP and EML as separate components and not jointly as TP (7).

Raab (13-15) has been the foremost advocate for interpreting the ICP as an indicator of sympathetic-adrenergic influence on ventricular contractility through the hormones epinephrine (released by the adrenal medulla and transported by the blood to the heart) and norepinephrine (discharged directly into the myocardial cells from the sympathetic nerve endings). As a cardiologist, Raab sees clinical implications for this relationship. His contention is that hypoxic, degenerative (ischemic) heart disease is not caused solely by a decrease in, or lack of, oxygen supply brought about by arteriosclerosis, but also by a parallel increase in oxygen consumption due to the adrenergic catecholamine interference (14). Hence, an individual with a shortened ICP would appear to be one who is sympathetically dominated and potentially prone to heart injury. Conversely, a lengthened ICP would be interpreted as providing the individual with a degree of anti-adrenergic sympatho-inhibitory counter-regulation. With this counter-regulation, the heart functions more efficiently and more economically in the utilization of its oxygen supply. These changes are deemed to be protective to the heart.

In addition to the probable significance of the ICP as a reflection of the sympathetic tone of an individual, it is also an indication of the level of adaptation to the stress of training (7). Generally, if the level of work in conditioning is too strenuous for an individual, the stress is indicated by a shortened ICP. However, as adaptation takes place, the organism is better able
to withstand stress and the ICP lengthens. In this regard, a longer post-exercise ICP would indicate that the standard exercise caused less stress in the individual as a result of the training.

The electromechanical lag is a specific factor and is the most inconsistent of the cardiac time components. It represents the time required for the spread of the electrical stimulation from the Sino-Atrial Node across the ventricles. Although the overall change at rest was a decrease of 3.7%, in almost half of the studies which measured this variable, an increase in length post training was found. In no case, however, was the mean change more than 8 milliseconds.

It appears that the shortening of EML would be a positive change, for a sluggish propagation of the electrical stimulus is often associated with pathological conditions (7).

CONCLUSION

In conclusion, it appears that following adaptation to physical training, the changes which occur in the cardiac time components at rest, that is a lengthened diastole, ICP, EP and a shortened EML, indicate a more efficient and economically functioning heart. The post submaximal exercise changes, specifically less shortening in the diastole, ICP, and EP, and no change in the EML, undoubtedly reflect a decrease in stress response.
BIBLIOGRAPHY


Cardiac Time Components: Sedentary versus Active Individuals

Sharon Ann Plowman
Northern Illinois University
DeKalb, Illinois

It is possible to consider comparison studies of athletes or "highly active" individuals versus normal sedentary or "low active" controls as a form of training study. In studies of this type, the investigator has no control over the type or duration of the training of the individuals, but merely decides by work activity, leisure time pursuits, or competitive status the activity category of each individual.

Less than a dozen of these studies which have been published have used the cardiac interval time components as variables (1-10). All have been concerned with male subjects. None have reported postexercise values for the various intervals. Mean resting values are reported in Table 2. As with the controlled training studies, all comparisons are made in terms of per cent change using the sedentary individuals' values as the baseline.

Active individuals were found to have a 42.2% longer diastole (1,2), a 20.9% longer cycle time (1,2,4,7-10), and a 6.9% slower rate (1,2,4,7-10) than the inactive controls (Figure 4).

The systolic intervals of ejection period, total systole, and mechanical systole measured 1.1% (4,5,8,9), 8.7% (1,2,3,5,9), and 9.6% (1,2,4,9) greater in active individuals than in sedentary ones (Figure 5).

Isovolumetric contraction period was 29.7% longer (2,4,9) and the tension period 15.2% longer (2,4-10) in the more active subjects. Only the electromechanical lag was found to be shorter among athletes and active individuals when compared to their non-athletic sedentary counterparts (1-4,9) (Figure 5). This difference was 2.2% and again a number of studies reported differences in the opposite direction.
<table>
<thead>
<tr>
<th>Factor</th>
<th>Variable</th>
<th>Sedentary Individuals</th>
<th>Active Individuals</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>milliseconds</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diastole</td>
<td>542</td>
<td>771</td>
<td></td>
</tr>
<tr>
<td>Cycle Time</td>
<td>847</td>
<td>1023</td>
<td></td>
</tr>
<tr>
<td>Heart Rate (b/min)</td>
<td>71</td>
<td>59</td>
<td></td>
</tr>
<tr>
<td>Ejection Period</td>
<td>279</td>
<td>282</td>
<td></td>
</tr>
<tr>
<td>Total Systole</td>
<td>367</td>
<td>399</td>
<td></td>
</tr>
<tr>
<td>Mechanical Systole</td>
<td>303</td>
<td>332</td>
<td></td>
</tr>
<tr>
<td>Isovolumetric Contraction Period</td>
<td>74</td>
<td>96</td>
<td></td>
</tr>
<tr>
<td>Tension Period</td>
<td>99</td>
<td>114</td>
<td></td>
</tr>
<tr>
<td>Electromechanical Lag</td>
<td>70</td>
<td>68</td>
<td></td>
</tr>
</tbody>
</table>
SEDENTARY VS. ACTIVE INDIVIDUALS

FACTOR 1
LEFT VENTRICULAR DIASTOLE

SEDENTARY

ACTIVE

DIASTOLE
S2-Q
CYCLE TIME
R-R'
HEART RATE
60/CT

MILLICORDS (1000-1 SL.C.)

HR B/MM

0
50
100
150
200
250
300
350
400
450
500
550
600
650
700
750
800
850
900
950
1000
SEDENTARY VS. ACTIVE INDIVIDUALS

FACTORS 2, 3 & 4
SYSTOLE, SYMPATHETIC TONE, SPREAD OF ELECTRICAL STIMULATION

SEDENTARY

ACTIVE

MILLISERIES (1000: 1 sec.)

ISOVOLUMETRIC CONTRACTION PERIOD MS-EP
TENSION PERIOD TS-EP
ELECTRO-MECHANICAL LAG Q-S1
EJECTION PERIOD C1-C3
TOTAL SYSTOLE Q-S2
MECHANICAL SYSTOLE S1-S2

+29.7%  +15.2%  -2.2%  +1.1%  +8.7%  +9.6%
Thus, the differences between active and sedentary males, a longer diastole, ejection period and isovolumetric contraction period and a shorter electromechanical lag in the actives, were seen to parallel precisely the changes in the left ventricular time components that training programs produced. The physiological explanations and significance of the differences are assumed to be equally parallel and applicable.
BIBLIOGRAPHY

Sedentary vs. Active Individuals


