Hemodynamics and Non-invasive Evaluation in Elderly

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CARDIOVASCULAR DISEASES IN ELDERLY

Endocardium :

- Valvular heart disease
- Infective endocarditis

Myocardium

- Ischemic heart disease
- Myocarditis
- Cardiomyopathy
- Pericardial disease
 - Pericarditis
 - Pericardial effusion
- Aortic disease :
 - Aortic aneurysm
 - Aortic dissection

Hypertension Carotid artery stenosis Renal artery stenosis Extremity artery stenosis Pulmonary artery embolism

Deep vein thrombosis

CARDIOVASCULAR AGING



Major Changes; LV Function

Decreased in rate of myocardial relaxation
⇒LV diastolic dysfunction
⇒LV becomes stiffer and takes longer to relax and fill in diastole



Major Changes; Vascular Structure

Decreased elasticity and compliance of aorta and great arteries

 ⇒ Increased systolic arterial pressure & increased impedance of LV contraction
 ⇒ Mild LV hypertrophy and interstitial fibrosis

Major Changes; Cardiac Structure

- LV thickness progressively increased with ages
- Increase in average myocyte size but decrease in number (apoptosis)
 Increase in intramyocardial fibrosis
 Decreased in overall LV mass

CONDUCTING SYSTEM



Major Changes; Conduction System
A 50-75% loss of pacemaker cells in SA node
⇒ decrease in intrinsic & maximum sinus rate

Preserved number of AV nodal cells
Increase in AV nodal delay (PR interval) ⇒ 1st
degree AV block
Fibrosis and loss of specialized cells in His bundle and bundle branches ⇒ bundle branch block

 Decreased responsiveness to beta-adrenergic receptor stimulation Autoregulation (Frank-Starling "Law of the Heart")

CARDIAC OUTPUT = STROKE VOLUME x HEART RATE (L/min) (beats/min)

Sympathetic _ Nervous System

> Parasympathetic Nervous System

LENGHT/ TENSION AND THE FRANK-STARLING RELATION

VENTRICULA



INITIAL MYOCARDIAL FIBER LENGHT VENTRICULAR END-DIASTOLIC VOLUME

Major Changes; Physiology Cardiac output remains normal at rest, but with slower HR (in elderly) \Rightarrow increased end diastolic volume (EDV) ⇒ increase stroke volume & keep cardiac output normal. • With exercise \Rightarrow decrease in ability to

- achieve maximum HR and O_2 consumption.
- However, ejection fraction (EF) is normal by the increased SV as above.



Cardiovascular Investigations

AIMS OF INVESTIGATIONS

FOR DIAGNOSIS
History + P.E. + Investigations

FOR ASSESSMENT OF
 DISEASE SEVERITY
 DISEASE PROGNOSIS

CARDIAC INVESTIGATIONS

NON-INVASIVE

Blood pressure measurement Oxygen saturation measurement Electrocardiogram (ECG) Chest X-ray (CXR) Laboratory testing 2D-3D Echocardiography Transesophageal echocardiography (TEE) Exercise stress test (EST) or Exercise treadmill⁻ test (ETT) Exercise stress- Echocardiographic study Dobutamine stress echocardiographic study Holter monitoring Tilt table test Carotid artery Doppler study Ankle-brachial index (ABI) study CT-angiography : coronary, pulmonary, aorta, renal artery, peripheral artery Cardiac MRI: rest CMR or stress CMR Stress cardiac nuclear study

INVASIVE

Coronary angiography Left sided cardiac catheterization Right sided cardiac catheterization Pulmonary artery, carotid artery, renal artery, extremity artery angiography Endomyocardial biopsy Electrophysiologic (EP) study

NONINVASIVE INVESTIGATIONS

THE ROLES OF NON-INVASIVE INVESTIGATIONS

- Diagnosis
- Risk/Prognostic Assessment
- Management

Anatomical (non-invasive imaging)
 Functional (non-imaging & imaging)
 Multimodality Noninvasive Testing

Prognostic Assessment Role Predictor of Long-Term Survival with CAD

 Extent of LV dysfunction
 Anatomical extent & severity of atherosclerotic involvement --> number of diseased arteries

Evidence of recent acute coronary syndrome
General health & non-coronary comorbidity

Prognostic Assessment Role Prognostic Information from Stress Testing

Detection of :
Ischemia threshold
Extent and severity of ischemia
Functional capacity

CARDIOVASCULAR INVESTIGATION OPTIONS







2Dimensional Echocardiography (2D-Echo)

FR 39Hz

2D 62% C 50 P Low HGen

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10

EXERCISE STRESS TEST EXERCISE TREADMILL TEST





Exercise-Stress Echocardiographic Study





Dobutamine-Stress Echocardiographic Study





CAVI 6.0-8.0







Gardiac Nuclear Study











Exercise or Dobutamine Stress Echocardiography

Rest SAX HR 59 BPM





50%

JPEG

Exercise or Dobutamine Stress Echocardiography

Rest LAX HR 62 RPI



Peak LAX HR 131 BPM T01 00:00:04

Post LAX HR 82 BPM

T01 00:06:43

50%

JPEG CR 10:1

Exercise or pharmacological stress SPECT myocardial perfusion imaging





Coronary Artery Calcium score (CACS) : LM=27.5, LAD=973, LCX469, RCA=4.7, RI=46

Total CAC score = 1521.4



Cardiac MRI (CMR)





Pharmacological Stress CMR







Male 69 yr, history of MI Contrast-enhanced delayed perfusion MR





Visualization of a stenosis in the LAD with whole-heart coronary MRA



Diagnostic accuracy in identifying significant CAD

~ 72-75%

J Am Coll Cardiol 2006;48:1946-50.

SUMMARY

Several hemodynamics and physiologic changes occur during aging process

 Many cardiovascular investigations [Multimodality investigation] are essential for cardiovascular disease diagnosis, severity & prognosis evaluation