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Normal Human Heart Anatomy Indicates Age-Related Adaptations

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ABSTRACT

Our understanding of age-related alterations in the normal heart has been expanded by recent findings. Age causes considerable increases in heart weight, ventricular septal wall thickness, and maybe left ventricular free wall thickness, as well as valve circumferences. There are increases in fat, collagen, elastin, and lipofuscin in the myocardium. Due to a decrease in base-to-apex dimension, a rightward shift and dilation of the aortic root, and left atrial dilation, the geometry of the heart also alters. Along their appositional surfaces, the aortic and mitral valves thicken and become fibrotic, and their annuli are the sites of collagen degradation, lipid buildup, and calcification. The coronary arteries become convoluted, dilated, and calcified at specific sites. The atria and ventricles exhibit atrophy and lack of specialised conduction tissue. Despite their ordinarily modest nature, these alterations may impair the ability of the ageing heart to adjust to the strains imposed by a number of cardiovascular disorders**.**

Keywords: Specimens, hypertension , evidence , coronary, ventricular, myocardium

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INTRODUCTION

Although anatomy is one of the oldest basic medical L sciences, the majority of information addressing age-related changes in the normal human heart, especially in octogenarians, is derived from older individuals and nonagenarians, only somewhat lately reported (1-10). Development of age-related normative data is crucial since ageing impacts a range of cardiovascular disease diagnostic parameters (11-17). Due to the high frequency of heart illness among the elderly (18), it can be difficult to define age-appropriate reference standards. Although subject screening is essential, overly severe criteria can produce in a superselected group whose results do not truly reflect the characteristics of the overall population (19). It has been asserted that fundamental biologic ageing processes should be absent in the very young and uniformly present in the very old, increase with age, and have no association with particular diseases (10). Despite the fact that knowledge of aging-related alterations in the heart has been considerably improved by recent studies, there are still significant areas of doubt.

Cardiac weight

In 1925, H. L. Smith analysed 1,000 normal hearts at autopsy from patients without a history of hypertension and discovered that male hearts were heavier than female hearts and did not increase with age (11).

However, the majority of subsequent research have indicated that heart weight increases with age, and the number of extremely elderly people has been small and atherosclerosis has not always been ruled out (12-14).

Recently, we investigated the association between age and many conventional cardiac parameters used by pathologists, such as heart weight (2). This study comprised 765 normal hearts dispersed evenly by age and gender between the ages of 20 and 99. Subjects with a history of hypertension or severe coronary atherosclerosis were excluded from the study. The mean heart weight, indexed to body surface area, was independent of age in men under the age of 100 and consistently higher in men than in women (Figure 1). Mean heart weight grew dramatically with age for women, however.

The majority of the increase in cardiac mass in women appears to occur between the fourth and seventh decades, probably due to a minor increase in blood pressure linked with menopause (15).

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In a companion study of 200 hearts from participants under 20 years old, the heart weights of females were initially bigger than those of boys, but this trend reversed around puberty (16). Thus, the effect of age and gender on heart weight index may be partially mediated by sex hormones**.**

Connective tissue

From middle age to old age, there is a rise in interstitial collagen and elastin (11-16) seen microscopically as diffusely dispersed foci of fibrosis within the subendocardium and heart muscle. Age-related fibrosis and atherosclerotic coronary disease have not been fully explored; nevertheless, given its widespread distribution and occurrence in other mammals (12), they are likely independent. These alterations would be expected to enhance myocardial stiffness and decrease compliance (13-15).

Myocyte degeneration

There is a decline in the quantity and an increase in the size of myocytes in the myocardium as a result of ageing (12-16). The frequent presence of nuclear expansion, irregularity, and replication is indicative of polyploidy (17). As a result of aberrant glycogenolysis, focal basophilic cardiac degeneration is nearly universal in old myocardium (17). The functional significance of these modifications has not been studied.

Age-related deposition of lipofuscin, a "wear and tear" pigment, gives the old myocardium a darkened appearance and is commonly referred to as brown atrophy. A classic study employing quantitative fluorescent staining techniques revealed that lipofuscin was absent in the hearts of very young individuals, increased with age, and was present in all hearts of elderly individuals, but did not correlate with the presence of clinically apparent heart disease, such as heart failure (20). As lipofuscin is a solid and occupied up to 10% of the myocardial volume in the very elderly, the authors hypothesised that it may contribute to subclinical cardiac dysfunction in some patients, despite the fact that this is likely a biological ageing phenomenon (20).

The net effect of the aforementioned variations in myocardial composition on human heart mass and wall thicknesses is unclear (12-19).

Amyloid - Unlike "classic" or primary systemic amyloidosis, senile cardiac amyloid is most likely part of a poorly known illness spectrum. Nonetheless, it is included in this debate due to its

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distinctive relationship to ageing. It is present in around 65% of the hearts of people over age 90, but is uncommon before age 60. (20). It is typically identified as an incidental finding during autopsy, where it appears as microscopic semitranslucent yellow deposits lining the endocardium of the left atrium.

In autopsy series (11-12) and, more recently, in living patients (13), extensive senile cardiac amyloid deposition has been associated with congestive heart failure and atrial fibrillation, despite the fact that focal deposition appears to have little functional significance in the majority of individuals.

Chambers

Due to a minor shortening of the base-to-apex (long-axis) dimension as a result of age, the heart's shape alters. It depicts a decrease in the internal systolic and diastolic dimensions of the left ventricle, dilatation and rightward displacement of the aortic root, and dilatation of the left atrium (1,12-14).

These alterations have not been precisely quantified, and their underlying mechanisms are unclear. Shortening the base-to-apex length may cause the mitral chordae tendineae to seem longer than required and permit modest hooding of the posterior leaflet, suggesting mitral valve prolapse (1-14).

Valves

Several age-related alterations in the heart valves may have functional significance. In normal hearts, the aortic and mitral leaflet thicknesses rise gradually with each decade (3), especially around their closure borders (15). Consequently, the weight and area of the individual cusps of the aortic valve increase with age (9). Collagen deposition and degeneration, lipid buildup, and localised dystrophic calcification impact the aortic and mitral valve leaflets and annuli histologically (16).

Aortic valve sclerosis, defined by valve thickness without hemodynamic impairment, is perhaps the most prevalent clinical manifestation of these alterations. Nonetheless, age-related degenerative calcification of an otherwise normal-appearing tricuspid aortic valve can lead to progressive aortic stenosis (17-18). Degenerative disease was the most common cause of aortic stenosis in a recent series of patients receiving aortic valve replacement. In previous surgical

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series, the frequency of degenerative disease as a cause of aortic stenosis had ranged from 10% to 30% (18). As the proportion of aged people in the general population rises, the incidence of symptomatic aortic stenosis may rise and have a major influence on health care expenses (18).

Pericardium

The pericardium is mostly made of collagen bundles grouped in undulating bands that can straighten out to permit a tiny degree of distensibility when pericardial pressure or volume rapidly increases. Age causes the collagen fibres to become straighter and the pericardium to become thicker and more rigid (16,14). It has been proposed that pericardial stiffness may lead to impaired left ventricular diastolic compliance in aged patients (14).

Foramen ovale

In approximately one-third of typical younger-than-30-year-old patients 33% - Percentage of patent 20 foramen ovale Age groups (years)

Age groups (years)

Patent foramen ovale. A. Prevalence of patent foramen ovale in 965 normal hearts per decade. B. Size distribution of patent foramen ovale (PFO) in 263 cases (m = diameter mean). From Hagen et al. I984 (6). with permission (6). Patency of the foramen ovale declines with advancing age, and the size of those that remain patent rises (6). The foramen ovale is potentially patent in around one-fourth of individuals aged 30 to 80 and one-fifth of those older than 80. (6). Paradoxical embolism over a patent foramen ovale may occur more frequently than previously believed and may result in brain infarcts (15).

Cardiac arteries

Independent of cardiac weight, coronary arteries grow convoluted and dilated with age (16), probably due to hemodynamic drag (17). Some data suggests that the number and size of coronary collaterals may grow with age (19), although it is unclear whether this is independent of atherosclerosis. Although atherosclerosis is always seen as a disease, Monckeberg's medial calcification is likely an age-related degenerative process (18). It is an almost universal observation among the elderly, regardless of gender. It probably contributes to the age-related increase in systolic blood pressure in peripheral vasculature. Due to the prevalence of

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degenerative calcification in the elderly, fluoroscopic detection of coronary artery calcification loses its prognostic usefulness for coronary atherosclerosis in persons over 65. (14).

Conduction system

Aging is characterised by fibrosis, lipid infiltration, and loss of specialised conducting proteins throughout the conduction system.tissues (19-21). The number of pacemaker cells in the sinus node may drop by 90% between the ages of 20 and 75. Simultaneously, but to a lesser extent, muscle fibres in the internodal tracts and left bundle fascicles emanating from the bundle of His are lost. These modifications have no relation to coronary artery disease (22). The relevance of these alterations in healthy adults is uncertain, although they may contribute to the age-related fall in maximal heart rate, along with decreasing catecholamine reactivity. These modifications likely contribute to the occurrence of heart block, bundle branch block, and sinus pauses in aged people. Age-related dilatation of the left atrium and senile cardiac amyloid deposition may contribute to the 200-fold increase in the prevalence of atrial fibrillation between the fourth and ninth decades of life (23).

Cardiac endocrine structures

Granules containing atrial natriuretic factor (ANF) have been detected in human atria and, under some conditions, in ventricular myocardium. ANF is a recently described volume-regulating hormone secreted by the heart (21-24).

No research to date have examined whether the number or distribution of ANF granules in the heart changes with ageing. Interestingly, however, it was recently shown that typical aged men have increased amounts of circulating ANF (25-27). Additionally, ANF may play a role in the development of one kind of senile cardiac amyloid (28).

CONCLUSION

The significance of the alterations in the heart's structure that accompany normal ageing varies. Some, such as the deposition of lipofuscin, are presumably not functional.Important and can be considered an epiphenomenon of ageing.

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Others, such as aortic sclerosis and ventricular septal thickening, may resemble illness in certain respects. [29-30]Certain features, such as senile amyloid and calcified mitral annulus, which are strongly correlated with advancing age and particularly prevalent in elderly hearts, are most likely owing to poorly understood disease processes rather than ageing. Given the information currently available, distinctions between the effects of ageing and the consequences of illness, particularly in extremely elderly hearts, are not always clear. Many of the discussed ageing changes may lower the threshold for clinical disease expression (31-32) and thus predispose the elderly to a variety of cardiovascular disorders, such as diastolic heart failure, hypertensive hypertrophic cardiomyopathy, stenotic and regurgitant valves, systolic hypertension, supraventricular arrhythmias, and conduction defects. Longitudinal studies of designated populations with developing technology could aid in elucidating relationships between normal ageing and cardiovascular disease .

ACKNOWLEDGMENTS

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