Trends in Systolic Blood Pressure in the Thousand Aviator Cohort over a Twenty-four-Year Period

By Albert Oberman, M.D., M.P.H., Norman E. Lane, LT MSC USN, William R. Harlan, M.D., Ashton Graybiel, M.D., and Robert E. Mitchell, CAPT MC USN

SUMMARY

A cohort of 1,056 normotensive, healthy, young men initially examined in 1940 at the mean age of 24 years was followed at three periodic intervals through 1964. The cohort demonstrated little rise in mean systolic blood pressure beyond age 35; a portion of this cohort showed no change of systolic blood pressure with age.

If the men are classified by quintile according to systolic blood pressure in 1940, those men in the upper quintile tend to remain high; when classified by quintile in 1951, those men at the extremes maintained their relative position through 1964. Predictive utility of a systolic blood pressure may be a function of its actual level as well as the age of the individual.

Two factors further influenced the systolic blood pressure of this cohort, namely, parental longevity and gain in weight. The significant effect of parental longevity became less important relative to gain in weight as time progressed and after 1940 affected primarily those men who had gained weight.

Additional Indexing Words:
Aviation medicine Cardiovascular epidemiology Hypertension
Parental longevity Genetics Obesity

Pickering and Platt, questioning the division of blood pressure into normotension and hypertension, have repeatedly stressed the need for longitudinal information. Previous studies of blood pressure have generally been retrospective without experimental control over the collection of data or have been cross-sectional whereby inferences about the significance of blood pressure at various ages must be generated from measurements in altogether different groups of persons. Prospective investigations directed toward the description of population characteristics related to blood pressure, elucidation of its inheritance, and the change of mean blood pressure with age for the most

From the Department of Epidemiology, University of Michigan School of Public Health, Ann Arbor, Michigan; the Naval Aerospace Medical Institute, Pensacola, Florida; and the Department of Medicine, Medical College of Virginia, Richmond, Virginia.

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Address for reprints: Dr. Albert Oberman, Department of Public Health and Epidemiology, University of Alabama Medical Center, Birmingham, Alabama 35233.
part have been based on single measurements, either initial or most recent. Armitage and Rose have enumerated the advantages and improvements in precision from taking multiple readings. These methodological difficulties have added to the conflicting interpretations of environmental influences, genetic factors, and longitudinal variation in blood pressure.

In the present study, an extension of the work previously reported by Harlan and associates, we have combined systolic readings from a cohort of naval aviators examined periodically from youth to middle age. This cohort, homogeneous in background and environmental circumstances, obviously does not lend itself directly to generalization but does provide an unusual opportunity for the longitudinal study of systolic blood pressure in a group of young men, with minimal extraneous sources of variability. We examined these data collected over a 24-year span for the purpose of determining whether individuals maintain their rank in the distribution of systolic blood pressure through middle age, and the extent to which certain factors, especially parental longevity and gain in weight, modify their position.

Methods

This population consists of survivors of a cohort participating in an investigation termed the “Thousand Aviator Study,” initiated in 1940 to determine important physiological and psychological measures for selection of pilots. The original 1,056 members were drawn from the population of aviation cadets and flight instructors who were at the Pensacola Naval Air Station at that time. All of the men were preselected in that they entered the study at optimal weight and with supine blood pressure of less than 132/86 mm Hg, and had qualified for flight training by passing rigorous medical and flight proficiency examinations. This cohort then in their mid-twenties was reexamined at each of the following times: 1951-52, 1957-58, and 1963-64. Details of the composition of the group, selected characteristics, and various aspects of the examinations are described in previously published monographs.

We present here the findings on blood pressure and related data obtained from these serial examinations at which, respectively, 703 (85%), 785 (96%), and 675 (85%) of the surviving members were examined. Of the original cohort, 213 men died in the Second World War. Of the survivors, 575 men had blood pressure data available from all four examinations; subjects on whom complete data were not available were excluded from some analyses.

The 1940 examination included a supine “casual” blood pressure and a “basal” blood pressure recorded as part of a cold pressor test performed in the manner of Hines and Brown. The blood pressures analyzed in this report are supine “casual” systolic pressures (S.B.P.) taken by the examiner midway through the physical examination. Although diastolic pressure is clinically the preferred index of hypertension, only the systolic pressure is used in the present analyses because of the statistical advantages of less error in the measurement, a wider range of values, and the more linear relationship with age. Moreover, systolic pressure correlates highly with diastolic pressure and is of equal or better value as a risk factor in coronary heart disease.

In this study systolic pressure correlated well with diastolic pressure in 1940 (r = 0.4), 1951 (r = 0.6), 1957 (r = 0.7), and 1963 (r = 0.7).

Because of differences in S.B.P. distribution at each examination, individual values could not be directly compared across time. In order to compare S.B.P. for an individual, T-scores were computed for S.B.P. at each of the four examinations:

\[ T = 10 \left( \frac{x - \bar{x}}{s} \right) + 50 \]

where \( x \) = systolic pressure at any given examination, \( \bar{x} \) = mean systolic pressure of the cohort for that examination, and \( s \) = standard deviation of the cohort for the examination. Even though the variance of S.B.P. was not greatly different at these four different points of time, the potential error from changes in the technique of blood pressure measurement could be minimized. This T-score conversion produces distributions with the same variance in each examination, and a subject’s standing in one examination can be directly compared to his standings in each of the other examinations. The mean of these four T values for an individual then indicates his overall or average position in the S.B.P. distribution for the entire 24-year period of the study. We shall refer to this mean T-score as the mean systolic score (M.S.S.).

Measurement of height, weight, and somatotype were available from the 1940 evaluation. We also used the 1951 weight and heart rate because these values were thought more representative in some ways than those recorded in
Results

Age

Harlan and associates\(^8\) have reported for this cohort the frequency distribution curves of S.B.P. at 5-mm intervals "smoothed" to correct for number bias for each 5-year age interval from 15 to 44 years. The configuration of the frequency distribution for the 40- to 44-age group as reported by Harlan and associates\(^8\) previously corresponds closely to the curves for the 45- to 49-, and the 50- to 54-age groups from the present study (fig. 1), the primary difference being the few more extreme values at the upper end of the distribution with increased age. Systolic blood pressure did not increase with age as expected and ran lower at all ages compared with the National Health Survey\(^17\) or the population of Hamilton and co-workers,\(^18\) but was higher than that in the insurance population studied by Robinson and Brucer\(^19\) (fig. 2). The slope of the curve from the "Thousand Aviator Study" is notably less steep after age 35 than prior to that age. Variance of S.B.P. increased with age as noted previously for this group\(^8\) and others.\(^17\)

Longitudinal Trends

We divided the cohort into quintiles at each examination according to their S.B.P. so that an individual's rank at two different examinations might be comparable even though the distribution of S.B.P. might vary.
or fifth quintile for the remainder of the period of follow-up. There was a striking tendency for those men in the fifth quintile in 1951 to remain there in 1957 and 1963.

We also compared subjects in the upper quintiles (fourth and fifth) in 1963 to the remainder of men with regard to movement among quintiles during the span of the study. Of the cohort in these upper quintiles 9% remained in the same quintile for all four examinations, whereas only 2% of those in the other quintiles did so. Only 8% of those in the upper quintiles were in a different quintile for each examination compared with 15% of those in the lower quintiles. A majority of the group of both the upper and lower quintiles absolutely from one time to the next. Next we traced separately each of the 1940 quintiles from 1951 to 1963 to determine the proportion of men falling into each quintile at subsequent examinations. For example (fig. 3), of those in the lowest quintile (I) in 1940, 16.5% were in the lowest quintile in 1951; 19.8% in the second quintile; 13.2% in the third, and so on. It is immediately apparent that the first four quintiles did not follow any definite pattern in succeeding evaluations, but those men in the fifth quintile, and, to some extent those in the fourth quintile, did maintain upper ranks throughout the study. If, however, the 1951 quintiles are used as the initial criteria for ranking (fig. 4), a more consistent pattern evolves; men in all quintiles tended to maintain their relative position as is evident from the distribution at each subsequent examination. More than one half of the men in the first quintile in 1951 were in either the first or second quintile in 1957 and 1963, and approximately two thirds of those in the fifth quintile in 1951 were in either the fourth

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Figure 3
Percentage distribution of initial quintile (1940) for succeeding examinations.

Figure 4
Percentage distribution of initial quintile (1951) for succeeding examinations.
tiles transgressed three or four quintiles during the four examinations.

Of those 134 men whose S.B.P. exceeded the arbitrary cutoff point of 140 mm Hg at any of the examinations, 2% had such a level in 1940, 50% by 1951, 75% by the mean age of 42 years in 1957, and the remainder exceeded this level in 1963.

Additional information on S.B.P. trends across time is given by the correlation matrix in Table 1. The correlations can in one sense be viewed as reliability coefficients, with varying intervals between retesting. From this standpoint, it is apparent that the prognostic value of a "casual" S.B.P. obtained early in adult life is limited. The S.B.P. in 1940 correlated poorly with that obtained 24 years later. Yet, it must be remembered that in 1940 the men were in optimal physical condition and had a narrow range of S.B.P. Better correlations between S.B.P. in 1940 and other examinations were obtained by using the "basal" S.B.P.20

Heredity

Morrison and Morris21 suggested that the inheritance of blood pressure be studied by dividing subjects into two groups—those persons, one or both of whose parents died at 40 to 64 years, and those whose parents lived to age 65 or beyond. Hypertension, presumably an important factor in shortening life, should be more frequent in those parents dying in middle age. The Kolmogorov-Smirnov test22 has been used to test statistically the difference in the distribution curves of S.B.P. between the men with different histories of parental longevity.5 If the groups have cumulative distributions which diverge significantly at any point, the samples may have emanated from different populations, indicating a possible genetic influence.

Our analyses for the difference in parental longevity were carried out for each examination for S.B.P. and for the M.S.S. representing all examinations. Although the curves for S.B.P. were not different at the 0.05-significance level for any one examination, there was a definite approach to significance with age. The M.S.S., a better index of S.B.P. because it represents the mean of scores in relation to variance over a period of years, did show that those men whose parents lived to old age had a consistently lower cumulative percentage for each M.S.S., significant (P < 0.02) at the most divergent portions of the curves (fig. 5). Furthermore, men whose parents died in middle age had higher S.B.P. at all four examinations and greater increments with age for successive examinations (table 2).

Significantly more men from the fifth quintile in 1940 had short-lived parents compared to those in the first quintile (Student's t-test P < 0.05). The men in these extreme quintiles did not differ significantly in any of the other variables measured in 1940. Yet, in 1951 heart rate and weight variables in quintile I were

Table 1

<table>
<thead>
<tr>
<th></th>
<th>1940</th>
<th>1951</th>
<th>1957</th>
<th>1963</th>
<th>Mean systolic score</th>
</tr>
</thead>
<tbody>
<tr>
<td>1940</td>
<td>1.0</td>
<td>0.19</td>
<td>0.15</td>
<td>0.52</td>
<td></td>
</tr>
<tr>
<td>1951</td>
<td></td>
<td>0.43</td>
<td>0.39</td>
<td>0.69</td>
<td></td>
</tr>
<tr>
<td>1957</td>
<td></td>
<td></td>
<td>0.58</td>
<td>0.79</td>
<td></td>
</tr>
<tr>
<td>1963</td>
<td></td>
<td></td>
<td></td>
<td>0.77</td>
<td></td>
</tr>
</tbody>
</table>

N = 444. Two-tailed significance values for correlation coefficients: r(0.05) = 0.09; r(0.01) = 0.12; r(0.001) = 0.16.

![Figure 5](http://circ.ahajournals.org/)

**Figure 5**

Cumulative percentage distribution of mean systolic scores by parental groups.

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Table 2

Mean Systolic Blood Pressure (mm Hg) and Mean Systolic Score by Parental Longevity Group

<table>
<thead>
<tr>
<th>Age of parents at death</th>
<th>Examination</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Both parents:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt; 64 yr</td>
<td>119.81</td>
<td>122.38</td>
<td>120.98</td>
<td>123.89</td>
<td>49.39</td>
<td></td>
</tr>
<tr>
<td>Either parent:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>40-64 yr</td>
<td>121.30</td>
<td>124.54</td>
<td>124.08</td>
<td>126.70</td>
<td>51.35</td>
<td></td>
</tr>
<tr>
<td>Difference</td>
<td>1.49</td>
<td>2.16</td>
<td>3.10</td>
<td>2.81</td>
<td>1.96</td>
<td></td>
</tr>
</tbody>
</table>

Table 3

Mean Values of Selected Variables for Five Levels of Mean Systolic Score and Correlation Coefficients Between Variables and Mean Systolic Score

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean systolic score</th>
<th>Correlation coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>34.4-44.2 N = 114</td>
<td>44.3-47.9 N = 114</td>
</tr>
<tr>
<td>Endomorphy, 1940</td>
<td>3.06</td>
<td>3.15</td>
</tr>
<tr>
<td>Heart rate, 1951</td>
<td>73.19</td>
<td>74.40</td>
</tr>
<tr>
<td>HT/WT, 1940</td>
<td>4.54</td>
<td>4.49</td>
</tr>
<tr>
<td>HT/WT, 1951</td>
<td>4.25</td>
<td>4.15</td>
</tr>
<tr>
<td>Δ HT/WT, 1940-57</td>
<td>1.63</td>
<td>1.60</td>
</tr>
<tr>
<td>WT, 1940 (lb)</td>
<td>154.81</td>
<td>157.67</td>
</tr>
<tr>
<td>WT, 1951 (lb)</td>
<td>165.48</td>
<td>171.36</td>
</tr>
<tr>
<td>WT, 1957 (lb)</td>
<td>168.59</td>
<td>172.99</td>
</tr>
<tr>
<td>Δ WT, 1940-57 (lb)</td>
<td>13.78</td>
<td>15.33</td>
</tr>
</tbody>
</table>

Two-tailed significance values for correlation coefficients: r(0.05) = 0.09; r(0.01) = 0.12; r(0.001) = 0.16. HT/WT represents 10 times the ratio of height to weight.

significantly different from those in quintile V, but the parental longevity groups showed no such differences.

Separation of groups by parental history of known vascular disease (hypertension, heart disease, or cerebrovascular accident) before the age of 60 revealed no significant differences for S.B.P. or the M.S.S. This discrepancy between parental age at death and history of vascular disease as a discriminant for S.B.P. may perhaps be explained by the inaccuracy of more subtle differences in the family history.

Other Influencing Factors

We divided the cohort into five approximately equal groups on the basis of the M.S.S. to determine the possible influence of pertinent variables on S.B.P. over the years. The gradation of M.S.S. for those variables with a significant correlation coefficient is demonstrated in table 3. Although heart rate in 1951 related to M.S.S., it is evident that those factors associated with weight were the most important. This is more than an artifact of arm size. Removing the effect of weight from arm circumference statistically resulted in a partial correlation of −0.01, indicating that arm circumference itself did not bear on S.B.P. Smoking habits and social status in 1963 were not related to S.B.P. over the period of the study.

Interaction of Heredity and Environment

The combined effect of a gain of more than 20 pounds and short-lived parents on S.B.P. and M.S.S. is shown in figure 6. To test the significance of these differences, we did an analysis of variance (table 4) for the data at each examination and for the M.S.S., using the method of expected cell frequencies to adjust for nonproportional N's. In order to evaluate the changes in the effects of the variables across time, we also estimated the
Systolic blood pressure and mean systolic score by weight gain and parental longevity.

Table 4
Significance of Parental Longevity and Weight Gain on Systolic Blood Pressure by Examination Year and Mean Systolic Score Analyzed by Variance and Estimation of the Components of Variance

<table>
<thead>
<tr>
<th>Source (N = 541)</th>
<th>1940</th>
<th>Examination</th>
<th>1951</th>
<th>1957</th>
<th>1963</th>
<th>Mean systolic score</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F*</td>
<td>P</td>
<td>F</td>
<td>P</td>
<td>F</td>
<td>P</td>
</tr>
<tr>
<td>P†</td>
<td>4.31</td>
<td>&lt; 0.05</td>
<td>3.19</td>
<td>&lt; 0.10</td>
<td>5.55</td>
<td>&lt; 0.025</td>
</tr>
<tr>
<td>W</td>
<td>0.03</td>
<td>NS‡</td>
<td>7.09</td>
<td>&lt; 0.01</td>
<td>9.34</td>
<td>&lt; 0.005</td>
</tr>
<tr>
<td>P × W</td>
<td>0.59</td>
<td>NS‡</td>
<td>0.94</td>
<td>NS‡</td>
<td>4.55</td>
<td>&lt; 0.050</td>
</tr>
</tbody>
</table>

Estimated components of variance (ECV)

<table>
<thead>
<tr>
<th>Source (N = 541)</th>
<th>1940</th>
<th>Examination</th>
<th>1951</th>
<th>1957</th>
<th>1963</th>
<th>Mean systolic score</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F*</td>
<td>P</td>
<td>F</td>
<td>P</td>
<td>F</td>
<td>P</td>
</tr>
<tr>
<td>P</td>
<td>0.91</td>
<td>1.26</td>
<td>2.82</td>
<td>1.99</td>
<td>1.36‡</td>
<td></td>
</tr>
<tr>
<td>W</td>
<td>0.00</td>
<td>3.52</td>
<td>5.54</td>
<td>8.49</td>
<td>1.76‡</td>
<td></td>
</tr>
<tr>
<td>P × W</td>
<td>0.00</td>
<td>4.40</td>
<td>5.48</td>
<td>5.48</td>
<td>0.58‡</td>
<td></td>
</tr>
</tbody>
</table>

*Sources of variance have one degree of freedom.
†P = parental longevity; W = weight gain; P × W = interaction of P and W.
‡NS = not significant.
§Not directly comparable to estimates of variance for years because of a different scale of values.

Both parental longevity (P) and weight gain (W) had significant effects on S.B.P., but the importance of these effects changed with the year of examination. In 1940, parental longevity was significant, weight gain was not, and there was no interaction between...
these variables. After 1940, the estimated component of variance for weight gain (ECV\textsubscript{w}) increased from 0 to 8.49 and exceeded the estimated component of variance for parental longevity (ECV\textsubscript{p}) at each examination. In 1957 and 1963, there was significant PW interaction; ECV\textsubscript{pw} exceeded ECV\textsubscript{p}, indicating that the effect of parental longevity might not exist independently of the interaction of P and W. The effect of parental longevity on S.B.P. was dependent upon which level of weight gain was being considered. While parental longevity had a significant effect in the analysis of variance beyond 1940, it was primarily with those men gaining more than 20 pounds; the mean levels of S.B.P. in 1963 were identical for those gaining less than 20 pounds with dissimilar parental longevity (122.8 versus 122.8). It is apparent both by analysis and by inspection that weight gain was more important than parental longevity as a determinant of S.B.P. From 1951 to 1963, a weight gain of more than 20 pounds superseded the influence of short-lived parents on S.B.P. The men with the highest M.S.S. had short-lived parents and a gain of over 20 pounds, whereas at the opposite end of the scale, although separated from the remainder of the cohort to a lesser extent, were those men with long-lived parents and a gain not exceeding 20 pounds.

**Discussion**

Several important aspects of longitudinal change in blood pressure are disclosed by studies of this cohort. Although the mean blood pressure of the entire group increases with age, blood pressure does not increase with age in every individual, and in fact, in the majority of the group there was a random variation in blood pressure during the 24 years of follow-up. However, a small group of individuals did experience a consistent rise in blood pressure as they grew older, and the increase of mean blood pressure for the group reflected the increase in these individuals. Furthermore, at least two factors that were associated with this increase could be delineated. The subjects with increasing blood pressure had greater increments in weight as they grew older and had short-lived parents. One might expect that a greater rise in blood pressure in particular members of this group would produce a bimodal distribution of blood pressure. However, this has not become evident thus far, perhaps because at least two factors, weight and parental longevity, are involved and because other unidentified factors may contribute significantly to the variation in blood pressure.

The concept of two populations, one with a definite rise in blood pressure during middle-age, and another with little or no rise, has been championed by Platt. A bimodal frequency distribution of blood pressure according to parental longevity would support this single gene hypothesis. On this basis Morrison and Morris in their study of busmen claimed the rise in mean blood pressure in middle age was caused by a minority of men from hypertensive families. These data have been subject to criticism because of small numbers; moreover, the findings could not be duplicated in Western Electric employees, longshoremen, or employees of an engineering firm.

Systolic blood pressure was consistently higher for those men in the cohort with short-lived parents. Furthermore, the difference became more marked with time and reached significance when the M.S.S., the overall index of S.B.P., was used to classify the cohort. Concerning bimodality of blood pressure, McKusick stated: "A study performed in a population as homogeneous as possible in racial background and environmental circumstances and designed to reduce extraneous sources of variability to a minimum might be ideal." Armitage and Rose have further stated that bimodality might be obscured on the basis of a single blood pressure reading. A higher rate of increase of S.B.P. over the duration of the study and a significantly higher M.S.S. in those men with short-lived parents suggests a real dichotomy in this cohort.

Age of parental death appeared to be operative in 1940 with minimal dependence on weight gain, whereas weight gain was of
greater significance from 1951 to 1963. As stated previously, it is not surprising that weight gain was unimportant in 1940 because the men were at that time in optimal physical condition with little excess weight; it is remarkable that the influence of parental longevity on S.B.P. can be shown in such a young group with a limited range of blood pressure. Those men with greater weight gain are even more likely to have high S.B.P. if their parents died in middle age, but for those with 20 pounds or less weight gain, parental longevity is relatively immaterial. The combined effect of weight gain, and parental longevity to a lesser extent, is clearly evident in the M.S.S.

For 24 years the men comprising the upper quintile in 1940 maintained their rank; others did not have a “fixed” rank until 1951 at which time they were still for the most part in their mid-thirties. This trend was discernible despite regression to the mean S.B.P. which undoubtedly occurred. These data suggest that a part of the cohort at the upper end of the S.B.P. distribution in 1940 have their pressure determined at an early age perhaps due to some auto-regulatory mechanism, such as setting of baroreceptors at a higher level.

This predisposition, if representing autonomic balance, is not manifest by significant differences between the extreme quintiles in 1940 in heart rate, weight, or somatotype. Parental longevity does differ between quintiles I and V. Those at the lower end of the S.B.P. distribution do not manifest consistent systolic levels until 1951 after which everyone in the cohort maintains his relative rank in the S.B.P. distribution, especially those in the first and fifth quintiles. Responsibility for this difference in age at attainment of a “fixed” rank between the upper and other quintiles might be attributed to an interplay of environmental and genetic factors shortly after the special circumstances in 1940 when all subjects at optimal physical condition and in a common environment exhibited more uniform systolic pressures. Delayed expression of genetic influences is another possibility.

Although not presented in this report, prediction of future blood pressure was best achieved with the “basal” recording in 1940 from which almost 20% of the variance through age 40 could be estimated; cold pressor data from the original examination was of little value. The M.S.S. related best to S.B.P. at age 40+, either in 1957 or 1963, rather than at earlier readings.

Lability of blood pressure has been considered a pre-hypertensive state or early phase of hypertension, yet the men in the upper portion of the S.B.P. distribution in 1963 had been slightly more stable than the remainder of the cohort. Frequent recordings of blood pressure by Sokolow and associates demonstrated little relationship between variability and mean level of pressure.

We hoped to obviate many of the shortcomings of the cross-sectional study by maintaining a cohort composition constant over time, and using blood pressures singly and in combination from four intervals over a span of 24 years. Moreover, our investigation began when the subjects were in their early twenties, rather than at a chance point in time, enabling direct observations to be made of secular trends and the interplay of heredity and environment.

Efforts were made to reduce error in measurements so that a more precise representation of trends could be made. With rare exception, blood pressures were obtained in a nonclinical setting familiar to the aviator. A variety of environmental and somatic stimuli may still, of course, influence these isolated recordings, but the assumption that the biological variation will be random and not bias the estimate of the population mean is a reasonable one. To eliminate variability among evaluations, T-scores and quintiles, as previously described, were employed. A distinct problem exists though with loss of subjects to follow-up whether by death or otherwise; available data indicated a slightly higher mean S.B.P. in this group.

The minimal increase of S.B.P., especially in the fourth decade of the cohort, might be attributed in some part to loss to follow-up of
the hypertensives, but no doubt in greater part to the initial selection procedure for these men. Addition of lost hypertensives to this population could raise the mean S.B.P. with age, especially since the range of blood pressure was limited at the onset of the study, but would in no way preclude the fact that a segment of this cohort shows no appreciable rise of S.B.P. with age. It is also conceivable that hypertension may become manifest at a later age in such a pre-selected cohort; these men may just now be entering this critical period. Recent studies\(^\text{17}\) have shown S.B.P. to change with age exponentially. On the other hand, Paul and Ostfeld\(^\text{29}\) have pointed out the fallacy of assuming blood pressure to be a function of age. Stampler\(^\text{8}\) reported that approximately 30% of a utility company employees with 20 to 30 years follow-up demonstrated little or no rise of blood pressure with age, and Miall\(^\text{30}\) demonstrated that age was not the primary determinant of rate of rise for S.B.P. in a general population from South Wales. The S.B.P. of insurance applicants studied by Robinson and Brucer\(^\text{10}\) remained stable until after age 40.

The main findings of this investigation, namely, S.B.P. does not necessarily rise with age; individuals at the extremes of the S.B.P. distribution tend to maintain their relative position; and the early influence of both weight gain and parental longevity, separately and combined, on S.B.P. have many possible therapeutic implications. Of further significance is whether there is a perceptible difference in "target organ" damage between those who have attained a given level of S.B.P. by weight gain alone as opposed to those with short-lived parents and weight gain. Tantamount to this latter problem, is the duration of elevated S.B.P. more critical than the age at onset? Early classification of subjects likely to sustain cardiovascular injury by mild elevation of blood pressure appears requisite before guides for therapy of mild hypertension can be structured.

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References


A Refreshing Opinion

The ballast of factual information, so far from being just about to sink us, is growing daily less. The factual burden of a science varies inversely with its degree of maturity. As a science advances, particular facts are comprehended within, and therefore in a sense annihilated by, general statements of steadily increasing explanatory power and compass—whereupon the facts need no longer be known explicitly, i.e. spelled out and kept in mind. In all sciences we are being progressively relieved of the burden of singular instances, the tyranny of the particular. We need no longer record the fall of every apple.—P. B. Medawar: The Art of the Soluble. London, Methuen & Co. Ltd., 1967, p. 114; also distributed by Barnes & Noble, Inc., New York.
Trends in Systolic Blood Pressure in the Thousand Aviator Cohort over a Twenty-four-Year Period
ALBERT OBERMAN, NORMAN E. LANE, LT, WILLIAM R. HARLAN, ASHTON GRAYBIEL and ROBERT E. MITCHELL, CAPT

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