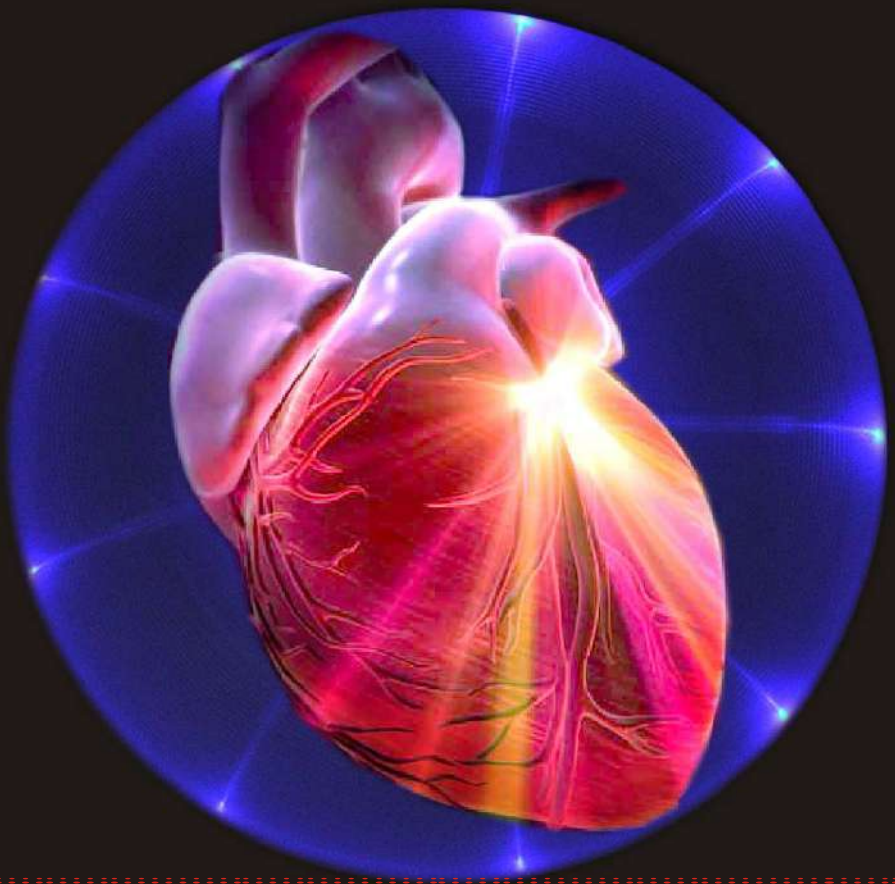
Abstract Book

3-C CON-2007

3RD ANNUAL SCIENTIFIC CONFERENCE
(12TH YEAR OF ACADEMICS)



Dates : **January 26-27, 2007**
Venue : **Tagore Hall, Paldi, Ahmedabad**

Date : **January 28, 2007**
Venue : **R-World, Gandhinagar and Rajpath Club, Ahmedabad**

Organizers and Faculty

Conference Chairman
Dr. Keyur Parikh
Scientific Committee Chairman
Dr. Milan Chag

Conference Director
Dr. Anil Jain
Conference Co-Director
Dr. Srinivas Mallya

Scientific Committee	Course Co-ordinators	Reception Commiittee	Advisory Committee
Dr. Hemang Baxi	Dr. Anish Chandarana	Dr. Urmil Shah	Dr. Ajay Naik
Dr. Dhiren Shah	Dr. Naman Shastri	Dr. Vishal Gupta	Dr. Chirag Mehta
Dr. Satya Gupta	Dr. Joyal Shah	Dr. Vineet Sankhla	Dr. Gunvant Patel

Local Organizing Team

Dr. Bharat Trivedi	Dr. Satish Patel	Dr. Rajan Modi	Dr. Pankaj Manoria
Dr. Kalpana Jain	Dr. Ravi Singhvi	Dr. Urmi Shah	Dr. Mihir Tanna
Dr. Niren Bhavsar			Dr. Jayesh Bhanushali

International Faculty

Banai, Shmuel - Israel	Hjalmarson, Ake - Sweden	Parikh, Manish - USA
Berrebi, Alain - France	Makkar, Raj - USA	Shah, Pravin - USA
Kar, Saibal - USA	Mehta, Sameer - USA	Wijns, William - Belguim
Krucoff, Mitchell - USA	Nair, Anuja - USA	

National Faculty

National Faculty	Joshi, Shashank	Manoria, P.C.	Rohit Manojkumar
Bhargava, Balram	Joshi, Shilpa	Mavalankar, Dileep	Sadikot, S.M.
Bhoraskar, Anil	Kaushik, S.K.	Parashar, S.K.	Singh, Manoj
Chandra, Pravin	Kaul, Upendra	Prakash, V.S.	Sinha, Nakul
Hiremath, Jagdish	Krishnakumar, R.	Prabhakaran, D.	Shrivastava, S.
Jain, P.K.	Kothari, S.S.	Radhakrishnan, S.	

Doctor's Team

Local Organizing Committee

Sitting (Left to Right) : Dr Niren Bhavsar, Dr Kalpana Jain, Dr Vishal Gupta, Dr Bharat Trivedi, Dr Chirag Mehta, Dr Anil Jain, Dr Srinivas Mallya, Dr Naman Shastri, Dr Dhiren Shah, Dr Rajan Modi, Dr Satish Patel

Standing (Left to Right) : Dr Gunvant Patel, Dr Joyal Shah, Dr Urmi Shah, Dr Anish Chandarana, Dr Hemang Baxi, Dr Milan Chag, Dr Keyur Parikh, Dr Urmil Shah, Dr Ajay Naik, Dr Pankaj Manoria, Dr Vineet Sankhla, Dr Mihir Tanna, Dr Satya Gupta

Good People Great Doctors

Message from Conference Chairman

From the desk of Dr. Keyur Parikh - Conference Chairman

Dear Friends,

I am glad to inform you that we have registered over 1100 delegates this year!

When you rule your mind, you rule your world

I will start the conference with my lecture on "Guidelines for Secondary Prevention for Patients with Coronary and Other Atherosclerotic Vascular Disease: 2006 Update" followed by "Principles of Atherogenesis and Pathobiologic Insights into Myocardial Ischemia & Infarction: State of Art"

It is very important to understand both the preventive aspect of Coronary disease as well as understand the insights of the pathogenesis of atherosclerosis.

50% of heart failure patients have Diastolic Dysfunction which contributes equally in the morbidity of heart failure patients. Hear the latest on Diastolic Heart Failure from Dr. Milan Chag.

Make sure you come on time on the first day so as not to miss the first session.

Do you rush so fast from one thing to another, perhaps from one appointment to another, that you fail to notice the advancement of medical science? Begin to bring about a positive change for good by shifting your awareness outside of yourself to learning more of medicine. Someone whose life you touch today can use your gift of kindness. Your sincere caring would be appreciated by patients in your practice.

Our goal as Doctors is restoring the joy of life and happiness to patients and improving quality of care. And by the way, we are confirming January 25, 26, 27 2008 (Republic Day weekend, as usual!) for our 3-C Con 2008, Scientific Session.

- Dr. Keyur Parikh

Message from Organizing Commitee

A Dozen Years in Practice of Cardiovascular Medicine
Welcome to our third international conference on Cardiovascular Medicine and Cardiac Surgery, the 3-C Con 2007, in our twelfth year of organizing academic activities in Cardiology. Our group is now averaging

- Over 5000 Invasive Percutaneous Cardiac procedures annually
- Over 1200 Percutaneous Coronary or Cardiac Interventions annually
- Over 1200 Bypass & Cardiac Surgeries Annually
- Highest EP studies & RF ablations and Device Placements in Gujarat
- Highest SVR and LV Restoration surgeries in India

The overwhelming response to our two previous international conferences, the JIC-2005 and the 3-C Con 2006 and the obligation to teach has inspired us to the next annual cardiology conference, the 3-C Con 2007.

Satellite Sessions : The informal atmosphere of the Satellite Sessions addressing both formal and informal topics have made them a regular event. This year we will have four Satellite Sessions with very interesting topics.

Break Away Sessions : On the last day the delegates will split into four parallel sessions. You may customise your 3-C Con 2007 schedule by choosing one of the four.

CME Certificate : A CME certificate of attendance will be issued.

New Echo Session : We are introducing a new Advanced Echo Symposium this time. Echo lectures for Basic Operators will be on January 27, 2007 evening. For intermediate and advanced operators we are having International and National Faculty for a full day on January 28, 2007.

Our eventual goal is excellent Care of the patient which we will always provide.

Dr. Milan Chag
Dr. Urmil Shah
Dr. Hemang Baxi
Dr. Anish Chandarana
Dr. Ajay Naik
Dr. Satya Gupta
Dr. Gunvant Patel
Dr. Vineet Sankhala
Dr. Pankaj Manoria
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Dr. Mihir Tanna
Dr. Ravi Singhvi
Dr. Jayesh Bhanushali

Dr. Anil Jain
Dr. Srinivas Mallya
Dr. Vishal Gupta
Dr. Bharat Trivedi
Dr. Dhiren Shah
Dr. Rajan Modi
Dr. (Mrs.) Urmi Shah

Cardiac Anaesthetists & Intensivists

Dr. Naman Shastri
Dr. Chirag Mehta
Dr. Kalpana Jain
Dr. Niren Bhavsar
Dr. Satish Patel

3 - C CON 2007
Day-1 Friday 26th January 2007
Tagore Hall, Paldi, Ahmedabad

Time	Programme & Sub-Topic
8:00 am	Registration & Breakfast
	Introduction
	Chair : Dr. Keyur Parikh, Dr. Milan Chag
	Moderator: Dr. Mitchell Krucoff, Dr. Anil Jain
8:30 am	Introduction to the Conference - AHA/ACC Guidelines for Secondary Prevention for Patients with Coronary and Other Atherosclerotic Vascular Disease: 2006 Update - *Dr. Keyur Parikh*
9:20 am	Principles of Atherogenesis and Pathobiologic Insights into Myocardial Ischemia & Infarction: State of Art - *Dr. Keyur Parikh*
9:40 am	Mystery of Diastolic Heart Failure : Is it as deadly ? - *Dr. Milan Chag*
10:00 am	PLENARY LECTURE - Cause for Passion or Cause for Panic: Where Are We With Drug Eluting Stents? - *Dr. Mitchell Krucoff*
10:25 am	Question & Answer
10:35 am	Refreshment Break
	Heart Failure
	Chair: Dr. Keyur Parikh, Dr. Anil Jain
	Moderator : Dr. Urmil Shah, Dr. Dhiren Shah
10:45 am	Coronary Artery Disease and Heart Failure: The Dangerous Intersection - *Dr. Dhiren Shah*
11:00 am	Utility of the Intra-aortic Balloon Pump in Heart Failure and Cardiogenic Shock - *Dr. Urmil Shah*
11:15 am	Percutaneous LV Assist Devices: Overview - *Dr. Raj Makkar*
11:30 am	Novel Therapies for Heart Failure - What has changed in the past 2 years ? - *Dr. Ajay Naik*
11:45 am	Implication of Left Ventricular size and shape on the Heart Failure - *Dr. Anil Jain*
12:05 pm	Question & Answer
12:25 pm	PLENARY LECTURE- Pharmacotherapy of the Heart Failure : Past, Present & Future - *Dr. Ake Hjalmarson*
12:50 pm	Lunch

Valvular Heart Disease

Chair: Dr. Srinivas Mallya, Dr. Anish Chandarana

Moderator: Dr. Vishal Gupta, Dr. Rajan Modi

01:20 pm Infective Endocarditis: 2007 Update - *Dr. Anish Chandarana*

01:40 pm A 35-year-old person with moderately severe MR What should be done ? - *Dr. Srinivas Mallya*

02:00 pm Percutaneous Mitral Valve repair - *Dr. Saibal Kar*

02:20 pm Management of Symptomatic/ Asymptomatic Severe Aortic Stenosis - *Dr. Vishal Gupta*

02:35 pm Question & Answer

02:45 pm PLENARY LECTURE - Cardiovascular Stem Cell Therapy for AMI and CHF: Progress, Pitfalls and Expectations - Dr. William Wijns

Coronary Artery Heart Disease

Chair : Dr. William Wijns, Dr. Hemang Baxi

Moderator: Prof. Shmuel Banai, Dr. Satya Gupta

03.10 pm Stable Angina : State of Art : 2007 - *Dr. Hemang Baxi*

03:30 pm From Vulnerable Plaque to Vulnerable Patient - *Dr. William Wijns*

03:50 pm Drug Eluting Stents - Results in the Real World - *Dr. Pravin Chandra*

04:10 pm Multi vessel Stenting - Has it replaced surgery? - *Dr. Pravin Chandra*

04.25 pm Vulnerable Plaque : Where are we with virtual histology ? - *Dr. Anuja Nair*

04:40 pm No options Therapy for CAD - *Dr. Keyur Parikh/Prof. Shmuel Banai*

04:55 pm Radial A. Vs Femoral A approach in Coronary Intervention -How ? When ? Why? - *Dr. Satya Gupta*

05:10 pm Question & Answer/Refreshment

Special Topics

Chair: Dr. Dileep Mavalankar, Dr. Jagdish Hiremath

Moderator: Dr. Bharat Trivedi, Dr. Vineet Sankhala

05:25 pm "Profits at bottom of pyramid in Cardiovascular Care" - *Prof. Dileep Mavalankar*

05:45 pm "Get Unstuck" - *Dr. Jagdish Hiremath*

06:05 pm Question & Answer

06:15 pm TRANSPORTATION FROM TAGORE HALL TO HOTEL INDER RESIDENCY

08:00 pm SATELLITE SYMPOSIUM

Guidelines Updated on Secondary Prevention for Atherosclerotic Vascular Disease
Dr. Keyur Parikh -Taken from AHA/ACC guidelines)

The American Heart Association (AHA)/American College of Cardiology (ACC) released updated guidelines on secondary prevention for patients with atherosclerotic

Important issues addressed by the committee based on recent evidence included findings from additional lipid reduction trials resulting in

1. new optional therapeutic targets;
2. specific recommendations for clopidogrel use in post–acute coronary syndrome or post–percutaneous coronary intervention–stented patients;
3. recommendations for lower-dose aspirin for chronic therapy;
4. confirmation of the benefit of aldosterone antagonist therapy for patients with impaired left ventricular function;
5. findings of a trial involving angiotensin-converting enzyme (ACE) inhibitor therapy in patients at relatively low risk with stable coronary disease and normal left ventricular function;
6. and a new recommendation regarding influenza vaccination.

Study Highlights

- Clinicians should ask patients with CHD about tobacco status at every visit and urge zero exposure to tobacco smoke. Patients who continue to smoke should be referred to special programs, and pharmacotherapy with nicotine replacement and bupropion should be considered.
- Blood pressure goals for patients with CHD are generally less than 140/90 mm Hg, although patients with concomitant diabetes or chronic kidney disease should have a blood pressure less than 130/80 mm Hg. The authors advocate components of the Dietary Approaches to Stop Hypertension diet, including increased consumption of fruits, vegetables, and low-fat dairy products, to lower blood pressure. Beta-blockers and ACE inhibitors are first-line agents for reducing hypertension in these individuals, and drugs such as thiazide diuretics can then be added to reduce blood pressure levels to goal.
- Dietary therapy for cholesterol control should be instituted for all patients with CHD. No more than 7% of daily calories should be derived from saturated fat, and total cholesterol intake should be less than 200 mg/day. Plant sterol intake of 2 g/day or more or intake of viscous fiber totaling more than 10 g/day can also reduce LDL-C levels. Fish consumption or omega-3 supplements of at least 1 g/day can also improve cholesterol levels.
- LDL-C levels should be less than 100 mg/dL in patients with CHD. Reduction of LDL-C to levels of less than 70 mg/dL is also reasonable. In patients with triglyceride levels of 200 to 499 mg/dL, the goal for non-HDL-C is less than 130 mg/dL. Fibrates or niacin is first-line therapy for patients with triglyceride levels higher than 500 mg/dL to reduce the risk for pancreatitis prior to statin therapy.
- The Adult Treatment Panel III guidelines to lower LDL-C to under 100 mg/dL (but not < 70 mg/dL) for patients with diabetes or with a 10-year CHD risk level of more than 20% have

not changed.

- Patients with CHD should partake in 30 to 60 minutes of moderate-intensity aerobic exercise at least 5 days per week, and the authors also recommend resistance training at least twice per week.
- Weight should be managed to achieve a body mass index between 18.5 and 24.9 kg/m^2 and a waist circumference less than 35 inches in women and less than 40 inches in men. The initial goal of weight loss therapy should be 10% of body weight.
- Diabetes should be managed to achieve a goal glycated hemoglobin level less than 7%.
- Aspirin at a dose of 75 to 162 mg/day should be continued indefinitely for patients with CHD unless contraindications to aspirin therapy exist. Treatment with aspirin should be initiated within 48 hours of coronary artery bypass grafting. Clopidogrel, 75 mg/day, can be used in combination with aspirin therapy for up to 1 year among patients with acute coronary syndrome. Clopidogrel and aspirin should also be used for at least 1 month after a bare metal coronary artery stent is placed, for at least 3 months among patients with sirolimus-eluting stents, and for at least 6 months among patients with paclitaxel-eluting stents.
- ACE inhibitors should be used for all patients with CHD and a left ventricular ejection fraction less than 40%, hypertension, diabetes, or chronic kidney disease. Angiotensin receptor blockers may be considered for patients with those same indications who can not tolerate ACE inhibitors. Aldosterone-blocking agents may be used with beta-blockers and ACE inhibitors among patients with congestive heart failure.
- Beta-blockers should be initiated and continued indefinitely among all patients with CHD with or without symptoms of heart failure.
- Patients with CHD should receive an annual influenza vaccination.

Pearls for Practice

- Patients with CHD should have an LDL-C level at least lower than 100 mg/dL and may have a target LDL-C level lower than 70 mg/dL. However, the current recommendations reiterate that the goal LDL-C level in patients with diabetes or at high risk for CHD remains less than 100 mg/dL but not less than 70 mg/dL.
- ACE inhibitors and beta-blockers are first-line antihypertensive medications among patients with CHD.

Principles of Atherogenesis and Pathobiologic Insights into Myocardial Ischemia & Infarction: State of Art - Dr. Keyur Parikh

Cardiovascular disease has long been the leading cause of death in developed countries, and it is rapidly becoming the number one killer in the developing countries.

Every year, about 5 million people in India,1 million people in the United States and more than 19 million others worldwide experience a sudden cardiac event (acute coronary syndromes and/or sudden cardiac death). Approximately fifty percent of this population has no prior symptom.

Despite major advances in treatment of CHD patients, approximately fifty percent of the victims of the disease who are apparently healthy, die suddenly without prior symptoms.
In three-fourths of patients who present with acute coronary syndromes (ACS), thrombotic occlusion of the coronary arteries occurs as a consequence of rupture of an atherosclerotic plaque. In the remaining one-fourth of cases, surface erosion of the plaque is responsible for luminal thrombosis.

Histopathologic characteristics of ruptured plaques and those vulnerable to rupture are well established Such plaques have thin fibrous caps and are termed *thin cap fibroatheroma (TCFA);* caps thinner than 65 μm are presumed to be vulnerable. Thin caps are often inflamed with macrophage infiltration, and the cap attenuation is directly related to the magnitude of macrophage infiltration.

It is essential to assess total vulnerability burden and not just search for a single, unstable coronary plaque. A composite risk score (e.g., a vulnerability index), that comprises the total burden of atherosclerosis and vulnerable plaque in the coronaries (and aorta, carotid, femoral…arteries), and that includes blood and myocardial vulnerability factors, should be a more accurate method of risk stratification. Such a vulnerability index would indicate the likelihood that a patient with certain factors would have a clinical event in the coming year. Use of the state-of-the-art bioinformatics tools, such as neural networks may provide substantial improvement for risk calculations.

The recognition of the role of the vulnerable plaque has opened new avenues of opportunity in the field of cardiovascular medicine.

1) Rupture-prone plaques are not the only vulnerable plaques. All types of atherosclerotic plaques with high likelihood of thrombotic complications and rapid progression should be considered as vulnerable plaques.
2) Vulnerable plaques are not the only culprit factors for the development of acute coronary syndromes, myocardial infarction, and sudden cardiac death. Vulnerable blood (prone to thrombosis) and vulnerable myocardium (prone to fatal arrhythmia) play an important role in the outcome. Therefore, the term "vulnerable patient" may be more appropriate.
3) A quantitative method for cumulative risk assessment of vulnerable patients needs to be developed .Recently developed assays (eg. C-Reactive Protein), imaging techniques (eg. CTangio and MRI), non-invasive electrophysiological tests (for vulnerable myocardium), and emerging catheters (to localize vulnerable plaque) in combination with future genotyping techniques will guide deployment of new therapies and help reduce the incidence of acute coronary syndromes and sudden cardiac death.

Diagnosis of Vulnerable Plaque / Artery

Major Criteria

The presence of one or a combination of these factors may warrant higher risk of plaque complication.

1. Active Inflammation-Plaques with active inflammation may be identified by extensive macrophage accumulation (especially with activated T-cells and mast cells).

2. A Thin Cap with a Large Lipid Core-These plaques have a cap thickness of <100 μ and a lipid core accounting for >40% of the plaque's total volume.
3. Endothelial Denudation with Superficial Platelet Aggregation-These plaques are characterized by superficial erosion and platelet aggregation or fibrin deposition
4. Fissured/Injured Plaque-Plaques with a fissured cap (most of them involving a recent rupture) that did not result in occlusive thrombi may be prone to subsequent thrombosis, entailing occlusive thrombi or thromboemboli
5. Severe Stenosis-On the surface of plaques with severe stenosis, shear stress imposes a significant risk of thrombosis and sudden occlusion

Minor Criteria

1. Superficial Calcified Nodule-These plaques have a calcified nodule within, or very close to, their cap, and this structure protrudes through and can rupture the cap.
2. Yellow Color (on angioscopy)-Yellow plaques, particularly glistening ones, may indicate a large lipid core and thin fibrous cap, suggesting a high risk of rupture.
3. Intraplaque Hemorrhage-Extravasation of red blood cells, or iron accumulation in plaque, may represent plaque instability.
4. Endothelial Dysfunction- Impaired endothelial vasodilator function occurs in a variety of acute and chronic disease states. Patients with cardiovascular risk factors, acute infections, postprandial hypertriglyceridemia, and hyperglycemia, a high homocysteine level, and a high prooxidant burden may have endothelial dysfunction.
5. Expansive (Positive) Remodeling: Many of the non-stenotic lesions undergo "expansive" or "positive" or "outward" remodeling, namely compensatory enlargement before impinging significantly on the vascular lumen. This phenomenon was considered as "positive" remodeling because the luminal area was not affected and stenosis was the only measure of risk. However, with the emphasis on plaque rupture in non-stenotic lesion, the "positive" remodeling may not be truly positive and beneficial.

Pan-Arterial Approach

Diagnostic methods may focus on the total burden of coronary artery disease
The coronary Calcium Score is a good example of using CT for this purpose.
It will be important in the future to identify plaques that are on a trajectory of evolution towards a vulnerable state, and to be able to target interventions to those plaques most likely to develop thrombosis. It is likely that local hemodynamic factors and 3-dimensional morphology may provide insight regarding the temporal course of an evolving plaque.
New studies are unraveling the role of the adventitia and peri-adventitial connective tissue in vulnerability of atherosclerotic plaques.
New Risk Assessment Strategies
A Cumulative Vulnerability Index based on:
" Vulnerable Plaque / Artery
" Vulnerable Blood (prone to thrombosis)
" Vulnerable Myocardium (prone to life-threatening arrhythmia)

" In Search of the Vulnerable Patient

The ideal method for screening vulnerable patients should be 1) inexpensive, 2) relatively noninvasive, 3) widely reproducible, 4) readily applicable to an asymptomatic population, and 5) capable of adding predicted value to measurements of established risk factors. Such a method should provide a cost-effective step-wise approach designed to further stratify risk and provide reliable diagnosis and pathways for monitoring therapy. Obviously, these goals are hard to achieve with today's tools.

Mystery of diastolic dysfunction : Is it as deadly ?
- Dr. Milan Chag, Ahmedabad, India

Mystery of Heart failure with preserved systolic function (Diastolic Heart Failure): an over-looked problem? Is it as deadly?

Epidemiologic studies have consistently shown that a meaningful percentage of patients with heart failure (HF) has a preserved left ventricular (LV) systolic function or, more properly, a normal LV ejection fraction (EF). This also named as Diastolic Heart Failure.

Epidemiology:

There is a wide variation in the prevalence of this condition. However, on average, these patients encompass 50-55% of all the patients hospitalized for HF. Compared to the patients with HF and a low LVEF, they are more likely to be elderly, to be females, to have a history of hyper-tension and less likely to have had a previous myocardial infarction. With regards to prognosis, they have a similar rehospitalisation rate and a similar or, more often, slightly lower mortality.

Diastolic Heart Failure: Effects of Age on Prevalence and Prognosis

	Age, y		
	<50	50-70	>70
Prevalence	15	33	50
Mortality	15	33	50
Morbidity	25	50	50

All values are percentages.

Prevalence indicates percentage of all heart failure patients presenting with diastolic heart failure; Mortality, 5-year mortality rate; and Morbidity, 1-year rate of hospital admission for heart failure. The percentage values given in this table are approximate and rounded figures based on multiple studies.

Definition of Diastolic Heart Failure:

Diastolic heart failure is a clinical syndrome characterized by the symptoms and signs of heart failure, a preserved ejection fraction (EF), and abnormal diastolic function. From a conceptual perspective, diastolic heart failure occurs when the ventricular chamber is unable to accept an adequate volume of blood during diastole, at normal diastolic pressures and at volumes sufficient to maintain an appropriate stroke volume. These abnormalities are caused by a decrease in ventricular relaxation and/or an increase in ventricular stiffness.

Definition of Diastolic Dysfunction:

Conceptually, diastole encompasses the time period during which the myocardium loses its ability to generate force and shorten and returns to an unstressed length and force. By extension, diastolic dysfunction occurs when these processes are prolonged, slowed, or incomplete.

Mechanism of Diastolic Heart Failure:

Heart failure with normal LVEF is characterized by: 1) symptoms and signs of heart failure, 2) a preserved LV ejection fraction, and 3) diastolic dysfunction.Systolic and diastolic function generally coexist and there is a continuum from normal LV function to LV with preserved LVEF, though with other abnormalities of systolic and diastolic function, to HF with low LVEF. The main pathophysiological mechanisms of HF with normal LVEF are three: inappropriate tachycardia, slow systolic relaxation, decreased LV compliance. Slow systolic relaxation may be caused by excessive preload or afterload, impaired inactivation at the cardiac myocyte level, non-uniformity of load and inactivation. The episodes of HF with normal LVEF occurring during a hypertensive crisis are typical examples. The excessive increase in afterload causes impaired relaxation and increased LV and aortic stiffness generally contribute.

Etiology:

The incidence of diastolic heart failure increases with age, and it is more common in older women.Hypertension and cardiac ischemia are the most common causes of diastolic heart failure *(Table)*. Common precipitating factors include volume overload; tachycardia; exercise; hypertension; ischemia; systemic stressors (e.g., anemia, fever, infection, thyrotoxicosis); arrhythmia (e.g., atrial fibrillation, atrioventricular nodal block); increased salt intake; and use of nonsteroidal anti-inflammatory drugs.

Causes of Diastolic Heart Failure

Common causes*

- Cardiac ischemia
- Hypertension
- Aging

- Obesity
- Aortic stenosis
- Uncommon causes

Myocardial disorders
- Myocardial diseases
  - Infiltrative disease (e.g., amyloidosis, sarcoidosis, fatty infiltration)
  - Noninfiltrative diseases (e.g., idiopathic and hypertrophic cardiomyopathy)
- Endomyocardial diseases
  - Hypereosinophilic syndrome
- Storage diseases
  - Glycogen storage disease
  - Hemochromatosis

Pericardial disorders
- Constrictive pericarditis
- Effusive-constrictive pericarditis
- Pericardial effusion

**—Common causes are listed in order of prevalence.*

Cinical profile:

These patients are older, more likely to be females, to have a history of arterial hypertension and/or to suffer of atrial fibrillation

Current European Diagnostic Criteria:

(1) Signs and symptoms of CHF (effort dyspnea, pulmonary rales/edema, CPX test VO2 max < 25 ml/kg/min
(2) Normal or preserved LV systolic function(LVEF>45%, LVEDDI <32mm/sq M, LVEDVI <102 ml/sq M
(3) Abnormal LV relaxation, filling, diastolic stiffness by echo or cardiac catheterization

Diagnosis:

It is difficult to distinguish diastolic from systolic heart failure based on physical findings or symptoms. Doppler echocardiography has assumed the primary role in the noninvasive assessment of cardiac diastolic function and is used to confirm the diagnosis.

Assessment of LV diastolic function requires a meticulous and systematic approach. The initial evaluation begins with echocardiographic observation of standard M-mode and 2-dimensional anatomic imaging.

Anatomic evaluation of LA diameter and volume, LV mass, LV relative wall thickness, and LV systolic function should be the first step in the evaluation of diastolic dysfunction. Mitral inflow interrogation is the cornerstone of initial physiologic evaluation of diastolic function. PV flow variables and the Valsalva maneuver add pivotal

information to characterize the stage of diastolic abnormality. Colour M-Mode Propagation Velocity of mitral inflow and Doppler Tissue Imaging of mitral annular motion provide supportive information to better stratify the degree of diastolic dysfunction.

The ratio of early to late transmitral flow velocities (E/A ratio) has a U-shaped relationship with LV diastolic function with its highest values in a normal left ventricle and with severe LV diastolic dysfunction. Their diagnostic accuracy may be increased by the use of the Valsalva manoeuvre. Newer indices allow a better assessment of LV diastolic function. Perhaps the most useful is the ratio of the mitral E velocity to the velocity of mitral annular motion during early diastole recorded by tissue Doppler echocardiography (E/Ea). This parameter is highly related to LV filling pressure. Another important parameter is the difference in duration during atrial contraction of antegrade flow through the mitral valve compared with retrograde flow into the pulmonary veins. Left atrial volume also allows an accurate estimate of LV filling pressure.The growing availability of echocardiography, as well as point-of-care biomarkers such as brain natriuretic peptide, probably increases the likelihood that patients with dyspnea will be diagnosed as having diastolic heart failure. The serum brain natriuretic peptide (BNP) test can accurately differentiate heart failure from noncardiac conditions in a patient with dyspnea, but it cannot distinguish diastolic from systolic heart failure.

Table-1 : Doppler echocardiographic patterns of current echocardiographic tools in relation to the grading of LV diastolic dysfunction

Parameter	Normal	Pattern of abnormal Relaxation (grade I)	Pseudonormal pattern (Grade-II)	Restrictive pattern (Grade-III-IV)
E/A	>1	<1	1-2	≥2
DT(ms)	160-210	>220	150-200	<150
IVRT(ms)	7-90	>95	6-95	<60
S/D	1.3-1.5	1.6-2.0	<1	0.40-0.60
AR(m/sec)	0.22-0.32	0.21-0.28	≥0.35	≥0.25
Em(cm/sec)	>8	<8	<8	<5
Vp(cm/sec)	>55	<45	<45	<35
E/Em	<8			>16E
E/Vp				>2.5

AR= Atrial retrograde velocity, DT=Deceleration time, E/A=Transmitral E/A ratio, E/Em=Transmitral early diastolic velocity to myocardial early diastolic velocity of lateral Mitral annulus by tissue Doppler, Em= myocardial early diastolic velocity by Tissue Doppler at lateral Mitral annulus, IVRT=Isovolumic relaxation time,=Systolic velocity to diastolic velocity ratio by pulmonary veins assessment, Vp=Velocity propagation

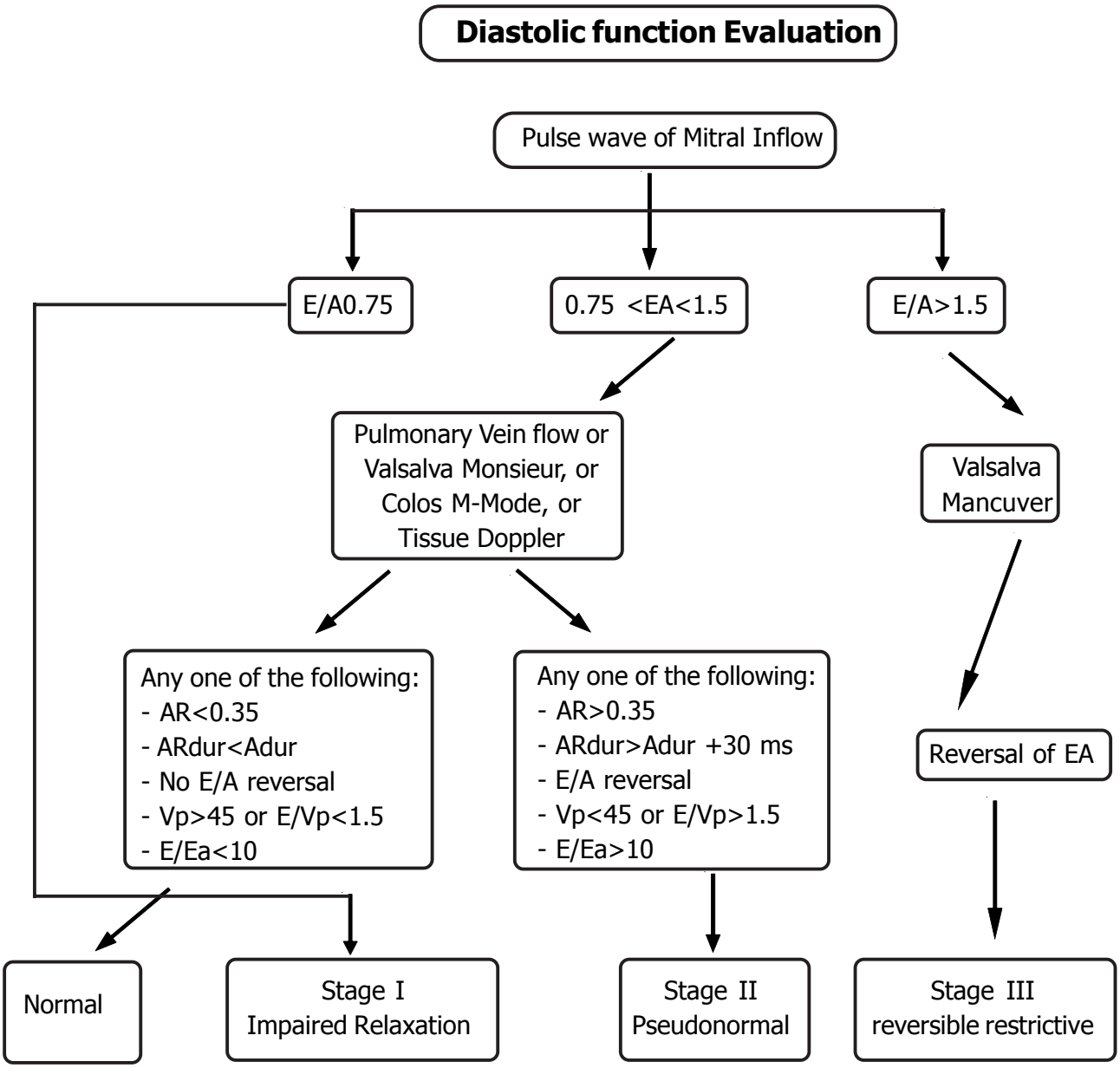


Diagram illustrates practical echocardiographic approach to evaluation of diastolic function. A, Peak late diastolic Transmitral flow velocity; Adur, duration of A wave; AR, peak pulmonary venous atrial reversal flow velocity; ARdur, ARduration; E, peak early diastolic transmitral flow velocity; Ea, peak early diastolic myocardial velocity; Vp, flow propagation velocity.

Prognosis and Treatment:

The nosology of heart failure has been the subject of much current debate, and some extreme positions have been taken. The observation that 22 to 29 percent of patients with diastolic heart failure die within one year of hospital discharge, and 65 percent die within five years, is a reminder that we are facing a lethal condition, regardless of its name. In recent years, there has been little improvement in survival rate among patients with diastolic heart failure, in contrast to the improvement in survival rate over time among patients with systolic heart failure. The news

is not all bad, however. The noted improvement in the survival rate of patients with systolic heart failure provides encouragement that emerging treatment strategies for diastolic heart failure, such as the use of angiotensin receptor blockers, might eventually have a clinical effect. We should also not neglect preventive measures with proven efficacy (such as antihypertensive therapy), given that there is no effective cure for aging. The prevention of a first or recurrent myocardial infarction is likely to be the best means we have to keep the ejection fraction "preserved." However, the development of specific, effective management approaches for diastolic heart failure must also become a high priority.

Management

The management of diastolic heart failure has two major objectives: to reverse the consequences of diastolic dysfunction (e.g., venous congestion and exercise intolerance) and to eliminate or reduce the factors responsible for the diastolic dysfunction.

Initial Management

The initial treatment of patients with diastolic heart failure, like that of patients with systolic heart failure, is aimed at reducing pulmonary venous pressure and congestion, and such treatment usually requires therapy with diuretics. Tachycardia causes an increase in demand for myocardial oxygen and a decrease in coronary perfusion time, which may lead to myocardial ischemia, even in the absence of obstructive coronary artery disease. In addition, there may be insufficient time for complete relaxation, with a resultant increase in diastolic pressure; ventricular filling may also be compromised. There are no data to support the use of a particular pharmacologic agent or strategy over another for rate control in patients with diastolic heart failure and atrial fibrillation, but beta-blockers[27] or nondihydropyridine calcium-channel blockers[38] can be used to prevent tachycardia or to slow the heart rate in patients who have diastolic heart failure.

Long-Term Management

Data from long-term investigations of any agent compared with placebo in patients with diastolic heart failure are lacking, as are data from studies comparing agents of different classes. Several small, short-term studies of patients with hypertensive disease, coronary heart disease, or both (and a normal or near-normal ejection fraction) indicate that calcium-channel blockers, angiotensin-converting–enzyme inhibitors, or angiotensin-receptor blockers may be useful in improving exercise capacity.

There are insufficient data from randomized trials to assess the effects of various pharmacologic agents on congestive heart failure and on other cardiovascular outcomes or to support a preference for one agent or class of agents over another. Certain pharmacologic agents have been proposed for use in patients with diastolic dysfunction because of their biologic effects, such as the elimination of tachycardia, ischemia, or both (e.g., beta-blockers and rate-lowering calcium-channel blockers) or the regression of left ventricular hypertrophy (e.g., diuretics and angiotensin-converting–enzyme inhibitors) and fibrosis (e.g., spironolactone). Agents that inhibit the renin–angiotensin–aldosterone system may have several of these effects. However, more data are needed to demonstrate that such biologic effects reduce the risk of heart failure.

TABLE

Goals for Treating Diastolic Heart Failure

Treat precipitating factors and underlying disease.
Prevent and treat hypertension and ischemic heart disease.
Surgically remove diseased pericardium.
Improve left ventricular relaxation.
- ACE inhibitors
- Calcium channel blockers

Regress left ventricular hypertrophy (decrease wall thickness and remove excess collagen).
- ACE inhibitors and ARBs
- Aldosterone antagonists
- Beta blockers
- Calcium channel blockers

Maintain atrioventricular synchrony by managing tachycardia (tachyarrhythmia).
- Beta blockers (preferred)
- Calcium channel blockers (second-line agents)
- Digoxin (controversial)
- Atrioventricular node ablation (rare cases)

Optimize circulating volume (hemodynamics).
- ACE inhibitors
- Aldosterone antagonists (theoretical benefit)
- Salt and water restriction
- Diuresis, dialysis, or plasmapheresis

Improve survival.
- Beta blockers
- ACE inhibitors

Prevent relapse by intensifying outpatient follow-up.
- Control blood pressure.
- Dietary counseling (sodium)
- Monitoring volume status (daily weights and diuretic adjustment)
- Institute exercise program.

Conclusive implications:

It is now recognised that HF with normal LVEF represent a frequent clinical presentation of HF. These patients are more often elderly, females and with a history of hypertension.
The diagnosis of diastolic HF can be considered in the presence of the signs of HF and normal EF (50% or more) but it should be usually supported by a Doppler examination. Diastolic HF is associated to four-fold increase mortality. If it is true that the mortality expectation is lower than in patients with systolic HF, it is even true that this difference has a trend to be blunted during long-

term follow-up, with possible overlapping after 5.5 years or more. The therapeutic management of diastolic HF is, at least partially, empirical and several studies, ongoing or completed, have been planned to test the effects of ACE inhibitors, angiotensin-inhibitors and â-blockers. The prevention of diastolic HF may be obtained by a better control of blood pressure values and of concomitant risk factors in hypertensive patients.

Cause for Passion or Cause for Panic: Where Are We With Drug Eluting Stents? - Dr. Mitchell Krucoff

Abstract Not received

Coronary Artery Disease and Heart Failure: The Dangerous Intersection - Dr. Dhiren Shah, Ahmedabad, India

Twenty first century will see the epidemic of heart failure . In India the situation is going to be very worse , as half of the cardiac patients of the world would be in India.
Coronary artery disease is the most common cause of heart failure in world.Almost 2/3 of heart failure patient have a past history of Myocardial infarction.

Heart failure is a heterogeneous syndrome, but having coronary disease is a prominent contributing factor. If you look at Framingham data, patients who have angina have a two- to three-fold increased risk of developing heart failure – and that's for men and women – over the general population. If you've had an infarct, that increased risk goes up to 10 times.

But the general mindset is "you've had an MI, the way you're going to develop heart failure is by having another MI," said Dr. Pfeffer. If one can avoid that next MI, then heart failure will be avoided too , this is secondary prevention.

That's a common misconception, he said. Abundant evidence suggests that 60-80%, of patients who develop HF post-MI do not experience another MI. "So there is some time-dependent process. We used to think most of that was remodeling, and that is true to some extent…but we don't have a single etiology that really ties the whole thing with a nice ribbon."

If a right road is taken after the intersection the probably we can change the survival and natural history of these patients with CAD and heart failure. This long term outcomes can be modified by various procedure like CABG ,SVR, MitralValve Repair, and some novel therapies like Stem cell injection . LVAD. There are various studies and reports and registry showing the importance of this modalities of treatment in improving the outlook of the patient.

Definitely patients with heart failure and CAD had bad long term prognosis in the past , but with the new understanding of heart failure syndrome and its pathophysiology the morbidity of these patients can be minimized and survival of the patients can be prolonged .

Utility of the Intra-aortic Balloon Pump in Heart Failure and Cardiogenic Shock
Dr. Urmil Shah, Ahmedabad, India

IABP- role in Cardiogenic shock and failure

History

- Realization that coronary perfusion mainly occurs during diastole -1950s
- Aspiration of arterial blood during systole with re infusion during diastole decreased cardiac work without compromising coronary perfusion – Harkin-1960s
- Intravascular volume displacement with latex balloons - early 1960s

Background

- Preload
- Afterload
- Coronary flow – in diastole
- Myocardial oxygen consumption in the heart is determined by:
  - Pulse rate
  - Transmural wall stress
  - Intrinsic contractile properties

Definition

- Heart's inability to meet metabolic needs
- Hypotension (SBP <80-90); cold, clamy and pale extremities; cyanosis, altered mentation, oliguria.
- CI <2.2 l/min/m2, PCWP >18 mmH, lactate >1.5 mM/l.

Types of Shock

- Hypovolemic, the most common
- Cardiogenic - systolic dysfx/mechanical dysfx -
- Obstructive - cardiogenic
  - Tamponade, LA myxoma, LA thrombus, PE
- Distributive
  - Sepsis, anaphylaxis, DOD, spinal shock

Systemic Inflammatory Response

- Pts with large MIs have LGT and ↑WBC
- ↑ serum complement, interleukins, and CRP
- ↑ inducible nitric oxide synthetase (iNOS)
- ↑ nitric oxide and peroxynitrite levels

High NO and Peroxynitrite Levels

- Direct inhibition of myocardial contractility
- ↓ mitochondrial respiration in non-ischemic cells
- ↓ glucose metabolism
- ↑ pro-inflammatory effects
- ↓ cathecolamine sensitivity
- ↑ ↑ systemic vasodilation

Contraindications

- Severe peripheral vascular disease
- Severe aortic incompetence
- Active bleeding
- Patients with contraindication to anticoagulation
- Thrombocytopenia (<50,000)
- Acute stroke

Guidelines

"Emergency high risk PCI such as direct PCI for acute MI can usually be performed without IABP or CPS."However, it should be noted that in patients with borderline hemodynamics, ongoing ischemia, or cardiogenic shock, insertion of an intra-aortic balloon just prior to coronary instrumentation has been associated with improved outcomes. Furthermore it is reasonable to obtain vascular access in the contralateral femoral artery prior to the procedure in patients in whom the risk of hemodynamic compromise is high…"

Recommendations for the use of IABP in the treatment of AMI

Class I

- Cardiogenic shock not quickly reversed with pharmacological therapy as a stabilizing measure for angiography and prompt revascularization
- Acute MR or VSD – as a stabilizing therapy for angio and repair/ revascularization
- Recurrent intractable ventricular arrhythmias with hemodynamic instability
- Refractory post –MI angina as a bridge to revascularization

Class IIa

Signs of hemodynamic instability, poor LV , or persistent ischemia in patients with large areas of myocardium at risk

Class IIb

Following successful angioplasty to prevent reocclusion

Large areas at risk w/o active ischemia

Indication summary

Unstable angina medically refractory or post MI

Complication of myocardial infarction

VSD

MR

Refractory arrhythmia

Cardiogenic shock

with PTCA/ CABG/ Thrombolytic Rx
Angioplasty
failure – pre CABG / complex high risk procedure
Post cardiotomy cardiac failure
Severe Lt main disease
Poor LV function with severe TVD
High risk non cardiac surgery

HEMODYNAMIC EFFECTS OF IABP

- EF
- diastolic BP
- CO
- LV stroke work index
- cerebral blood flow
- renal perfusion
- myocardial contractility

Cardiogenic Shock

Prognosis

- Overall in-hospital mortality 60%
- Higher mortality in early CS (<24 hours)
- Similar mortality in pts with STEMI vs NSTEMI
- Higher mortality in those with mechanical complications vs pump failure
- Higher mortality in elderly (>75), women

Prognosis

- 40% periop mortality in those with severe MR
- 95% mortality in those with unoperated VSD
- 80% mortality in those with operated VSD

Timing

- On arrival: 10%-15%
- In hospital: 85%-90%
- Median of 6 hours after AMI
- Early shock (<24 hours) in 75%

Non-ST-elevation MI (17%)

- More common in patients >65 y/o
- Prior MI, heart failure, CABG, or PVD
- More often have 3 vessel CAD
- Post-MI angina or MI extension are common

Post-MI acute severe MR (7% - 8%)

- Occurs early (median 13 hours)
- More common in women
- More common with infero-posterior MI

- More common with NSTEMI (60% vs 40%)

Post-MI VSD (4% - 5%)

- Occurs early (median of 16 hours)
- More common in those >65 y/o
- More common in women
- Twice more common if no DM or prior MI
- Most common in RCA or LAD - MIs (88%)

***Post-MI free wall rupture/tamponade* (2% - 3%)**

- More common with STEMI
- More common without prior MI
- More common without diabetes

Causes in patients post-CABG/VR

- Ischemia or MI
- Prolonged bypass/reperfusion injury
- Tamponade (frequently localized)
- Prosthesis dehiscence or thrombus

Cardiogenic Shock – Patients at risk

- Age >65 y/o
- Women
- Previous angina, MI, CHF
- DM
- Stroke or PVD
- Persistently occluded IRA
- LAD-MI
- Multivessel CAD
- LVEF <35%
- High CPK-MB/troponin

General measures

- Team approach - be the leader
- Start Rx before full evaluation completed
- Assess volume, ventilation, pump function
- Restore/maintain sinus rhythm
- Correct acid-base abnormalities
- Improve O2 carrying capacity (Hct $\geq$33)
- *"CARDIAC CATH - REVASCULARIZE"*

LV and RV function and mechanics

- Beside echocardiography
  - LV and RV ejection fraction
  - LV and RV wall motion

- MR, VSD, pseudoaneurysm
- Pericardial effusion/tamponade

Pharmacotherapy

- Phosphodiesterase inhibitors
  - amrinone: 0.75-3 mg/kg, 5-10 ug/kg/min
  - milrinone: 20 ug/kg over 10' then 0.5 ug/kg/min
- Dobutamine, fluids, A-V pacing in RV-MI
- Do not use digoxin

- **Nitric oxide synthase inhibitor (L-NAME)**
  - Dose: 1mg/Kg bolus, 1mg/Kg/hour x 5 hours
  - ↓ vasodilation
  - ↑ mean arterial pressure
  - ↑ CO after transient decrease
  - ↑ urine output, ↓ time on IABP/mechanical ventilation
  - ↓ mortality at 1 month
  - Does not affect LV systolic or diastolic fx
- **Levosimendan (infusion – 0.1 ug/kg/minx24 h)**
  - Calcium sensitizer
  - ↑ contractility without ↑ in intracellular Ca or c-AMP
  - Vasodilator, open ATP sensitive K channels
  - Compared to or in addition to cathecolamines, improves hemodynamics and 1, 6 month mortality and less arrhythmogenic

Intraaortic Balloon Pump

- Improves survival (40% to 60%) if used with lytics/PCI/CABG/VR
- Used in >80% of patients undergoing PCI/CABG
- Similar benefit when compared to VADs

Thrombolytic therapy

- Success rate of lytics is low (30-50%)
- Improves survival up to 60% if successful
- TPA and SK have similar efficacy
- Higher rate of wall rupture in pts $\geq$75 y/o

Coronary angiography

- Performed in ≅ 60% of patients
- 3 vessel CAD: 50% - 55%, 1-2 vessel CAD:20% -25%, and $\geq$50% left main: 15% - 20%
- LAD culprit vessel in 40% of pts
- LAD or RCA culprit vessel in those with VSD
- RCA culprit vessel in pts with severe MR/RV-MI
- Cx culprit in those with free wall rupture

Percutaneous Coronary Interventions

- Low use (≈30%) and lower success rate (≈ 75%) than in those without cardiogenic shock

- ↑success and survival when stents, 2b/3a inhibitors, and asa & clopidogrel used
- ↑ survival when done within 16 hrs
- ↑ success in pts <70 y/o, 1 vessel CAD, no previous MI, and had no lytic Rx
- ↑ survival with TIMI 3 flow

CABG

- Only 12-50% of pts undergo CABG
- Half of pts have had lytics, IABP, or PCI
- High perioperative/overall mortality (40-50%)
- Lower mortality for MR/VSD (30%/40%)
- Highest mortality in pts with LAD, 3V CAD or >70 years old

Myocardial Oxygen Consumption

- Has a linear relationship to:
  - Systolic wall stress
  - Intraventricular pressure
  - Afterload
  - End diastolic volume
  - Wall thickness

Indications for IABP

- Cardiac failure after a cardiac surgical procedure
- Refractory angina despite maximal medical management
- Perioperative treatment of complications due to myocardial infarction
- Failed PTCA
- As a bridge to cardiac transplantation

IABP in Myocardial Infarction and Cardiogenic Shock

- Improves diastolic flow velocities after angioplasty
- Allows for additional intervention to be done more safely

IABP During or After Cardiac Surgery

- Patients who have sustained ventricular damage preoperatively and experience harmful additional ischemia during surgery
- Some patients begin with relatively normal cardiac function an experienced severe, but reversible, myocardial stunning during the operation

IABP As a Bridge to Cardiac Transplantation

- 15 to 30 % of endstage cardiomyopathy patients awaiting transplantation need mechanical support
- May decrease the need for more invasive LVAD support

Other Indications for IABP

- Prophylactic use prior to cardiac surgery in patients with:
  - Left main disease

- Unstable angina
- Poor left ventricular function
- Severe aortic stenosis

Contraindications to IABP

- Severe aortic insufficiency
- Aortic aneurysm

Insertion Techniques

- Percutaneous
  - sheath less
- Surgical insertion

Positioning

- The end of the balloon should be just distal to the takeoff of the left subclavian artery
- Position should be confirmed by fluoroscopy or chest x-ray

Timing of Counterpulsation

- Electrocardiographic
- Arterial pressure tracing

Weaning of IABP

- Decreasing inotropic support
- Decreasing pump ratio

IABP Removal

- Discontinue heparin six hours prior
- Check platelets and coagulation factors
- Deflate the balloon
- Apply manual pressure above and below IABP insertion site
- Remove and alternate pressure to expel any clots
- Apply constant pressure to the insertion site for a minimum of 30 minutes
- Check distal pulses frequently

Guidelines

"Emergency high risk PCI such as direct PCI for acute MI can usually be performed without IABP or CPS.

…

However, it should be noted that in patients with borderline hemodynamics, ongoing ischemia, or cardiogenic shock, insertion of an intra-aortic balloon just prior to coronary instrumentation has been associated with improved outcomes.

Furthermore it is reasonable to obtain vascular access in the contralateral femoral artery prior to the procedure in patients in whom the risk of hemodynamic compromise is high…"

Percutaneous LV Assist Devices: Overview - Dr. Raj Makkar, USA

Abstract Not received

Novel Therapies for Heart Failure - What has changed in the past 2 years ? - Dr. Ajay Naik, Ahmedabad, India

Over the past decade, Heart Failure (HF) management has had a major paradigm shift, particularly so over the past 2 years.

A. Pharmacologic innovations:

Cardiac Myosin Activators directly target the force-generating cardiac enzyme, myuocardial myosin ATPase, accelerating its activity in order to enhance contractility.

Istaroxime (PST-2744) a novel Na/K ATPase inhibitor chemically unrelated to cardiac glycosides augments myocardial contractility by stimulating calcium entry via the sarcolemmal Na/Ca exchanger.

Urodilatin a novel atrial natriuretic peptide (ANP) analogue is under investigation for its diuretic role that is superior to the conventional thiazide and loop diuretics.

Nesiritide (Recombinant B-type Natriuretic peptide) significantly reduces PCWP via pulmonary vasodilatation and diuresis; ongoing clinical data should provide mortality data.

Adenosine antagonists and Vasopressin antagonists (Tolvaptan) also show promise in decompensated heart failure management.

Perhexiline, a modulator of myocyte energetics significantly increases exercise tolerance.

B. Implantable Device Innovations:

Cardiac Resynchronization Therapy (CRT, Biventricular Pacemaker Implantation) has dramatically improved the quality of life of patients in NYHA class III and IV HF. The CARE-HF trial data has shown reduction in mortality in patients with CRT.

CRT-D: Cardiac Resynchronization Therapy with Defibrillator is recommended for patients with HF at risk of Sudden Cardiac Death (SCD) due to ventricular arrhythmias.

CRT devices with transthoarcic impedance sensors monitor and warn against occurrence of pulmonary congestion – these help in fine tuning drug therapy before overt heart failure

occurs.

C. Surgical Innovations:

LV Assist Devices are now real options to support LV function in patients with transient severe LV dysfunction (such as myocarditis). They serve as a bridge to heart transplants.

LVADs have been miniaturized and work on smaller, more powerful battery packs that can help in patient mobilization.

Surgical Ventricular Restoration (SVR) attempts to achieve normal LV topology in patients with dyskinetic and aneurysmal myocardial segments. This improves the efficiency of the heart.

Implication of Left ventricular size and shape on Heart failure - Dr. Anil Jain, Ahmedabad, India

Heart Failure, especially left ventricular is always associated with changes in ventricular size and shape, just as stated in the Frank Starling Law. Progressive remodeling always leads to worsening of heart failure and ultimately death of the patient. The factors that play a role in remodeling and the role of surgical ventricular restoration in treating such patients shall be discussed. Also shall be presenting the outcome of the largest national series of patients undergoing this surgery

Pharmacotherapy of the Heart Failure : Past, Present & Future - Dr. Ake Hjalmarson

Pharmacotherapy in Heart Failure
Ake Hjalmarson, MD.PhD, The Wallenberg Laboratory for Cardiovascular Research, Sahlgrenska
University Hospital, Göteborg University, Sweden

Introduction

In the 1980´s the standard treatment of heart failure was diuretics, digitalis and vasodilators. The impact of these treatments on mortality and morbidity was unclear. Since that time new therapeutic regimens have been introduced including inotropic agents, angiotensin-converting enzyme-inhibitors (ACE-inhibitors), betareceptor-blockers, aldosterone antagonists , angiotensin II type 1-receptor blockers (AT1-receptor-blockers), endothelin-antagonists and many others. Among these therapies only those which involve neurohormonal blockade have been demonstrated to improve long-term mortality and morbidity in large prospective trials.

The purpose of this presentation is to describe the role of neurohormonal blockade in heart failure therapy with focus on placebo-controlled survival studies.

Betablockers.

Already in 1975 it was first reported by Waagstein, Hjalmarson and co-workers (1) that patients with idiopathic dilated cardiomyopathy (IDC) and heart failure could be improved by betablockers given at a low starting dose and with step-wise up-titration. Our group demonstrated later that long-term betablockade resulted in improvement of cardiac function and symptoms and later betablocker withdrawal caused deterioration. In 1979,. Swedberg and co-workers suggested that chronic betablockade could improve survival in patients with IDC (2). However, this was not a randomised and placebo-controlled study, but used historical controls for comparison.. These reports were often referred to as the "Swedish experience" and were not widely accepted in the medical society. During the late 70´s and early 80´s our research group in Göteborg, Sweden reported in several papers the beneficial effects of chronic betablockade not only in non-ischemic but also in ischemic cardiomyopathy. Since betablockad at this time in general was considered contraindicated the cardiologists were not willing to believe in our results. However, we think that this was the very first step of the important establishment of neurohormonal blockade in heart failure.

In the early 1980´s a growing number of cardiologists got an interest in the studies of betablocked in heart failure and a prospective, randomised and placebo-controlled trial on the beta1-receptor-selective agent metoprolol was planned. This Metoprolol in Dilated Cardiomyopthy (MDC)-trial (3) randomised 383 patients in 33 centres in the US and in Europe to metoprolol or placebo with an 18-months follow-up. The primary objective was the combined and-point death or need for cardiac transplantation. In a total of 383 patients with IDC there was a 34% reduction of the primary end-point ($p=0.058$), with the major effect on reduction of need for cardiac transplantation (19 vs 2, $p<0.001$).

During the 1990´s were published 3 trials, which supported a beneficial effect of betablockade in patients with heart failure with ischemic and non-ischemic cardiomyopathy. These were the CIBIS-I on bisoprolol, the US Carvedilol program and the AZ/New Zealand study on carvedilol . In 1999 were published to very large place-controlled trials with total mortality as primary objective, the CIBIS-II (4) on bisoprolol and the MERIT-HF (5) on metoprolol CR/XL (controlled release) in 2647 and 3991 patients, respectively, with symptomatic heart failure and ejection fraction (EF) <0.40. Both demonstrated a 34% reduction in total mortality ($p<0.0001$), a marked reduction in sudden deaths and deaths due to worsening of heart failure and in hospital admissions.

In 2001 was published the CAPRICORN trial (6) which included 1959 patients with left ventricular dysfunction after a myocardial infarction. Carvedilol reduced total mortality by 23% ($p=0.031$). After this report a subgroup analysis of post-infarction patients (n=1926) of the MERIT-HF population was done and there was a 40% reduction in total mortality and 50% reduction of sudden cardiac deaths ($p=0.0004$). In 2002 the COPERNICUS trial (7) in 2289 patients with more severe heart failure (EF <0.25) showed that carvedilol reduced total mortality by 35%. In subsets of similar patients of the MERIT-HF and CIBIS-II trials the same results were seen for metoprolol and bisoprolol.

When comparing the trials on bisoprolol, carvedilol and metoprolol CR/XL very consistent data were seen in the total populations and in subgroups (elderly, females, hypertensives, more severe heart failure, post-MI, diabetics). Recently the COMET trial (8) was published stating that carvedilol extends survival in a study of 3029 heart failure patients compared to metoprolol. However, the two dose regimens in this trial were not comparable since short-acting metoprolol tartrate was given (9). There is no study showing and no reason to believe that the effects on mortality differs between bisoprolol, carvedilol and long-acting metoprolol CR/XL as used in the large placebo-controlled survival trials.

ACE-inhibitors

In the early 1980´s the ACE-inhibitors were introduced in the treatment of hypertension and evaluated for use in heart failure. The first prospective randomised survival study was the CONSENSUS (10) in 253 patients in NYHA class IV, published in 1987. Enalapril caused a 44% reduction in mortality. The results received wide acceptance and enalapril was used for treatment of heart failure around the world. This trial was followed by a large number of studies demonstrating important effects of ACE-inhibitors. The most important was the SOLVD project (Studies On Left Ventricular Dysfunction) in 6797 patients (11). In the treatment arm 2569 patients were randomised and enalapril reduced mortality by 16% (p= 0.0036) and deaths due to worsening heart failure was reduced by 22% and hospitalisations for heart failure by 26%.

In the metaanalysis by Garg and co-workers (12) of 32 placebo-controlled trials including 7105 patients ACE-inhibitors caused a 23% reduction in total mortality and in the combination of deaths and hospitalisations a 35% reduction.

Aldosterone-antagonists

Since long time it is known that ACE-inhibitors did not attenuate the activation of the renin-angiotensin system completely. Plasma level of aldosterone falls acutely with introduction of an ACE-inhibitor, but returns towards pre-treatment levels with chronic therapy. In spite of increasing doses of ACE-inhibitors higher plasma levels of aldosterone have been reported. Aldosterone has a number of possible harmful cardiovascular effects, including effects on electrolytes, sympathetic activity, endothelial function and collagen turnover.

This is the background to the RALES study (13) , which was a placebo-controlled trial of the aldosterone antagonist in 1663 patients with severe heart failure (NYHA III-IV). Spironolactone when added to ACE-inhibitors reduced mortality by 30% ($p<0.0001$) and had favourable effects on sudden cardiac deaths and morbidity. In the EPHESUS study (14) the effects of the aldosterone antagonist eplerenone were compared to placebo in patients who had sustained a recent myocardial infarction and had an EF <0.40 and with evidence of heart failure (90%) or diabetes mellitus (32%). Eplerenone added to ACE-inhibitors and betablockers reduced mortality by 15% and cardiovascular deaths or hospitalisations by 13% ($p=0.008$ and 0.002, respectively). In patients with EF < 0.30 eplerenone reduced sudden death by 33%.

Angiotensin-receptor blockers

The first large placebo-controlled survival study of AT1-receptor-blocking agents was the Val-HeFT (15) in 5010 heart failure patients.. In this study valsartan when added to conventional treatment, ACE-inhibitors (93%), betablockers (35%) and spirolactone (5%) reduced the risk of the composite primary endpoint death or cardiovascular morbidity (admission for CHF, i.v. treatment for CHF or resuscitation) by 13.2% In the 1610 patients treated with both ACE-inhibitors and betablockers at baseline, valsartan was associated with a worse outcome.

The very recent and largest trial on AT1-receptor-blockers was the CHARM-Overall programme in 7601 patients with symptomatic heart failure and NYHA Class II-IV (16). Candesartan reduced total mortality by 10% (adjusted, $p=0.032$) and hospital admissions for CHF ($p<0.0001$). In patients EF 0.40 or lower (n=2548) candesartan added to ACE-inhibitors reduced the composite primary objective, cardiovascular deaths or hospital admissions for CHF, by 15% ($p=0.010$). In patients intolerant to ACE-inhibitors and EF 0.40 or lower, candesartan reduced the composite endpoint (as above) by 30% (adjusted, $p<0.0001$). In patients with EF above 0.40 (n=3025) candesartan reduced the composite endpoint (as above) by 14% (adjusted, $p=0.051$).

Conclusions

The activation of the neurohormonal system including the sympathetic pathway and the renin-angiotensin and aldosterone system is of great importance in the normal circulatory regulation, e.g. during bleeding or dehydration. Chronic activation of the system in cardiovascular diseases seems to have an opposite effect and causes progression of the disease and higher mortality. It has therefore been more and more evident that blockade of the neurohormonal activation ought to be useful in the treatment of cardiovascular diseases.

In the present review is shown that betablockers, ACE-inhibitors, aldosterone antagonists and AT1-receptor blockers, each by itself compared to placebo, reduce mortality and morbidity in patients with heart failure. The combination of these therapies should be the standard therapy for a broad range of patients with heart failure and left ventricular dysfunction. This will have a marked impact on the mortality and morbidity as well as on quality of life for patients with heart failure. It is very important that the medications prescribed will be given at dosages and in formulations as were used in the placebo-controlled large trials which gave the approval by regulatory authorities. This is also what is recommended by the international and national guidelines. Unfortunately, there is a marked underutilization of medications with proven effects on mortality and morbidity in patients with heart failure. This is true both what concerns proportion of patients treated and the dosages prescribed.

References

1. Waagstein F., Hjalmarson A, Varnauskas E et al. Effect of chronic beta-adrenergic receptor blockade in congestive cardiomyopathy. Br. Heart J. 1975; 37: 1022 - 1036.

2. Swedberg K., Hjalmarson A, Waagstein F. et al. Prolongation of survival in congestive cardiomyopathy by beta-receptor blockade. Lancet 1979; 2: 1374 - 1376.
3. Waagstein F., Bristow M.R., Swedberg K. et al. Beneficial effects of metoprolol in idiopathic dilated cardiomyopathy. Metoprolol in Dilated Cardiomyopathy (MDC) Trial Study Group. Lancet 1993; 342: 1441 - 1446.
4. CIBIS-II Investigators and Committees. The Cardiac Insufficiency Bisoprolol Study II (CIBIS-II). A randomized trial. Lancet 1999; 353: 9 - 13.
5. MERIT-HF Study Group. Effect of metoprolol CR/XL in chronic heart failure: Metoprolol CR/XL Randomized Intervention Trial in congestive Heart Failure (MERIT-HF). Lancet 1999; 353: 2001-2007.
6. The CAPRICORN Investigators. Effect of carvedilol on outcome after myocardial in patients with left-ventricular dysfunction: the CAPRICORN randomized trial. Lancet 2001; 357: 1385 - 1390.
7. Packer M., CoatsA.J.S., Fowler M.B. et al. for the Carvedilol Prospective Randomized Cumulative Survival Study Group (COPERNICUS). Effect of carvedilol on Survival in Severe Chronic Heart Failure. N.Engl. J.Med. 2001; 344 (22): 1651 - 1658.
8. Poole-Wilson P.A., Swedberg K., Cleland J.G.F. et al for the COMET investigators. Comparison of carvedilol and metoprolol on clinical outcomes in patients with chronic heart failure in the Carvedilol Or Metoprolol European Trial (COMET): randomized controlled trial. Lancet 2003; 362: 7 - 13.
9. Hjalmarson A., Waagstein F. COMET: A proposed mechanism of action to explain the results and concerns about dose. Letter to the editor. Lancet 2003; 3562: 1077.
10. The CONSENSUS Trial Study Group. Effects of enalapril on mortality in severe congestive heart failure. Results of the Cooperative North Scandinavian Enalapril Survival Study (CONSENSUS). N.Engl. J. Med. 1987; 316: 1429 - 1435.
11. The SOLVD Investigators. Effect of enalapril in patients with reduced left ventricular ejection fraction and congestive heart failure. N.Engl.J. Med. 1991; 325: 293 - 302.
12. Carg R., Yusuf S. for the Collaborative Group on ACE Inhibitor Trials. Overview of randomized trials of angiotensin-converting enzyme inhibitors on mortality and morbidity in patients with heart failure. JAMA 1995; 273: 1450 - 1456.
13. Pitt B., Zannad F., Remme W.J. et al. for the Randomized Aldactone Evaluation Study Investigators. The effect of spironolactone on morbidity and mortality in patients with severe heart failure. N.Engl.J.Med. 1999; 341: 709 - 717.
14. Pitt B., Remme W.J., Zannad F.et al. Eplerenone Post-Acute Myocardial Infarction Heart Failure Efficacy and Survival Study Investigators. Eplerenone, a selective aldosterone blocker, in patients with left ventricular dysfunction after myocardial infarction. N.Engl.J.Med. 2003; 348 (14): 1309 - 1321.
15. Cohn J.N., Tognoni G. A randomized trial of angiotensin-receptor blocker valsartan in chronic heart failure. N.Engl.J.Med. 2001: 345: 1667 1673.
16. Pfeffer M.A., Swedberg K., Granger C.B. et al for the CHARM Investigators and Committees. Effects of candesartan on mortality and morbidity in patients with chronic heart failure: the CHARM-Overall programme. Lancet 2003; 362: 759- 781.

Infective Endocarditis: 2007 Update - Dr. Anish Chandarana, Ahmedabad, India

Infective endocarditis (IE) is a microbial infection of the endothelial surface of the heart. The vegetation, characteristic lesion, is a variably sized amorphous mass of platelets and fibrin in which abundant microorganisms and moderate inflammatory cells are incorporated. Heart valves are most commonly involved; but infection can occur at the site of septal defect, chordae tendineae, arteriovenous shunt or mural endocardium. Many species of bacteria and fungi, mycobacteria, rickettsiae, mycoplasmas and chlamydiae cause IE. Streptococci, staphylococci, enterococci and gram-negative fastidious coccobacilli cause most of cases of IE.

Acute IE arises with marked toxicity and progresses over days to several weeks to valvular destruction and metastatic infection. It is typically, although not exclusively, caused by Staphylococcus aureus. Sub acute IE evolves over weeks to months with only modest toxicity and rarely causes metastatic infection. Such infection is more likely to be caused by viridans streptococci, coagulase-negative staphylococci, enterococci, or gram-negative coccobacilli.

Fever, chills, sweats, anorexia, malaise, weight loss, dyspnoea, stroke, murmur, sleenomegaly, clubbing, peripheral embolism, petechie are few of the common features. Though diagnosis is made with the help of modified Duke criteria into definite, possible or rejected IE, a high index of suspicion to start with is the key not to miss IE. In addition to routine laboratory evaluation, blood culture and echocardiogram are the main diagnostic tools.

In most of the cases, after determining causative organism and its susceptibility to antimicrobial medicines, a prolonged course of the same in intravenous form during hospitalization is needed. The infecting microorganism in the vegetation, as well as, in the invasive destruction must be eradicated to achieve cure. Invasive, destructive intracardiac and focal extracardiac complications of infection must be resolved to minimize high morbidity and mortality usually seen with this. Later may require cardiac or other surgical intervention in addition to effective antimicrobial treatment. Effective therapy leads to defeverescence within a week in almost 75 percent patients. Reported mortality rates in large series of treated patients range from 6 to 35 percent, decided by many variables.

Though antecedent history of instrumentation or surgical procedure in recent past is not found in majority of patients with IE, morbidity and mortality associated with IE justifies chemoprophylaxis before and after certain defined procedures in people with high or intermediate risk preexisting cardiac disorders.

A 35-year-old person with moderately severe MR What should be done ?- Dr. Srinivas Mallya, Ahmedabad, India

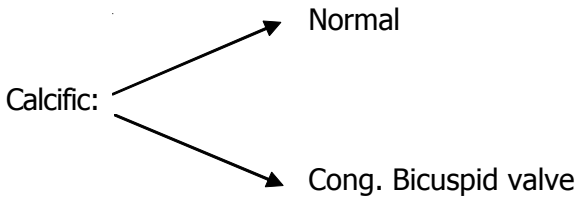
Abstract Not received

Percutaneous Mitral Valve repair - Dr. Saibal Kar

Abstract Not received

Management of Symptomatic/ Asymptomatic Severe Aortic Stenosis - Dr. Vishal Gupta, Ahmedabad, India

Cause Management of Symptomatic/ Asymptomatic Severe Aortic Stenosis



Grading

Mild Area	<1.5 cm^2	MG <25 mmHg
Moderate	1.0-1.5 cm^2	MG 25-40 mmHg
Severe	<1.0 cm^2	MG > 40 mmHg

Therapeutic Decision

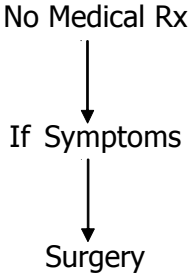
+NCE / -NCE of Symptoms
Once symptoms – Survival 2-3 year & sudden death

Diagnostic ECHO:

- Diagnostic & severity
- LV Wall thickness
- LVEF
- Mild: 3-5 years
- Moderate: 1-2 years Reevaluation

Medical Therapy

IE Prophylaxis
Anti Hypertension



Cath

- Risk for CAD
- Homodynamic – Inconclusive

Indication of AVR

- Symptoms + Severe Aortic Stenosis
- Asy. Sev Aortic Stenosis going for CABG

Surgery on Aorta other valves

- Severe Aortic Stenosis & LV dysfunction <50%
- Moderate Aortic Stenosis undergone Surgery (CABG Aortic Valve)
- Mild Aortic Stenosis - Severe Calcification

Treatment

- Balloon aortic valvotomy
- Aortic valve repair
- Aortic Valve Replacement: Mechanical Valve / Tissue Valve
- Ross Procedure

Stable Angina : State of Art : 2007
- Dr. Hemang Baxi , Ahmedabad, India

Angina is a common clinical manifestation of ischaemic heart disease. It is the initial presentation in approximately 50% of patients with ischaemic heart disease. Due to ageing population worldwide the incidence of chronic stable angina is expected to increase by 50% over the next 3 decades. In Recent past there have been substantial advances in understanding of pathophysiologic mechanisms of various subsets of angina and their pharmacologic and nonpharmacologic management. Newer and investigational agents which can potentially benefit patients with refractory angina are discussed alongwith nonpharmacological interventions for chronic refractory angina.

From Vulnerable Plaque to Vulnerable Patient
- Dr. William Wijns

Cardiac imaging is at the cornerstone of diagnosis, prognostic stratification and treatment of virtually all cardiovascular diseases only to name coronary disease and heart failure as well as valvular disorders (1).

Imaging modalities that measure global and regional cardiac function or depict valvular anatomy and function non invasively cannot be missed. Despite the many options, the detection of residual myocardial viability in poorly contracting ventricles is technically challenging but continues to play a central role in the evaluation of patients with ischemic cardiomyopathy.

While these established techniques of proven usefulness will remain and a few new indications are emerging, it should be recognized that the demands of the clinical cardiologist on non invasive imaging, myocardial perfusion imaging in particular, are currently in an evolutionary state. This is because accepted diagnostic and therapeutic practices are being challenged with the nearly simultaneous emergence of two breakthrough technologies, drug-eluting stents for percutaneous treatment of coronary stenoses (DES) and multisclice computed tomography (MS-CTA) for non invasive coronary angiography.

The availability of DES has conferred durability to the results of percutaneous coronary intervention (PCI), the most convenient and "flexible" mode of mechanical revascularization. With reduced restenosis rates, follow-up interrogation is often restricted to anginal symptoms. Patients with acute presentation (unstable angina, non ST and ST segment elevation myocardial infarction) are preferentially treated by PCI. Anatomic subsets such as multivessel disease or left main stenosis that used to require bypass surgery are now increasingly often treated percutaneously. Overall the use of bypass grafting has been greatly reduced, the majority of remaining bypass procedures being conducted in patients with associated valvular disease, prior operation (redo) or extensive history of previous interventional procedures.

MS-CTA, another technological breakthrough, has for the first time made coronary anatomy accessible non invasively, albeit with the burden of significant exposure to radiation and iodine contrast. It already appears that MS-CTA will not be very useful in patients with known or very high likelihood of disease, who should be referred for invasive angiography followed by ad hoc percutaneous treatment, as required. Instead MS-CTA will likely be used most often in patients with intermediate likelihood. Non invasive imaging now unravels coronary anatomy and calcium scoring in many subjects who would normally not undergo the invasive angiogram. Indeed, the majority of these subjects are found to have non obstructive plaque without evidence of stress-inducible ischemia, as evidenced by normal MPI. Although it is conceptually appealing to believe that these subjects are at higher risk than their asymptomatic counterparts who have no plaque and normal or low calcium score, prognostic data are being accumulated and have not been released yet. Likewise, it remains to be shown that our capability to risk stratify the individual patient improves by adding non invasive coronary angiography to the Framingham or the ESC risk scores. The results of applying "secondary" prevention measures to such subjects screened for pre-clinical coronary artery disease are awaited as well.

Because sudden ischemic cardiac death and acute myocardial infarction remain the first manifestation of coronary disease in many instances, the interventional community is investing in invasive methods searching for the rupture-prone plaque that is likely to cause the next acute event. Although it is acknowledged that atherosclerosis is an ubiquitous and systemic disease that affects large groups within the population, several vascular territories in any diseased subject and multiple segments within a diseased vascular territory, still acute forms of coronary atherosclerosis are caused by exquisitely focal events. These result from a burst of disease activity that superimposes on the background of a systemic, slowly progressive, chronic disease process. Therefore, it is postulated that preventing sudden ischemic cardiac death and myocardial infarction will likely

require combined systemic and focal therapies. The concept of "plaque sealing" that was put forward prematurely on a theoretical basis in 1995 (2) can now be tested if indeed MS-CTA, in combination with targeted invasive approaches, can be used to identify amongst the asymptomatic subjects who are the "healthy carriers" of rupture-prone plaque.

Keeping this subjective picture of the evolving clinical background in mind, a number of unmet needs and wishes can be listed.

1. Redefining the role of functional imaging (MPI, stress echocardiography, dobutamine MRI).

While functional testing, and specifically MPI, used to serve as the gatekeeper that governed access to invasive angiography, it is likely that in the near future, coronary anatomy will be available in larger patient populations, albeit non invasively. With this new paradigm, functional testing will serve as a guide for therapy, helping to make the choice between medical therapy only or mechanical revascularization. In the ideal scenario, patients shown to have plaque on MS-CTA will only be referred for invasive treatment in the presence of critical stenoses and/or objective signs of inducible ischemia of significant size upon functional testing. However, it is to be expected that many patients will undergo invasive catheterization in the absence of any prior functional test, even more often than nowadays, increasing the likelihood that non hemodynamically significant stenoses are being dilated and stented. Under these conditions, pressure-derived Fractional Flow Reserve measurements can be used as a useful surrogate for functional testing (3). Likewise, in patients with multiple stenoses, there is a need for identifying which of the many lesions are flow-limiting and deserve treatment with the expensive DES. Conventional functional imaging, in particular MPI, often falls short in this subset of patients with a proportion of cases showing normal MPI and other studies failing to identify less critical but significant stenoses. Here again, the invasive Fractional Flow Reserve method may prove useful, as currently tested in a prospective randomised trial (FAME).

From the above, it is clear that addressing these new challenges requires rethinking of some of the currently proposed options for non invasive functional testing in order to make them compatible in practical terms with the new clinical scenarios. One interesting approach might be the development of hybrid technologies that combine SPECT or PET with MS-CT, the so-called "one stop shop" approach (4).

2. Imaging activity of atherosclerotic vascular disease.

An increasing number of sophisticated invasive techniques is being developed in order to identify vulnerable plaque based on anatomic or functional features, as known from histopathology. These tools are being evaluated in prospective natural history studies for their ability to predict sites of recurrence in patients recovering from an acute episode. At the same

time, invasive measurements of plaque vulnerability are being used as surrogate endpoints for ongoing, prospective drug-therapy trials. These studies may be relevant for further development of plaque imaging. Although challenging, it may prove rewarding to invest on translating the most successful invasive imaging methods into clinically useful non invasive modalities for application to coronary or non coronary territories.

Successful examples of cross-talk between invasive and non invasive imaging have emerged in the electrophysiology laboratory by the use of MS-CT of the left atrium and pulmonary veins during ablative treatment of atrium fibrillation, in combination with catheter-based Noga imaging (5).

3. Providing evidence for the use of imaging modalities in specialized populations, in particular elderly patients.

Specialized populations are currently actively investigated and receiving increased attention, which is appropriate. Women and diabetics often are underdiagnosed and undertreated, yet their prognosis is poor. Likewise, little is known about the relative performance of non invasive imaging in elderly patients, an expanding target group with the expected worldwide ageing of the population. As an illustration, the BASKET trial has recently demonstrated that DES do prevent in-stent restenosis, as expected from trial-based evidence, but at the same time are associated with an increased risk of infarction and death when used in elderly patients (6). It is likely that several imaging modalities suffer from specific limitations in the elderly, calling for an appraisal of the optimal diagnostic strategies in this specific subset.

4. Continue to develop molecular imaging, imaging of gene expression and modalities for in vivo cell tracking.

Although this remains a research-oriented and long term goal, the emergence of cell therapy with bone-marrow derived stem cells for the acute myocardial infarction indication (7) has created a need for imaging modalities that allow non invasive cell tracking in vivo. These approaches will be needed to evaluate optimized solutions for the therapy with the use of homing factors or enriched cell populations as well as to provide a better understanding of the fate of the transplanted cells.

In summary, a number of take-home statements can be proposed.

- The issue is not whether Anatomy "or " Physiology will prevail. I need both.
- With the availability of an increasing number of imaging options, the emphasis will be placed more than ever on cost/effectiveness (on a population basis) and predictive accuracy (in the individual subject).
- I expect my non invasive adviser to master multiple imaging modalities and to help me in selecting the most appropriate path for a given patient.

Relevant References.

1. Bax JJ, Kramer C, Marwick TH, Wijns W. Cardiovascular Imaging. A Handbook for Clinical Practice. ESC Education Series. Blackwell Publishing, 2005, 312 p.
2. Meier B, Ramamurthy S. Plaque sealing by coronary angioplasty. Cathet Cardiovasc Diagn. 1995;36:295-7.
3. Berger A, Botman KJ, MacCarthy PA, Wijns W, Bartunek J, Heyndrickx GR, Pijls NH, De Bruyne B. Long-term clinical outcome after fractional flow reserve-guided percutaneous coronary intervention in patients with multivessel disease. J Am Coll Cardiol. 2005;46:438-42.
4. Namdar M, Hany TF, Koepfli P, Siegrist PT, Burger C, Wyss CA, Luscher TF, von Schulthess GK, Kaufmann PA. Integrated PET/CT for the assessment of coronary artery disease: a feasibility study. J Nucl Med. 2005;46:930-5.
5. Tops LF, de Groot NM, Bax JJ, Schalij MJ. Fusion of electroanatomical activation maps and multislice computed tomography to guide ablation of a focal atrial tachycardia in a Fontan patient. J Cardiovasc Electrophysiol. 2006;17:431-4.
6. Kaiser C, Brunner-La Rocca HP, Buser PT, Bonetti PO, Osswald S, Linka A, Bernheim A, Zutter A, Zellweger M, Grize L, Pfisterer ME; BASKET Investigators. Incremental cost-effectiveness of drug-eluting stents compared with a third-generation bare-metal stent in a real-world setting: randomised Basel Stent Kosten Effektivitats Trial (BASKET). Lancet. 2005;366:921-9. Erratum in: Lancet. 2005;366:2086.
7. Bartunek J, Dimmeler S, Drexler H, Fernandez-Aviles F, Galinanes M, Janssens S, Martin J, Mathur A, Menasche P, Priori S, Strauer B, Tendera M, Wijns W, Zeiher A; task force of the European Society of Cardiology. The consensus of the task force of the European Society of Cardiology concerning the clinical investigation of the use of autologous adult stem cells for repair of the heart. Eur Heart J. 2006;27:1338-40.

Drug Eluting Stents - Results in the Real World
- Dr. Pravin Chandra

Abstract Not received

Multi vessel Stenting - Has it replaced Surgery?
- Dr. Pravind Chandra

Abstract Not received

Vulnerable Plaque : Where are we with virtual histology ?
- Dr. Anuja Nair

Abstract Not received

No options Therapy for CAD
- Dr. Keyur Parikh/Dr. Shmuel Banai

No Options Therapy-Coronary Sinus Reducer-Stent for the Treatment of Chronic Refractory Angina Pectoris

A prospective, open label, multi-center safety feasibility first-in-man study

Shmuel Banai, Shmuel Ben Muvhar, **Keyur H Parikh**, Aharon Medina, Horst Sievert, Ashok Seth, Jonathan Tsehori, Yoav Paz, Ami Sheinfeld, Gad Keren

From the Cardiology Department Tel Aviv Medical Center, Tel Aviv, Israel (SB, GK), **The Heart Care Clinic / SAL Hospital, Ahmedabad, India (Keyur H.Parikh**), Max Devki Devi Heart and Vascular Institute, New Delhi, India (AS), Cardio Vascular Center Frankfurt, Sankt Katharinen, Frankfurt, Germany (HS), Bikur Cholim Hospital, Jerusalem, Israel (AM)

Abstract:

Background: Increased Coronary Sinus (CS) pressure can reduce myocardial ischemia by redistribution of blood from non-ischemic to ischemic territories. The CS Reducer is a percutaneous implantable device, designed to establish CS narrowing and to elevate CS pressure. In pre-clinical experiments, implantation of the Reducer was safe and was associated with improved ischemic parameters. In the present study the safety and feasibility of the CS Reducer was evaluated in patients with refractory angina who are not candidates for revascularization.
Methods: Fifteen CAD patients with severe angina, and reversible ischemia were electively treated with the Reducer.**10 of the 15 patients were done through The Heart Care Clinic ,Ahmedabad.** Clinical evaluation, dobutamine-echo, thallium SPECT, and angina questionnaire were performed before and 6 months after implantation. Cardiac CT was performed 2 days and 6 months after implantation.

Results:

All procedures were completed successfully. No procedural-related adverse events have occurred during the peri-procedural and the follow-up periods. Angina score improved in 12 of 14 patients. Average CCS was 3.07 at baseline and 1.64 at follow-up ($n=14$, $p<0.0001$). Stress induced ST Segment depression was reduced in 6 of 9 patients and was eliminated in 2 of these 6 ($p=0.047$). The extent and severity of myocardial ischemia by dobutamine-echo and by thallium SPECT was reduced, $p=0.004$, $n=13$, and $p=0.042$, $n=10$, respectively.

Conclusions:

Implantation of the CS Reducer is feasible and safe. These findings along with the clinical improvement observed support further evaluation of the Reducer as an alternative treatment for patients with chronic refractory angina who are not candidates for coronary revascularization(No options Patients)

Radial A. Vs Femoral A approach in Coronary Intervention - How ? When ? Why ? - Dr. Satya Gupta, Ahmedabad, India

The femoral artery has traditionally been the preferred access site for coronary interventions, but this approach has some limitations. It is relatively contraindicated in the presence of severe peripheral vascular disease and patient receiving anticoagulation treatment. A period of post procedure recumbency is needed to avoid disruption of the arterial puncture site. This may be poorly tolerated by patients with pulmonary congestion, lung disease, or back or hip pain. Despite bed rest, the rate of complications at the femoral access site (haematoma, pseudoaneurysm, a-v fistula, need for blood transfusion or surgical arterial repair) is 2-8% after transfemoral interventions. These factors together with patients satisfaction, morbidity, length of hospital stay, and cost have driven the development of alternate vascular access for coronary procedure.

The radial artery is a potentially safer entry site for percutaneous interventions. It was pioneered by Lucien Campeau who performed first hundred transradial cardiac catheterization procedures. The first transradial angioplasty as well as stenting was performed by Pro Kiemeneij and his colleagues at Amsterdam in 1992. Subsequently this approach has been widely practice worldwide.

The superficial course of radial artery facilitates hemostasis by simple application of a pressure bandage over the puncture site. Bleeding from puncture site is immediately noticed by patient, who can at least temporarily address this problem himself. The absence of major veins makes the incidence of arteriovenous fistula rare. The most striking advantages is immediate ambulation of the patient.

However, transradial coronary angioplasty technique appeared to be technically more demanding compared to the traditional femoral technique, since smaller guiding catheters have to be used. In addition, these catheters are approaching the coronary arteries from the right side instead of the left side making adequate coronary cannulation a greater challenge. Downsized angioplasty equipment has to be selected, require more delicate handling. Anatomy of the arm and subclavian arteries is less well known to interventional cardiologist and abnormal origin of vessel can be obstacles some time.

Though there are several advantages of radial approach, it is not appropriate in the minority of patients viz , patients with abnormal Allen's test, Raynauds disease, Hemodynamically unstable patient, patient with complex procedure need IABP, multiple stenting or large size catheters. Furthermore, the risk of asymptomatic occlusion of radial artery after transradial procedure is 2-5%, and the cardiac surgeon who uses radial graft frequently may not prefer transradial angiography.

Transradial technique is still evolving, awaiting newly designed physician's friendly catheters to combat difficult engagement. Although I doubt that transradial access will ever replace transfemoral intervention, I do believe that this type of access has an important role for diagnostic and interventional procedures.

"Profits at bottom of pyramid in Cardiovascular Care "
- Prof. Dileep Mavalankar, Ahmedabad, India

Profits at the Bottom of the Pyramid in Cardiovascular Care - An public health approach - Prof. Dileep Mavalankar IIM, Ahmedabad

Bottom of the pyramid is a very famous book and approach developed by well known Management Guru Prof. C.K. Prahalad. The basic idea in this approach is that many companies globally have developed products and services which meet the needs of lower middle class and the poor. These companies have made substantial profits by serving the needs of the lower segments of the community. Generally in most countries and especially in developing countries, the income pyramids have very small tip but very large bottom. Hence when a company starts serving the needs of the bottom of the pyramid their potential market expands very rapidly. Each unit of the product may not generate much profit but they can sell large number of units and hence make substantial profit.

This presentation will take the concepts of bottom of pyramids and see how they could be applied using basic public health and epidemiological understanding of cardiovascular diseases. In epidemiology, it is well known that the disease distribution in a community is like an iceberg with a very small tip which is visible clinically and a large bottom which is invisible. We argue that by trying to address the invisible disease in the community and the disease among the poor and lower middle class, the individual cardiologists and cardiac hospitals can make sizeable profits by increasing their overall market. This requires reduction in prices, increasing efficiency and systematization of work as well as developing a preventive orientation so that diseases can be identified at much earlier stage through large scale screening. This will not only help hospitals and doctors but would be a major saving to the patient and to the community.

Get Unstuck - Dr. Jagdish Hiremath
GET UNSTUCK

Convention is something that was convenient in the "past". Conventional wisdom was contemporary, which means it was situational. As situations change, wisdom should change and one should not be scared of re-defining tradition. Following a convention or a tradition blindly leads to stuck behaviours. Despite the intellectuals prowess as doctors we are stuck with variety of aspects of life.

We get stuck to fixed ideas, responsibilities, images and faiths. We tend to follow instincts

based on egos, past and future rigidness and emotions. This leads to many disadvantages. If a river stops flowing it becomes a pond. Our lives should flow like rivers and should stop getting stuck and becoming ponds.

Advantages of getting unstuck are many. Applying traditions in today's modern world, examining conventions with our current wisdom leads to wider fields, horizon, new ideas, friends, enriched life. In order to lead unstuck life we need to have a good body, good mind, we should learn to rise above the taught culture, learn to live in the present tense and enjoy the present moment. In order to do this, we need to have correct diet, regular exercise, practices like yoga and meditation. You must refrain from addictions and look at life in a spiritual way so that we as intellectuals remain flowing, remain full of energy and in turn help our family, social circle and the society. This means "Getting Unstuck".

Satellite Symposia
Day-1 Friday 26th Jan. 2007 (8.00 pm to 10.00 pm)
Venue : Hotel Inder Residency

Recent updates in General Cardiovascular Medicine

Chair : Dr. Milan Chag, Dr. Pravin Chandra
Panelist : Dr. S. Kaushik, Dr. Saibal Kar, Dr. Raj Makkar, Dr. Jagdish Hiremath, Dr. Joyal Shah, Dr. Niren Bhavsar

- Chest X-Ray in CaSatellite Symposia - *Dr. Milan Chag*
- Risk Stratification in ACS - *Dr. Pravin Chandra*
- Ten Important points to remember about diet and cardiovascular health. - *Dr. S. Kaushik*
- Closure of PFOs for stroke-Where are we in 2007 - *Dr. Saibal Kar*
- Stem Cell Therapy: Future directions - *Dr. Raj Makkar*
- Differential impact of blood pressure lowering drugs on central aortic pressure and clinical implications - *Dr. Jagdish Hiremath*

Recent updates in ECG & Arrhythmia

Chair : Dr. Ajay Naik, Dr. V. S. Prakash
Moderator : Dr. Manojkumar Rohit, Dr. Vineet Sankhala

- My worst nightmare in the ICU : Real Life Clinical Scenarios - 60 mins
- Interactive ECG session - 60 mins

Recent updates in Cardiac Pharmacology

Chair : Dr. Ake Hjalmarson, Prof. Nakul Sinha
Panelist : Dr. P. C. Manoria, Dr. Anish Chandarana, Dr. Mihir Tanna, Dr. Satish Patel

- Aldosterone Inhibitors : For Whom ? How Long ? - *Dr. Ake Hjalmarson*
- Aspirin and Clopidogrel Resistance : Fact OR Fiction? - *Dr. P. C. Manoria*
- Let's Raise Some HDL - *Dr. Joyal Shah*
- ARBs vs. ACE Inhibitors: Which Are Most Effective? - *Dr. Anish Chandarana*
- Antocoagulation in Patients with Heart Failure,Who,When and Why? - *Prof. Nakul Sinha*
- Lipid-lowering after acute coronary syndrome: Is it how low you go or how you go low? - *Dr. Mihir Tanna*

Recent updates in ACS/CAD

Chair : Dr. William Wijns , Dr. Keyur Parikh
Panelist : Dr. Anuja Nair, Prof. Shmuel Banai,Dr. Satya Gupta, Dr. Mitchell Krucoff Dr. Urmil Shah, Dr. Hemang Baxi

- Vulnerable Plaque : Can We Find it and Fix it ? - *Dr. Anuja Nair*
- Myocardial Preservation After AMI : Will Novel Therapies Change Future Treatment Paradigms - *Dr. William Wijns*
- Antithrombotic Approaches in Acute Coronary Syndrome - Prof. Shmuel Banai
- Predictive Accuracy of 64 Slice CT Angiography for the At-Risk Lesion and Patient - A Cardiologist's perspective - *Dr. Satya Gupta/Dr. Keyur Parikh*
- High Tech in the Cath Lab—Is That All You Have to Offer? Music, Imagery, Touch and Prayer as Adjuncts to Interventional Cardiac Care: The MANTRA Study Project - *Dr. Mitchell Krucoff*

A. Recent updates in General Cardiovascular Medicine

a) Chest X-Ray in Cardiology - Dr. Milan Chag

Abstract not received

b) Risk Stratification in ACS - Dr. Pravin Chandra

Abstract not received

c) Ten important points to remember about diet and cardiovascular health. - Dr. S. Kaushik

Abstract not received

d) Closure of PFOs for stroke-Where are we in 2007 - Dr. Saibal Kar

Abstract not received

e) Stem Cell Therapy : Future directions - Dr. Raj Makkar

Abstract not received

f) Differential impact of blood pressure lowering drugs on central aortic pressure and clinical implications.- Dr. Jagdish Hiremath

Traditionally blood pressure is measured over the brachial artery and reduction in brachial artery blood pressure is proven to reduce the cardiovascular event risk in hypertensive and coronary artery disease patients. However numerous trials like HOPE and EUROPA have shown that only blood pressure lowering does not explain the reduction in cardiovascular events and there are benefits beyond BP reduction.

The recently published ASCOT trial showed that the reduction in brachial artery blood pressure was similar in amlodipine + peindopril group and the atenolol + thiazide group, however the reducing in cardiovascular events was significantly superior in the group receiving amlodipine + perindopril. One of the probable reason suggested was the superior reduction in the central aortic blood pressure, which was evaluated in the CAFÉ study. The CAFÉ study evaluated the central aortic blood pressure of 2199 ASCOT patients using a non-invasive technique, called radial applanation tonometry. Throughout the study period the central aortic blood pressure was significantly lower in the group receiving amlodipine + perindopril. CAFÉ study also suggested that reduction in central aortic blood pressure is a better predictor of Cardiovascular outcomes.

Central aortic blood pressure (CABP) differs from the brachial as it is dependent on a number of factors like the pulse wave velocity and arterial stiffness. ACE inhibitors as proven to alter these parameters and are known to reduce the central aortic blood pressure. Apart from the CAFÉ study, the RESAON study with perindopril + indapamide has also shown an improvement in parameters of central aortic blood pressure. Calcium channel blockers are also likely to improve the CABP whereas Beta-blockers could increase the CABP.

CAFÉ along with ASCOT trial is influencing the management of hypertension. Based on the results of these trials the latest BHS/NICE guidelines recommend to initiate the management of hypertension with ACE inhibitors in patients below 55 years and if required add a diuretic or a CCB as necessary.

In conclusion, a brachial artery systolic pressure may not be a good estimate of central aortic pressure. CABP appears to be a better predictor of cardiovascular outcomes. In clinical practice more evidence is required till we base our anti hypertensive treatment on CABP.

B. Recent updates in ECG & Arrhythmia

a) My worst nightmare in the ICU : Real Life Clinical Scenarios

b) Interactive ECG session

C. Recent updates in Cardiac Pharmacology

a) Aldosterone Inhibitors : For Whom ? How Long ? - Dr. Ake Hjalmarson

Abstract not received

b) Aspirin and Clopidogrel Resistance : Fact OR Fiction? - Dr. P. C. Manoria

Aspirin and clopidogrel resistance has emerged as a real reality. It is now documented that the phenomenon of aspirin and clopidogrel resistance seen in the haematology laboratory is associated with adverse clinical events. Aspirin resistance is diagnosed if arachidonic acid (.5 mg/ml) induced maximal platelet aggregation is >20% or adenosine diphosphate (5/20 mmol/1) induced maximal platelet aggregation is >70%. Clopidogrel resistance is diagnosed, if the adenosine diphosphate (5/20 mmol/l.) induced platelet aggregation shows <10% reduction compared to pretreatment values. In semiresponders there is 10-29% reduction in platelet aggregation. Aspirin resistance is believed to be due to synthesis of thromboxene A_2 by nucleated cells even in phase of aspirin treatment, over expression of cox-2 pathway as atherosclerosis is an inflammatory disease and genetic polymorphism involving the cox-1 pathway. Aspirin resistance is treated by increasing dose of aspirin in pharmacokinetic aspirin resistance and adding clopidogrel in pharmacodynamic and psuedo aspirin resistance. Clopidogrel resistance is caused by several extrinsic and intrinsic mechanisms. Clopidogrel resistance is treated by strict compliance in using the drug, avoiding drug interactions decreasing clopidogrel action, increasing dose and use of new antiplatelate

agents like prasugrel and others.

c) Let's Raise Some HDL - Dr. Joyal Shah

Till recent time more attention in majority has been directed at lowering "bad cholesterol" than at raising "good cholesterol," and this is true in both layperson's and healthcare professional circles. Patients with lipid and lipoprotein abnormalities are commonly referred to as having *hyper*lipidemia; treatments for dyslipidemia are commonly termed *lipid-lowering* therapies; and lipoprotein targets of consensus guidelines are based on lowering LDL-C.

HDL is instrumental in mediating reverse cholesterol transport, has antioxidant properties, and exerts other potentially vasculoprotective effects. The risk of CAD increases as circulating HDL-C levels decrease. **HDL-C increases of only 6% have been associated with significant reductions in coronary morbidity and mortality in patients with low HDL-C**
The importance of the metabolic effects of HDL has been reflected in consensus guidelines for nearly a decade.[1,2,10] In the second National Cholesterol Education Program (NCEP) Adult Treatment Panel II (NCEP ATP-II) consensus guidelines, a high HDL-C (> 60 mg/dL) was classified as a "negative" or "inverse" risk factor, and hence protective against future cardiovascular events.[1] The third NCEP consensus guidelines (ATP-III) increased the number of patients eligible for treatment by raising the upper limit of "low HDL-C" from 35 mg/dL to 40 mg/dL.

By introducing the concept of 10-year absolute coronary risk ("global risk"), the ATP-III also indirectly increased the focus on HDL-C. When computing global risk, 1 point is added to the risk score if HDL-C is between 40 and 49 mg/dL, and 2 points are added if HDL-C is < 40 mg/dL; 1 point is subtracted from the risk score if HDL-C is >/= 60 mg/dL. Finally, the ATP-III also identified metabolic syndrome, of which low HDL-C is component (defined as HDL < 50 mg/dL in women, < 40 mg/dL in men).

Although the ATP-III recognized HDL-C as a potential secondary target for therapy after high levels of LDL-C and non-HDL-C, it did not set HDL-C "goals" as they exist for LDL-C and non-HDL-C. However, inasmuch as the guidelines are explicit as to the desirability of high HDL-C levels — or raising HDL-C levels that are too low .

Every 1-mg increase in HDL-C, there is a 2% to 3% decrease in cardiovascular risk.[7] Individuals in the 80th percentile of HDL-C are at 50% lower coronary risk compared with those in the 20th percentile... For every 1-mg increase in HDL-C, there is a 2% to 3% decrease in coronary risk HDL particles are highly heterogeneous in structure, intravascular metabolism and antiatherogenic activity. HDL particles possess multiple antiatherogenic activities, which include reverse cholesterol transport from the arterial wall to the liver for excretion, and antioxidative, anti-inflammatory, antiapoptotic, antithrombotic, anti-infectious and vasodilatory actions Small, dense HDL possesses potent antioxidative activity but this is compromised under conditions of atherogenic dyslipidemia. HDL functional deficiency frequently coincides with reductions in HDL-cholesterol concentration and alterations in HDL metabolism and structure. Formation of small, dense HDL particles with attenuated antiatherogenic activity can be mechanistically related to HDL enrichment in triglycerides and in serum amyloid A, depletion of cholesteryl esters, covalent modification of HDL apolipoproteins and attenuated antiatherogenic function of apolipoprotein AI. Low circulating levels of HDL cholesterol might, therefore, be associated with the defective functionality of small HDL particles of abnormal structure and composition. In common metabolic diseases, such as

type 2 diabetes and metabolic syndrome, deficiency of HDL particle number and function favor accelerated atherosclerosis. Therapeutic normalization of the quantity, quality and biological activities of HDL particles thus represents a novel approach to attenuating atherosclerosis in dyslipidemic individuals with metabolic disease. Cholesteryl ester transfer protein inhibitors, nicotinic acid, reconstituted HDL and other HDL-raising agents are being investigated. Induction of selective increase in the circulating concentrations of small, dense HDL3 particles with increased antiatherogenic activity seems especially promising, particularly for therapy of atherogenic dyslipidemia.

Increasing concentrations of HDL progressively reduce cardiovascular risk and protect against CHD even in the presence of elevated LDL levels. Routine clinical measurements of plasma HDL-cholesterol primarily reflect levels of large, cholesterol-rich particles and frequently lack the sensitivity to detect small, cholesterol-poor HDL. For example, small HDL3c represents a minor subfraction, accounting for about 6% of total HDL mass, 10% of apo AI and 13% of HDL particles.[8,9,10,12] The clinical significance of circulating levels of individual HDL subfractions is nonetheless unclear. Small, dense HDL3 displays high cholesterol efflux capacity, affords potent protection of LDL against oxidative stress and possesses strong anti-inflammatory properties.
Therapeutic normalization of both the quantity and quality of HDL particles, and especially of the function of small, dense HDL thus constitutes a novel approach to attenuating atherosclerosis in dyslipidemic individuals with metabolic disease. This innovative concept implies that small, dense HDL3 represents a new therapeutic target in atherogenic dyslipidemia.

d) ARBs vs. ACE Inhibitors: Which Are Most Effective? - Dr. Anish Chandarana

Abstract not received

e) Antocoagulation in Patients with Heart Failure,Who,When and Why? - Prof. Nakul Sinha

Abstract not received

f) Lipid-lowering after acute coronary syndrome: Is it how low you go or how you go low ? - Dr. Mihir Tanna

Clinical trials have shown that the lowering of total cholesterol or low-density lipoprotein cholesterol levels substantially reduces the risk of morbidity and mortality due to coronary heart disease. Previously, the benefits of lipid lowering in patients with acute coronary syndromes was less well studied. The majority of statin trials excluded patients who had experienced recent unstable angina or acute myocardial infarction and, therefore, were not able to evaluate the potential beneficial effects that may result from early statin therapy. However, new evidence is emerging that statins may be effective immediately after an acute coronary event. Recent observational studies indicate that patients who are treated with a statin early after a coronary event have a more favourable outcome than those who are not, and retrospective analyses of clinical trial databases of patients with acute coronary syndromes have also shown this pattern. Statin therapy favourably impacts interrelated pathophysiologic mechanisms that are intimately involved

in the pathogenesis of acute coronary syndromes, most notably endothelial function, platelet thrombus deposition and inflammation.The following important findings and their practical implications are highlighted:

1. When to start lipid lowering therapy: Effect of early vs. Late onset therapy: The MIRACL Study
2. Statins: Are they all same
3. Single drug or multiple drugs?
4. Intensive vs. moderate lipid lowering: PROVE IT-TIMI22 Study
5. Effect of Discontinution of lipid lowering therapy after ACS
6. Safety and Tolerability of Single and Multiple drugs
7. Important Drug Interactions
8. Future Directions

D. Recent updates in ACS/CAD

a) Vulnerable Plaque : Can We Find it and Fix it ? - Dr. Anuja Nair

b) Myocardial Preservation After AMI : Will Novel Therapies Change Future Treatment Paradigms - Dr. William Wijns

William Wijns, MD, PhD
Cardiovascular Center, OLV Ziekenhuis Aalst, Belgium

The use of bone marrow-derived stem cells is a promising approach to facilitate myocardial regeneration of acutely infracted myocardium or in developed congestive approach. Current clinical data seem to indicate that this approach is safe and not associated with overall increase in adverse clinical events. Nevertheless, the level of safety confidence is limited due to limited number of treated patients and absence of long-term clinical follow-up. The further process of establishing the clinical safety should include further collection of the clinical data from previous and ongoing clinical data in various subsets of patients relative to their background risk. In parallel, several conceptual safety concerns should be actively scrutinized. They relate to a number of operational mechanisms like myocardial biological effects on differentiation, remote homing, progression/regression of atherosclerosis and arrhythmias. The pro-active scrutiny of these phenomenon could facilitate the safe translation of the promise of cardiac regeneration to an effective therapy

The presence of stem cells in the bone marrow, their versatility and homing at the site of the injuried myocardium as well as experimental evidence that stem cells may biologically and functionally regenerated infarcted myocardium make bone marrow-derived cells one of the most promising cell types for cardiac regeneration. Results of initial trials sparked enthusiasm and a number of ongoing intermediate-size randomized trials evaluate therapeutic effects in patients with recent myocardial infarction or congestive heart failure. Here, we review the current evidence for the clinical safety of the therapy and highlight conceptual risks that need to be scrutinized in further translational and clinical research.

Current evidence for the clinical safety of the stem cell therapy remains overall optimistic.

Nevertheless, given the limited number of patients and only short to medium term follow-up as well as heteregoneus design of published trials, class I confidence level is not yet reached. Instead, cautious optimism should be strengthen by the long-term follow-up of cell-treated patients and by designing specific, hypothesis-driven clinical or basic studies to address conceptual concerns. The process towards greater confidence in the clinical setting could be greatly facilitated by creation of registries monitored by independent committees like ESC Task Force on Stem Cell Therapy.

Bartunek J, Dimmeler S, Drexler H, Fernandez-Aviles F, Galinanes M, Janssens S, Martin J, Mathur A, Menasche P, Priori S, Strauer B, Tendera M, Wijns W, Zeiher A; task force of the European Society of Cardiology. The consensus of the task force of the European Society of Cardiology concerning the clinical investigation of the use of autologous adult stem cells for repair of the heart. Eur Heart J. 2006;27:1338-40.

c) Antithrombotic Approaches in Acute Coronary Syndrome - Dr. Shmuel Banai

d) Predictive Accuracy of 64 Slice CT Angiography for the At-Risk Lesion and Patient - A Cardiologist's perspective - Dr. Satya Gupta/Dr. Keyur Parikh

Recent developments in computed tomography technology have made imaging of the coronary arteries possible. With the latest generation 64 slice computed tomography and adequate patient preparation (which includes lowering of the heart rate), rates of sensitivity ranging from 83% to 99% and specificity between 93% to 98% have been reported for the detection of coronary artery stenosis in comparison with invasive coronary angiography.

The high negative predictive value (95% to 100%) reported in various studies suggests that coronary CT angio may be a useful diagnostic technique to rule out the presence of coronary stenoses in selected patients, especially those with a rather low pretest likelihood of disease. Imaging of coronary artery bypass grafts is reliable, but clinical application can be hampered by difficulties in assessing the native coronary arteries in patients after undergoing bypass because of their often severe calcification. The detection of in-stent restenosis is made difficult by artifacts caused by metal, especially in smaller stents.

Based on current scientific studies, multislice CT angio is clinically useful to rule out the presence of coronary stenoses in patients who have clinically suspected coronary artery disease with the need for further workup, but a low to intermediate pre-test likelihood of diease. Furthermore, CT angio can not replace conventional diagnostic coronary angiography but it is an alternative diagnostic test in certain clinical situations.

e) High Tech in the Cath Lab—Is That All You Have to Offer? Music, Imagery, Touch and Prayer as Adjuncts to Interventional Cardiac Care: The MANTRA Study Project - Dr. Mitchell Krucoff

3 - C CON 2007
Day-2 Saturday 27th January 2007
Tagore Hall, Paldi, Ahmedabad

7:30 am	Registration & Breakfast
	Cardiac Arrhythmias
	Chair : Dr. Ajay Naik, Dr. V. S. Prakash
	Moderator: Dr. Manojkumar Rohit, Dr. Anish Chandarana
8:00 am	Masters at your Mercy - *Dr. V. S. Prakash / Dr. Ajay Naik / Dr. Manojkumar Rohit*
8:30 am	Syncope - Common problems, vexing issues - *Dr. Manojkumar Rohit*
9:00 am	How I cure Atrial Fibrillation & VF - *Dr. V. S. Prakash*
9:30 am	Promises beyond Amio & Warf - *Dr. Ajay Naik*
10:00 am	Question & Answer
10:10 am	PLENARY LECTURE - "Revisiting Clinical Examination in 21st Century" - *Dr. Pravin Shah*
10:35 am	Refreshment Break
	Hypertension, Lipids & Preventive Cardiology
	Chair : Dr. Pravin Shah, Dr. Milan Chag
	Moderator: Prof. Nakul Sinha, Dr. Gunvant Patel
10:45 am	Cardiogenic Stock - State of the Art 2007 - *Dr. Jagdish Hiremath*
11:05 am	Controversies in Lipid-Lowering Therapy - *Prof. Nakul Sinha*
11:25 am	Hypertension 2007: Controversies and Treatment Challenges - *Prof. S. K. Kaushik*
11:45 am	Treating the Metabolic Syndrome in Clinical Practice : Multidisciplinary Care - *Dr. P. C. Manoria*
12:05 pm	ACC/AHA Guidelines 2006 - Management of Patients With Asymptomatic Valvular Heart Disease - *Dr. Pravin Shah*
12:25 pm	Mitral valve in heart failure and ischemic disease - *Dr. Alain Berrebi*
12:45 pm	Question & Answer
12:55 pm	Lunch
1:25 pm	3-C Con Oration - Practical Insights into Primary PCI: What all physicians should know? - *Dr. Sameer Mehta*
1:50 pm	**Unique Question & Answer Session with FACULTY**
	Myocardial Ischemia , Infarction & Intervention
	Chair: Dr. Keyur Parikh, Dr. Sameer Mehta
	Moderator: Dr. Raj Makkar, Dr. Balram Bhargava
2:20 pm	Guideline based medicine : Can we implement it in India ? - *Dr. D. Prabhakaran*
2:40 pm	Use of Bivalirudin in ACS, AMI & PCI - *Dr. Sameer Mehta*

3:00 pm Acute Myocardial Infarction: Current State of the Art - The first 24 hours - *Prof. Shmuel Banai*

3:20 pm Effect of door-to-Balloon or door to thrombolytic time on Mortality in Patients with ST-Segment Elevation Myocardial Infarction - *Dr. Manish Parikh*

3:35 pm Biomarkers in Ischemic Heart Disease - *Dr. Manish Parikh*

3:50 pm Benefit of Early Invasive Therapy in Acute Coronary Syndromes - A Meta-Analysis of Contemporary Randomized Clinical Trials - *Dr. Upendra Kaul*

4:10 pm Enoxaparin Vs other LMWH Vs Unfractionaled heparin ? Where are we in 2007? - *Dr. Balram Bhargava*

4:30 pm Patients with Left Main Disease: Will the Interventionalists and Surgeons Ever Get on the Same Page? - *Dr. Raj Makkar*

4:45 pm Medical Malpractice in Cardiovascular Medicine - *Dr. Sameer Mehta*

5:00 pm Delegates may stay at Tagore Hall to learn Basic Echocardiography in Parallel Session-A

Parallel Session-A

Basic Echocardiography (Time : 5.00 PM to 7.30 PM)

Venue : Tagore Hall, Ahmedabad

Chair : Dr. Pravin Shah, Dr. Hemang Baxi

Panelist : Dr. Milan Chag, Dr. S. K. Parashar, Dr. Alain Berrebi

- Assessment of Left Ventricular systolic function : 2-D and Doppler parameters. (ejection fraction, LV volumes, cardiac output) - *Dr. S. K. Parashar*
- Doppler Haemodynamics Assessment : Principles, Formulae and Calculations - *Dr. Pravin Shah*
- Diastology : A practical approach to evaluation of diastolic function of Heart by Doppler Echocardiography - *Dr. S. K. Parashar*
- Aortic & Mitral Stenosis: 2-D / Doppler assessment & Quantitation of severity - *Dr. Hemang Baxi*
- Echocardiographic Assessment of Tricuspid & Pulmonic disease: Right Heart pressure & Right Ventricular function - *Dr. Vineet Sankhala*
- Rapid Fire Recorded Cases

5.00 PM TRANSPORTATION FROM TAGORE HALL TO RAJPATH CLUB FOR PARALLEL SESSION-B

Parallel Session-B

Real Life Cases (Time : 6.00 to 8.00 PM)

Venue : Diamond Hall, Rajpath Club, Ahmedabad

Chair : Dr. Keyur Parikh, Dr. William Wijns

Panelist : Dr. Manish Parikh, Dr. Ajay Naik, Dr. Anish Chandarana, Dr. Urmil Shah, Dr. Mitchell Krucoff, Dr. Upendra Kaul

- 44-year-old male was prompted to have a cholesterol evaluation following his father's coronary surgical procedure and post-operative coma from a thrombo-occlusive stroke. His lipid profile revealed a total cholesterol of 207 mg/dl, LDL cholesterol of 156 mg/dl, triglycerides of 125 mg/dl, and an HDL cholesterol of 26 mg/dl. - *Dr. Upendra Kaul*
- ECG - A middle aged man is referred for a second opinion regarding seizure disorder - *Dr. Ajay Naik*
- Syncope in Young lady : Benign or Malignant ? - A young lady presents with syncope - *Dr. Manojkumar Rohit*
- Echo - A 72-year-old man has NYHA functional class 3 heart failure and a systolic ejection murmur in the "aortic" position - *Dr. Anish Chandarana*
- Twenty-Year-Old Man with Severe Chest Pain Sudden severe left-sided chest pain while playing cricket - *Dr. Urmil Shah*
- 62-Year-Old Woman With Hypoxia With Standing and Exercise - The patient was a 62-year-old woman with past medical history of gastroesophageal reflux disease, daytime somnolence, and fatigue. She was noted to have hypoxemia at rest and with exercise. - *Dr. Manish Parikh*
- Post-Myocardial Infarction Syncope - A 64-Year-old female presented with syncope not preceded by chest pain, dyspnea, or palpitaion. During the night, she climbed out of bed, felt dizzy and passed out. Two minutes later, she regained consciousness but was dizzy and diaphoretic - *Dr. Mitchell Krucoff*
- Real life case - *Dr. William Wijns*

ONLY FOR DELEGATES

At 8.00 PM all delegates meet at The Heart Care Clinic for appetizers/snacks followed by dinner at Diamond Hall, Rajpath Club

ONLY FOR DELEGATES

An excellent entertainment program will be presented by SPIN Academy at Diamond Hall, Rajpath Club from 9.30 PM-10.30 PM

Cardiac Arrhythmias

Masters at your Mercy - Dr. V. S. Prakash / Dr. Ajay Naik/ Dr. Manojkumar Rohit

Abstract not received

Syncope - Common problems, vexing issues. – Dr. Manojkumar Rohit

Abstract not received

How I cure Atrial Fibrillation & VF - Dr. V. S. Prakash

Abstract not received

Promises beyond Amio & Warf - Dr. Ajay Naik

NEW ANTIARRHYTHMIC AGENTS

Beta blockers and Amiodarone have been the mainstay of therapy in arrhythmia management. Novel antiarrhythmic agents are now under clinical scrutiny for promises of effective therapy without the drawbacks of proarrhythmia and adverse effects.

Dronedarone is an Amiodarone analogue without the Iodine moiety. EURIDIS and ADONIS trials have revelead that It is as effective as Amiodarone but without attendant thyroid or pulmonary toxicity. It has once a day dosage and minimal drug interactions compared to Amiodarone.

RSD 1235 is an atrial selective agent that prolongs repolarization. CRAFT and ACT 1 studies have shown that this drug prevents AF relapses post Cardioversion significantly compared to Placebo. **There is no QT prolongation or ventricular proarrhythmia with this drug.**

AVE 0118 has also shown prevention of AF relapses without ventricular proarrhythmia. **SSR149744C** is a multiple-channel blocker drug like Amiodarone but with a better safety profile.

Candesartan, Statins and omega-3 fatty acids are examples of **"Upstream Therapies in AF"**, these drugs have intriguing beneficial effects in reduction in incidence of AF.

NEW ANTITHROMBOTICS and ANTICOAGULANTS.

Warfarin (Coumadin) has been the mainstay of therapy in prevention of Stroke and Systemic embolism episodes in Atrial Fibrillation. It is an effective drug, however regular monitoring of the Prothombin Time, INR values and variation in dosage requirement are the main drawbacks.

Direct Thrombin Inhibitors have the advantage of fixed oral dosing without coagulation

monitoring. Ximelagatran was the first direct thrombin inhibitor proved to be non-inferior to Warfarin in preventing systemic and cerebral embolism while having less bleeding complications. However elevated liver enzymes in 6% of patients have prevented its clinical use.

Dabigatran is the newer analogue that is currently in Phase 3 study worldwide, including India. The Heart Care Clinic is currently one of the top recruiters for the trial in the country.

Apixaban and Rivoroxaban are other analogues that will shortly be entering Phase 3 clinical studies.

PLENARY LECTURE - "Revisiting Clinical Examination in 21st Century" - Dr. Pravin Shah

Abstract not received

Hypertension, Lipids & Preventive Cardiology

Cardiogenic Stock - State of the Art 2007 - Dr. Jagdish Hiremath

Commonest cause of cardiogenic shock is acute myocardial infarction. The focus is shifting from hemodynamic management to multidisciplinary viewpoints encompassing basic molecular aspects.

Myocardiocytes have an obligate aerobic functioning. With acute thrombotic occlusion of the coronary artery, it has neither facultative capability, nor functional channels. It leads to 'pasteur effect' (myocardial adaptive resources against energy crisis) lysosomal autophagy, apoptosis and cell death.

Myocardial infarction research unit (MIRU) criteria for cardiogenic shock include:

1) Systolic BP of 90mm of Hg or less or acute drop by 30mm of Hg.
2) Hypo perfusion manifestation.
3) Hypotension due to drugs, hemorrhage, vasovegal excluded.

Treatment strategies include general measures like O_2, inotropes, heparins, aspirin, fluids, thrombolytics. Recently CD40 ligand is used as proinflammatory markers. Clinical implication of which is clinical effectiveness of platelet inhibition by GP IIb/IIIa inhibitor. Delayed reperfusion may act as a double-edged sword due to ROA (reactive oxygen species). Early revascularisation by PCI, CABG & liberal use of intra aortic balloon pump support is advocated.

Apart from revascularisation strategies, overall shock management is changing from importance of B1 receptors to alfa adrenergic receptors, tissue paracrine axis. Shock is considered generalized ATP deficiency hence uses of exogenous ATP compound ATP-$MgCl_2$ is attempted. Novel approaches also used are promotion of collateral channels by growth factors like VGEF, implantation of stem cells and new genomics paradigm associated with myocardial regeneration.

Controversies in Lipid-Lowering Therapy - Dr. Nakul Sinha

Hypertension 2007: Controversies and Treatment Challenges - Prof. S. K. Kaushik

Abstract not received

Treating the Metabolic Syndrome in Clinical Practice : Multidisciplinary Care - Dr. P. C. Manoria

Metabolic syndrome (MS) in characterized by intrabdominal obesity (waist >90 cm in male and >80 cm in females in South Asians) coupled with atherogenic dyslipidemia (triglycende >150 mg/ dl and HDl cholesterol <40 mg/dl in men and<50 mg/dl in women), hypertension(BP>130/85 mm kg) and raised fasting glucose (>100 mg/dl) or previously diagnosed type-2 diabetes. Besides this, it is also associated with impaired fibrinolysis, increased suceptiblity to thrombotic events and raised inflammatory markers. India has the largest number of type-2 diabetics in the world and so it can be extrapolated the country also has the largest number of patients with metabolic syndrome; the prevalence being 24.9% overall, with 18.4% in men and 30.9% in women.

Life style modification is an essential component of management and benefits all components of M.S. Prior to the era of Rimonabant, a panoply of drugs has to be used to target individual components of MS. Anti-obesity drugs such as subutramine and orlistat are used to reduce weight and central obesity and also control obesity related cormorbidities. Statins, ezetimibe, fibrates, niacin and omega-3 fatty acids are used in isolation or combinations to modulate atherogenic dyslipidemia. The trials of CETP inhibitor, torcetrapib has been stopped because of adverse effects. For insulin resistance, insulin senstizers like metformin and thiazolidinediones are utilized. Available evidence suggests that angiotensin converting enzyme (ACE) inhibitors and angiotenson receptor blockers (ARBs) may be more beneficial for treatment of hypertension in patients with MS compared to other class of drugs as they also prevent development of diabetes. Patients with MS also have elevations fibrinogen and other coagulation factors leading to prothombotic state and aspirin/clopidogrel is useful for targeting this altered state. The cannabinoid CB-1 blocker, Rimonabant has revolutionized the therapy of MS and with this multi impact drug, the cardio metabolic risk factors can be substantially modified favorably. It has opened the flood gates of pharmacological reduction of cardio-metabolic risk factors. The polypill for MS is also an emerging viable concept that needs to be evaluated in long term prospective trials.

ACC/AHA Guidelines 2006 - Management of Patients With Asymptomatic Valvular Heart Disease - Dr. Pravin Shah

Abstract not received

Mitral valve in heart failure and ischemic disease - Dr. Alain Berrebi

Abstract not received

3 C CON ORATION- Practical Insights into Primary PCI : What all physicians should know ? - Dr. Sameer Mehta

Practical Insights into Primary PCI Gained from the SINCERE Primary PCI Registry

Sameer Mehta, MD, Rosanna Briceno, MD, Martine Lifleur. Memorial Regional Hospital, Hollywood, Florida

Stenting of culprit lesions has been demonstrated to be clearly superior to thrombolytic therapy for treating acute myocardial infarction (AMI). Several large randomized trials have proven that early revascularization with stenting for AMI (Primary PCI) results in improved outcomes - better preservation of left ventricular function and decreased incidence of MI, revascularization, stroke and death. Early discharge from the hospital has also been conclusively demonstrated with Primary PCI. These trials have also emphasized on the need to lower the Door to Balloon time – a benchmark of 90 minutes is presently being recommended as per the ACC/AHA Guidelines for Primary PCI.

The SINCERE (Single INdividual Community Experience REgistry) Primary PCI experience provides pivotal lessons that are critical in performing Primary Stenting. It demonstrates the critical mandates of skills and speed in achieving very high success rates for Primary PCI. The following important Practical Insights are emphasized:

1. The importance of a team approach between ambulance services, emergency rooms and the cardiovascular laboratory to predictably achieve sustained door to balloon times of less than 90 minutes
2. Triple anti-platelet therapy
3. Use of bolus-only strategy of Direct Thrombin Inhibitor, Bivalirudin
4. Aggressive use of Aspiration Atherectomy via low-profile aspiration catheters to debulk thrombus prior to stenting
5. Intracoronary vasodilators, mainly Nitroprusside, for enhanced TIMI flow and Blush score
6. Universal application of Vascular Closure Devices
7. Early ambulation and hospital discharge

A compendium of illustrative cases is used to highlight the Practical Insights from SINCERE

Myocardial Ischemia , Infarction & Intervention

Guideline based medicine : Can we implement it in India ? - Dr. Prabhakaran

Guideline based medicine: Can we implement it in India?

Cardiovascular disease (CVD) is the leading cause of global mortality resulting in nearly 17.5

million deaths worldwide in 2005. Whereas the causes of this epidemic involve the same risk factors, the approaches to its control and prevention can differ in each geographic region because of cultural, social, medical and economic circumstances.

The results of recent clinical trials indicate that a clinical strategy, which incorporates careful risk stratification in conjunction with novel therapeutic agents and revascularization in appropriately selected patients, may improve both immediate and long-term outcome. Despite major therapeutic advances and proven benefits from the early management of CHD patients, significant challenges remain in improving clinical practice. Several European Surveys such as the Euroheart Survey, Grace Study and EuroAspire, have identified large treatment gap among CHD patients. In recognition of the need for effective implementation of guidelines to curb the growing worldwide mortality from CVD, the World Heart Federation (WHF) has set forth principles upon which every nation can develop a policy on CVD prevention. The recommended strategies involve treatment of high-risk patients using medical therapies whose benefits have been confirmed by large randomized clinical trials, and the parallel institution of behavioral modification for the general population focused on diet, physical activity and prevention of smoking.

Data on practice patterns in India is limited. A study by WHO, on prevention of recurrence of myocardial infarction and stroke in developing countries including India, reported underutilization of evidence based, cost effective, and appropriate medications. In a study from Kerala (carried out by us) we noted some barriers in implementation of guideline based care in the management of ACS. Lack of physician knowledge and awareness, familiarity, agreement with practice guidelines and uncertainty about relevance of treatment recommendations for diverse patients in actual clinical practice are often suggested as impediments to implementation of guideline based care. Later we conducted a quality improvement programme in management of acute coronary syndromes in Kerala. The quality improvement program (standing instructions, discharge summaries, review of evidence based practices) helped to guide efforts designed to promote evidence-based care and ultimately determined the effect of widespread implementation of practice guidelines on clinical outcomes. The methods used to implement this programme are readily available and would be expected to be able to be implemented in any similar hospital settings and community. Therefore quality improvement and monitoring of adherence to practice guidelines should be considered components of optimal clinical practice in the detection and management of ACS.

Use of Bivalirudin in ACS, AMI & PCI - Dr. Sameer Mehta

Use of Bivalirudin in ACS, AMI & PCI

Sameer Mehta, MD, Rosanna Briceno, MD, Martine Lifleur. Memorial Regional Hospital, Hollywood, Florida

Thrombin plays a major role in acute coronary artery occlusions during percutaneous coronary interventions. Unfractionated heparin has been traditionally used during invasive coronary procedures to reduce the risk of thrombotic occlusion. Bivalirudin, a direct antithrombin inhibitor, has several advantages over unfractionated heparin: it acts independently of antithrombin and

inhibits both free and clot-bound thrombin; it is not neutralized by circulating inhibitors; exhibits consistent dose-response characteristics, and does not cause thrombocytopenia.

Bivalirudin is a Direct Thrombin Inhibitor that has been extensively studied during the past decade. This clinical research conducted with a multitude of very large, double blind, placebo-controlled, randomized trials has lead to the extensive application of Bivalirudin in the treatment of Acute Coronary Syndromes (ACS), Percutaneous Coronary Interventions (PCI) and Acute Myocardial Infarction (AMI).

A review of these clinical trials is essential to understanding the applications of Bivalirudin in Cardiology.

Bivalirudin versus Unfractionated Heparin in PTCA Results of three randomized, clinical trials of bivalirudin in PTCA have been published.[22, 27, 28] An initial dose-escalation trial in 291 patients evaluated abrupt coronary artery closure after five different dosages of bivalirudin (a bolus dose of 0.15-0.55 mg/kg followed by a continuous infusion dosage of 0.6-2.2 mg/kg/hour for 4 hours).[27] Abrupt vessel closure was lowest in the patients receiving the two highest dosages of bivalirudin. This study was limited because no control group with unfractionated heparin was included and the number of patients evaluated was small. In addition, application of the findings to contemporary practice is difficult owing to increased application of intracoronary stents and GP IIb-IIIa receptor inhibitors, neither of which were included in the trial. Nonetheless, the results suggest efficacy of bivalirudin in prevention of abrupt closure after PTCA.

The results of two large bivalirudin comparative trials were published in 1995 in a single article.[28] These parallel clinical trials with identical inclusion and exclusion criteria compared bivalirudin with unfractionated heparin in patients undergoing PTCA. The original manufacturer, Biogen (Cambridge, MA), sponsored the trials with the intention of combining the results, for more than 4000 patients, in preparation for submission to the FDA for approval in PTCA. At 121 medical centers in North America, patients with severe, accelerating or rest angina within the last month who had coronary artery stenoses suitable for PTCA were randomly assigned to double-blind treatment with high-dose unfractionated heparin (2151 patients, 175 U/kg followed by an 18-24-hr infusion of 15 U/kg/hr) or bivalirudin (2161 patients, 1-mg/kg bolus followed by a 4-hr infusion of 2.5 mg/kg/hr followed by a 14-20-hour infusion of 0.2 mg/kg/hr). Both treatments were targeted at achieving an ACT of 350 seconds. The median duration of treatment was 16 hours. All patients were treated with aspirin 300-325 mg before PTCA and then daily thereafter. The primary end point of the study was a composite of in-hospital death, myocardial infarction, abrupt vessel closure or rapid clinical deterioration of cardiac origin requiring bypass surgery, intraaortic balloon counterpulsation, or repeat PTCA.

In the original publication, the unadjudicated results of 4098 of the 4312 patients were reported.[28] These preliminary results indicated no statistically significant difference between bivalirudin and unfractionated heparin with respect to the intent-to-treat primary end point (11.8% vs 12.9%, respectively, $p=0.26$). However, the frequency of the secondary end point, death or myocardial infarction (2.0% vs 5.1%, $p=0.04$), as well as major hemorrhage, defined as at least a 3-g/dl decrease in hemoglobin, need for transfusion, intracranial hemorrhage, or retroperitoneal bleed (3.8% vs 9.8%, $p<0.001$), were significantly reduced. Based on the results of this initial analysis, Biogen abandoned drug development. Subsequently, the development and marketing rights were purchased by The Medicines Company, which reanalyzed the data from the trial, now

known as the Bivalirudin Angioplasty Trial (BAT), using contemporary definitions of adjudicated end points such as revascularizations and enzymatic or clinical evidence of myocardial infarction with complete follow-up. These trials formed the basis for the drug's approval by the FDA for use in PTCA in 2001. The results were audited and reanalyzed by statisticians from the FDA before approval.

Final data from the BAT trial were published recently and indicated superiority of bivalirudin compared with unfractionated heparin, with a 22% reduction in death, myocardial infarction, or revascularization at 7 days (6.2% vs 7.9%, p=0.039).[10, 22] These differences were sustained at 3 months (15.7% vs 18.5%, p=0.012) but were not statistically significant at 6 months (23% vs 24.7%, p=0.15).[10] There was a 62% reduction in major bleeding (3.5% vs 9.3%, p<0.001), as well as lower transfusion rates (p<0.001).[10, 22] A prespecified subgroup analysis of 741 patients undergoing PTCA within 2 weeks of myocardial infarction also demonstrated lower rates of procedural failure, defined as acute vessel closure, death, myocardial infarction, or revascularization during hospitalization (5.1% vs 10.8%, p=0.004); death or myocardial infarction (0.5% vs 4.3%, p=0.001); death (0% vs 0.8%, p=0.047); myocardial infarction (0.5% vs 3.8%, p=0.001); and revascularization (3.0% vs 6.5%, p=0.028).[29] Some believe that because the definitions of the study end points changed after the completion of the trial, these results should be viewed only as hypothetical.[30]

Bivalirudin versus GP IIb-IIIa Plus Unfractionated Heparin in PCI

Application of the results of BAT to contemporary practice is difficult because intracoronary stent placement now accounts for greater than 70% of PCI procedures.[5] Typical therapy in such procedures consists of unfractionated heparin or LMWH in combination with a GP IIb-IIIa receptor inhibitor, rather than unfractionated heparin alone. In addition, current practice patterns advocate lower dosages of unfractionated heparin and discontinuance of unfractionated heparin immediately after the procedure.[5] However, bivalirudin is being studied in intracoronary stent placement procedures as an alternative to unfractionated heparin and as a potential replacement for GP IIb-IIIa receptor inhibitors.

Preliminary information is available from two clinical trials, as yet unpublished, that have compared bivalirudin with abciximab (standard dosage of 0.25-mg/kg intravenous bolus followed by a 12-hr 0.125-µg/kg/min, maximum 10-µg/min, infusion) plus low-dose unfractionated heparin (70-U/kg bolus) in patients undergoing PCI. In the Comparison of Abciximab Complications with Hirulog and Back-up Abciximab Events Trial (CACHET) parts B-C, 208 low-risk patients undergoing elective PCI were randomly assigned to receive either low-dose unfractionated heparin (70-U/kg bolus) plus abciximab (0.25 mg/kg followed by a 12-hr infusion of 0.125 µg/kg/min, maximum 10 µg/min) or two different dosages of bivalirudin (either a 0.5-mg/kg bolus followed by an infusion of 1.75 mg/kg/hr in the first 85 bivalirudin-treated patients or a bolus of 0.75 mg/kg followed by an infusion of 1.75 mg/kg/hr in the next 59 bivalirudin-treated patients) continued until the end of the procedure.[28, 31-33] All patients received aspirin and clopidogrel. Administration of provisional abciximab was permitted in the bivalirudin-treated patients at the clinician's discretion if the PCI results were not adequate as defined by diminished flow, greater than 40% residual stenosis, or dissection. Eighty-eight percent of patients received intracoronary stents, and abciximab was given in 24% of patients randomly assigned to receive bivalirudin. Results indicate that the combined bivalirudin groups experienced a lower combined frequency of death, myocardial infarction, revascularization, or major hemorrhage occurring at 7 days when compared with the group receiv-

ing unfractionated heparin plus abciximab (3.1% vs 14.1%, p=0.013).[33] Death, myocardial infarction, or revascularization was lower in the bivalirudin-treated patients (2.8% vs 7.8%, p=0.137), and major hemorrhagic events were reduced (1.4% vs 6.3%, p=0.074).
A second clinical trial, Randomized Evaluation in PCI Linking Angiomax to Reduced Clinical Events (REPLACE) part 1, compared bivalirudin (0.75-mg/kg bolus followed by a 1.75-mg/kg/hr infusion continued to the end of the procedure) with unfractionated heparin (60-70 U/kg) in 1056 patients undergoing PCI.[34] Eighty-five percent of patients underwent intracoronary stent placement, and 72% received concomitant GP IIb-IIIa receptor inhibitor therapy with either abciximab, tirofiban, or eptifibatide in combination with aspirin and clopidogrel. Preliminary results of REPLACE part 1 indicate a 19% reduction (7.1% vs 8.8%, not statistically significant) in the primary composite end point of death, myocardial infarction, revascularization, or clinically significant bleeding occurring at 48 hours or at hospital discharge, whichever occurred first. The frequency of the triple composite end point of death, myocardial infarction, or revascularization also was reduced by 19% (5.6% vs 6.9%, not statistically significant). Major bleeding, defined as intracranial, retroperitoneal, intraocular, or clinically overt bleeding associated with a decrease in hemoglobin of 3 g/dl or more from baseline, was reduced by 22% (2.1% vs 2.7%, not statistically significant). Major bleeding rates were identical in the subgroups of patients receiving bivalirudin or heparin in combination with a GP IIb-IIIa receptor inhibitor (29%). However, the combination of bivalirudin or unfractionated heparin with GP IIb-IIIa receptor inhibitor therapy showed similar or slightly lower major bleeding rates with unfractionated heparin than with bivalirudin, despite significantly higher ACTs with abciximab plus bivalirudin compared with unfractionated heparin (371 vs 304 sec, p<0.001) at the start of the procedure.[34] This was a pilot study and was not powered to detect statistically significant differences in outcomes between the groups.[34]

In the ongoing REPLACE trial part 2, 6000 patients receiving aspirin and clopidogrel will be randomly assigned to receive either unfractionated heparin with the routine GP IIb-IIIa receptor inhibitor administered at each study site, or bivalirudin (0.75-mg/kg bolus followed by 1.75-mg/kg/hr infusion to the end of the procedure) with provisional abciximab.[32] The primary end point is death, myocardial infarction, revascularization, or major hemorrhage occurring at 30 days. A 1-year pharmacoeconomic analysis also is planned. The expected date of study completion is December 2002.

The decision to use major bleeding as a part of the primary composite end point in REPLACE parts 1 and 2 is unique among PCI trials.[32, 34] Preliminary data suggest that bivalirudin is safer than both unfractionated heparin alone and unfractionated heparin with abciximab, without a loss of efficacy. However, major bleeding rates observed in the GP IIb-IIIa receptor inhibitor arms of both the CACHET and REPLACE trials are higher than those observed in other contemporary clinical trials such as Enhanced Suppression of the Platelet IIb-IIIa Receptor with Integrilin Therapy (ESPRIT, 1%) and the Do Tirofiban and ReoPro Give Similar Efficacy Trial (TARGET, 0.9% and 0.7%) despite similar definitions of bleeding.[35, 36] Furthermore, the use of bleeding end points may introduce bias because they often include transfusion rates.

Two dosages of bivalirudin were tested: 1.0-mg/kg bolus followed by 2.5-mg/kg/hour infusion (21 patients) or a 0.75-mg/kg bolus followed by a 1.75-mg/kg/hour infusion (11 patients) for 4 hours. Unfractionated heparin (11 patients) was dosed to an ACT of greater than 200 seconds. Eptifibatide was dosed as a double bolus of 180 µg/kg separated by 10 minutes and followed by an infusion of 2 µg/kg/minute begun after the first bolus (ESPRIT dosing) and continued for 18-24

hours.[35, 37] The small sample prohibits making any definitive conclusions.[37]

Heparin-Induced Thrombocytopenia

Because bivalirudin has no structural similarity to unfractionated heparin, it may be administered safely to patients with HIT or heparin-induced thrombotic thrombocytopenia syndrome (HITTS) or a history of HIT or HITTS. The efficacy and safety of bivalirudin have been reported in 50 patients with HIT.[38] The first 39 patients received bivalirudin for a variety of indications, but most received treatment in the setting of PCI (17 patients). Of these 39 patients, 17 had acute HIT and 22 had a history of HIT. Mortality in these patients was 10%, and all four deaths were related to complications of HIT. Minor bleeding complications occurred in nine patients.[38] Preliminary information on 11 of a planned 50 patients with acute HIT or HITTS or a history of HIT or HITTS from an ongoing clinical trial (Bivalirudin to Assist in the Performance of Percutaneous Coronary Intervention in Patients with Heparin-Induced Thrombocytopenia [ATBAT] trial) also has been reported. All patients had Thrombolysis in Myocardial Infarction (TIMI) grade 3 flow during PCI. After PCI, no clinical events or major bleeding occurred, and two patients experienced minor bleeding.[38]

Other Investigations With Bivalirudin

A small, randomized, double-blind pilot trial of bivalirudin versus unfractionated heparin in patients with ST segment elevation myocardial infarction who presented within 12 hours of symptom onset was performed in 45 patients treated with intravenous streptokinase and aspirin.[39] Bivalirudin (0.5 mg/kg/hr for 12 hrs reduced to 0.1 mg/kg/hr) or unfractionated heparin (1000 U/hr titrated to maintain an aPTT of 2-2.5 times control) was administered just before or simultaneously with streptokinase 1,500,000 U administered over 60 minutes. Anticoagulant therapy was continued until the time of angiography, which occurred, on average, at day 4. The TIMI grade 3 flow, indicating complete reperfusion, was present in 77% of patients receiving bivalirudin compared with 40% of patients treated with unfractionated heparin ($p<0.02$).[39] Serious bleeding complications were similar between the two groups (unfractionated heparin 27%, bivalirudin 13%, not statistically significant).

In a second study in streptokinase-treated patients with ST segment elevation myocardial infarction performed by the same group of investigators, 70 patients were randomly assigned to two different dosages of bivalirudin (either 0.5 or 0.1 mg/kg/hr) versus unfractionated heparin (dosed as described above). At 90 minutes, the rate of TIMI grade 3 flow was highest in patients treated with the lower bivalirudin dosage ($p=0.04$).[40] The rate of blood transfusions was lower in the bivalirudin-treated patients (5%) compared with that in patients treated with unfractionated heparin ($p<0.02$).[40]

Another dosing strategy for bivalirudin was evaluated in a larger pilot trial of 412 patients with ST segment elevation myocardial infarction who were undergoing thrombolysis with streptokinase, the Hirulog versus Heparin in Patients Receiving Streptokinase and Aspirin for Acute Myocardial Infarction 1 (HERO-1) trial. The higher dosage of bivalirudin in this trial, a 0.25-mg/kg bolus followed by 0.5 mg/kg/hour for 12 hours followed by 0.25 mg/kg/hour for up to 60 hours, was associated with a higher frequency of TIMI grade 3 flow at 90-120 minutes as compared with unfractionated heparin (48% vs 35%, $p=0.03$). Major bleeding was significantly lower with bivalirudin than with unfractionated heparin (27% vs 19%, $p<0.01$).[41] These TIMI grade 3 flow rates are not as high as those of more fibrin-specific thrombolytics in combination with

unfractionated heparin, as reported in the Global Use of Strategies to Open Occluded Coronary Arteries in Acute Coronary Syndromes (GUSTO)-1 trial with alteplase (48-60%) and the Reteplase (r-PA) versus Alteplase Patency Investigation during Myocardial Infarction (RAPID)-2 with reteplase (59.9%).[42, 43]

This same dosage of bivalirudin was being administered in the Hirulog/Early Reperfusion Occlusion-2 (HERO-2) study.[44] This study evaluated 17,073 patients with ST segment elevation myocardial infarction from 46 countries. Patients receiving streptokinase were randomly assigned to receive either unfractionated heparin (5000-U bolus followed by a weight-based infusion titrated to an aPTT of 50-70 sec for 48 hrs) or bivalirudin (0.25-mg/kg bolus followed by 0.5 mg/kg/hr for 12 hrs and 0.25 mg/kg for 36 hrs) administered just before or simultaneously with streptokinase.[28] Both treatment groups received aspirin. The study sample was selected to have an 80% power to detect a 15% difference in 30-day mortality. There was no significant difference in the primary end point, 30-day mortality. Mortality as an independent measure was not significantly reduced (10.5% for bivalirudin vs 10.9% for heparin, p=0.46); however, the combined 30-day rate of death or reinfarction was 12.9% for bivalirudin and 14.2% for unfractionated heparin (p=0.023). The results showed a significant reduction in a secondary end point, nonfatal reinfarction, with bivalirudin at 96 hours compared with unfractionated heparin (2.3% vs 1.6%, p=0.001) and was sustained at 30 days (3.5% vs 4.5%, p<0.001).[45] The frequency of severe bleeding between the two treatment groups was not different. However, the rates of moderate and mild bleeding were increased (bivalirudin 0.7%, unfractionated heparin 0.5%, p=0.07). This trial demonstrates that bivalirudin is at least as effective as unfractionated heparin and not inferior when combined with streptokinase for the treatment of ST segment elevation myocardial infarction. Application of the results of this clinical trial to practices in the United States will be limited, however, since more fibrin-specific thrombolytic agents are preferred and no data on the combination of bivalirudin with thrombolytics other than streptokinase are available.[46]

The TIMI-8 trial was terminated prematurely after 133 of a planned 5320 patients with non-ST segment elevation acute coronary syndromes were enrolled because the original sponsor, Biogen, decided to halt drug development. In TIMI-8, unfractionated heparin, administered as a bolus of 70 U/kg followed by an infusion of 15 U/kg/hour titrated to an aPTT of 55-85 seconds, was compared with bivalirudin administered as a bolus of 0.1 mg/kg followed by an infusion of 0.25 mg/kg/hour for a minimum of 72 hours.[47] The primary efficacy end point, a composite of death or myocardial infarction through hospital discharge or 14 days, whichever came first, was 9.2% in the unfractionated heparin group versus 2.9% in the bivalirudin group (p=0.16). Major bleeding occurred in none of the bivalirudin-treated patients versus three patients (4.6%) in the unfractionated heparin group (p=0.11). Although the results of this study appear promising, it is unclear whether the current study sponsor, The Medicines Company, plans to pursue additional bivalirudin clinical trials in patients with non-ST segment elevation acute coronary syndromes.[47]

Un-Fractionated Heparin vs. Bivalirudin During PCI (ISAR-REACT 3)

The purpose of this study is to determine whether bivalirudin given during PCI is associated with better outcomes compared to un-fractionated heparin. Previous studies have shown that use of bivalirudin among patients undergoing percutaneous coronary interventions is associated with better outcomes (death, myocardial infarction, urgent repeat revascularization or in-hospital major bleeding) as compared with unfractionated heparin and adjunctive use of glycoprotein IIb/IIIa platelet receptor inhibitors. However, previous studies have included patients treated with plain

balloon angioplasty or stenting after inadequate pre-treatment with thienopyridines (ticlopidine or clopidogrel). Recent guidelines recommend that all patients undergoing percutaneous coronary interventions must receive a loading dose of 300 -600 mg of clopidogrel. A 600 mg loading dose of clopidogrel eliminates the need for glycoprotein IIb/IIIa platelet receptor inhibitors in adjunct to heparin. According to existing evidence antithrombotic regimens based on either bivalirudin or pre-treatment with 600 mg of clopidogrel in addition to UFH intraprocedurally, are effective strategies to reduce ischemic and hemorrhagic complications in patients with coronary artery disease undergoing PCI. At present, it is not known whether bivalirudin is superior to UHF in patients who have been optimally pre-treated with a loading dose of clopidogrel.

Bivalirudin in Patients with Acute Myocardial Infarction (AMI) Undergoing Primary PCI

In this ongoing Phase 3 Trial in Germany, the purpose of this study is to demonstrate the benefit of bivalirudin in combination with clopidogrel with provisional G IIb/IIIa inhibitor use, in reducing the bleeding complications associated with early invasive management of patients presenting with an ST Elevation Myocardial Infarction (STEMI) and undergoing primary PCI, while providing similar rates of ischemic events when compared to published results of relevant trials.

Bivalirudin for Early Removal of Angioplasty Sheaths (AFRICA Trial)

In the Angiomax Facilitates Rapid and Complete Ambulation (AFRICA) Trial, Sameer Mehta, et al, studied 200 consecutive patients and demonstrated the feasibility of early sheath removal after the use of Bivalirudin during PCI

Acute Myocardial Infarction: Current State of the Art - The first 24 hours - Dr. Shmuel Banai

Acute Myocardial Infarction: Current State of the Art – The first 24 hours
Shmuel Banai MD

Significant recent advances have been made in strategies for managing ST-segment elevation myocardial infarction (STEMI).

The prehospital care of patients with STEMI is crucial for survival. Most deaths associated with STEMI occur within the first hour of its onset and are usually due to ventricular fibrillation. Immediate implementation of definitive resuscitative efforts and rapid transport to a hospital are extremely important.

Major components of the delay from the onset of symptoms consistent with acute myocardial infarction to reperfusion include the following:

Time for the patient to recognize the seriousness of the problem and seek medical attention, prehospital evaluation, treatment, and transportation, time for diagnostic measures and initiation of treatment in the hospital (e.g., "door-to-needle" time for patients receiving a thrombolytic agent and "door-to-balloon" time for patients undergoing a catheter-based reperfusion strategy), time from initiation of treatment to restoration of flow.

Reperfusion of the infarct-related artery is by far the most important therapy administered to patients suffering from acute MI.

Fibrinolysis is the most common method of reperfusion used worldwide for patients with ST-elevation MI. Primary PCI is the preferred reperfusion therapy for STEMI. Compared with fibrinolytic treatment primary PCI reduces short- and long-term mortality and reinfarction rate by 30% to 40%, shortens hospital stay and reduces later need for hospital care. Therefore, if available, primary PCI today is the treatment of choice for STEMI.

The Reperfusion therapy should be combined with antithrombotic agents, aspirin and unfractionated heparin, with a movement toward lower doses of long-term aspirin and lower doses of unfractionated heparin (UFH). Studies have also been completed on the use of enoxaparin and fondaparinux as very effective alternatives to heparin. Clinical trial data support the early use of glycoprotein IIb/IIIa inhibitors (along with aspirin, UFH, and clopidogrel) in patients undergoing primary percutaneous coronary intervention for acute STEMI. Clopidogrel has also been shown to improve angiographic and clinical outcomes in patients with STEMI who are undergoing thrombolysis or being treated medically. The new ACC/AHA recommendations support clopidogrel pretreatment and long-term therapy.

Effect of door-to-Balloon or door to thrombolytic time on Mortality in Patients with ST-Segment Elevation Myocardial Infarction - Dr. Manish Parikh

Abstract not received

Biomarkers in Ischemic Heart Disease - Dr. Manish Parikh

Abstract not received

Benefit of Early Invasive Therapy in Acute Coronary Syndromes - A Meta-Analysis of Contemporary Randomized Clinical Trials - Dr.Upendra Kaul

Benefit of Early Invasive Therapy in Acute Coronary Syndromes with NSTEMI

Upendra Kaul *,MD,DM,FCSI,FACC,FAMS,FSCAI*
Foris Hospital, New Delhi and NOIDA

Acute coronary syndrome with NSTEMI is a common presentation of ischemic heart disease. These patients often are seen in the emergency rooms with chest pain at rest. The recognition of this syndrome and risk stratification is very important to manage this potentially serious problem.

Patients presenting with classical recurrent rest angina, Associated ECG changes of ST segment deviation, elevated serum bio markers (Troponins , CPK MB etc), hemodynamic(Left ventricular failure, shock) or electrical (Ventricular tachycardia non sustained) instability are all high risk patients needing immediate admission. The 30 day adverse events (death, MI or heart failure) can occur in up to 40% of these patients.

These patients need immediate hospitalization and aggressive medical management, which consists of aspirin, clopidogrel , administration of low molecular weight heparin , nitroglycerine, beta blockers or heart rate lowering calcium blockers along with statins. There is also a role of starting these patients on small molecule Gp iib/iiia blockers (Eptifibatide or tirofiban) as upstream treatment before taking them to the cath lab for angiography.

There has been an ongoing debate regarding the utility of early invasive therapy for patients with intermediate and high risk ACS . The issues for discussion are whether all patients need to undergo early invasive strategy or selected patients with ongoing angina / stress studies demonstrable ischemia need it after an initial aggressive medical treatment. Earlier studies done in the era when PCI was confined to balloon angioplasty alone with limited medical treatment (no clopidogrel and Gp iib/iiia blockers) showed that early conservative treatment was marginally better than the early invasive treatment. [VANQUISH Study]. However studies carried out in the last 5 years after the widespread use of stents instead of plain balloon angioplasty
Showed improved results with the early invasive therapy.

A number of randomized studies done in the recent times have addressed this issue. A recent meta analysis of studies using the current methods of PCI and aggressive medical treatment [FRISC II, VINO, TIMI 18, RITA3 TRUCS, ICTUS, ISAR COOL] has concluded that the early invasive therapy results in a reduction of the relative risk of non fatal MI by 27%,and 31% reduction of recurrent unstable angina resulting in hospitalization. The composite end point of death, MI and need for re hospitalization was reduced by 25%. (**Bavry, A. A. et al. J Am Coll Cardiol 2006;48:1319-1325).**

Conclusion:

Early invasive therapy along with aggressive medical treatment is the preferred mode of treatment with ACS with NSTEMI in intermediate an high risk psatients.

Enoxaparin Vs other LMWH Vs Unfractionaled heparin ? Where are we in 2007 ? - Dr. Balram Bhargava

Abstract not received

Patients with Left Main Disease: Will the Interventionalists and Surgeons Ever Get on the Same Page? - Dr. Raj Makkar

Abstract not received

Medical Malpractice in Cardiovascular Medicine - Dr. Sameer Mehta

Abstract not received

4 Parallel Sessions

Break Away Session | 1

Advanced Echocardiography

Sunday, 28 January, 2007

Venue : Banquet Hall, Rajpath Club, Ahmedabad

07:00 am Registration & Breakfast

Chair : Dr. Hemang Baxi, Dr. Anish Chandarana

Moderator : Dr. S.K. Parashar, Dr. Alain Berrebi

07:30 am Early bird case review session

08:30 am Mitral Regurgitation : Echocardiography assessment - *Dr. Alain Berrebi*

08:50 am Aortic Regurgitation : Echocardiography assessment - *Dr. Urmil Shah*

09:10 am Quantitative Assessment of Valvular Regurgitation case based format - *Dr. S. K. Parashar*

09:30 am Clinical Decision making in Valvular Heart Diseases - *Dr. Alain Berrebi*

09:45 am (a)Assessment of Prosthetic Valves and (b) Assessment of Infective Endocarditis - *Dr. S. K. Parashar*

10:25 am Echo in coronary artery disease, Myocardial infarction including its complications - *Dr. Anish Chandarana*

10:55 am Refreshment Break

11:10 am Echocardiographic assessment of Dilated Cardiomyopathy : How will I select patient for CRT ? - *Dr. Ajay Naik*

11:25 am Echocardiography in adult congenital heart disease - *Dr. S. Radhakrishnan*

11:45 am Rapid fire Cases - *Dr. Pravin Shah, - Dr. Alain Berrebi*

12:30 pm Role of Echocardiography in the evaluation of rheumatic MR - *Dr. Alain Berrebi*

12:50 pm Hypertrophic cardiomyopathy: 2-D & Doppler assessment of systolic & diastolic function - *Dr. Pankaj Manoria*

01:05 pm Cardiac tamponade, Constrictive Pericarditis & Restrictive Cardiomyopathy : Echocardiography assessment by 2-D & Doppler - *Dr. Pravin Shah*

01:35 pm Echocardiography in aortic diseases - *Dr. Satya Gupta*

01:50 pm Factfile : Guide to applying new technologies in Echocardiography - *Dr. Pravin Shah*

02:20 pm Lunch

03:00 pm Demonstration

03:30 pm Hands On Breakout Session at The Heart Care Clinic (3.30 pm to 6.00 pm)

Advanced Echocardiography

Echocardiography is one of the most frequently used techniques for diagnosing cardiovascular diseases. It is now considered an *extension of the physical examination*. Echocardiography has come a long way since its beginnings in the mid-1950s. Although there are many new, highly sophisticated imaging technologies being developed, there is every reason to believe that the clinical utility and popularity of echocardiography will continue to grow. This diagnostic tool is amazingly versatile. It is still very cost-effective compared with competing technologies and has many new possibilities as to how this examination can be improved and provide more and better information.

Proper technical and cognitive skills are required for the optimal application of echocardiography and the interpretation of its results. It is an operator-dependent technique, more so than other cardiovascular techniques. Echocardiography should be used as a *"definitive" rather than a "screening" diagnostic tool*. After a significant lesion that requires surgical treatment is identified by echocardiography, the patient should be able to receive that treatment without having to undergo other confirmatory diagnostic tests. To do so requires not only the expertise of an echocardiographer but also an understanding of the capabilities and limitations of echocardiography by other physicians involved in the care of the patient, including primary care physicians and surgeons. For e.g., before advent of Doppler echocardiography, all patients with severe aortic stenosis underwent homodynamic cardiac catheterization before aortic valve replacement. However presently only 20% of such patients require invasive hemodynamic studies.Rarely is cardiac catheterization required for the diagnosis of congenital heart disease.

To have clinical impact, a new diagnostic modality should replace an existing procedure, not be an additional procedure, and it should do so without increasing the cost or risk of the examination and without sacrificing the accuracy of the results. Echocardiography meets these criteria, and it has had considerable clinical impact on the diagnosis and management of various cardiovascular diseases. The future of echocardiography should be as productive and exciting as have been the previous five decades.

In 3C con 2007 we have two sessions – basic and advanced echocardiography. Some of sessions will be taken by stalwarts in echocardiography which will be helpful to physicians in their cardiology practice. It will be also useful to cardiologists as it will update the recent advances and future in echocardiography.

We are sure the echocardiography sessions would be of immense help to physicians, cardiologist and paediatricians interested in this simple, cost effective and very useful noninvasive imaging for cardiovascular disease.

4 Parallel Sessions

Break Away Session | 2

National Conference on Pediatric Cardiology

Sunday, 28 January, 2007,

Venue : R-World Multiplex

07:30 am Registration & Breakfast

Chair :Dr. Milan Chag, Dr. Srinivas Mallya

Moderator : Dr. S. Radhakrishnan, Dr. S. S. Kothari, Dr. R. Krishnakumar, Dr. S. Shrivastava

08.00 am Simplified Classification of Congenital Heart Disease and prevalence of common CHD - *Dr. S.S. Kothari*

08.20 am When to suspect and How to Detect a child with CHD - *Dr. Milan Chag*

09.00 am Persistent Pulmonary Hypertension of Newborn: Etiopathology, Diagnosis and Management - *Dr. R Krishnakumar*

09.20 am Echo evaluation of common Congenital Heart Diseases - *Dr. S. Radhakrishnan*

09.50 am Foetal echocardiography: When to do and how to do? - *Dr. R Krishnakumar*

10:10 am Refreshment Break

10.40 am A neonate presents with cyanosis - *Dr. S. Shrivastava*

11.00 am A neonate presents with respiratory distress - *Dr. R Krishnakumar*

11.20 am Infective Endocarditis in children……: Diagnostic difficulties and basic principles of management. - *Dr. S. S. Kothari*

11.40 am Acute Rheumatic Fever : Current Concepts - *Dr. S. Shrivastava*

12.00 pm 2 year old child has murmur on routine examination... - *Dr. S.S. Kothari*

12.20 pm Timing of surgery and long term outcome - *Dr. Srinivas Mallya*

01.00 pm Non-surgical treatment of CHD: Present & Future - *Dr. S. Radhakrishnan*

01.30 pm Arrhythmias in children….. What Pediatricians should know - *Dr. Ajay Naik*

01.50 pm Q & A session - *All Faculty*

02.10 pm Lunch & Farewell

When To Suspect and How To Detect Patients with Congenital Heart Disease ? - Dr. Milan Chag

Introduction:

- Every year, 200,000 children are born with CHD in our country;
- Of these, only 5000 get treatment because of lack of awareness, under diagnosis or late diagnosis
- CHD accounts for 20% of Infant Mortality Rate:
  - in half of these cases parents never even get to know the problem

Prevalence of CHD:

1 % of all live births; 33% of them need intervention or die by one year age.

Natural history of CHD:

- 25% of them die in neonatal period
- 33% of them die by 1 year
- 75% of them die by 5 years
- Majority of the surviving patients may become inoperable

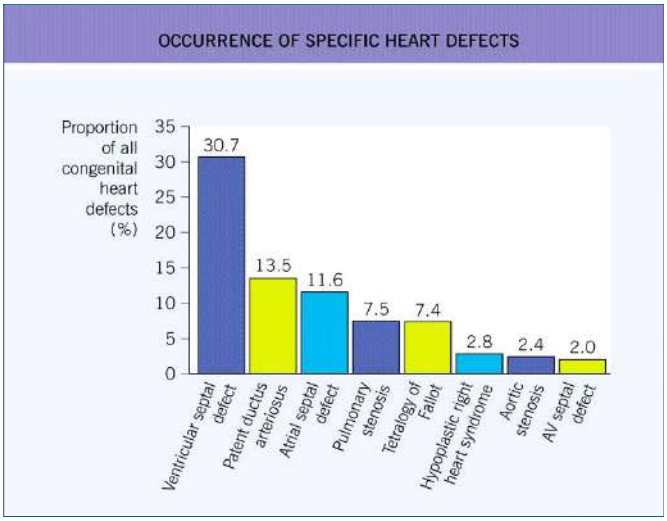
Therefore, early diagnosis is mandatory for safe management.

Few Common *(but potentially LETHAL)* Myths …

- " Hole in heart usually closes spontaneously, one may wait till 5 yrs age"
- " Open heart surgery is not possible till pt's weight is 10 kg"
- "Bluish discoloration is due to cold (peripheral cyanosis), nothing to worry!"
- "Murmur in children are common: they are functional and disappears as child grows"

Without confirmed diagnosis, such assumptions lead to inoperability or death.

Prevalence of CHD at time of birth:



Pre-natal History: Certain maternal conditions can cause CHD in newborn.

- Diabetes Mellitus -LVOTO, TGA, Cardiomyopathy
- SLE -Congenital CHB
- Infection:-Rubella -PDA, Peripheral PS, Myocaditis
- Drugs:-Alcohol, -Phenytoin -VSD, PS, AS, PDA, Coarct
- Consanguity -ASD(o.s.)
- Folate deficiency -TOF, Truncus

Simplified Classification of CHD:

- STENOTIC Lesions:
- PS, AS, Coarctation
- SHUNT Lesions:
- *Acyanotic (LàR shunt):*
- ASD, VSD, PDA, AP-window
- *Cyanotic (RàL shunt):*
- TOF
- Complex CHD: TGA, TAPVC, Tricuspid Atresia, Truncus, Single Ventricle
- Eisenmenger (Reversal of shunt)

Neonatal Murmurs:

They are not heard in > 75 % of newborns with CHD, as found in …

- Shunt lesions (ASD,VSD,PDA)
- D-TGA
- Coarctation
- TAPVC
- Pulmonary Atresia
- HLHS
- Cardiomyopathies

Clues to Cardiac Disease in Neonates:

- Cyanosis
- Tachypnea
- Tachycardia
- Diaphoresis
- Gallop
- Abnormal splitting of S2
- Abnormal precordial activity
- Hepatomegaly
- Abnormal femoral pulses, cool extremities

CHD: Modes of Presentation:

1. CHF
2. Hypoxemia:
3. Impaired Growth
4. Pulmonary Hypertension
5. Low cardiac output

Modes of Presentation :

(1) CHF:

- Usually occurs within first 6 months
- Onset after 1 year age is rare unless complicated by :
- Endocarditis
- Anemia
- Infection
- *As a rule, the earlier the onset of CHF, the more likely the need for surgery*
- Features of Heart Failure in Infants
- Poor feeding, Failure to thrive
- Excessive perspiration
- Respiratory distress: Tachypnea (> 40-60 / min)
- Tachycardia (160-180 bpm)
- Pulmonary wheeze or rales
- Color : pale or faintly cyanotic
- Gallop
- Cardiomegaly, PVH on X-ray
- Hepatomegaly
- Diminished urine output
- CHF on Day 1 of life :
- Regurgitant lesions:TR, MR, AR, PR
- Cerebral or Hepatic AV-fistula
- Myocarditis
- Congenital CHB
- SVT
- CHF in remainder of first week (1-7 days) is due to obstructive lesion of systemic circulation:
- Coarctation of aorta
- Aortic stenosis
- Hypoplastic left heart syndrome (HLHS)

CHF in 3rd week and beyond is due to increased pulmonary blood flow or LVOT obstruction

- VSD, AV canal defect
- PDA, AP window
- Coarctation
- TGA
- TAPVC

(2) Hypoxemia: presents with Cyanosis, Clubbing, Polycythemia, Squatting, and Hypoxic spell

- Complications:
  - Cerebral thrombosis (usually < 2 yr age)
  - Paradoxic embolus
  - Brain abcess (Rare < 18 mon, Incidence: 2% Mortality : 30-40%)
  - Hemoptysis

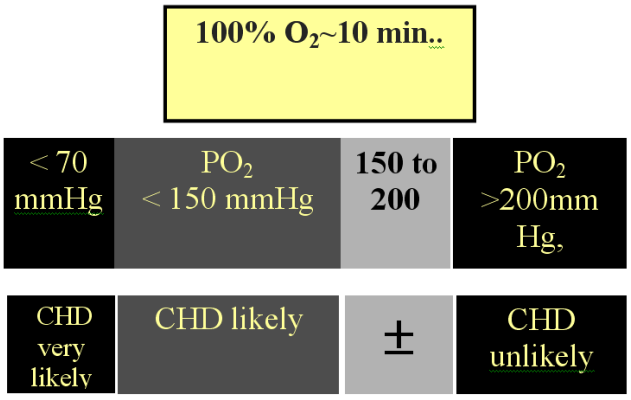
Causes of Cyanosis in Newborn

- CHD: R à L shunt
- Respiratory diseases
- Central hypoventilation
- PPHN (Persistent Fetal Circulation)
- Methaemoglobinemia (Low SaO2, Normal PaO2)

Cyanosis is clinically detected when-

- SaO2 < 85%
- PaO2 : 45-55 mm Hg
- Reduced Hb > 3 gm%
- *Physiological : First 20 min of birth*

Hyperoxia Test: done to differentiate cardiac from non-cardiac causes of hypoxia.
à Give 100% oxygen for 10 minutes and check pO2.



Non-cardiac causes of Cyanosis in Newborn:

Central and Hematological causes:

- Sedative to mother ® Hypoventilation
- ICH due to birth trauma
- Meningitis, Encephalitis
- Methemoglobinemia (SaO2:low, PaO2:N)
- Hypoglycemia (BSL < 30 mg%)
- High Hb level
- Septicemia

Pulmonary causes:

Intra-pulmonary causes:

- Hyaline Membrane Disease (HMD)
- Atelectasis
- Pneumonitis

Airway obstruction:

- Choanal atresia
- Vascular ring
- Tracheomalacia, vocal cord peresis
- Diaphragmatic hernia
- Pneumothorax

(3) Impaired Growth:

In patients with large left to right shunt, weight retardation is more than height retardation. In patients with cyanotic CHD, height and weight are equally affected. In both groups, mental development is normal. In contrast, patients with Genetic syndromes like Noonan's, Turner's, Down's and Rubella,
Height and weight are equally affected and they may have Mental retardation.

(4) Pulmonary Hypertension:

- At birth: ~ Identical pressures on both sides
- Within 2-3 weeks, PVR falls to adult level
- *With VSD or PDA, PVR fall is delayed:*
  - Takes 3-6 weeks in Pre-term
  - Takes 6-10 weeks in Full-term

Types of Pulmonary Hypertension:

- Hyperkinetic PAH
  - due to large Là R shunt
  - Associated with Cardiomegaly, S3, Flow MDM
- Obstructive PAH
  - due to Pulmonary vascular obstructive disease
  - No CM, No S3/MDM, loud P2, PEC
- *Normal PVR < 3 Wood units*
- *Normal PVR/SVR < 0.2:1*
- *Inoperable - if…*
  - PVRI > 10 WU
  - PVR/SVR > 0.7: 1
  - Qp/Qs < 1.5:1

Conditions with High Risk for PVOD:

(May develop PVOD within 6-12 months age)

- *TGA*
- *AV canal defect*
- *Truncus arteriosus*
- *Large L à R shunt (VSD, PDA, APW) with or without Down's syndrome*

(5) Low cardiac output:

- Pallor(Poor perfusion), bradycardia, hypotension
- Acidosis
- Cyanosis
- Arrhythmias
- Altered sensorium
- Temperature instability
- Renal and Liver dysfunction

Conditions causing shock-like clinical findings in the neonate:

- HLHS
- Critical AS
- Co A
- Myocarditis
- Tachyarrhythmias
- Inturrupted Aortic Arch
- Complex CHD & LVOTO
- AV Malformation
- Tumor

Summary: When to Suspect?

Rule 1: Tell parents to watch for:

1. Poor feeding
2. Fast breathing
3. Persistent cough
4. Cyanosis or pallor
5. Sweating, even while sleeping
6. Irritability, excessive crying
7. Lethargy
8. Failure to thrive, decreased motor mile stones

Rule 2: Have a VERY LOW threshold for checking a neonate's SaO2

Rule 3: Keep a HIGH index of suspicion

Rule 4: Perform careful cardiac examination

Rule 5: NEVER presume diagnosis
Rule 6: When abnormal, get investigated by Pediatric Cardiologist
Rule 7:NEVER ask parents to wait for Treatment till a particular "hypothetical" age or weight
Rule 8: In present era, child can be operated on Day 1 of life or at any wt –if required

Future is in YOUR hands ...
Let Us Give Chance To Every Child
To Live
In This Beautiful World !

Timing of surgery for Congenital heart diseases - Dr. Srinivas Mallya

Most of the congenital heart diseases that we see in our practice are essentially correctable provided they are operated at the right time Many of the simpler forms of the CHD become inoperable by the time they reach the hospital mostly because of lack of awareness among the general public & to an extent in the physicians themselves. Hence an attempt is made to discuss the natural history of some common chd,ideal time for surgery so that outcomes are optimized.

4 Parallel Sessions

Break Away Session | 3

Symposium on Critical Care Medicine

Sunday, 28 January, 2007

Venue : R-World Multiplex

08:00 am Registration & Breakfast

Heart Failure & Management in I.C.U.

Chair : Dr. Anil Jain, Dr. Naman Shastri

Moderator : Dr. Vishal Gupta, Dr. Chirag Mehta

09.00 am Diastolic Heart Failure & Critical Illness - *Dr. Naman Shastri*

09.25 am Non Interventional management of severe heart failure - *Dr. Anish Chandarana*

09.50 am Is this a right time to send patient for CABG? - *Dr. Vishal Gupta*

10.15 am Identification of 'RIGHT' heart failure and management - *Dr. Naman Shastri*

10:40 am Refreshment Break

Shock and Resucitation

Chair : Dr. Tibriwale, Dr. Hemant Antani

Moderator : Dr. P. K. Jain, Dr. Manoj Singh, Dr. Mukul Oza

10.50 am What is current trend in CPR? - *Dr. Manoj Singh*

11.15 am Blood Gas Analysis : Is that so easy? - *Dr. P. K. Jain*

11.40 am Functional haemodynamic monitoring : A practical approach for volume responders - *Dr. Naman Shastri*

12.05 pm Non Invasive Ventilation in ICU. Is that really helpful? - Dr. Chirag Mehta

Beside Care

Chair : Dr. Bharat Trivedi, Dr. Hemant Antani

Moderator : Dr. Urmish Chudgar, Dr. P. K. Jain, Dr. Samuel Johnson

12.40 pm Thrombocytes : Are they worth monitoring? - *Dr. Urmish Chudgar*

01.05 pm Hyponatraemia & Hypokalemia : Silent nightmare for Physicians - *Dr. P. K. Jain*

01.30 pm Question & Answer Session

Panel Discussion

Moderator : Dr. Naman Shastri

Panelists : Dr. Chirag Mehta, Dr. Ajay Naik, Dr. Anil Jain, Dr. Manoj Singh Dr. Dhiren Shah

01.40 pm Managing Acute emergency in Intra-operative period

02:00 pm Lunch & Farewell

Critical care Sessions

This session comprises of many issues regarding Critical care aspects faced by physicians in the ICU.

Diastolic Dysfunction in the ICU

Systolic dysfunction is very common in the ICU, but we often neglect Diastolic Dysfunction in the ICU. Bedside evaluation is mandatory in sick patients. Tachycardia and Ischemia is something which we need to control urgently. Volume response in this category is very tricky. What is the ideal time and up to what limit should it be given should be titrated.

Nonintervention management of Heart failure

In the era of the urgent PCI this approach still has a definitely role in the managing a case of acute LVF. In this case the Ionotropes and titrated volume play the main role. Haemodynamic monitoring plays an equal and important role. All physicians practicing in peripheral centres have to be masters in this approach so as to stabilize these patients before referring to the tertiary centres in a better condition.

Right time to send the patient for CABG

If a patient with recent MI turns out to be a candidate for CABG on angiography, the cardiologist may put the patient for primary CABG in view of the urgency. But is he really a fit candidate for the surgical intervention? There are many studies being done on this issue. The surgeon's perspectives will be discussed.

Right ventricular Dysfunction

Almost 70% patients of admitted to the ICUs are having definite evidence of right ventricular dysfunction. The old myth is that in RV failure, give volume. Is that right? If "no" then till what extent it is wise to give volume? If we need to add an ionotrope, which one is ideal in the scenario? There are some new molecules in the basket, like Enoximone, levosemendan along with our old friendly molecules.

Weaning patient from mechanical ventilator

Weaning from the mechanical ventilator is the most important aspect of ICU care. In recent times with the advent of new and sophisticated ICU ventilators and availability of new imaging devices, the management has become simpler. But, more the knowledge; more the confusion! To make a right decision about weaning selection of the correct mode is much necessary.

Non Invasive Ventilator

This has become a part of routine care in ICU. Its use has reduced the necessity to re intubate a patient weaned from a mechanical ventilator. Complete dependency on the invasive or non inva-

sive ventilator has created complications to some extent, so ventilators should be used only when it is prudent.

Blood gas analysis and electrolyte imbalance is monitored by machines but it is interpreted by the physician. Appropriate diagnosis and management by the physician is necessary. Sometimes they may be manipulated in interpretation. An exact understanding of the electro-metabolic disorders will help in the reduction of overall morbidity and mortality.

Thrombocyte count is very much confusing in many conditions and it has direct relation to not only bleeding disorders but also infection processes. What is the target platelet count? In which conditions should we consider replenishment? The approach in the diagnosis of high or low count is equally important.

The Symposium on Critical Care will cover these issues.

Dr.Naman Shastri
Course Director

Non Interventional management of severe heart failure
- Dr. Anish Chandarana

Acute heart failure is appreciated as rapid onset of symptoms and signs secondary to cardiac dysfunction. Cardiac dysfunction may be secondary to systolic and/or diastolic dysfunction, preload and afterload mismatch or rhythm abnormalities. Variety of etiologies like acute coronary syndrome, rheumatic and nonrheumatic valvular heart disease, hypertensive heart disease including acute hypertensive crisis, primary disease of myocardium like myocarditis or cardiomyopathy, congenital heart diseases, pericardial diseases, diseases of aorta and certain high output status like thyrotoxicosis etc. can cause acute heart failure. It may be a first time presentation or acute deterioration of an already existing chronic stable heart failure. Infection and fever, anaemia, pregnancy, undue exertion, fluid excess etc. can lead to acute deterioration.

Management strategies are decided by clinical, laboratory and hemodynamic assessment. All attempts should be made to find out precipitating and etiologic factors. Better and judicious use of variety of laboratory tests, noninvasive and invasive hemodynamic monitoring including systemic and pulmonary arterial and central venous pressure monitoring, echocardiography, cardiac catheterization etc. can provide significant insight into diagnosis and perfection and adequacy in management. Goal is to improve symptoms and clinical outcomes.

Treatment of the route cause e.g. primary PTCA in acute myocardial infarction or intravenous antihypertensive medicines in acute hypertensive crisis and correction of precipitating factor like antibiotic medicines for pneumonitis or RBC replacement for anaemia can do a lot to stabilize the patient very effectively and rapidly. By the time definite measures are taken and improvement

occurs, optimizing afterload and preload along with improving myocardial contractility can help to bridge the condition. Intravenous intermittent boluses or infusion of loop diuretics, intravenous infusion of nitroglycerine, sodium nitroprusside, or nesiritide and intravenous infusion of dopamine, dobutamine, adrenaline, or milrinone depending upon clinical and hemodynamic conditions. Variety of surgical and percutaneous interventions(like intra aortic balloon counterpulsation, non invasive and invasive ventilatory support, urgent surgery for mechanical complications of acute myocardial infarction, percutaneous coronary intervention for acute coronary syndrome, ventricular assist device etc.) can be of great value in supporting and treating patients with acute heart failure.

4 Parallel Sessions

Break Away Session | 4

Symposium on Diabetes

Sunday, 28 January, 2007

Venue : R-World Multiplex

08:00 am Registration & Breakfast

SESSION I

Chair : Dr. Ashok Parekh, Dr. Sunil Wadhwani

Moderator : Dr. Anil Bhoraskar, Dr. Mahadev Desai

09:00 am Inaugural Lecture

Epidemiology of Diabetes and Cardiovascular diseases - *Dr. O.P. Gupta*

09:15 am Dietician plays a central role in managing Diabetes in Indian Setup - *Mrs. Shilpa Joshi*

09:24 am Dietician can play very little role in managing diabetes in Indian setup - *Dr. Anil Bhoraskar*

RAPID FIRE LECTURE

09:40 am Glycemic Index-Best Or Waste? - *Dr. Tiven Marwah*

09:46 am Micronutrients and Diabetes - Do we know all about it? - *Mrs. Shilpa Joshi*

09:52 am HbA1C- Time to update? - *Dr. Mukul Oza*

09:58 am Glucometers- Reliable or Liable? - *Dr. Mahadev Desai*

10:10 am GUEST LECTURE - Prevention of Diabetes- A Reality? - *Dr. S.M.Sadikot*

10:25 am Refreshment Break

SESSION II

Chair : Dr. Kirti Shah, Dr. Nitin Parikh

Moderator : Dr. Mayur Patel, Dr. Shashank Joshi

10:40 am Oral Hypoglycemic agents are overused - *Dr. Shashank Joshi*

10:50 am Oral Hypoglycemic agents are underused - *Dr. Urman Dhruv*

RAPID FIRE LECTURE

11:00 am 2-Hr PG Controversy Continues - *Dr. Mayur Patel*

11:08 am Need to set our own targets? - *Dr. Vivek Arya*

11:16 am Cardiac Autonomic Neuropathy in Diabetes - *Dr. Manoj Vithalani*

11:24 am Are We Happy with Future Drugs in Pipeline? - *TBA*

11:35 am GUEST LECTURE - AMI and Diabetes-Have we reached a consensus? - *Dr. Anil Bhoraskar*

SESSION III

Chair : Dr. Bipin Amin, Dr. Deepak Dantara

Moderator: Dr. Urman Dhruv, Dr. Kirit Akhani

Panelists : Dr. Anil Bhoraskar, Dr. Shashank Joshi, Dr. S.M.Sadikot, Mrs. Shilpa Joshi

12:00 pm PANEL DISCUSSION ON Long Live Metabolic Syndrome- The Metabolic Syndrome is Dead

SESSION IV

Chair : Dr. Navin Khimesara,Dr. Paras Doshi

Moderator : Dr. S.M.Sadidkot, Dr. Vishal Mehta

12:30 pm What Matters More? RAAS Inhibition V/S Blood Pressure Control? - *Dr. Anil Bhoraskar*

12:40 pm Inflammatory Markers V/S Biochemical Markers? - *Dr. S.M.Sadikot*

12:50 pm Impaired Fasting Glucose V/S Impaired Glucose Tolerance? - *Dr. Shashank Joshi*

SESSION V

Chair : Dr. Sunil Mehta, Dr. Gajanan Mohata

Moderator : Dr. Sanjeev Phatak, Dr. Bansi Saboo

1:00 pm Diabetes is a Cardiovascular Disease - *Dr. Anish Chandarana*

1:09 pm Diabetes is a Metabolic Disease - *Dr. S.M.Sadikot*

RAPID FIRE LECTURE Evidence Based Medicine

1:20 pm Insulin Infusion Pumps - *Dr. Sanjeev Phatak*

1:26 pm Continuous Glucose Monitoring System - *Dr. Bansi Saboo*

1:32 pm Inhaled Insulin - *Dr. Navneet Shah*

1:38 pm Glitazones- Expanding Horizons - *Dr. Ramesh Goyal*

1:45 pm GUEST LECTURE - Impact of recently concluded trials on Clinical Practice - *Dr. Shashank Joshi*

02.00 pm Lunch & Farewell

Oral Hypoglycemic agents are underused
Dr. Urman Dhruv

We are not utilizing Oral Hypoglycemic Agents Optimally Debate

Overuse of OHA is well known and I am sure will be targeted at by my opponent speaker. So I will leave it for him.

Underuse of OHA is at several stages and include

1. Underuse for primary prevention especially Metformin and Glitazones in view of recently concluded Dream Trial
2. Underuse in IGT/IFG i.e. in Prediabetes.
3. The definite subsets of diabetics where one can salvage pancreas is Prediabetics. We do not have an insulin which can prevent b cell regeneration/reguvination but we definitely have OHA which can salvage b cells in time.
4. Fine tuning with OHA's
5. Omitting OHA as soon as one starts with Insulin.
6. Use in pregnancy.

It is said that we do not have any ideal OHA but do we have any ideal insulin?

It is said that it is the result [control of glycemia] and not the tool that is important tor managing glycemia

Diabetes is only a subset of a less understood broad disease which leads to high cardiovascular mortality and we have failed to make use of OHA for this broad disease and restricted their use only to managing glycemia.

Glycemic Index – Is it Best or a Waste?
Dr. Tiven Marwah, M D (Medicine), D I S (Endocrinology) France, D I S (Nutrition) France

Diet plays a major role for prevention & management of several metabolic disorders like obesity, diabetes and dyslipidemia. Diet planning can be done by calories distribution, macronutrient composition, carbohydrate counting and a new concept called glycemic index. Glycemic index is defined as the incremental area under the glucose response curve after a standard amount of carbohydrate from a test food like glucose. The Glycemic Index (G I) of a specific food or meal is determined by the nature of the carbohydrate consumed and by other dietary factors.

Food with high G I (like potato & rice) produce hyperglycemia and hyperglycemia followed by hypoglycemia inducing hunger, food intake & eventually weight gain. On other hand foods with low G I (like legumes and non-starchy vegetables) produce lesser hyperglycemia and hyperglycemia and increase satiety. They do not induce weight gain.

Several studies have confirmed that low G I foods in comparison to high G I foods facilitate weight loss, improvement in blood sugar & lipid values in diabetic and non-diabetic subjects.

In this brief talk, I will discuss the practical utility of concept of G I for management of obesity and diabetes in Indian scenario.

Cardiac Autonomic Neuropathy in Diabetes
Dr Manoj Vithlani

- Represents a serious complication and leads to an approximately five fold risk of mortality.
- Prevalence of CAN in diabetes, approximately 17% in Type 1 Diabetes, 22% in Type 2 Diabetes. An additional 9% of type 1 & 12% of type 2 patients have borderline dysfunction.
- In asymptomatic diabetic patients, CAN appears to be a better predictor of major cardiac events than silent myocardial ischaemia.
- Once CAN identified in a patient with diabetes, health care providers may consider altering the prescribed exercise regime, increased surveillance for cardiac ischaemia, check list of prescribed medications and aggressive treatment of CV risk factors.

Continuous Glucose Monitoring: The New Glzmos
Dr. Banshi Saboo

Continuous glucose monitoring is the newest revolution in the technology crusade that has brought an "artificial pancreas" tantalizingly close.

A disposable sensor probe is inserted subcutaneously and then connected to a battery-powered transmitter. The transmitter sends a radio signal to a receiver that displays the glucose concentration in the subcutaneous interstitial fluid at 1 or 5 minute intervals. The transmitter and receiver are expected to last a year or more.

The sensors are smaller than an insulin infusion catheter and there is minimal discomfort associated with their introduction. After attaching the transmitter, a2-10-hour warm-up period begins. Routine capillary blood glucose measurements are then required to calibrate the system monitors serve as adjuncts and do not replace meters. Therefore, sensor readings need to be confirmed with a meter before corrective actions are taken. We will peep into the evidence based data about the usefulness of CMBG in the management of diabetes.