The Relation of Heart Rate to Cardiovascular Dynamics

Pacing by Atrial Electrodes

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The effects of heart rate on cardiovascular dynamics have been investigated in both human and animal studies.1–8 When the heart rate is altered by a change in state such as exercise, during ventricular pacing, or with the use of pharmacological agents, there are marked changes in cardiac output and other hemodynamic parameters.

The purpose of this study was to investigate the effect of controlled heart rates on the hemodynamics in man at rest and during exercise. The neurohumoral stimuli to the heart remained constant in each of the states tested, rest and exercise, and ventricular activation was unaltered by controlling the heart rate with atrial pacing.

Methods

Ten normal male volunteers ranging from 25 to 45 years of age were studied at cardiac catheterization at rest and during exercise. Right heart catheterization was performed in the postabsorptive, nonsedated state. A bipolar pacemaker catheter was positioned in the right atrium and a Cournand lumen catheter was positioned in the pulmonary artery. The brachial artery was cannulated with an arterial needle.

The right atrial electrode catheter was connected to a cardiac pacemaker and the heart rate was controlled by atrial pacing. Cardiac outputs were determined by the indicator-dilution technique, using indocyanine green and a Gilford densitometer. The dye was injected into the pulmonary artery and the blood was sampled from the brachial artery. The brachial artery pressure was recorded by means of a Statham strain gauge.

A standard lead electrocardiogram, brachial artery pressure, and cardiac outputs were recorded at rest and during the fourth to sixth minute of constant-load, supine leg exercise which was performed on a bicycle ergometer (Collins Electronic Ergometer, 40 to 60 watts, 1,770 to 2,523 foot-pounds per minute). In each state pressures and cardiac outputs were obtained at sinus rhythm, and at various paced heart rates up to 160 beats per minute.

Left ventricular work, peripheral resistance, stroke index and mean systolic ejection rate were calculated using the standard formulae. The tension-time index was calculated from high speed brachial artery pressure curves as described by Sarnoff and associates.11 All dye-dilution curves were recorded in duplicate, and the areas under the curves were measured by planimetry. Careful attention was paid to the grounding of all equipment in order to prevent the accidental induction of atrial fibrillation. All records were taken on a multichannel oscillographic photographic recorder, and the brachial artery pressures were recorded at a paper speed of 100 mm/second.

Results

Cardiac Index (C.I.) (Fig. 1)

There was no significant change in the C.I. when the heart rate was increased by atrial pacing at rest or during exercise. At paced heart rates comparable to the resting state, exercise increased the C.I. up to 98% with a mean increase of 68%. Patient 4 was anxious throughout the study, and was not in a resting state; this accounts for his high C.I. at rest.

Tension-Time Index (T.T.I.) (Fig. 2)

The T.T.I. was almost linearly related to increases in heart rate in both the resting and exercise states. At rest when the heart rate was increased above the sinus rate by a mean...
Changes in heart rate did not significantly alter cardiac index at rest or during exercise. Exercise increased the cardiac index by a mean of 68%. Patient 4 was not in a basal state because of anxiety. Each number represents a single patient.

Exercise increased the cardiac index by a mean of 68%. The increase in L.V.W. during exercise primarily reflects the increased cardiac output.

Peripheral Resistance (P.R.) (Fig. 4)

There was no significant change in P.R. when the heart rate was increased at rest or during exercise. Exercise resulted in a mean decrease of 38% in the P.R.

Stroke Index (S.I.) (Fig. 5)

The S.I. decreased linearly as the heart rate was increased at rest and during exercise. At rest, when the heart rate was increased by a mean of 83%, there was a mean decrease in the S.I. of 45%. During exercise when the heart rate was increased by a mean of 43% there was a mean decrease in the S.I. of 30%. At comparable heart rates exercise increased the S.I. by a mean of 67%.

Mean Systolic Ejection Rate (M.S.E.R.) (Fig. 6)

There was close to a linear decrease in the
M.S.E.R. as the heart rate was increased at rest or during exercise. At rest when the heart rate was increased by a mean of 83%, there was a mean decrease in the M.S.E.R. of 26%. During exercise when the heart rate was increased by a mean of 43%, there was a mean decrease in the M.S.E.R. of 13%. At comparable heart rates exercise increased the M.S.E.R. by a mean of 52%.

Discussion

The effects of heart rate on cardiovascular dynamics have been previously studied. In those studies heart rate was controlled by ventricular pacing, exercise, or pharmacological agents. Ventricular pacing resulted in aberrant ventricular conduction with an abnormal relationship between atrial and ventricular systole; pharmacological agents and exercise resulted in a change in state. In all of those heart block studies cardiac output was found to vary when the heart rate was altered.

Rushmer measured cardiac outputs in dogs at exercise and found that cardiac output was increased predominantly by accelerated heart rate without much increase in stroke volume. In studies of heart block in humans and animals in which the heart rate was controlled by ventricular pacing, initial
increases in heart rate resulted in an increased cardiac output; however, further increases in heart rate resulted in a decline in cardiac output. Wiggers,\textsuperscript{13} Gilmore,\textsuperscript{14} and Lister\textsuperscript{4} and their associates showed that cardiac performance during ventricular pacing is related to the pacemaker site, and that the optimum heart rate for ventricular pacing will vary with different pacemaker sites.

The results of the present study are in agreement with the findings of Linhart and associates,\textsuperscript{15} who showed that cardiac output was not significantly altered by changes in heart rate, and that at comparable heart rates at rest and during exercise, exercise markedly increased stroke volume. In the present study cardiac output did not change significantly when heart rate was increased by atrial pacing at rest or during exercise. In both states the stroke volume was inversely related to heart rate. At comparable heart rates, exercise resulted in a normal increase in cardiac output by increasing the stroke volume. The T.T.I. was directly related to heart rate and at comparable heart rates was increased by exercise. The S.I. was inversely related to heart rate and at comparable heart rates was increased by exercise. L.V.W. and P.R. did not change significantly when the heart rate was increased; exercise increased L.V.W. and decreased P.R. The M.S.E.R. was inversely related to heart rate. At comparable heart rates

\textit{Figure 3}

Relationship between heart rate and left ventricular work at rest and during exercise. Left ventricular work was not significantly altered by heart rate at rest or during exercise. Exercise increased the left ventricular work by a mean of 70%.
the M.S.E.R. was higher during exercise than at rest. This agrees with the findings of Levine and associates\textsuperscript{16} who found that the exercising, nonfailing left ventricle consistently exhibited a rise in the M.S.E.R.

Sarnoff and associates\textsuperscript{11} have demonstrated that the T.T.I. reflects myocardial oxygen consumption independent of wide fluctuations in cardiac output and coronary blood flow. Katz\textsuperscript{17} stated that blood pressure times heart rate determines myocardial oxygen consumption. It has been generally accepted that the T.T.I. reflects myocardial oxygen consumption at rest. Frank and associates\textsuperscript{19} found that the T.T.I. is an accurate estimate of myocardial oxygen consumption during exercise in patients with mitral stenosis.

The validity of the T.T.I. during exercise has been questioned. Sonnenblick and associates\textsuperscript{19} stated that velocity of contraction is an important determinant of myocardial oxygen consumption. One of the authors\textsuperscript{20} has measured the T.T.I. and the maximal rate of rise of the left ventricular pressure (dp/dt max.) in anesthetized dogs at various paced atrial heart rates with and without an epinephrine infusion. During the control period there was a constant relationship between the T.T.I. and dp/dt max. at all heart rates tested. When the state of the animals was altered by the administration of epinephrine, a new relationship was established between the T.T.I. and dp/dt max.; this new relationship between dp/dt max. and the T.T.I. again re-

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**Figure 4**

Relationship between heart rate and peripheral resistance at rest and during exercise. There was no significant change in peripheral resistance at all heart rates tested at rest or during exercise. Exercise decreased the peripheral resistance by a mean of 38%.
mained constant at all heart rates tested. In any given state the T.T.I. reflects changes in myocardial oxygen consumption when the heart rate is increased by atrial pacing. However, it may not reflect changes in myocardial oxygen consumption when there is a change in state (rest to exercise).

Our results demonstrate that cardiac output is unchanged when heart rate is altered by atrial pacing. Since the T.T.I. is augmented with increased heart rate, a rise in myocardial oxygen consumption is inferred. Therefore, an increase in heart rate unaccompanied by a change in state does not increase cardiac output but may increase myocardial oxygen consumption.

**Summary**

Hemodynamic studies were carried out as heart rates were controlled by atrial pacing on 10 normal patients at rest and during exercise. Each patient served as his own control. In both states tested the cardiac index, left ventricular work, and peripheral resistance were not significantly altered by changes in heart rate. The stroke index and mean systolic ejection rate decreased linearly with heart rate. During exercise the cardiac index increased up to 98% and again remained constant at all paced heart rates tested. At comparable heart rates the tension-time index, left ventricular work, stroke index, and mean systolic ejection rate were higher during exercise than at rest. At comparable heart rates...
peripheral resistance was 38% lower during exercise than at rest. Myocardial oxygen consumption, as inferred from the tension-time index, increased with heart rate in both states.

References
7. Levinson, D. C., Gunther, L., Meehan, J. P., Griffith, S. C., and Spritzler, R. J.: Hemo-

Electroshock Cardiac Resuscitation, 1774

In the register (apparently transactions) of the Royal Humane Society for the year 1774, it is reported that a child, three years old, fell from one pair of stairs window upon the pavement, and was taken up without signs of life. A medical practitioner being sent for, declared that nothing could be done, and the child was irrevocably dead; but a gentleman having proposed a trial of electricity, the parents consented. At least twenty minutes elapsed before he could apply the shock, which he gave to various parts of the body without any appearance of success. On directing a few shocks through the chest, a small pulsation became perceptible, and soon after the child began to sigh, and to breathe though with great difficulty; in about ten minutes, it vomited. A kind of stupor remained for some days; but it was restored to perfect health and spirits in about a week.—WILLIAM W. L. GLENN: The Pacemaker Team. Ann NY Acad Sci 3: 815, 1964.
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