Capacity of Human Coronary Arteries
A Postmortem Study

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With the technical assistance of Olga F. Connolly

A postmortem study of the capacity of the coronary arteries of 100 human hearts is presented. The volume of Schlesinger's barium sulfate-gelatin injection mass entering the coronary arterial tree under standard conditions was taken as a measure of its capacity. The factors analyzed include the weight of the heart in the normal and diseased state, the cross-sectional dimensions of the main coronary arteries near their origins, and the sex, age, and nutrition of the subjects.

The information available concerning the capacity of the coronary arterial tree is derived from such collateral data as the perfusibility of its bed and the dimensions of its main components. Direct observations on the total capacity of the human coronary arteries are reported here. The relationship to the weight of the normal and the diseased heart, and possible variations attributable to sex, age, and nutrition are the aspects dealt with in the analyses. The volume capacity of the coronary arterial tree is also correlated with the cross-sectional dimensions of its main components near their origins.

Method and Materials

An elaboration of a method devised by Schlesinger was used in this investigation. The coronary arteries of hearts obtained at autopsy were injected with a radiopaque mass, laid out in 1 plane, visualized roentgenographically, and dissected. The radiopaque mass consists of barium sulfate in a menstruum of gelatin; it remains confined to the arterial side of the circulation and penetrates regularly to arterioles 40 to 50 μ in diameter. According to Schlesinger and from our own observations it advances only inconstant into vessels of smaller size. By leaving the capillaries and veins uninjected the primary distribution and ultimate terminations of the coronary arterial tree were delimited. A pneumatic apparatus in which the pressure may be accurately controlled was employed for injection. The injection pressure was gradually raised to 200 mm Hg, was maintained at that level for 5 minutes, and then the injection was terminated. Clumps placed about the cut ends of the great vessels prevented leaks.

The capacity of the coronary arteries was determined by the amount of injection mass taken up under the standard conditions outlined above. Burets were used as reservoirs. In preliminary experiments it was found that the barium sulfate-gelatin mixture tended to adhere to the sides of the buret rendering volumetric readings difficult to take. To obviate this difficulty, a long column of silicone of low viscosity (Dow Corning, 5 c.s.) was superimposed on the injection mass in the burets, and its fall was taken as a measure of the amount of injection mass entering the coronary arteries. Roentgenograms of the ventricles sliced in coronal fashion established the completeness of injection of the fine arterial branches.

The mean internal diameters of the left descending, left circumflex, and right coronary arteries were determined from the angiogram of the "unrolled" heart. For this purpose, a measuring magnifier calibrated at 0.01-cm. intervals was used. The "unrolling" of the heart was accomplished by a series of incisions which leaves the interventricular septum intact. The x-ray values employed for all hearts were 100 ma., 42 kv., 0.75 second, and 40 inches (tube to film). Measurements were confined to the first centimeter of each artery. The sum of the cross-sectional areas of all 3 vessels was then derived from the diameters obtained.

The analysis of the capacity of the coronary arteries was limited to 100 out of a total of over 200 hearts processed. Absence of leaks during injection and complete filling of the coronary arteries constituted the 2 criteria for selection. The cross-sectional areas of the 3 coronary arteries were obtained in 50 of these 100 hearts.

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Distortion of the lumen or mural calcification in the arterial segments studied precluded reliable measurements in the others.

**Observations**

The capacity of the coronary arteries in cubic centimeters is expressed both as actual values (absolute capacity), and in terms relative to 100 Gm. of heart weight (relative capacity). The latter was obtained by dividing the capacity of the coronary arteries by the weight of the heart and multiplying the ratio so derived by 100. The recorded weight of the heart excludes the injection mass within the coronary arteries and includes usually less than 1.5 cm. of the aorta and pulmonary artery.

The results of the analyses are presented in the form of comparisons of group means, variability, and frequency distributions. The level of significance adopted was a probability of 0.05.

**Heart Weight and Sex**

Figure 1 presents the distribution of the absolute and relative capacities of the coronary arteries of 100 human hearts grouped according to weight and sex.

There were 57 male hearts weighing from 208 to 620 Gm. and 43 female hearts weighing between 182 and 552 Gm. Thirty-seven hearts (17 males, 20 females) exceeded the upper limits of normal size arbitrarily set at 399 Gm. for male, and 349 Gm. for female hearts.

The capacity of the coronary arteries varied greatly from heart to heart. The absolute capacities ranged from 5.3 to 17.7 ml. among males, and from 4.7 to 17.7 ml. among females. The corresponding values for the relative capacities were from 1.88 to 4.50 ml./100 Gm. for males and from 1.95 to 6.42 ml./100 Gm. for females, respectively.

In both males and females, a rise in the weight of the heart was associated with an increase in the capacity of the coronary arteries. However, the enlargement of the coronary arteries appeared not to keep pace with that of the cardiac mass. Although heavy hearts gave larger absolute capacities than small hearts (fig. 1A), the capacity of the coronary arteries per 100 Gm. of heart weight was actually less in the heavy hearts than in the small ones (fig. 1B).

Within each weight class, differences in the absolute and relative capacities of the coronary arteries between males and females were negligible.

**Cardiac Pathology**

All hearts in the series were grouped into 4 pathologic categories:

**A. Myocardial Infarcts: Fourteen Hearts (8 Males, 6 Females).** This group contained
13 healed and 5 recent infarcts. Twenty-five separate occlusions were demonstrated in the coronary arteries of 12 hearts. In 2, infarction was associated with stenosing coronary atherosclerosis without occlusion.

B. Chronic Valvular Disease: Seven Hearts. Conspicuous deforming lesions of the valves were present in 3 hearts with aortic stenosis (1 male, 2 females) and 4 with mitral stenosis (1 male, 3 females).

C. Miscellaneous Forms of Cardiac Pathology: Ten Hearts. Comprising this group are 4 hearts with acute bacterial endocarditis (1 male, 3 females), 2 with nonbacterial endocarditis (2 females), and 4 with cardiac mural thrombosis not associated with valvular disease or myocardial infarction (2 males, 2 females).

D. Hearts Not Belonging to Any of the Above Categories: Sixty-Nine Hearts. (44 Males, 25 Females). This control group comprised normal hearts as well as hearts with such abnormalities as cardiac hypertrophy; coronary atherosclerosis not associated with severe stenosis or occlusion of the coronary arteries; focal fibrosis near the annulus fibrosus, in the tips of papillary muscles, or the vicinity of blood vessels; nondeforming atherosclerosis of the valves or valve rings; and patches of epicardial thickening.

The distribution of the absolute and relative capacities of the coronary arteries of all hearts, rearranged according to weight and pathologic category, is shown in figure 2.

The presence of cardiac pathology other than hypertrophy appeared not to alter appreciably the capacity of the coronary arteries. Within each weight class no significant difference in the mean absolute or relative capacity of the coronary arteries was established between hearts with and those without pathology, or between hearts with and those without myocardial infarction (table 1).

Age

The mean ages for males and females in the series were 80.9 years and 68.4 years, respectively. Eighty-nine subjects (48 males, 41 females) fell between the sixth and ninth decades. Thirty-eight (20 males, 18 females) were in the eighth decade. The youngest heart was from a boy of 13 years; the oldest, from a man of 95 years.

The age distribution of all subjects grouped according to heart weight is listed in table 2. The differences in mean age of subjects distributed among the various (heart) weight classes were statistically negligible.

In order to evaluate further whether or not age has any influence on the capacity of the
coronary arteries, a group of 5 normal hearts representing one extreme of age in our series was contrasted with a similar group of comparable weight representing the other extreme (table 3). No significant difference in mean absolute and relative capacities of the coronary arteries was observed between the 2 groups.

Nutrition

Figure 3 presents the distribution of the absolute and relative capacities of the coronary arteries of all hearts, regrouped according to heart weight and nutritional status of the subjects.

Twenty-eight subjects were emaciated, 18 were obese, and 54 were nutritionally normal at the time of death.

Among hearts weighing from 300 to 349 Gm., the coronary arteries of those from emaciated subjects had significantly larger absolute and relative capacities than the coronary arteries of those from the nonemaciated. Within each of all other weight classes, the
mean absolute and relative capacities of the coronary arteries of hearts belonging to the 3 nutritional categories were of the same order of magnitude (table 4).

**Cross-sectional Areas of the Coronary Arterial Lumens**

The 50 hearts on which measurements of the coronary arterial lumens were made were from 33 males, age 13 to 95 years, and 17 females, age 16 to 88 years. The average age for males was 63.2 ± 20 years (standard deviation) and for females, 62.4 ± 13 years.

Coronary atherosclerosis was slight or absent in all cases. There were no hearts with myocardial infarction.

The heart weights ranged from 182 to 550 Gm. There were 37 normal-sized hearts among which were 2 with acute bacterial endocarditis, 2 with nonbacterial endocarditis, 1 with cardiac mural thrombus, and 1 with mitral stenosis. Hypertrophy was present in 13 hearts, attributed to aortic stenosis in 2, to mitral valvular disease in 1, and to hypertension in 10.

The sums of the cross-sectional areas of the coronary arterial lumens ranged from 18.05 to 45.89 mm². The corresponding values for the capacity of the coronary arteries were 6.5 to 17.7 ml.

The sums of the cross-sectional areas of the major coronary arteries of hypertrophic hearts were significantly larger (mean ± st. dev. : 36.76 ± 8.8 mm²) than those of normal-sized hearts (mean ± st. dev. : 29.54 ± 6.0 mm²) (fig. 4).
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TABLE 4.—The Capacity of the Coronary Arterial Tree in 100 Human Hearts Grouped According to Heart Weight and Nutritional Category

<table>
<thead>
<tr>
<th>Absolute capacity of the coronary arterial tree (ml.)</th>
<th>Heart weight (Gm.)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>182 to 249</td>
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<tr>
<td></td>
<td>250 to 299</td>
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<tr>
<td></td>
<td>300 to 349</td>
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<tr>
<td></td>
<td>350 to 399</td>
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<tr>
<td></td>
<td>400 to 449</td>
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<tr>
<td></td>
<td>450 to 499</td>
</tr>
<tr>
<td></td>
<td>500 to 549</td>
</tr>
<tr>
<td></td>
<td>550 and over</td>
</tr>
<tr>
<td>Nutritional category</td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>No. of cases</td>
<td>6</td>
</tr>
<tr>
<td>Mean</td>
<td>10.1</td>
</tr>
<tr>
<td>St. Dev.</td>
<td>2.9</td>
</tr>
<tr>
<td>No. of cases</td>
<td>8</td>
</tr>
<tr>
<td>Mean</td>
<td>8.2</td>
</tr>
<tr>
<td>St. Dev.</td>
<td>1.5</td>
</tr>
<tr>
<td>Emaciated</td>
<td></td>
</tr>
<tr>
<td>No. of cases</td>
<td>0</td>
</tr>
<tr>
<td>Mean</td>
<td>11.6</td>
</tr>
<tr>
<td>St. Dev.</td>
<td>2.1</td>
</tr>
<tr>
<td>Obese</td>
<td></td>
</tr>
<tr>
<td>No. of cases</td>
<td>14</td>
</tr>
<tr>
<td>Mean</td>
<td>9.0</td>
</tr>
<tr>
<td>St. Dev.</td>
<td>2.4</td>
</tr>
<tr>
<td>All cases</td>
<td></td>
</tr>
<tr>
<td>No. of cases</td>
<td>25</td>
</tr>
<tr>
<td>Mean</td>
<td>8.8</td>
</tr>
<tr>
<td>St. Dev.</td>
<td>2.2</td>
</tr>
</tbody>
</table>

A significant positive rectilinear correlation was established between the sums of the cross-sectional areas of the major coronary arteries and the capacities of the coronary arterial tree (coefficient of correlation: 0.64) (fig. 5).

DISCUSSION

Data provided by this investigation seem to establish the fact that the capacity of the human coronary arteries increases with the weight of the heart regardless of cause of hypertrophy, associated cardiac abnormality, or sex, age, and nutritional state of the subject. The exact mechanism by which enlargement of the coronary arteries is brought about remains to be investigated. At least in effect the phenomenon appears to be adaptive, the capacious coronary arterial tree satisfying, to some extent, the demands of the hypertrophied heart for an augmented blood supply.

The evidence seems to indicate that the requirements of the hypertrophic heart may not be fully met in every instance. The ability of the coronary arterial tree to enlarge appeared to vary from heart to heart, and on the average, such enlargement did not keep
pace with the increase in cardiac mass. It has been contended that the potentialities of the heart both for work and for growth vary directly with the available blood supply.\(^5\) If this be granted, then the suggestions may be made: (a) that the disproportion between the capacity of the coronary arteries and the size of the heart may constitute part of the anatomic substrate of the reduced functional reserve in cardiac hypertrophy; and (b) that the degree of enlargement which the coronary arterial tree is capable of attaining may help predetermine the maximum limits of hypertrophy allowed a given heart. It should be pointed out, however, that others have denied the fundamental importance of vascular insufficiency as a factor in the production of myocardial failure in hypertrophic hearts without coronary atherosclerosis.\(^1,^2\)

Our findings relating to hearts with normal vessels confirm the observation that the main coronary arteries enlarge with the heart,\(^2\) and also justify the assumption that the dimensions of the coronary arteries near their origins constitute a fair index of the capacity of the coronary arterial tree as a whole.\(^3\)

An altogether different situation seems to prevail among hearts with severe coronary atherosclerosis. Although there can be little doubt that the lumens of the main coronary arteries were reduced in hearts of our series with coronary narrowings and occlusions, nevertheless, the capacity of the entire coronary arterial tree in these hearts was of the same magnitude as that of hearts in the same weight class with normal vessels. Thus, it would seem that some sort of balance may be struck between the diminution in size of the large coronary arteries and an increase in capacity of other components of the coronary arterial tree. Enlargement of preexisting arterial anastomoses and the development of new ones are known to follow nonfatal restrictions imposed on the coronary arteries.\(^4\) It is not clear from our study whether the presence of such anastomoses accounts in full or only in part for the maintenance of the status quo with respect to coronary arterial volumes in hearts that develop coronary atherosclerosis.

Our findings relating to a decrease in the relative capacity of the coronary arteries in cardiac hypertrophy appear to have been anticipated by the kerosene perfusion experiments on dead hearts performed by Dock.\(^1\) His conclusions and ours are at variance, however, with respect to the influence of age.  

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**Fig. 4 Left.** The distribution of the sums of the cross-sectional areas of the lumens of the main coronary arteries in 37 normal-sized and 13 hypertrophic human hearts.

**Fig. 5 Right.** Regression of the capacity of the coronary arterial tree on the sum of the cross-sectional areas of the lumens of the main coronary arteries for 50 human hearts.
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On the basis of a diminished perfusibility of small old hearts he concluded that the coronary bed is reduced in these hearts. In our study the capacity of the coronary arteries in the senile was no smaller than that in the young.

To our view, the above discrepancy in our respective results may be more apparent than real. There seem to be some grounds for the suspicion that the diminished perfusibility of old hearts may not be due to a reduction in the capacity of the coronary bed. It is well known that with age, the coronary arteries became increasingly tortuous. In moving liquids, deviations from a straight-line motion occasion some loss in energy and pressure. The loss is greater in vessels with sharp bends than in vessels of the same caliber with easy ones. It is more in a series of reflex bends than in one in which the curvature is continuous. It would appear that a heightened resistance to flow attributable solely to the exaggerated tortuosity of the coronary arteries in the aged heart can, to a large extent, account for the diminished perfusibility of these hearts despite the fact that the volume capacity of the coronary arteries may not be reduced.

Indeed, since senescence generally involves arterial dilatation, elongation, and increased tortuosity (senile vascular ectasia), we are constrained to account for the fact that the coronary arterial capacities in the aged heart appeared to be no larger than in the young. Two possible reasons may be cited. First, the capacity of the coronary arteries as determined in this study represents in part the ratio between the resisting powers of the coronary arterial walls and the pressure to which they were subjected from within. Inasmuch as identical injection pressures were used for all hearts, the thinner-walled and more distensible coronary arteries of young hearts may have expanded more than those of the old, thus minimizing whatever differences in capacity might be due to age. A second and perhaps more important reason applies also to the influence of nutrition and is discussed below.

Atrophy of the blood vessels may occur in connection with atrophy of a particular organ or as part of the generalized atrophy of malnutrition or senescence. The contention has been made that the coronary arteries do not atrophy with the heart and are therefore exceptions to the rule. Our findings seem to indicate otherwise. Evidence has been presented that in cachexia the heart mass is reduced to a greater degree than the body mass and that in atrophy the heart is reduced in size frequently to two thirds or one half the normal. It is probably safe to assume therefore that in our series the hearts of emaciated subjects were included in the (heart) weight classes smaller than that they were in prior to emaciation. If the capacity of the coronary arteries remains unaffected by the nutritional state of the subject, the values for the coronary arterial capacities of emaciated hearts should be larger than those of the nonemaciated in the same (heart) weight class. This in general we found not to be true. Thus it would seem that despite the presence of senile vascular ectasia in some hearts from old cachectic subjects, in most instances the loss in cardiac mass was accompanied by a proportionate reduction in the capacity of the coronary arterial tree.

SUMMARY AND CONCLUSIONS

A postmortem study of the volume capacity of the coronary arteries in 57 men and 43 women is presented.

The amount of Schlesinger's barium sulfate-gelatin injection mass entering the coronary arterial tree under standard conditions was taken as a measure of its capacity.

1. The capacity of the coronary arteries increases with the weight of the heart.

2. The relationship between the capacity of the coronary arteries and the cardiac weight is not directly influenced by (a) the presence or absence of cardiac abnormalities other than hypertrophy, or (b) the sex, (c) age, and (d) nutritional status of the subject.

3. In cardiac hypertrophy the increase in the coronary arterial capacity does not keep pace with the cardiac mass.
4. In hearts with normal coronary arteries the capacity of the coronary arterial tree varies in direct proportion to the dimensions of the main coronary arteries near their origins.

5. In hearts with severe coronary atherosclerosis the over-all capacity of the coronary arterial tree may be maintained despite the diminution in size of its larger components.

6. In hearts from emaciated subjects the capacity of the coronary arteries is generally reduced in proportion to the loss in cardiac mass.

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SUMMARIO IN INTERLINGUA

Es presentate un studio post morte del capacitate volumetric del arterias coronari de 57 homines e 43 feminas.

Le proportion del massa inicie de sulfato de barium a gelatina de Schlesinger que penetra le vasculatura coronari sub conditiones standard eseva acceptate como mesura de su capacitate.

1. Le capacitate del arterias coronari cresce con le peso del corde.

2. Le relation inter le capacitate del arterias coronari e le peso del corde non es influentiate directemente per (a) le presentia o absensia de anormalitates cardiae altere que hypertrophia, (b) le sexo del subjecto, (c) su etate, e (d) su stato nutritional.

3. In hypertrophia cardiae, le augmento del capacitate del arterias coronari non es proportional al augmento del massa cardiae.

4. In cordes con normal arterias coronari, le capacitate del vasculatura coronari varia directemente in proportion con le dimensiones del major arterias coronari presso al sito de lor origine.

5. In cordes con sever atherosclerosis coronary, le capacitate total del vasculatura coronary pote esser mantenite in respecto de dimensiones in su componentes major.

6. In subjectos emaciate, le capacitate del arterias coronari es generalmente reducite in proportion al perdita de massa cardiae.

REFERENCES


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