The Aging Cardiovascular system

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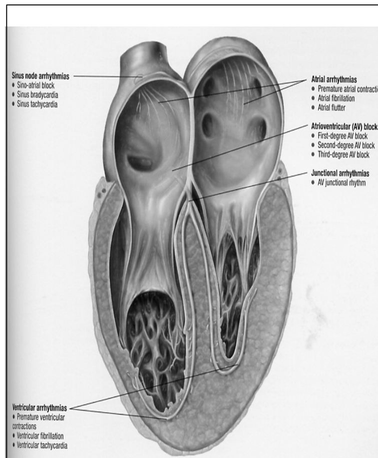
Cardiovascular system

- Heart
- Blood vessel
- Blood



Myocardium: Cardiac muscle *syncytium* (multi-nucleated)

Endocardium: Internal layer of heart

Pericardium: External connective tissue layer of heart

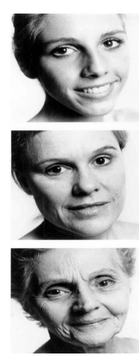
Valves: openings between cardiac chambers (atrial ventricular) or between heart the arteries (aorta and pulmonary)

Conduction system: sinoatrial node (SA) is the pacemaker; also atrial ventricular node (AV), Bundle of His, Purkinje system

Aging & Cardiovascular physiology

- A continuum progressing throughout the individual's life
- A process that is genetically programmed but modified by environment
- Physiologic aging occur more rapidly or more slowly than the chronologic age

Aging & Cardiovascular physiology



- Physical condition can radically affect cardiovascular function in the elderly
- Changes in physical activity can profoundly change cardiovascular function

Miller S J et al. Am J Physiol Heart Circ Physiol 2007;293:H2634-H2643

Cardiac output at rest and with exercise

- Increase in circulating norepinephrine and epinephrine
- Decreased responsiveness to adrenergic stimuli
- Delay in arterial relaxation in response to exercise increases vascular impedance with increasing age
- Increased plasma catecholamines can help compensate for the decrease of β-adrenergic responsiveness

Frank starling mechanism

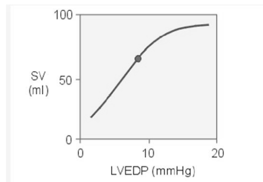


Figure 1. Frank-Starling mechanism. Increasing venous return to the left ventricle increases left ventricular end-diastolic pressure (LVEDP) and volume, thereby increasing ventricular preload. This results in an increase in stroke volume (SV). The "normal" operating point is at a LVEDP of ~8 mmHg and a SV of ~70 ml/beat.

- ↑EDV, dut to :
- Longer diastolic interval(↓HR)
- ↓EF – ↑amount of blood in LV at start of diastole

Response to upright exercise

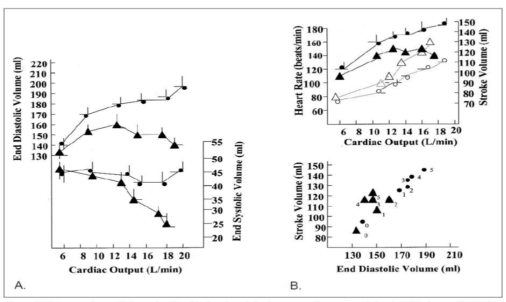


Figure 3. Response to upright exercise in subjects where latent coronary disease is ruled out. Subjects age 65–80 (circles); age 25–44 (triangles). Panel A: In the elderly patients the end-diastolic volume increases with exercise and there is little change in the end-systolic volume. In the younger subjects, the end-diastolic volume changes little and the end-systolic volume falls. Panel B: Heart rate (dotted lines) and stroke volume (solid lines) response to exercise. In the elderly subjects the heart rate increases less than in the younger subjects and the stroke volume increases more. The greater stroke volume in the elderly results from the increased end-diastolic volume (Panel A).
Adapted with permission from Geokas MC, Lakatta EG, Makinodan T, et al. The aging process. Ann Intern Med. 1990;113:455–466.

Changing Myocardial Function & Structure

1. Myocardial contractility
2. Preload
3. Afterload

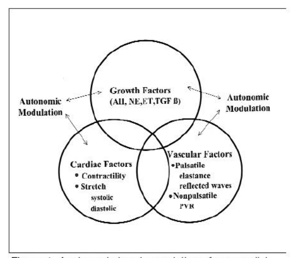
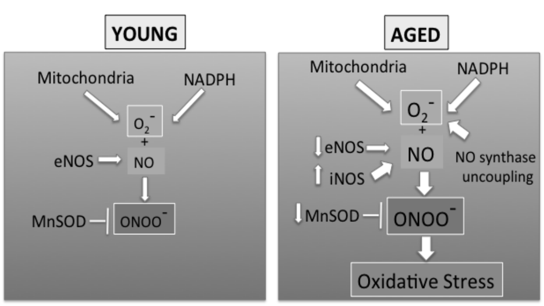


Figure 1. Acute and chronic regulation of myocardial function and structure
AII=angiotensin II; NE=norepinephrine; ET=endothelin; TGF β=transforming growth factor β; PVR=peripheral vascular resistance
Adapted with permission from Lakatta EG, et al. The aging heart. In: Eugene Braunwald, ed. Heart Disease. 5th ed. Philadelphia, PA: WB Saunders; 1997:1691.

Aging & Oxidative Stress



Maximal Oxygen Consumption

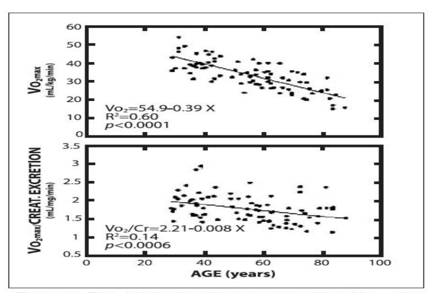


Figure 2. Top: Maximal oxygen consumption (VO_{2max}) as a function of age
Bottom: VO_{2max} normalized for creatinine (Cr) excretion (as a measure of muscle mass) as a function of age. When the decreased muscle mass is taken into account, the age-associated decrease in VO_{2max} rate is markedly attenuated.
Reprinted with permission from J Appl Physiol. 1988;65:1147–1151.[16]

Aging and B-Blockade

Table II. Similarities Between Aging and β Blockade During Aerobic Exercise

	Aging		β Blockade	
Maximal aerobic capacity	↓		↓	
Maximal heart rate	↓		↓	
Maximal end-diastolic volume	↑		↑	
Maximal ejection fraction	↓		↓	
Maximal cardiac output	↓	←→	↓	←→
Maximal plasma catecholamines	↑		↑	

↓=decrease; ↑=increase; ↔=no change
Reproduced with permission from Fleg JL, Gerstenblith G, Lakatta EG. Pathophysiology of the heart and circulation. In: Messerli FH, ed. *Cardiovascular Disease in the Elderly*. 3rd ed. New York, NY: Kluwer Academic Publishers; 1993:53.

Conclusion

The major effects of aging on the cardiovascular system

- decreased compliance of the arterial system, a dropout of myocytes and atrial pacemaker cells
- increasing fibrosis of the cardiac fibrous skeleton
- decreased responsiveness to β-adrenergic stimuli

(which is in part compensated by an increase to be little decrease in myocardial contractility, but there is slowing of ventricular relaxation, an increase in the contribution of atrial contraction to end-diastolic volume, and maintenance of the cardiac output during exercise mainly through the Frank-Starling mechanism)