Effect of Age on Pulse Wave Velocity and "Aortic Ejection Time" in Healthy Men and in Men with Coronary Artery Disease

By Ernst Simonson, M.D., and K. Nakagawa, M.D.

THE INCREASE of pulse wave velocity with age is well established and reflects a loss of the elastic properties of the arterial wall. The age trends of pulse wave velocity vary for different arteries. In view of early localization of atherosclerotic changes in the abdominal aorta, we have compared age trends of the aortic pulse wave velocity, measured by impedance plethysmography in normal subjects and in patients with coronary artery disease. Since arterial hypertension as well as age increases the pulse wave velocity, only normotensive patients were selected for this series. Impedance plethysmography is a convenient method for inertia-free recording of the aortic volume pulse from skin electrodes and eliminates the necessity of intrapolation of records from the subclavian or carotid arteries as in earlier investigations.

We have also measured the interval from the earliest QRS deflection in any of simultaneous 3 standard electrocardiographic leads to the foot point of the aortic volume pulse recorded by impedance plethysmogram. This interval consists of the electrical latent period, the period of isometric contraction, and the time from the opening of the aortic valve to the arrival of the pulse wave in the vicinity of the aortic arch. The isometric contraction time is probably the most important of these components. The term "aortic ejection time" is used for abbreviation. The effects of age and of coronary artery disease on this interval was studied for the first time.

From the Laboratory of Physiological Hygiene, University of Minnesota, Minneapolis, Minn.

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Method

We used an impedance plethysmograph developed by Dr. O. H. Schmitt, Department of Biophysics, University of Minnesota. With this instrument, variations of the base line due to varying blood content between a pair of electrodes are electronically eliminated. The record gives an inertia-free recording of the volume pulse due to variation of impedance in an alternating current circuit of 30,000 c.p.s., based on different conductivity of blood and surrounding tissue.

The aortic volume pulse was recorded from an electrode in the area of auscultation of the aortic valve with the second electrode opposite it on the back. Some exploration for the best location was needed, the chest electrode being varied slightly in the area of auscultation of the aortic valve. A satisfactory record could be obtained within a few minutes. The position of the back electrode was not critical. The tracings from the aorta were taken simultaneously with the 3 electrocardiographic standard leads on a 4-channel Sanborn recorder.

For the plethysmographic recording of the femoral pulse, one electrode was placed on the palpable femoral artery at the femoral triangle. The second electrode was placed opposite it on the thigh. The femoral pulse was also recorded simultaneously with electrocardiographic leads I, II, and III.

Impedance plethysmography is an indirect method; the calibration, in terms of units of electrical resistance (1/100 Ohm), cannot be converted into units of flow. This difficulty is not relevant to the present study, however, in which only the foot points of the aortic and femoral pulses were measured. The interval from the earliest QRS deflection in any of the 3 standard leads to the foot point of the aortic pulse ("aortic ejection time") and to the foot point of the femoral pulse was determined. The aortic pulse wave velocity (M/sec) was calculated from the anatomic distance between the electrode positions, divided by the interval between the aortic and femoral pulse. The film speed was 50 mm/sec. (fig. 1).

Fifty-one healthy men were compared with 42 men with coronary artery disease. In the normal group, absence of detectable disease was assured.
Pulse wave velocity also increases with age in the patients with coronary disease, but the patients' values are higher in all age groups. The difference is greatest (and statistical significance highest) in the age group over 60. Since the age distribution in the healthy men and in the patients is similar, comparison of the mean values of the total groups over 40 is justified. It is 6.73 M./sec. in the normal group, and 8.41 M./sec. in the patients. The difference of 1.68 M./sec. is statistically highly significant. If the curves are arbitrarily smoothed out and age differences for the same pulse wave velocity compared, an average difference of 18 years is found between the normal group and the patients.

Table 1 shows the means (M) and standard deviations (SD) of the "aortic ejection time" in the age groups of normal men and of patients with coronary artery disease. There is a slight continuous increase of the ejection time with age in the normal subjects as well.

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**Table 1**

*Age Trends of Aortic Ejection Time in Normal Subjects and in Patients Recorded with an Impedance Plethysmograph*

<table>
<thead>
<tr>
<th>Age group</th>
<th>Normal subject</th>
<th>Coronary patient</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>18 to 29</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>40 to 49</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>50 to 59</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>60 and over</td>
<td>18</td>
</tr>
<tr>
<td>N</td>
<td>18</td>
<td>—</td>
</tr>
<tr>
<td>M</td>
<td>0.087</td>
<td>0.119</td>
</tr>
<tr>
<td>S.D.</td>
<td>0.016</td>
<td>0.025</td>
</tr>
<tr>
<td></td>
<td>40 to 49</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>50 to 59</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>60 and over</td>
<td>18</td>
</tr>
<tr>
<td>N</td>
<td>12</td>
<td>0</td>
</tr>
<tr>
<td>M</td>
<td>0.098</td>
<td>0.1229</td>
</tr>
<tr>
<td>S.D.</td>
<td>0.013</td>
<td>0.016</td>
</tr>
<tr>
<td></td>
<td>40 to 49</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>50 to 59</td>
<td>16</td>
</tr>
<tr>
<td></td>
<td>60 and over</td>
<td>15</td>
</tr>
<tr>
<td>N</td>
<td>16</td>
<td>—</td>
</tr>
<tr>
<td>M</td>
<td>0.103</td>
<td>1.271</td>
</tr>
<tr>
<td>S.D.</td>
<td>0.018</td>
<td>0.015</td>
</tr>
</tbody>
</table>

N, number; M, mean; S.D., standard deviation.

Time in seconds from the onset of the earliest QRS complex of standard limb leads to the foot point of the plethysmogram recorded at the aortic area.

*P < 0.05.

**Results**

Figure 2 shows the mean pulse wave velocity in comparable age groups of healthy men and of those with coronary disease. The lines with asterisks connecting the group means show the statistical significance of differences between age groups and the vertical lines show the statistical significance of differences between patients and normal subjects of the same age. The statistical significance was evaluated by means of the t-test (* = p < 0.05; ** = p < 0.01; *** = p = 0.001). The changes in the slopes of the curves are undoubtedly due to the relatively small number of subjects in each age group. The increasing pulse wave velocity in the normal men confirms previous results and the validity of the method. The

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*The patients were part of a group used for other studies by Dr. Henry Blackburn, Jr., at this Laboratory.*

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Figure 2

Increase of mean aortic pulse wave velocity in normal subjects and in normotensive patients with coronary disease with age. Lines with asterisks show statistical significance between age groups (** = p<0.05; *** = p<0.01; **** = p<0.001). The aortic pulse-wave velocity is significantly higher in the patients.

Discussion

The higher aortic pulse wave velocity in patients with coronary disease than in normal men of the same age probably reflects degenerative changes in the aorta with resultant exaggerated age trends. It is possible that an increase of the aortic pulse wave velocity may precede coronary insufficiency, since the aorta is one of the earliest locations of atherosclerosis. In view of the simplicity of the method—the aortic and femoral impedance plethysmogram takes about the same time as a conventional electrocardiogram—it is suitable for large-scale clinical or epidemiologic exploration for early detection of atherosclerosis. The present study, though encouraging, is only the first step in this direction.

The "aortic ejection time" is physiologically not related to the pulse wave velocity but is presented for the sake of brevity, since the results were obtained with the same procedure on the same subjects.

Prolongation of the "aortic ejection time" with age cannot be due to the interval from the opening of the aortic valve to the arrival of the pulse wave beneath the electrode; the age trend of the aortic pulse wave velocity is opposite, and the time interval is too short to be measured with precision. We may also assume that the electrical latent period is fairly constant. The prolongation is therefore probably due to a prolongation of the isometric contraction time with age. This information could be directly obtained with cardiac catheterization, but impedance plethysmography has the great advantage of simplicity, and the avoidance of discomfort, inconvenience, or risk for the patient. The larger intervals in patients with coronary artery disease may be interpreted as some interference of the isometric contraction by myocardial ischemia or degeneration.

The changes of pulse wave velocity as well as those of the "aortic ejection time" in the patients with coronary artery disease are in the direction of normal age trends. This observation conforms, in general, to earlier experience in studies of peripheral circulation and the electrocardiogram.

Summary

Impedance plethysmograms were taken in 51 healthy men and 42 normotensive men with coronary heart disease from the aorta near the place of auscultation of the aortic valve and from the femoral artery in the femoral triangle recorded simultaneously with the 3 electrocardiographic standard leads.

The aortic pulse wave velocity was determined from the time interval between the foot points of the aortic and femoral volume pulse, and the anatomic distance between the electrodes. It increases with age in normal subjects as well as in patients, but the values are significantly higher in the patients. These re-
Pulse wave velocity

Results are suggestive of accelerated degenerative changes in the aorta, probably due to atherosclerosis.

The interval from the earliest detectable QRS deflection to the foot point of the aortic volume pulse ("aortic ejection time") becomes longer with age in the normal group. In patients with coronary artery disease, the prolongation is significantly larger for each of 3 age subgroups. The results suggest that ventricular ischemia affects ventricular isometric contraction.

Summario in Interlingua

In 51 homines normal e in 42 homines con normotension in le presentia de morbo coronari, plethysmogrammas de impedantia esseva obtenite ab le aorta proxime al sito de auscultation del valvula aortica e ab le arteria femoral in le triangulo femoral, in simultaneitate con le obtention del tres derivationes electrocardiographic standard.

Le velocitate del unda del pulso aortico esseva determinate super le base del intervallo de tempore inter le punctos de base del aortico e del femoral pulso de volumine, insimul con le distantia anatomic inter le electrodos. Iste velocitate cresce con le etate, tanto in patientes como etiam in normales, sed le valores es significativamente plus alte in le patientes. Iste resultatos suggere le presentia de accelerate alterations degeneratorii in le aorta, probabimelemente in consequentia de atherosclerosis.

Le intervallo inter le prime detegibile deflexion QRS e le puncto de base del pulso de volumine aortico ("tempore de ejection aortico") cresce con le etate in le grupo normal e in le patientes, sed in istes—i.e. le grupo de patientes con morbo de arteria coronari—le prolongation es significativamente plus marcate in tres sub-gruppos de etate. Iste resultatos suggere que ischemia ventricular affice le contraction isometric ventricular.

References


Age

To be seventy years young is sometimes far more cheerful and hopeful than to be forty years old.—(To Julia Ward Howe on her seventieth birthday) By Oliver Wendell Holmes, M.D.

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ERNST SIMONSON and K. NAKAGAWA

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