

# 3-C Con 2006

NOVELTIES AND ADVANCES IN CARDIOVASCULAR MEDICINE



## *Abstract & Program Book*



**February 11-12, 2006**  
**Nirma University, Village Chharodi,**  
**S. G. Highway, Ahmedabad**

**Organized by**



**The Heart Care Clinic**

Care Cardiovascular Consultants

## Organizers and Faculty

### Conference Chairman

Dr Keyur Parikh (M) 98250 26999

### Conference Director

Dr Anil Jain (M) 98250 63284

### Scientific Committee

Dr Milan Chag  
(M) 98240 22107  
Dr Hemang Baxi  
(M) 98250 30111  
Dr Dhiren Shah  
(M) 98255 75933

### Course Co-ordinators

Dr Anish Chandarana  
(M) 98250 96922  
Dr Ajay Naik  
(M) 98250 82666  
Dr Naman Shastri  
(M) 98250 16676

### Reception Committee

Dr Urmil Shah  
(M) 98250 66939  
Dr Vishal Gupta  
(M) 98250 11239  
Dr Chirag Mehta  
(M) 98250 16956

### Advisory Committee

Dr Srinivas Mallya  
(M) 93761 49769  
Dr Guntant Patel  
(M) 98240 61266  
Dr Ramesh K Goyal  
(M) 98254 48588  
Dr Ashit Jain (USA)

### Local Organizing Committee

Dr Bharat Trivedi  
(M) 98250 09518  
Dr Kalpana Jain  
(M) 93270 63284  
Dr Satya Gupta

Dr Joyal Shah  
(M) 98253 19645  
Dr A. Nagesh  
(M) 98792 59672

Dr Urmil Shah  
(M) 98240 88997  
Dr Niren Bhavsar  
(M) 98795 71917

Dr Mihir Tanna  
(M) 98242 14770  
Dr Satish Patel  
(M) 98980 99079

### International Faculty

Banai, Shmuel, Israel  
Makkar, Raj, U.S.A.  
Rozenman, Yoseph, Israel

Donato, Marisa Di, Italy  
Nair, Anuja, U.S.A.  
Shah, Bharat, U.S.A.

Parikh, Ashish, U.S.A.  
Waagstein, Finn, Sweden

### National Faculty

Rozenman, Yoseph, Israel  
Bhargava, Balram, New Delhi  
Kamath, Prakash, Cochin  
Manoria, P. C., Bhopal  
Parashar, S. K., New Delhi

Shah, Bharat, U.S.A.  
Hiremath, Jagdish, Pune  
Kapoor, Sunil, Hyderabad  
Sinha, Nakul, Lucknow

Waagstein, Finn, Sweden  
Hiremath, M. S., Pune  
Kaushik, S. K., Udaipur  
Mavlankar, Dileep, Ahmedabad



### Local Organizing Committee

**Sitting (Left to Right) :** Dr. Urmil Shah, Dr. Dhiren Shah, Dr. Bharat Trivedi, Dr. Vishal Gupta, Dr. Srinivas Mallya, Dr. Anil Jain, Dr. Naman Shastri, Dr. Chirag Mehta, Dr. Kalpana Jain, Dr. Niren Bhavsar  
**Standing (Left to Right) :** Dr. Joyal Shah, Dr. Anish Chandarana, Dr. Ajay Naik, Dr. Keyur Parikh, Dr. Milan Chag, Dr. Hemang Baxi, Dr. Urmil Shah, Dr. Guntant Patel, Dr. A. Nagesh

## From the desk of Dr. Keyur Parikh Conference Chairman

I have great pleasure in presenting a seven star conference with a menu of over 80 lectures delivered by 40 Medical Scientists from all over the world on the 11th and 12th February 2006, the 3-C Con 2006!

At the outset let me state that the credit for this event goes to the **superhuman** effort put in by my **superhuman partners and colleagues**, at the **'The Heart Care Clinic' (Care Cardiovascular Consultants)!**

An equal in effort has been put in by staff, family and friends.

Why do we put in so much effort over and above our hectic lives as heart specialists? I quote an ancient proverb: **'When there is no vision, the people perish!** This conference is a must for people who want to glimpse into the vision of Cardiovascular Medicine in 2006 and beyond.

Einstein has said: 'Imagination... is more important than knowledge'. I invite you to an intellectual feast of **'Novelties and Advances in Cardiology'** which may have few parallels in 2006. There are more advances in cardiology than we can imagine! We will reveal many of them in the 3-C Con 2006.

Above all, the 3-C Con 2006 is for creating, developing and renewing friendship through our common ethical goals with a touch of kindness, goodness, faithfulness, gentleness, self control and smiles.

Progress requires change. We hope to lead by progress and change.

### Welcome Message

Our 11<sup>th</sup> year of academics brings us to another confluence of thought leaders from all over the world. We are pleased to welcome you to the **3-C Con 2006** on behalf of The Heart Care Clinic and the joint organizers – Association of Physicians, Ahmedabad, to be held on 11<sup>th</sup> and 12<sup>th</sup> February 2006 at Nirma University.

Our previous major conference with multiple international faculty members was the JIC-2005 held in December – January 2004 – 2005. The success of JIC – 2005 with more than 800 registered delegates, triple parallel sessions and additional academic gatherings like Satellite Symposia has given us a legacy of excellent educational meetings.

We modestly hope to maintain the high standards and if possible better them.

This time the focus will be on 'Novelties and Advances' in each subject with over 40 Faculty Members from all over the world, more than 85 lectures and 18 hours of CME.

A CME attendance certificate for 18 hours will be issued to those delegates who attend all the sessions.

Apart from the whole day seminars, we have organized Satellite Symposia in which delegates can choose the subject of their interest. Prior registration for satellite symposia is necessary as seats in each of the separate symposium are limited. Only registered delegates of the 3-C Con 2006 can attend the Satellite Symposia.

Hoping to meet you for an educational odyssey at Nirma University on 11<sup>th</sup> and 12<sup>th</sup> February, 2006...

#### Dr. Milan Chag

##### Scientific Committee

Dr Hemang Baxi  
Dr Dhiren Shah  
Dr Joyal Shah

#### Dr. Anil Jain

##### (Conference Director)

##### Course Co-ordinators

Dr Anish Chandarana  
Dr Ajay Naik  
Dr Naman Shastri

#### Dr. Srinivas Mallya

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Dr Vishal Gupta  
Dr Chirag Mehta

##### Advisory Committ

Dr Gunvant Patel  
Dr Ramesh K Goyal  
Dr Ashit Jain (USA)

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Dr Niren Bhavsar

Dr Urmi Shah  
Dr Mihir Tanna

Dr Kalpana Jain  
Dr Satish Patel

Dr A. Nagesh  
Dr Satya Gupta

# 3-C Con 2006

**Saturday 11<sup>th</sup> February 2006- DAY-1**

**REGISTRATION & BREAKFAST (7:30 AM to 8:30 AM)**

**Novelties and Advances in General Cardiovascular Medicine  
(8:30 AM to 10:30 AM)**

**Hall-A**

**Chair : Dr. Keyur Parikh, Dr. Naman Shastri  
Moderator: Dr. Milan Chag, Dr. Chirag Mehta**

**Hall-B**

**Chair : Dr. Anish Chandarana  
Moderator : Dr. Vishal Gupta**

- 08.30 AM Cardiology in 2006: Past, Present & Future  
*Dr. Keyur Parikh , Ahmedabad, India*
- 09.00 AM Clinical Decision Making in Cardiology: What to do ? What not to do?  
*Dr. Keyur Parikh , Ahmedabad, India*
- 09.20 AM The Principles of Drug Therapy-Importance of Correct Drug Use: What to give? What not to give?  
*Dr. Milan Chag, Ahmedabad, India*
- 09.40 AM Can We Still Improve Pre-Operative Care for Critically Ill Patients ?  
*Dr. Naman Shastri, Ahmedabad, India.*
- 09.50 AM Can We Still Improve Post-Operative Care for Critically Ill Patients ?  
*Dr. Chirag Mehta, Ahmedabad, India.*
- 10.10 AM CT Angio, CMR, Radionuclide Studies & Future of Imaging Technology 2006  
*Dr. Urmil Shah, Ahmedabad, India.*

**PLENARY LECTURE - Women and Heart Disease  
Prof. Marisa Di Donato, Firenze, Italy  
(10:30 AM to 10:50 AM)**

**REFRESHMENT BREAK (10:50 AM to 11:00 AM)**

**Novelties and Advances in Heart Failure (A)  
(11:00 AM to 12:20 PM)**

**Hall-A**

**Chair: Prof. Finn Waagstein, Dr. Vishal Gupta  
Moderator: Dr. Ajay Naik, Dr. Dhiren Shah**

**Hall-B**

**Chair : Dr. Urmil Shah  
Moderator : Dr. A. Nagesh**

- 11.00 AM An overview in Heart Failure & 2005 ACC Guidelines for CHF  
*Prof. S.Kaushik, Udaipur, India*
- 11.20 AM Drugs in the treatment of Heart Failure and Resistant Heart Failure  
*Prof. Marisa Di Donato, Firenze, Italy*
- 11.40 AM Critical Survey of Trials concerning Chronic Heart Failure  
*Prof. Finn Waagstein, Sweden*
- 12.00 PM Device Management of Heart Failure: 2006  
to & Beyond
- 12.20 PM *Dr. Ajay Naik, Ahmedabad, India*

**PLENARY LECTURE - Peripheral Vascular Disease : What all Physicians should know ?  
Dr. Ashish Parikh, Delaware, U.S.A.  
(12:25 PM to 12:45 PM)**

**LUNCH (12:45 PM to 01:15 PM)**

**Novelties and Advances in Heart Failure (B) (01:15 PM to 02:15 PM)****Hall-A****Chair: Prof. Marisa Di Donato, Dr. Anish Chandarana,  
Moderator: Dr. Anil Jain, Dr. Gunvant Patel****Hall-B****Chair : Dr. Hemang Baxi  
Moderator: Dr. Niren Bhavasar**

- 01.15 PM Should I send my Patient with severe MR and LV Dysfunction for surgery?  
*Dr. Dhiren Shah, Ahmedabad, India*
- 01.30 PM To STICH or not to STICH: We know the answer, but do we understand the question?  
Surgical Ventricular Restoration (SVR) In End Stage Ischemic Heart Disease: Long Term Result  
*Prof. Marisa Di Donato, Firenze, Italy*
- 01.45 PM Surgical Management of Heart Failure: 2006 & Beyond  
*Dr. Anil Jain, Ahmedabad, India*
- 02.00 PM Alternative-Non pharmacologic treatment of dilated cardiomyopathy  
*Prof. Finn Waagstein, Sweden*

**Hall-A****Chair : Dr. Milan Chag, Dr. Keyur Parikh  
Moderator: Dr. Nakul Sinha, Dr. Urmil Shah****Hall-B****Chair : Dr. Naman Shatri  
Moderator : Dr. Haridas****3 C CON ORATION - Stem Cell Therapy for Myocardial Repair  
Dr. Raj Makkar, Los Angeles, USA (2:20 PM to 2:40 PM)****(A)****Achieving very low LDL (50-70) Levels and to very high HDL (>60) Levels  
Prof. Nakul Sinha, Lucknow, India (2.45 PM to 3.00 PM)****(B)****REGISTRATION & BREAKFAST (3:00 PM to 3:10 PM)****Novelties and Advances in Valvular Heart Disease & in General Cardiology (3:10 PM to 5:40 PM)****Hall-A****Chair : Dr. Raj Makkar, Dr. Srinivas Mallya  
Moderator: Dr. Milan Chag, Dr. Hemang Baxi****Hall-B****Chair : Dr. Joyal Shah  
Moderator : Dr. Vishal Gupta**

- 03.10 PM In Aortic and Mitral Valve Disease: Percutaneous Valve Repair and Replacement  
*Dr. Raj Makkar, Los Angeles, USA*
- 03.30 PM In Aortic and Mitral Valve Disease: Surgical Repair & Replacement: 2006 & Beyond  
*Dr. Srinivas Mallya, Ahmedabad, India*
- 03.50 PM The Growing Body of Drug-Eluting Stent Data: Clinical Trials and Real World Results  
*Dr. Keyur Parikh / Dr. Anish Chandarana, Ahmedabad, India*
- 04.05 PM Why my father should under go angioplasty for Heart Attack ?  
*Dr. Milan Chag, Ahmedabad, India*
- 04.15 PM What is Cryptogenic strokes/PFO's, LAA ? What should we do about it ?  
*Dr. Satya Gupta, Paris, France*
- 04.25 PM Diabetes and CAD  
*Dr. Hemang Baxi, Ahmedabad, India*
- 04.35 PM Multivessel CAD  
*Dr. Urmil Shah, Ahmedabad, India*
- 04.45 PM Hypertrophic Obstructive Cardiomyopathy: Clinical Pearls and Latest Management  
*Dr. Sunil Kapoor, Hyderabad, India*
- 05.00 PM Pulmonary Arterial Hypertension (PAH):  
to Clinical Pearls & Latest Management
- 05.15 PM *Dr. Jagdish Hiremath, Pune, India*

**TRANSPORTATION FROM NIRMA TO HCC (5:30 onwards)****RECEPTION with SNACKS at The Heart Care Clinic (6.00 PM to 7.30 PM)****SATELLITE SYMPOSIUM (8:00 PM ONWARDS)**

## Morning Session : 8.30 AM to 10.50 AM

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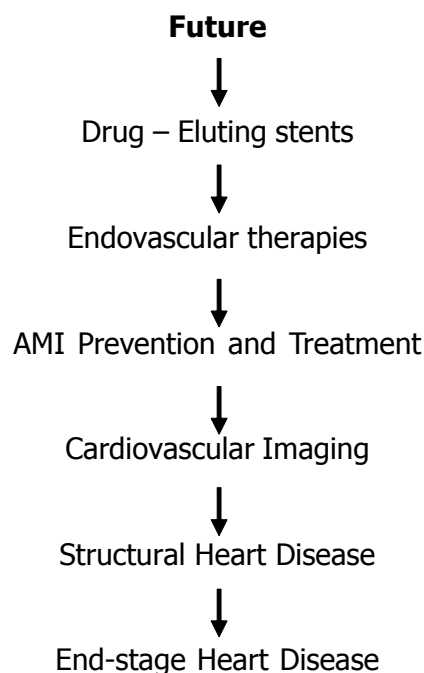
### Cardiology in 2006: Past Present Future for Abstract

*Dr. Keyur H. Parikh, Ahmedabad, India*

He will discuss many of these trials presented in 2005-2006

ACTIVATE	ACTIVE-W	ASSENT-4	ASTAMI	CAFE
FIELD	IDEAL	JELIS	MEGA	OmniHeart
PREVENT	PROactive	REPAIR	REVIVE II	SURVIVE
TITAN	Vareniciline			

This will be followed by a discussion of future



### Clinical Decision Making in Cardiology: What to do ? What not to do?

*Dr. Keyur Parikh , Ahmedabad, India*

#### **Process of Medical Decision-Making**

- ▶ Skilled Clinical Decision-making is the cornerstone of Medical Practice despite continuing significant advances in medical knowledge and technology.
- ▶ How the Medical decision-making becomes complicated?
- ▶ How the Medical decision-making becomes complicated?
- ▶ Probabilistic Reasoning
- ▶ Causal Reasoning
- ▶ Deterministic (categorical) reasoning
- ▶ Heuristic Reasoning
- ▶ Behavioral and Environmental Influences
- ▶ Diagnostic Test Performance

- ▶ Selection and Use of Diagnostic Tests
- ▶ Screening
- ▶ Using Tests in Combination
- ▶ Measures of Quality for Acute Myocardial Infarction
- ▶ Measures of Quality for Heart Failure
- ▶ Stages of Practice Development
  - The Honeymoon Stage
  - The Reputation Development Stage
  - The Industry Relationship Stage
  - The New Technology Growth Stage
  - The Sustaining Vision Stage
- ▶ Do you provide good service?
- ▶ Tidbits on saving and using time
- ▶ Places where I save time
- ▶ Are you disorganized?
- ▶ Getting organized
  - Keep data on cases and outcomes
  - Put good cases in PowerPoint format
  - Finish today's work today
  - Leave time for unexpected consults
  - On slow days, prepare a talk
  - Take one day per month to strategize
  - Learn practice flow
- ▶ Reasons for Organizing Cases
- ▶ Educate, Educate, Educate
- ▶ Don't complain, make a difference
- ▶ Don't have a bad attitude
- ▶ Final list of "Pearls"
- ▶ Timeline of Sickness

## **The Principles of Drug Therapy-Importance of Correct Drug Use: What to give? What not to give?**

***Dr. Milan Chag, Ahmedabad, India***  
***Abstract not received***

## **Can We Still Improve Pre-Operative Care for Critically Ill Patients ?**

***Dr. Naman Shastri. Ahmedabad, India***

The cost of providing pre-operative care would be offset by the the reduction in subsequent complications and length of hospital stay.

- ▶ "If resources are made available to invest in high-dependency beds and skilled medical intervention it is quite likely that..

it would prevent many patients from developing complications and ending up back in intensive care in big trouble and costing a lot of money."

### **Definition of perioperative optimisation**

It is the preventive manipulations of physiological parameters during the pre operative period. Studies assessing the effectiveness of specific strategies exists in the following areas

- ▶ Increasing tissue oxygen delivery
- ▶ Prevention of myocardial ischaemia
- ▶ Maintenance of normothermia
- ▶ Maintenance of normoglycaemia

### **Preoperative haemodynamic optimization of high-risk surgical patients**

- ▶ Preoperative optimization of oxygen delivery CI greater than  $4.5 \text{ litre min}^{-1} \text{ m}^{-2}$ ,
- ▶  $\dot{V}O_2$  greater than  $600 \text{ ml min}^{-1} \text{ m}^{-2}$ ,
- ▶ pulmonary artery occlusion pressure (PAOP) below 18 mm Hg,
- ▶ CVP below 15 mm Hg,
- ▶ Systemic vascular resistance (SVR) above  $1450 \text{ dyne s cm}^{-5}$  and
- ▶  $\dot{V}O_2$  above  $170 \text{ ml min}^{-1} \text{ m}^{-2}$ .

Perioperative optimization and right heart catheterization:

### **what technique in which patient?**

Crit Care. 2003; 7(3): 201–202

- ▶ pulmonary artery catheterization should not be performed routinely on all patients, when used correctly by trained personnel in selected patients the pulmonary artery catheter continues to provide valuable information.
- ▶ Based on the available data, it appears appropriate and safe to withhold transfusion based on the hemoglobin or hematocrit level until the patient's hemoglobin is 7 g/dl or less.  
(British Journal of Anaesthesia, 2003, Vol. 90, No. 6 719-722)

Perioperative haemodynamic optimisation of high risk surgical patients seems to be associated with a reduction in morbidity and mortality

## **Can We Still Improve Post Operative case in critically ill patients**

***Dr. Chirag Mehta, Ahmedabad, India.***

Many of the services provided to critically ill patients in post-operative period are the same no matter the underlying disease. While critical care Anesthesiologist often provide care to post-operative patients in surgical ICUS, the diversity to their skills provide an opportunity to care for patients with both medical and surgical illness. The roster of services that is required by these complex patients, often requires a multidisciplinary approach to care to ensure that the patient receives the services necessary to optimize the clinical outcome and most effectively utilize costly resources. Proper ventilatory care, fluid and electrolyte management, adequate pain relief nutrition & sound sleep are few services that form a part of improving post operative care in critically ill.

## **CT Angio, CMR, Radionuclide Studies & Future of Imaging Technology 2006**

***Dr. Urmil Shah, Ahmedabad, India***

***Abstract not received***

# Women and Heart Disease

***Prof. Marisa Di Donato, Firenze, Italy***

During the past 3 decades, numerous and remarkably consistent studies have reported sex differences in the epidemiology, prevention, diagnosis, and clinical manifestations of coronary artery disease, and, especially noteworthy, in the sex differences in patients undergoing coronary revascularization, where a higher mortality rate has been noted in women. In fact, the thesis that women are different from men is not argued in any more important venue than the surgical theatre. The questions of whether and why women have higher probabilities of poor outcomes after coronary artery bypass grafting (CABG) have been repeatedly asked.

The difference in mortality has been explained by the fact that female CABG surgery candidates frequently present with older age, smaller body size, smaller coronary arteries, more urgent status, and more comorbid factors than their male counterparts. The difference in outcomes experienced by women after CABG surgery or after coronary revascularization could also be related to delayed referral or a different pathogenesis of the progression in coronary atherosclerosis. Or, can it simply be related to biological factors specific to women that make them unmatched with men? The randomized controlled trial is considered the gold standard when examining outcomes between groups but on the question of gender, however, a randomized controlled trial is impossible: one cannot randomize patients to sex!

The most provocative explanation for the sex difference in in-hospital mortality in patients undergoing coronary revascularization relates to the higher incidence of left ventricular hypertrophy and hypertensive heart disease in women compared with men. Women have been reported to have a higher incidence of congestive heart failure despite better left ventricular function (with fewer previous infarctions) than men. This is best illustrated in the Bypass Angioplasty Revascularization Investigation (BARI), in which, despite the similar incidence of in-hospital death and myocardial infarction, there was a significantly higher incidence of congestive heart failure or pulmonary edema after both CABG and PCI in women compared with men.

Furthermore, it is noteworthy that although 25% to 30% of patients undergoing revascularization are women, in the randomized SHould we emergently revascularize Occluded Coronaries for cardiogenic shock (SHOCK) trial, women comprised 36% of the study population! Moreover, the increasing age in the western population leads to a higher prevalence of women presenting with heart failure and there is a need to improve treatment effectiveness and to better understand gender differences. We report our experience in the surgical treatment of ischemic heart failure in women treated with surgical ventricular restoration. The technique consists in excluding the scarred tissue, reducing and reshaping the left ventricle through an endoventricular patch repair with the use of a Mannequin (Chase Medical, Richardson, Texas).

Complete coronary revascularization is almost always associated and mitral repair is performed if needed. Our patient population comprises 109 women (mean age 68+/-9 years) who have had a myocardial infarction and presented with symptoms of heart failure. Seventy-nine were in NYHA class III/IV; 45% had moderate to severe mitral regurgitation which was repaired in 71%. After surgery EF improved significantly from 31+/-7 to 39+/-6%  $p=0.001$  as well as NYHA class (from 3.1+/-0.6 to 1.9+/-0.8) and ventricular volumes decreased significantly (EDV -20%, ESV -25%  $p=0.0001$ ). Ten years survival rate was 69%, not different from men.

## Pre Lunch Session : 11.00 AM to 12.45 PM

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### An overview in Heart Failure & 2005 ACC Guidelines for CHF

*Prof. S.Kaushik, Udaipur, India*

*Abstract not received*

### Drugs in the treatment of Heart Failure and Resistant Heart Failure

*Prof. Marisa Di Donato, Firenze, Italy*

*Abstract not received*

### Critical Survey of Trials concerning Chronic Heart Failure

*Prof. Finn Waagstein, Sweden*

*Abstract not received*

### Device Management of Heart Failure: 2006 and Beyond

*Dr. Ajay Naik, MD, DM, DNB, FACC*

Over the past few years, Heart Failure (HF) management has evolved at a tremendous pace. **Cardiac Resynchronization Therapy (CRT, Biventricular Pacemaker Implantation)** has dramatically improved the quality of life of patients in NYHA class III and IV HF. Large multicenter trials MUSTIC and MIRACLE have demonstrated reduction in morbidity of HF after CRT implants. The latest CARE-HF trial data has shown reduction in mortality in patients with CRT. Patients who have improved from NYHA class III and IV HF and those who are in NYHA class II are at greatest risk of Sudden Cardiac Death (SCD) due to ventricular arrhythmias. These patients are likely to benefit by add-on defibrillator function (**CRT-D: Cardiac Resynchronization Therapy with Defibrillator**). **CRT devices now have sensors that monitor and warn rise in left atrial pressure** – these help in fine tuning drug therapy before overt heart failure occurs.

Surgical therapy of HF has also evolved remarkably. Patients who have improvement in LV function after Surgical Ventricular Restoration are protected against risk of SCD by implantation of **AICD (Automatic Implantable Cardioverter Defibrillator)**. **LV Assist Devices** are important to support LV function in patients with transient severe LV dysfunction (such as myocarditis). They serve as a bridge to heart transplants. **Total Artificial Heart (TAH)** is therapy of immense promise. There is continuous refinement in the TAH technology and this may become commonplace therapy in the future.

### Peripheral Vascular Disease : What all Physicians should know ?

*Dr. Ashish Parikh, Delaware, U.S.A.*

*Abstract not received*

## Afternoon Session : 1.15 PM to 3.00 PM

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### Should I send my patient with mitral regurgitation and LV dysfunction for surgery

**Dr. Dhiren Shah, Ahmedabad, India**

#### **Introduction**

Mitral regurgitation secondary to the LV dysfunction is also called as Functional or Ischaemic mitral regurgitation. This MR occurs secondarily to myocardial infarction or in the presence of acute ischaemia or due to dilated cardiomyopathy. Steven Bolling says "Functional mitral regurgitation is a ventricular disease, not a valvular disease"

#### **Incidence**

Occurs in approximately 20% of patients after MI and 56% patients with congestive heart failure due to ischaemic or non ischaemic cardiomyopathy. It occurs more commonly in patients with posterior than anterior infarction. It may appear acutely in case of acute myocardial infarction with ruptured papillary muscle or chordae rupture, presenting in cardiogenic shock. Or it may appear after months of insult, and this attributed to the remodeling of LV.

#### Pathophysiology

1. Remodelling of the LV with the increased sphericity.
2. Annular dilatation
3. Papillary muscle displacement and new wall motion abnormalities
4. Tethered chordae

It is the result of insult to the well-coordinated interconnection between the mitral leaflets, mitral annulus, the chordae, and papillary muscle.

#### **Diagnosing**

Clinical evaluation, high level suspicion on getting low intensity systolic murmur, and along with that 2D echo, dobutamine stress echo, angiography, MRI will help in timely diagnosing mitral regurgitation in LV dysfunction.

#### **Therapy**

##### **Treatment options are:**

- ▶ Coronary revascularization
- ▶ Ring mitral annuloplasty
- ▶ Mitral valve repair/replacement
- ▶ Surgical ventricular restoration (TRISVR)
- ▶ Resynchronization therapy
- ▶ Medical therapy

The ultimate treatment would be permutation and combination of the above options for that particular patient, i.e. custom made. The operative mortality is acceptable 10% for this very sick patient. This improves the quality of life, NYHA class, symptomatology, ejection fraction, reduces the recurrent hospitalization, and gives intermediate term mortality benefit. Menicanti and Steve Bolling found that SVR can improve the severity of mitral regurgitation and benefit clinical parameters and intermediate results. Donald Glower et al have concluded that long term patient survival is more influenced by baseline characteristics and comorbidity than the cause of MR.

# To STICH or not to STICH: We know the answer, but do we understand the question? Surgical Ventricular Restoration (SVR) In End Stage Ischemic Heart Disease: Long Term Result

**Prof. Marisa Di Donato, Firenze, Italy**

The STICH trial (Surgical Treatment of ischemic heart failure) is an international, randomized, controlled clinical trial that will address 2 primary hypothesis: *Revascularization hypothesis* states that CABG plus optimal medical treatment improves survival at 3 years compared to medical treatment alone in pts with ischemic heart failure, EF= $\leq$ 35% and coronary vessels amenable for grafting.

**Restoration hypothesis** states that in pts with EF= $\leq$ 35% and coronary vessels amenable for grafting, surgical ventricular restoration (SVR) plus CABG plus optimal medical treatment improves survival free of cardiac hospitalization at 3 year. The need for a randomized trial for both hypothesis comes from the fact that 1) data on the superiority of surgery in respect to medical therapy are old and obtained in small series of pts from the CASS study where pts with a depressed EF were very few and 2) data on the effectiveness of adding SVR to CABG are observational and need to be validated. Interestingly, despite a lack of evidence proving the superiority of one strategy over all others, cardiologists and surgeons secretly-or not so secretly-believe they know the best way to care for their pts.

Some investigators are hesitant to randomize because they believe that all pts should be by-passed and others believe that none of these pts should have surgery. This happens within the same countries and sometimes within the same cities. Interestingly in both sides, people are equally convinced that they are right! Instead, large number of patients fall into a gray zone without clear evidence for benefit from MED or CABG or CABG and SVR. Answers from the Stich will confirm whether pts presenting with HF should be evaluated for an ischemic etiology or simply treated with medical therapy, will provide the only data about survival benefit, if any, of redo CABG and will provide the importance of CMR, Nuclear Cardiology Testing, ECHO Neuro-hormonal factors, costs and benefits. It will definitely demonstrate whether CABG plus SVR in pts with a post-infarction severe asynergy will improve survival free from hospitalization. Confirming the STICH revascularization hypothesis will dramatically increase the use of CABG among the million of patients now being medically treated without evaluation for an ischemic etiology

We report our experience of SVR on more than 2000 pts operated at San Donato Hospital between 1989 and 2005 and at the Cardiothoracic Center of Monaco between 1985 and 2005. Since 1998 a sizing device was introduced in both centers to allow a residual near normal ventricular volume (50-60 ml/m<sup>2</sup>). Ninety % of pts have had an anterior myocardial infarction; indications for surgery were heart failure, angina or a combination of the two. Overall, the results showed a thirty-day mortality rate of 5.7%. Mitral repair was associated to SVR and CABG in 20% of pts for a severe degree of mitral regurgitation or for a dilated mitral annulus in the absence of a severe MR. End diastolic and end systolic volumes decreased significantly (EDV from 211 $\pm$ 68 to 147 $\pm$ 50 and ESV from 146 $\pm$ 60 to 91 $\pm$ 38 ml p 0.0001) and EF improved from 33 $\pm$ 9 to 38 $\pm$ 9% p 0.001. NYHA functional class improved significantly with very few pts in class III/IV late after surgery. Need for cardiac re-hospitalization was 7% and implantable cardioverter defibrillator was used in 5 to 10% of the pts. At the Cardiothoracic Center of Monaco an electrophysiologic study is routinely performed preoperatively, at 1 week and at 1 year after SVR to induce ventricular arrhythmias. Before surgery 40% to 70% of pts have inducible ventricular tachycardia; after surgery both early and at 1 year ventricular tachycardia is inducible only in 5 to 10% of pts with a dramatic significant reduction.

Conclusions: if the trial will confirm our data (the Restoration hypothesis), and if SVR procedure becomes a standard of care, millions of individuals who currently have few options, will benefit in terms of survival and quality of life.

## **Surgical Management of Heart Failure: 2006 & Beyond**

*Dr. Anil Jain, Ahmedabad, India*  
*Abstract not received*

## **Alternative-Non pharmacologic treatment of dilated cardiomyopathy**

*Prof. Finn Waagstein, Sweden*  
*Abstract not received*

## **Stem Cell Therapy for Myocardial Repair**

*Dr. Raj Makkar, Los Angeles, USA*  
*Abstract not received*

## **Achieving very low LDL (50-70) and to very high HDL (>60) Levels**

*Prof. Nakul Sinha, Lucknow, India*  
*Abstract not received*

## **Evening Session : 3.10 PM to 5.15 PM**

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### **In Aortic and Mitral Valve Disease: Percutaneous Valve Repair and Replacement**

*Dr. Raj Makkar, Los Angeles, USA*  
*Abstract not received*

### **In Aortic and Mitral Valve Disease: Surgical Repair & Replacement: 2006 & Beyond**

*Dr. Srinivas Mallya, Ahmedabad, India*  
*Abstract not received*

### **The Growing Body of Drug-Eluting Stent Data: Clinical Trials and Real World Results**

*Dr. Keyur Parikh / Dr. Anish Chandarana, Ahmedabad, India*  
*Abstract not received*

### **Why my father should under go angioplasty for Heart Attack ?**

*Dr. Milan Chag, Ahmedabad, India*  
*Abstract not received*

### **What is Cryptogenic strokes/PFO's, LAA ? What should we do about it ?**

*Dr. Satya Gupta, Paris, France*

In the past, left atrial appendage (LAA) has been consider to be a relatively insignificant portion of the cardiac anatomy. Recently, it has been recognised that LAA has a unique developmental, anatomical and physiological properties. It is the structure with important physiological and pathological associations. LAA is a long, tubular, hooked trabeculated structure. It is a source of atrial natriuretic peptide and it shortens to a greater extent than the rest of left atrium during atrial contraction phase. Anatomical and physiological properties of LAA can be assessed with transesophageal echocardiography. The flow pattern in subjects with sinus rhythm has been found to be quadriphasic which comprises two filling and two emptying phases.

The LAA is the site most commonly associated with the thrombus formation, particularly in patients with non-valvular atrial fibrillation. Relative stasis of blood which occure in the appendage owing to its shape, presence of trabeculations, altered flow and, contraction pattern play major role in thrombus formation. About 15% of all ischemic strokes arises as a result of atrial fibrillation and approximately 90% of atrial thrombi in non-rheumatic atrial fibrillation and 60% of such thrombi in patients with rheumatic mitral valve disease are seen within the LAA.

The most effective current prophylaxis of stroke in atrial fibrillation is oral anticoagulant

(OAC) therapy. OAC may be contraindicated in many patients, particularly in the elderly in whom the risk of stroke is highest and therefore alternative treatment is required for these patients.

Recently, a novel percutaneous approach for the prevention of embolic episode in these high risk patients who are not candidate for oral anticoagulant has been introduced. The percutaneous left atrial appendage transcatheter occlusion (PLAATO) device seals the communication between the LA and its appendage. The PLAATO system consists of an implant and a delivery catheter. The implant(PLAATO Device) is a self-expanding nitinol cage covered with an occlusive expanded polytetrafluoroethylene membrane, which is laminated directly to the frame structure so that the perimeter has intimate contact with the inner wall of the appendage. The purpose of the membrane is both to occlude the orifice of the left atrial appendage and to allow tissue incorporation into the device. Small anchors along the struts and the occlusive membrane help device anchoring and encourage healing response. The device is delivered through a 14 Fr trans septal sheath. Feasibility and safety of PLAATO and other devices like Watchman, Amplazer have been published recently.

At present, the implantation of these devices is technically challenging and is not the substitute for OAC to prevent stroke, but it is an alternative in patients who are not suitable for anticoagulant therapy. Furthermore, this technique will not prevent all episodes of thromboembolism, particularly in patients with mitral valvular heart disease, in whom the appendage is the sole location of thrombus in only 60% of patients.

## **Diabetes & CAD**

***Dr. Hemang Baxi, Ahmedabad, India***

People with diabetes have an increased prevalence of atherosclerosis & coronary heart disease (CHD) and experience higher morbidity and mortality after ACS and myocardial infarction (MI) than people without diabetes. CV complications of diabetes are especially concerning not only because of emerging epidemic of diabetes but also earlier onset of CAD in individuals who have diabetes. Also, diabetes dictates certain canonical approaches to disease, such as preference of CABG in diabetics with multivessel to CAD over PTCA. However advent of Drug Eluting Stent (DES) seems to be an important breakthrough as far as multivessel PTCA/stenting in diabetics with good long term results are concerned.

## **Multivessel CAD**

***Dr. Urmil Shah, Ahmedabad, India***

***Abstract not received***

## **Hypertrophic Obstructive Cardiomyopathy: Clinical Pearls and Latest Management**

***Dr. Sunil Kapoor, Hyderabad, India***

***Abstract not received***

# **Pulmonary Arterial Hypertension (PAH): Clinical Pearls & Latest Management**

***Dr. Jagdish Hiremath, Pune, India***

Pulmonary hypertension (PH) is defined as PA systolic pressure more than 35mm of Hg, diastolic more than 15mm of Hg or mean more than 25mm of Hg.

Valvular heart disease, LA pathology (membrane) thrombo-embolism are correctable causes but primary pulmonary hypertension is a relentlessly progressively fatal disease.

Clinical examination, ECG, X-ray, Colour Doppler, HRCT, CT-angio, MRI, V/Q Scan are all useful in arriving at correct etiology and severity of PH. Biopsy is not performed often (No yield).

Five years mortality of advanced PH is very high. Diuretics, CCB for vasodilatation and anticoagulants are main stay of treatment. Sildenafil is now accepted useful therapy for PH. Use of prostacycline like Treprospinil sodium are being tried.

Heart lung transplantation is like definitive treatment at present.

**Saturday 11<sup>th</sup> February 2006**  
**Satellite Symposia at TGB**

**BANQUET HALL-1**

**Novelties and Advances in Practice Management (8:00 PM to 10:00 PM)**

**Chair: Dr. Ashish Parikh**  
**Dr. Keyur Parikh**

**Moderator: Dr. Bharat Shah**  
**Dr. Sunil Kapoor**

- ▶ Group Practice for Physicians  
*Dr. Anish Chandrana, Ahmedabad, India.*
- ▶ How to start a solo/group Practice  
*Dr. Sunil Kapoor, Hyderabad, India*
- ▶ An overview of large Physician/Cardiologist Group Practice (A)  
*Dr. Bharat Shah, California, U.S.A.*
- ▶ An overview of large Physician/Cardiologist Group Practice (B)  
*Dr. Ashish Parikh, Delaware, U.S.A.*
- ▶ How to merge a Medical Practice  
*Dr. Keyur Parikh, Ahmedabad, India*

**BANQUET HALL-2**

**Novelties and Advances (8:00 PM to 10:00 PM)**

**Chair: Dr. Prof. Shmuel Banai**  
**Dr. Hemang Baxi**

**Moderator: Dr. Jagdish Hiremath**  
**Prof. Yoseph Rozenman**

- ▶ Pregnancy and Cardiac Drugs  
*Prof. S. Kaushik, Udaipur, India*
- ▶ Role of & How to develop "Cardiac Rehab & Exercise Programme" in 2006  
*Dr. Hemang Baxi, Ahmedabad, India.*
- ▶ How to Load & Manage "Antiplatelet Agents" in ACS & Chronic CAD  
*Prof. Yoseph Rozenman, Tel-Aviv, Israel*
- ▶ What Cardiac Markers & How to interpret and Manage in 2006  
*Dr. Jagdish Hiremath, Pune, India*
- ▶ How to Manage Heparins, Bivaluridin, Thrombolytics in ACS  
*Prof. Shmuel Banai, Tel-Aviv, Israel*
- ▶ The Endocannabinoid System: A novel Therapeutic Target For the management of Multiple Cardiovascular Risk Factors  
*Dr. Joyal Shah, Ahmedabad, India*

**BANQUET HALL-3**

**Novelties and Advances in ECG and Arrhythmia (8:00 PM to 10:00 PM)**

**Chair: Dr. Ajay Naik**  
**Dr. A. Nagesh**

**Moderator: Dr. Prakash Kamath**  
**Dr. Mihir Tanna**

- ▶ Culprit Artery Localization in Acute Coronary Syndrome  
*Dr. Ajay Naik / Prakash Kamath*
- ▶ Heat, Shock, Battery, Maze - Tools of the trade  
*Dr. Ajay Naik / Prakash Kamath*
- ▶ Quizzing the Masters - Interactive ECG Sessions  
*Dr. Ajay Naik / Prakash Kamath*

## At the 'Heart Care Clinic' across the TGB

### Novelties and Advances in Echocardiography (8:00 PM to 10:00 PM)

**Chair :** **Dr. (Col.) S. Parashar**  
**Dr. Milan Chag**

**Moderator:** **Prof. S. Kaushik**  
**Dr. Urmil Shah**  
**Prof. Marisa Di Donato**

- ▶ What is the basic Echo/Doppler Evaluation of Diastolic Function ?  
*International / National Faculty*
- ▶ Advanced Evaluation of LV Wall Motion Analysis  
*International / National Faculty*
- ▶ Echo Assessment of Mitral Valve  
*International / National Faculty*
- ▶ Echo Assessment of Aortic Valve  
*International / National Faculty*

## BANQUET HALL-4

### Practice Guidelines and Quality Standard (8:00 PM to 10:00 PM)

**Chair:** **Dr. M. S. Hiremath**  
**Dr. Niren Bhavsar**

**Moderator:** **Prof. Nakul Sinha**  
**Dr. Satya Gupta**

- ▶ Methods to identify and manage individuals in Stages A and B pre-HF  
*Dr. S. K. Parashar, New Delhi, India*
- ▶ Chronic Heart Failure in the Adult 2006: What is the future ?  
*Dr. Anil Jain, Ahmedabad, India*
- ▶ Systemic Hypertension - What drugs & how to dose in 2006  
*Dr. Satya Gupta, Paris, France*
- ▶ Chronic Stable Angina: 2006  
*Dr. K. Haridas, Kochi, India*
- ▶ Current Overview of FDA & CE approved DES (Drug Eluting Stents) & future of DES  
*Dr. M. S. Hiremath, Pune, India*
- ▶ Understanding the Vulnerable Plaque - Role of VH-IVUS  
*Dr. Anuja Nair, Cleveland, U.S.A.*
- ▶ How to manage Cardiovascular Disease in the Elderly ?  
*Prof. Nakul Sinha, Lucknow, India*

## **Group Practice for Physicians**

*Dr. Anish Chandrana, Ahmedabad, India.  
Abstract not received*

## **How to start a solo/group Practice**

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## **An overview of large Physician/Cardiologist Group Practice (A)**

*Dr. Bharat Shah, California, U.S.A.  
Abstract not received*

## **An overview of large Physician/Cardiologist Group Practice (B)**

*Dr. Ashish Parikh, Delaware, U.S.A.  
Abstract not received*

## **How to merge a Medical Practice**

*Dr. Keyur Parikh, Ahmedabad, India*

### **Association Practice**

Most group practices fall in to one of the following three categories:

- ▶ Professional corporation
- ▶ Partnership
- ▶ Proprietorship- One person ownership

### **FINANCIAL MANAGEMENT OF THE MEDICAL PRACTICE**

#### **UNDERSTANDING FINANCIAL STATEMENTS**

1. UNDERSTANDING THE THEORIES.
2. UNDERSTANDING THE PURPOSE
3. UNDERSTANDING THE PRACTICALITY
4. UNDERSTANDING THE EFFECT

#### **IMPORTANCE OF MERGING**

- ▶ TO CONSOLIDATE INDEPENDENT PRACTICES THAT MAY OR MAY NOT HAVE COMMON VISION OF CULTURES
- ▶ FOR DEVELOPING A BARRIER TO COMPETITION
- ▶ IMPROVEMENT OF THE QUALITY OF LIFE

## **BENEFITS OF A WELL-PLANNED MERGER**

LEVERAGE  
BARRIER TO COMPETITION  
ECONOMIES OF SCALE  
SECURITY  
MARKETING  
PARTNERSHIPS

ACCESS TO CAPITAL  
LEADERSHIP AND MANAGEMENT  
RESOURCES  
NEW SOURCE OF REVENUE  
QUALITY OF LIFE  
STAFF COHESION

### ***THE DOWNSIDE OF MERGERS***

- ▶ Essential Items in a Contract-I
- ▶ What to cover in a practice agreement
- ▶ Key Revenue and Expense Ratios
- ▶ Discussion during the initial meeting
- ▶ Information needed when planning a successful merger

## **Pregnancy and Cardiac Drugs**

***Prof. S. Kaushik, Udaipur, India***  
***Abstract not received***

### **Role of & How to develop “Cardiac Rehab & Exercise Programme” in 2006**

***Dr. Hemang Baxi, Ahmedabad, India.***

Coronary artery disease is one of the major cause of cardiovascular morbidity & mortality. CHD is responsible for about one out of every five deaths, with sudden death the most common presentation,. Survivor of acute stages of myocardial infarction are at markedly high risk of another MI, heart failure or stroke than general population. Contemporary comprehensive, multifaceted, exercise-based cardiac rehabilitation programs offer patients with CHD an effective way to reduce subsequent cardiovascular mortalling and morbidity. The services provided by such programmes can slow the progression, stabilize or partially reverse the underlying atherothrombotic process by multiple mechanisms. In addition they improve patient’s quality of life by optimizing physical, psychological and social functioning.

### **How to Load & Manage “Antiplatelet Agents” in ACS & Chronic CAD**

***Yoseph Rozenman, MSc, MD, FACC, Director, Heart Institute,  
The E. Wolfson Medical Center, Sackler faculty of medicine, Israel***

Platelets have a major role in the pathophysiology of atherosclerotic complications in patients with vascular disease. Aspirin is a weak antiplatelet agent and is not sufficient in patients with ACS and after PCI. The addition of clopidogrel is of critical importance to improve the outcome of these patients.

Most ACS patients (ST elevation and non ST elevation) are currently treated with PCI (usually with stents). The CLARITY and CURE studies demonstrated the benefit of addition of clopidogrel to aspirin in patients with ACS (ST elevation and non ST elevation respectively), some of whom were already on aggressive antithrombotic therapy (including thrombolysis - CLARITY). The benefit is evident already few hours after administration and is very effective in preventing ischemic complications before PCI (PCI-CURE, PCI-CLARITY). It is thus concluded that clopidogrel should be administered as early as possible (even in the ambulance) to ACS patients. The additional benefit of clopidogrel to patients who are already treated with "upstream" GP IIb/IIIa antagonists is not clear. Because of the increase rate of bleeding in patients who undergo CABG while on clopidogrel therapy, clopidogrel should probably be avoided from ACS patients in whom the likelihood of CABG is high (only if these patients are treated with "upstream" GP IIb/IIIa antagonists and are scheduled for early coronary angiography).

It is well known from multiple studies that clopidogrel is essential in patients who undergo stent implantation to prevent stent thrombosis (one month for bare stents and 3-6 months in drug eluting stents). The ARMYDA-2 trial demonstrated the advantage of 600mg loading dose, as compared to the standard 300mg, prior to PCI.

It is not entirely clear for how long after ACS (PCI) clopidogrel and aspirin should be administered together? Obviously too aggressive therapy is associated with an increase risk of bleeding complications. The CURE and CREDO studies concluded that for 9-12 months the benefit outweighs the risk. The conclusion from MATCH (patients with TIA or ischemic stroke) is that the bleeding risk is too high so that clopidogrel should not be administered with aspirin. It is hoped that the results from CHARISMA (expected this year) will clarify this issue.

**Summary:** Clopidogrel should be administered as soon as possible to most patients with ACS and those in whom PCI is planned. Loading dose is 600mg (? 300mg after thrombolysis). Treatment should be definitely continued as long as there is a risk for stent thrombosis. Longer duration should be considered in patients with high risk for vascular events and low bleeding risk.

## **What Cardiac Markers & How to interpret and Manage in 2006**

*Dr. Jagdish Hiremath, Pune, India*

Cardiac markers, till date are used in diagnosis of minimal/marked myocardial necrosis. Most such markers could give a semi quantitative assessment as well. As a by-product, prognostication (though crude) was possible.

CPKMB, myoglobin, Troponin T/I fall in this category. Their variability of positivity in blood gives time projection of infarction and serial estimation are more diagnostic (though expensive).

Their routine use in frank ST elevation MI can be debated.

History, ECG and above markers put together gives high sensitivity, specificity and prognosis in a patient of ACS. It also can prompt aggressiveness of therapy.

Markers of ischemia like the new IMA (ischemic modified albumin) is useful to decide whether coronary ischemia took place or not. More often such a marker is required in clinical practice than the markers of necrosis.

Markers of heart failure like BNP are remarkably useful in emergency room to diagnose

cardiac cause of breathlessness. Especially useful when a known airway disease patient presents with breathlessness. BNP also has some prognosis value.

Markers of inflammation (CRP) are not very diagnostic but are more prognostic.

2006 should see arrival of marker of plaque instability (whole blood choline). This can revolutionize approach to multi vessel CAD.

## **How to Manage Heparins, Bivaluridin, Thrombolytics in ACS**

*Prof. Shmuel Banai, Tel-Aviv, Israel*

*Abstract not received*

## **The Endocannabinoid System: A novel Therapeutic Target For the management of Multiple Cardiovascular Risk Factors**

*Dr. Joyal Shah, Ahmedabad, India*

*Abstract not received*

## **Culprit Artery Localization in Acute Coronary Syndromes**

*Dr Ajay Naik, Ahmedabad, India*

Acute Coronary Syndromes (ACS) are almost always the result of pathology in a single culprit vessel. ST elevations and T wave inversions point towards acutely ischemic territory. In contrast, **ST depressions are of poor localizing value.** "Ischemia at a distance" always needs to be considered when analyzing ECGs in ACS.

A large myocardial area is at jeopardy if ECG during angina reveals ST depression in >7 leads, ST elevation is seen in aVR or V1. This occurs in severe **Left Main Coronary Artery (LMCA) stenosis or severe Triple Vessel Disease (TVD).**

If ECG during in absence of angina reveals symmetrical deep T inversions in V2 – 4 that become upright during pain, it is a pointer towards **critical Proximal LAD stenosis.**

In **Acute Myocardial Infarction (AMI)**, the ECG may be normal in upto 10% of patients, especially with posterior wall involvement. Hence serial ECGs and extended leads V7 – 9, V4R should be obtained when clinical suspicion is high.

In **Anterior Wall MI**, the diagnosis of LAD occlusion is made from **ST elevation in precordial leads**, whereas the site of LAD segment involved can be obtained by analyzing combinations of ST elevation and depression in limb leads aVR, aVL and aVF. If LAD occlusion occurs proximal to first septal artery, ST elevation is seen in aVR. If it is proximal to first diagonal, ST elevation is seen in aVL. *ST elevation* can occur in inferior leads if mid or distal vessel occurs in a type A LAD.

**Inferior Wall MI** can occur due to RCA as well as LCx occlusion; these can be differentiated by analyzing **ST changes in leads II, III, V5-6 and V4R.** It is important not to miss RV and posterior wall infarction. The significance of ST depression in anterior leads during IWMI should be carefully analyzed. **Arrhythmias** during MI need to be recognized and analyzed.

ECG signs are an important clue to **prognosis of ACS**. The sensitivity and specificity ranges from 60 - 90 %. Anatomic variations and previous coronary events will modify the correlation.

## **Heat, Shock, Battery, Maze - Tools of the trade**

***Dr. Ajay Naik / Prakash Kamath***  
***Abstract not received***

## **Quizzing the Masters - Interactive ECG Sessions**

***Dr. Ajay Naik / Prakash Kamath***  
***Abstract not received***

## **What is the basic Echo/Doppler Evaluation of Diastolic Function ?**

***Dr. (Col.) S. Parashar, New Delhi, India***  
***Abstract not received***

## **Advanced Evaluation of LV Wall Motion Analysis**

***International / National Faculty***  
***Abstract not received***

## **Echo Assessment of Mitral Valve**

***Prof. Marisa Di Donato, Firenze, Italy***

Functional mitral regurgitation (MR) "broadly" denotes abnormal function of normal leaflets in the context of depressed ventricular function. It typically occurs in globally dilated ventricles or with regional abnormal motion that affects valve closure. It occurs in about 20 to 30% of pts following myocardial infarction (MI) and in 50% of those presenting with congestive heart failure. In this setting MR is frequently mild but conveys adverse prognosis doubling mortality after MI, in chronic heart failure and after surgical or percutaneous coronary revascularization. A graded relationship exists between severity and reduced survival. It is therefore extremely important to assess its presence and severity and to understand its mechanisms in view of treatment options. The equilibrium position of the mitral leaflets is determined by the balance of forces acting on them, including annular and papillary muscle tethering forces and LV-generated closing forces.

Normally, little force is required to close the thin leaflets, so MR is not produced by global dysfunction without tethering. However, once tethering is increased, leaflet closure is further impaired when less force is available to oppose tethering. Other components of mitral functioning are the driving forces given by the difference between the LV and the left atrial systolic pressures, annular dilatation and contraction and annulus tethering. All the components may change under different loading conditions making MR a dynamic lesion. Two dimensional and more recently the three dimensional echocardiography as well as the transesophageal approach have the potential of assessing the exact mechanisms leading to MR and help in tailoring the ideal combination of annular, ventricular and chordal approaches to achieve the best result in each patient.

The following measurements need to be done by parasternal long axis, two and four chamber views in order to define the subvalvular mitral geometry: Annular size, height of coaptation point (distance between the annulus and the point of coaptation), tenting area (area between the annulus and the closed leaflets), papillary muscle tethering (distance between the head of the papillary muscle and the mitral annulus) evidence of restricted motion, distance between the two papillary muscles (in short axis view). MR should also graded with the PISA method to calculate the effective regurgitant orifice or with vena contracta measure or with the ratio between regurgitant area and left atrium area. Provocative tests should also be performed such as: exercise test, dobutamine test or others in order to exactly quantify the severity of MR which can be masked by loading conditions and/or by LV dysfunction.

Therapy should therefore focus on reducing the geometric culprits. Among the several surgical approaches to reduce MR (mitral annuloplasty, posterior suture, scar resection with papillary muscle re-implantation, infarct plication, percutaneous annular reduction, constraint devices, re-synchronization therapy etc) we report our experience with ventricular patch reconstruction which by addressing remodeling restores a more physiologic shape and leads to an improved subvalvular mitral geometry and to a better functioning of the mitral valve.

## **Echo Assessment of Aortic Valve**

***International / National Faculty***  
***Abstract not received***

### **Methods to identify and manage individuals in Stages A and B pre-HF**

***Dr. Milan Chag, Ahmedabad, India***  
***Abstract not received***

### **Chronic Heart Failure in the Adult 2006: What is the future ?**

***Dr. Anil Jain, Ahmedabad, India***  
***Abstract not received***

### **Systemic Hypertension - What drugs & how to dose in 2006**

***Dr. Satya Gupta, Paris, France***

Evidence based medical practice is the hallmark of cardiac practice worldwide. Clear guidelines are available for almost all cardiac investigation and treatment, and these guidelines are based in part on the available information from clinical trials . Since the publication of JNC VI guideline for treatment of hypertension in 1997, several new landmarks trial have published which gave new insight for the management of systemic hypertension. To incorporate the results of these trails in the clinical practice, JNC<sup>1</sup> provided a new guideline in 2003. British Hypertension Society<sup>2</sup> (BSH) and European Society of Cardiology<sup>3</sup> (ESC) also provided a clear guideline for management of systemic hypertension. Whatever may be the guideline, the basic purpose of all guidelines is optimal control of blood pressure and reduction of cardiovascular risk. Here I have highlighted the key points of JNC VII guideline. Its worth reading full article of JNC VII as well as other guideline (BSH and ESC ) for

those physician who deals hypertensive patients and encounter challenging cases in day to day practice.

**Salient Features of JNC VII:**

1. In persons older than 50 years, systolic BP of more than 140 mmHg is a much more important cardiovascular disease risk(CVD) factor than diastolic BP.
2. The risk of CVD, beginning at 115/75 mm Hg, doubles with each increment of 20/10 mm Hg; individuals who are normotensive at 55 years of age have a 90% lifetime risk for developing hypertension.
3. Individuals with a systolic BP of 120 to 139 mm Hg or a diastolic BP of 80 to 89 mm Hg should be considered as prehypertensive and require health-promoting lifestyle modifications to prevent CVD.
4. Thiazide-type diuretics should be used in drug treatment for most patients with uncomplicated hypertension, either alone or combined with drugs from other classes. Certain high-risk conditions are compelling indications for the initial use of other antihypertensive drug classes (angiotensin-converting enzyme inhibitors, angiotensin-receptor blockers,  $\beta$ -blockers, calcium channel blockers).
5. Most patients with hypertension will require 2 or more antihypertensive medications to achieve goal BP (<140/90 mm Hg, or <130/80 mm Hg for patients with diabetes or chronic kidney disease).
6. If BP is more than 20/10 mm Hg above goal BP, consideration should be given to initiating therapy with 2 agents, 1 of which usually should be a thiazide-type diuretic.
7. The most effective therapy prescribed by the most careful clinician will control hypertension only if patients are motivated.

In presenting these guidelines, the committee recognizes that the responsible physician’s judgment remains paramount.

**JNC VII Classification of BP:**

<b>BP Classification</b>	<b>Systolic BP(mmHg)</b>		<b>Diastolic BP(mmHg)</b>
Normal	<120	and	<80
Prehypertension	120-139	or	80-89
Stage 1 hypertension	140-159	or	90-99
Stage 2 hypertension	> 160	or	> 100

**Treatment:**

**1. Goal:**

- For all patients < 140/90 mmHg
- For patients with diabetes and renal disease < 130/80 mmHg

**2. Life style modification:**

Recommended for all patients and should be encouraged in patients with normal BP.

Component of life style modification

1. Diet rich in fruits and green vegetables.
2. Dietary sodium reduction
3. Quiet Smoking
4. Moderate exercise and weight reduction if obese
5. Avoid Alcohol
6. Stress free life

### 3. Pharmacological Treatment:

#### JNC VII Recommended Treatment:

Hypertension Stage	Lifestyle Modification	Initial Drug Therapy	
		Compelling indication No	Yes
Normal	Encourage	-	-
Prehypertension	Yes	No drug	Drugs for compelling indications
Stage 1 hypertension	Yes	Thiazide –type diuretics for most; may consider ACEI, ARB, B-blocker, CCB or combination	Drugs for compelling indications, Other drugs (diuretics, ACEI, ARB, B-blocker, CCB) needed
Stage 2 hypertension	Yes	2-drugs combination for most( Usually thiazide and ACEI or ARB or B-blocker or CCB)	Drugs for compelling indications, Other drugs (diuretics, ACEI, ARB, B-blocker, CCB) as needed

Excellent clinical trial outcome data prove that lowering BP with several classes of drugs, including ACE inhibitors, angiotensin-receptor blockers (ARBs),  $\beta$ -blockers, calcium channel blockers (CCBs), and thiazide-type diuretics, will all reduce the complications of hypertension.

Thiazide-type diuretics have been the basis of antihypertensive therapy in most outcome trials. In these trials, including the recently published Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT), diuretics have been virtually unsurpassed in preventing the cardiovascular complications of hypertension.

The exception is the Second Australian National Blood Pressure trial (ANBP2) that reported slightly better outcomes in white men with a regimen that began with an ACE inhibitor compared with one starting with a diuretic. Diuretics enhance the antihypertensive efficacy of multidrug regimens, can be useful in achieving BP control, and are more affordable than other antihypertensive agents.

Thiazide-type diuretics should be used as initial therapy for most patients with hypertension, either alone or in combination with 1 of the other classes (ACE inhibitors, ARBs,  $\beta$ -blockers, CCBs) demonstrated to be beneficial in randomized controlled outcome trials. If a drug is not tolerated or is contraindicated, then 1 of the other classes proven to reduce cardiovascular events should be used instead.

### 4. Compelling Indications and Treatment:

Patients with hypertension and certain co-morbidities requires special attention and follow-up by the clinician. The drug selections for these compelling indications are based on favorable outcome data from clinical trials. Combination of agents may be required. Other management considerations include medications already in use, tolerability, and desired BP targets. In many cases, specialist consultation may be indicated.

## Recommended Treatment for Compelling Indications:

High risk conditions with compelling indication	Recommended Drugs	Clinical Trial Basis
Heart Failure	Diuretic,B-blocker,ACEI, ARB,Aldosterone antagonist	ACC/AHA HF guideline, MERIT-HF,COPERNICUS,CIBIS, SOLVD,AIRE,TRACE,ValHEFT, RALES
Post-myocardial Infarction	B-blocker,ACEI,Aldosterone antagonist	ACC/AHA Post-MI guideline BHAT,SAVE,CAPRICON, EPHEBUS
High coronary disease risk	Diuretic,B-blocker,ACEI,CCB	ALLHAT,HOPE,ANBP2,LIFE, CONVINCENCE
diabetes	B-blocker,ACEI,ARB,CCB	NKF- Guideline,ALLHAT,UKPDS
Chronic kidney disease	ACEI,ARB	NKF Guideline,Captopril Trial,RENAAL,IDNT,REIN,AASK
Recurrent stroke prevention	Diuretic,ACEI	PROGRESS

### 5. Dose of Drugs and its Titration:

Most patients with hypertension will require 2 or more antihypertensive medications to achieve their BP goals. Addition of a second drug from a different class should be initiated when use of a single drug in adequate doses fails to achieve the BP goal. When BP is more than 20/10 mm Hg above goal, consideration should be given to initiating therapy with 2 drugs, either as separate prescriptions or in fixed-dose combinations. The initiation of drug therapy with more than 1 agent may increase the likelihood of achieving the BP goal in a more timely fashion, but particular caution is advised in those at risk for orthostatic hypotension, such as patients with diabetes, autonomic dysfunction, and some older persons. Use of generic drugs or combination drugs should be considered to reduce prescription costs.

### 6. Patients follow up and monitoring:

Once antihypertensive drug therapy is initiated, most patients should return for follow-up and adjustment of medications at approximately monthly intervals until the BP goal is reached. More frequent visits will be necessary for patients with stage 2 hypertension or with complicating comorbid conditions. Serum potassium and creatinine should be monitored at least 1 to 2 times per year. After BP is at goal and stable, follow-up visits can usually be at 3- to 6-month intervals. Comorbidities, such as HF, associated diseases, such as diabetes, and the need for laboratory tests influence the frequency of visits. Other cardiovascular risk factors should be treated to their respective goals, and tobacco avoidance should be promoted vigorously. Low-dose aspirin is increased in patients with uncontrolled hypertension.

### 7. References:

1. Aram V. Chobanian; George L. Bakris; Henry R. Black; William C.ushman; Lee A. Green; Joseph L. Izzo Jr; Daniel W. Jones; Barry J. Materson; Suzanne Oparil; Jackson T. Wright Jr; Edward J. Roccella. **The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: The JNC 7 Report.** *JAMA.* 2003;289:2560-2571.
2. Bryan Williams, Neil R Poulter, Morris J Brown, Mark Davis, Gordon T McInnes, John F Potter, Peter S Sever, Simon McG Thom. British Hypertension Society guideline for

- hypertension management 2004(BHS-IV). BMJ 2004;328:634-640.
3. Guidelines Committee. 2003 European Society of Hypertension-European Society of Cardiology guidelines for the management of arterial hypertension. Journal of Hypertension 2003;21:1011-1053.

## **Current Overview of FDA & CE approved DES (Drug Eluting Stents) & future of DES**

*Dr. M. S. Hiremath, Pune, India*  
*Abstract not received*

## **Understanding the Vulnerable Plaque - Role of VH-IVUS**

*Dr. Anuja Nair, Cleveland, U.S.A.*  
*Abstract not received*

## **How to manage Cardiovascular Disease in the Elderly ?**

*Prof. Nakul Sinha, Lucknow, India*  
*Abstract not received*

# 3-C Con 2006

Sunday 12<sup>th</sup> February 2006- DAY-2

**BREAKFAST (7:30 AM to 8:00 AM)**

## Novelties and Advances in Cardiac Arrhythmia (8:00 AM to 9:25 AM)

### Hall-A

**Chair: Dr. Anish Chandarana, Dr. Hemang Baxi**

**Moderator: Dr. Prakash Kamath, Dr. Ajay Naik**

- 08.00 AM ECG, Arrhythmias  
*All Faculty*
- 08.30 AM Ablation Therapy in Arrhythmia  
*Dr. Ajay Naik, Ahmedabad, India*
- 08.45 AM Drug Therapy in Arrhythmia  
*Dr. Prakash Kamath, Cochin, India*
- 09.00 AM Device Therapy in Arrhythmia  
*Dr. Ajay Naik, Ahmedabad, India*

### Hall-B

**Chair: Dr. Urmil Shah**

**Moderator: Dr. Chirag Mehta**

**PLENARY LECTURE - The Role of the Renin - Angiotensin Aldosterone System in Atherosclerosis & Atherothrombosis - Prof. Shmuel Banai, Tel-Aviv, Israel (9:20 AM TO 9:40 AM)**

## Novelties and Advances in ACS & CAD (A) (9:40 AM TO 10:40 AM)

### Hall-A

**Chair : Prof. S. Kaushik, Dr. Keyur Parikh**

**Moderator: Dr. P.C. Manoria, Dr. Urmil Shah**

- 09.40 AM Vulnerable Plaque: What all Physicians should know about Virtual Histology in 2006 ?  
*Dr. Anuja Nair, Cleveland, U.S.A*
- 09.55 AM Role of Echo Cardiography in Evaluation in Acute Chest Pain/ACS  
*Dr. S. K. Parashar, New Delhi, India*
- 10.10 AM Intricacies and complexities in use of Oral hypoglycaemic drugs in diabetics with CAD  
*Dr. P. C. Manoria, Bhopal, India*
- 10.20 AM An Update In Management of ACS  
*Dr. Anish Chandarana, Ahmedabad, India*

### Hall-B

**Chair : Dr. Gunvant Patel**

**Moderator : Dr. Mihir Tanna**

### Hall-A

**Chair: Prof. Shmuel Banai, Dr. Milan Chag**

**Moderator: Dr. Bharat Shah, Dr. Vishal Gupta**

### Hall-B

**Chair :Dr. Hemang Baxi**

**Moderator : Dr. Chirag Mehta**

**Inauguration with unique interactive question & answer session (10:40 AM TO 11:30 AM)**

**Mini Plenary Lecture-Stress Management for Physicians - Dr. Jagdish Hiremath, Pune, India (11:30 AM TO 11:50 AM)**

**Mini Plenary Lecture - Quality of services in Health Sector - What Physicians should do ? Prof. Dileep Mavalankar, Ahmedabad, India (11:50 AM TO 12:10 PM)**

**Global Challenges in health care. Are we prepared? Mr. Tony Parmar (12:10 PM to 12:25 PM)**

## Novelties and Advances in ACS & CAD (B) (12:25 PM TO 1:15 PM)

- 12.25 PM AMI Adjunct Therapy  
*Dr. Bharat Shah, California, U.S.A*
- 12.40 PM PCI following Thrombolysis  
*Dr. M. S. Hiremath, Pune, India*
- 12.55 PM Cox-2 Inhibitors and heart-What to do in 2006?  
*Dr. P. C. Manoria, Bhopal, India*

**PLENARY LECTURE - Approach to therapy of diabetic patients with Cardiovascular Disease -  
Prof. Yoseph Rozenman, Tel-Aviv, Israel (1:15 PM TO 1:35 PM)**

**LUNCH (1.35 PM TO 2.05 PM)**

**Hall-A**

**Chair: Prof. Yoseph Rozenman, Dr. Anish Chandarana**

**Moderator: Dr. Ashish Parikh, Dr. Bharat Trivedi**

**Hall-B**

**Chair : Dr. Anish Chandarana**

**Moderator : Dr. Guntant Patel**

**Novelties and Advances in Betablocker in Heart Failure (2:05 PM TO 2:20 PM)**

02.05 PM Critical Survey of Trials of Beta Blockers in Heart Failure  
*Prof. Finn Waagstein, Sweden*

**Novelties and Advances in Everyday "Real Life Cases" (2:20 PM TO 4:35 PM)**

02.20 PM Heart Failure  
*Dr. Keyur Parikh, Ahmedabad, India*

02.30 PM Chronic Stable Angina : No Options Therapy  
*Dr. Keyur Parikh, Ahmedabad, India*

02.40 PM Cardiac Surgery  
*Dr. Vishal Gupta, Ahmedabad, India*

02.55 PM Renovascular Hypertension  
*Dr. Ashish Parikh, Delaware, U.S.A*

03.10 PM Acute Coronary Syndrome  
*Prof. Yoseph Rozenman, Tel-Aviv, Israel*

03.25 PM Geriatric (Elderly) Heart Disease  
*Dr. Shmuel Banai, Tel-Aviv, Israel*

03.40 PM Cardiogenic Shock  
*Dr. Hemang Baxi, Ahmedabad, India*

03.55 PM Ischemic Stroke  
*Dr. Ashish Parikh, Delaware, U.S.A*

04.10 PM "Take home message" from recent  
to trials of dyslipidemia

04.25 PM *Dr. Milan Chag, Ahmedabad, India*

**REFRESHMENT BREAK (4:35 PM TO 4:45 PM)**

**New - Novelties and Advances with Interactive Question & Answer Sessions  
"Real Life Cases from the World" (4:45 PM to 6:00 PM)**

**Hall-A**

**Chair: Dr. Milan Chag, Dr. Anish Chandarana**

**Moderator: Dr. Urmil Shah, Dr. Hemang Baxi**

► Review of more interesting ECGs, Arrhythmias, Chest X-Rays, Echocardiograms from Real World By The International Faculty.....An Experience of Lifetime.

- ◆ *Prof. Shmuel Banai, Tel-Aviv, Israel*
- ◆ *Prof. Marisa Di Donato, Firenze, Italy*
- ◆ *Dr. Saibal Kar, Los Angeles, U.S.A.*
- ◆ *Dr. Raj Makkar, Los Angeles, U.S.A.*
- ◆ *Dr. Anuja Nair, Cleveland, U.S.A.*
- ◆ *Dr. Ashish Parikh, Delaware, U.S.A.*
- ◆ *Prof. Yoseph Rozenman, Tel-Aviv, Israel*
- ◆ *Dr. Bharat Shah, California, U.S.A.*
- ◆ *Prof. Finn Waagstein, Sweden*

**Hall-B**

**Chair: Dr. Joyal Shah**

**Moderator : Dr. Mihir Tanna**



**CONCLUSION (6:00 PM)**

## Morning Session : 8.15 AM to 12.05 PM

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### ECG, Arrhythmias

*All Faculty*

*Abstract not received*

### Ablation Therapy in Cardiac Arrhythmias

*Dr Ajay Naik, Ahmedabad, India*

**Catheter based therapy** has revolutionized the management of Cardiac Arrhythmias. Over the past two decades, continuous refinement in mapping technology and Ablative therapy has promised permanent cure to patients with Cardiac arrhythmias.

**Radiofrequency Ablation** is the treatment of choice for **Supraventricular Tachycardia (SVT), WPW syndrome and AVRT mediated by Accessory Pathways, AV Nodal Reentrant Tachycardia (AVNRT), Atrial Flutter and Atrial Tachycardia** have a high success rate of ablation therapy. Over the past few years, even Atrial Fibrillation is being managed with ablation therapy in the Pulmonary veins and Left Atrium.

**Ventricular Tachycardia (VT)** occurring in structurally normal hearts can be due to **RVOT tachycardia (RVOTT) or Idiopathic Left Ventricular Tachycardia (ILVT)**. These can be cured in >90% of patients. **VT occurring in scars** due to Coronary Artery Disease and other myocardial diseases can also be treated with a reasonable (50 – 80%) success rates.

**3-dimensional mapping techniques** such as **Carto Biosense, RPM and Navix systems; non-contact mapping by Endocardial Solutions technique** are a major advance in treatment of complex arrhythmias.

**Cryoablation** holds much promise in treatment of arrhythmias occurring from foci in close proximity to critical anatomical and conduction system regions. **Microwave energy** is also in early clinical studies for arrhythmia therapy.

**Stereotaxis and Robotic therapy** are exciting frontiers being explored. In future, cardiac arrhythmias will be treated by remote steering of catheters by computers and manipulating them on 3-dimensional consoles in the control room of the catheterization laboratory.

### Drug Therapy in Arrhythmia

*Dr. Prakash Kamath, Cochin, India*

*Abstract not received*

### Device Therapy in Arrhythmias

*Dr Ajay Naik, Ahmedabad, India*

**Implantable Devices** were a major paradigm shift in cardiac therapy from their first introduction during the 1950s.

**Permanent Pacemakers** represented the first devices implanted to treat cardiac arrhythmias. Over the past 5 decades pacemakers have evolved from simple ventricular demand pacing to **Dual Chamber and Multisite Pacemakers** with prophylactic and therapeutic capabilities for cardiac arrhythmias.

**Automatic Implantable Cardioverter Defibrillators (AICD)** are implanted to detect and treat malignant ventricular arrhythmias in patients who have survived or are at risk of Sudden Cardiac Death.

**Cardiac Resynchronization Therapy (CRT)** is a device with triple chamber pacing capability (RA, RV and LV; biventricular pacemaker). This remarkable device has resulted in tremendous improvement in Quality of Life and reduction in hospitalizations, morbidity and mortality of patients with Congestive Heart Failure in NYHA class III and IV.

**Cardiac Resynchronization Therapy with Defibrillator (CRT-D)** holds promise for further reduction in mortality of patients with CHF. Patients who have improved from NYHA class III and IV HF and those who are in NYHA class II are at greatest risk of Sudden Cardiac Death (SCD) due to ventricular arrhythmias. These patients are likely to benefit by add-on defibrillator function.

**Implantable Loop Recorder (ILR)** is the final resort in diagnosis of infrequent but serious episodes of syncope and palpitations in selected patients.

The ever-growing repertoire of **Implantable Cardiac Devices** is revolutionizing the management of Cardiac Arrhythmias.

## **The Role of the Renin - Angiotensin Aldosterone System in Atherosclerosis & Atherothrombosis**

*Prof. Shmuel Banai, Tel-Aviv, Israel*  
*Abstract not received*

## **Vulnerable Plaque: What all Physicians should know about Virtual Histology in 2006 ?**

*Dr. Anuja Nair, Cleveland, U.S.A*  
*Abstract not received*

## **Role of Echo Cardiography in Evaluation in Acute Chest Pain/ACS**

*Dr. S. K. Parashar, New Delhi, India*  
*Abstract not received*

## **Intricacies and complexities in use of Oral hypoglycaemic drugs in diabetics with CAD**

*Dr. P. C. Manoria, Bhopal, India*  
*Abstract not received*

# **An Update In Management of ACS**

***Dr. Anish Chandarana, Ahmedabad, India***

Atherosclerotic Coronary Artery Disease (CAD) has been a serious disease affecting almost all populations all over the globe. Out of all patients presenting with Acute Coronary Syndrome (ACS), which is a more serious form of CAD, nearly there-quarter have either Unstable Angina (UA) or Non ST Elevation Myocardial Infarction (NSTEMT). UA / NSTEMT is usually associated with sever subtotal coronary obstruction. Among patients with UA / NSTEMT, approximately half have evidence of myocardial necrosis with elevated serum cardiac markers.

Exact understanding of path physiology of ACS has led to better and more precise treatment strategies. Because UA / NSTEMT is a syndrome and not a disease, patients vary widely in their mode of presentation, precipitating factors, associated illnesses and so the risk imposed by this condition. It is now very well accepted that treatment strategy should be formed in view of risk: higher the risk, more aggressive the treatment. Advanced age, presence of Diabetes, history of prior Myocardial Infarction / cerebrovascular disease / peripheral vascular disease, rest angina, hypotension, heart failure, serious ventricular arrhythmia, certain ECG changes, elevated serum levels of cardiac markers, presence of thrombus or three vessel diseases on angiogram are some of the important risk factors. Their presence gives an individual higher risk of death or STEMI in short and long term.

Based on variety of clinical research it is very clear that each such patient, after confirming diagnosis, should receive Aspirin, Unfractionated Heparin or Enoxaparine, Beta-Blockers, Nitrates and Clopidogrel. Further line of treatment should be based on magnitude of risk a given patient has. With introduction of intervention for revascularization (Percutaneous Coronary Intervention-PCI or Coronary Artery Bypass Graft Surgery-CABG), a debate started about what is the optimal treatment strategy for such Patient- early invasive or early conservative where invasive approach is reserved for patients with recurrent severe angina or those with high-risk stress test. Several randomized trials have assessed the relative merits of these two strategies. Most of the recent trials have suggested reduction in rates of MI and / of death at 6 – 12 months post event if early invasive strategy is accepted, particularly in intermediate to high risk patient subsets. Low risk patients do equally well with either strategy. The answer to the question 'how early' is not very clear; but available data suggests it to be within first 48 hours of presentation.

Variety of new medicines have found very important place in management of UA / NSTEMI. Glycoprotein IIb / IIIa receptor inhibitors have been studied extensively and have showed significant reduction in death, MI or severe refractory ischemia in short term and death or reMI in intermediate / long term. Though there are some differences; all three IV antiplatelet agents- abciximab, eptifibatide, tirofiban-have shown benefits in patients managed either with medicines only or by early intervention. In addition it has been seen that earlier initiation of this agent leads to more benefits. Use of this agent has received class I recommendation in patients undergoing cardiac catheterization and PCI; while use of eptifibatide or tirofiban is class IIa recommendation in high risk patients kept on medicines only. Direct thrombin inhibitors are another new class of drugs under research.

In addition to this, lifestyle changes and pharmacological interventions to modify variety of CAD risk factors like diabetes, high blood pressure, high lipids, tobacco use etc. is must. Proper use of cardiac rehabilitation programme is very rewarding.

## **Stress Management for Physicians**

***Dr. Jagdish Hiremath, Pune, India***  
***Abstract not received***

## **Quality of services in Health Sector - What Physicians should do ?**

***Prof. Dileep Mavalankar, Ahmedabad, India***

Quality is rapidly emerging as an important competitive tool in business and industry. The same thing is happening in health. The customers, regulators, insurance companies and professionals are becoming more and more demanding and quality conscious. Higher the quality, lesses the complications and more the satisfaction of the patients. Higher quality also allows for attractiing high paying clients. This presentation will provide a few key concepts of quality of services in health sector. These concepts are taken from wider management literature on quality of service sector, which includes not only health but also other sector. The presentation will bring out key lessons which physicians can implement at three levels - in their individual practices, as head of small to medium hospitals and as heads of large and growing hospitals and health systems. The presentation will show the distinction between technical and quality of services. The presentation will largely focus on quality of services as application in the health sector.

## **Global Challenges in health care. Are we prepared?**

***Mr. Tony Parmar***  
***Abstract not received***

## **Pre Lunch Session : 12.25 PM to 1.35 PM**

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### **AMI Adjunct Therapy**

***Dr. Bharat Shah, California, U.S.A***

***Abstract not received***

### **PCI following Thrombolysis**

***Dr. M. S. Hiremath, Pune, India***

***Abstract not received***

### **Cox-2 Inhibitors and heart-What to do in 2006?**

***Dr. P. C. Manoria, Bhopal, India***

***Abstract not received***

### **Approach to therapy of diabetic patients with Cardiovascular Disease**

***Yoseph Rozenman, Tel-Aviv, Israel***

Vascular disease progresses silently in patients with diabetes and epidemiological studies showed convincingly that the risk of vascular complications of diabetics without vascular disease is equivalent to that of nondiabetics with established disease. Among those with established disease diabetics are high risk subset that should be treated aggressively. The central dogma is that therapies which are effective in patients with CAD are also effective in the high risk diabetic subset. Since the relative risk reduction by therapy is similar in diabetics and nondiabetics the absolute benefit is higher in diabetics.

Aggressive medical therapy is essential to slow the rate of disease progression and should include antihypertensive and antihyperlipidemic therapy (low target goals). Patients should also be treated with antiplatelet agents, ACE inhibitors (ARB's). HbA1C should be use to titrate therapy for hyperglycemia (which should probably include "glitazones). Patients with ACS (both ST and non ST elevation) should be treated with early catheterization and revascularization with a liberal use of GP IIb/IIIa antagonists.

There is still an ongoing debate regarding the optimal method of revascularization: PCI versus CABG. This debate started with the publication of the outcome of diabetics enrolled in the BARI trial (higher mortality in PCI as compared to CABG). Critical analysis suggests that this difference in mortality is only partly explained by restenosis. The major difference is in the clinical significance of disease progression in patients after PCI as compared to those after CABG. Drug eluting stents virtually eliminate restenosis in nondiabetics and are also very effective in diabetic patients. Aggressive medical therapy is highly effective to prevent disease progression and should be given to all diabetic patients regardless of revascularization. Ongoing trials, BARI-II and FREEDOM will clarify the role of revascularization in diabetics, the preferred approach to glycemic control (insulin sensitization or insulin provision) and the impact of drug eluting stents on the outcome of PCI as compared to CABG.

## Afternoon Session : 2.05 PM to 4.35 PM

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### Critical Survey of Trials of Beta Blockers in Heart Failure

*Prof. Finn Waagstein, Sweden*

*Abstract not received*

### Heart Failure

*Dr. Keyur Parikh, Ahmedabad, India*

First-in-man study using the REMON Im Pressure TM ultrasonic intravascular sensor to measure Pulmonary Artery Pressure in patients with Chronic Congestive heart failure

#### **Background**

This scientific trial was conducted to help monitor patients of Congestive Heart Failure (CHF). CHF is a gradually progressive disease. It is very important to monitor how far the heart is damaged by CHF. Understanding the extent of heart damage in CHF decides the therapy. Knowing the pressure within the Pulmonary Artery (PA) is an important parameter for monitoring CHF patients.

- ▶ **The Goal:** Reduction in hospitalization by early prediction of decompensation at home.
- ▶ **The Solution:** The Remon ImPressure TM implant enables
  - frequent,
  - non-invasive
  - home monitoring of the PA pressure. PA diastolic is used as a surrogate for LV filling pressure (LVEDP)

#### **Implant**

- ▶ Miniature (3.0x2.4x17mm)
- ▶ Leadless
- ▶ Long term durability
- ▶ All Titanium Encapsulation
- ▶ Direct measurement of PA pressure waveform

Implantation Procedure with real life cases will be discussed with movie presentation

### Chronic Stable Angina : No Options Therapy

*Dr. Keyur Parikh, Ahmedabad, India*

Despite maximal available therapies, number of patients with CAD remains severely symptomatic with no suitable treatment

Patients with symptomatic coronary artery disease who are not candidates or are at high risk for revascularization procedures with either CABG or PCI need newer approaches to improve their quality of life.

These "no option" patients with refractory angina pectoris include patients with

- ▶ diffuse or distal coronary atherosclerosis
- ▶ small vessel coronary disease
- ▶ and patients with failure of previous revascularization procedures

Experimental data provide evidence that increased CS pressure causes redistribution of collateral blood flow into ischemic territories of the myocardium such redistribution of arterial blood significantly reduces

- ▶ **myocardial ischemic damage and**
- ▶ **infarct size**

In the obstructive CAD, by increasing CS pressure, perfusion of the myocardium in ischemic areas will be enhanced, and consequently hemodynamic parameters will improve  
A stent like device, designed to establish a permanent narrowing of the coronary sinus. The Reducer is implanted using a percutaneous trans-venous approach, of an over the-wire, balloon expandable system

Case examples of real cases and summary will be discussed.

## **Cardiac Surgery**

***Dr. Vishal Gupta, Ahmedabad, India***

1. **Endomyocardial Fibrosis** presenting as multiple episodes of giddiness & syncope. Patient was evaluated in form of CT & MRI & he changed a couple of Neurologist with no relief of symptoms. Finally wisdom prevailed & he was referred to a Cardiologist & Echo evaluation showed cardiac(LV) tumor. To our surprise, intraoperatively it was endomyocardial fibrosis & resection of fibrotic rind was done.
2. **Unruptured Sinus of Valsalva Aneurysm(SOVA):**  
A 28- year old man presented with multiple episodes of Ventricular Tachycardia. Echo evaluation showed tumor in LV wall, which on further evaluation by MRI showed unruptured SOVA burrowing in to LV wall with calcification. It was repaired with Gortex patch.
3. **Post MI, Severe MR & impending LV rupture :**  
A 44-year-old male came to hospital in Cardiogenic Shock. Patient was put on Ventilator & Intraaortic Balloon support was given. Trans Esophageal Echocardiography (TEE) showed severe Mitral Regurgitation with aneurismal dilatation of lateral wall & increase in interpapillary muscle area. Coronary Angiography showed normal LDA, Normal RCA & LCX total occlusion. Intraoperative Ring Annuloplasty & Linear Repair of lateral wall was done.  
Post correction intraoperative Transesophageal Echocardiography (TEE) showed mild MR. IAB removed on 3<sup>rd</sup> post-operative day & he was extubated on 5<sup>th</sup> day.
4. **Dissection of ascending aorta extending to aortic valve causing severe AR :**  
A 34-year-old non-hypertensive patient presented with sudden breathless. No history of Rheumatic Heart Disease. Echo showed severe AR with LV dysfunction(EF: 25%). CT angio showed aortic dissection extending from ascending aorta to aortic bifurcation (Stanford A) involving the aortic valve.

Emergency Beentall Procedure (Aortic root replacement with coronary implant) was done.

## 5. **LV Aneurysm :**

A 55-year old patient admitted with symptoms of Congestive Heart Failure(CHF). Patient had PTCA to LAD one year back. Echo showed large apicoanterior aneurysm with LVEF of 10% . Coronary Angiography showed normal LCX, normal RCA & significant in-stent restenosis in LAD. Intraoperatively, IVS & anterior wall was scarred, dyskinetic with 5\*4\*4 cm aneurysm. SVR was done & LV sized to 90 cc. Patient was extubated on 3<sup>rd</sup> day & discharged on 10<sup>th</sup> day.

Atherosclerotic Coronary Artery Disease (CAD) has been a serious disease affecting almost all populations all over the globe. Out of all patients presenting with Acute Coronary Syndrome (ACS), which is a more serious form of CAD, nearly three-quarter have either Unstable Angina (UA) or Non ST Elevation Myocardial Infarction (NSTEMI). UA / NSTEMI is usually associated with severe subtotal coronary obstruction. Among patients with UA / NSTEMI, approximately half have evidence of myocardial necrosis with elevated serum cardiac markers.

Exact understanding of pathophysiology of ACS has led to better and more precise treatment strategies. Because UA / NSTEMI is a syndrome and not a disease, patients vary widely in their mode of presentation, precipitating factors, associated illnesses and so the risk imposed by this condition. It is now very well accepted that treatment strategy should be formed in view of risk: higher the risk, more aggressive the treatment. Advanced age, presence of Diabetes, history of prior Myocardial Infarction / cerebrovascular disease / peripheral vascular disease, rest angina, hypotension, heart failure, serious ventricular arrhythmia, certain ECG changes, elevated serum levels of cardiac markers, presence of thrombus or three vessel disease on angiogram are some of the important risk factors. Their presence gives an individual higher risk of death or STEMI in short and long term.

Based on variety of clinical research it is very clear that each such patient, after confirming diagnosis, should receive Aspirin, Unfractionated Heparin or Enoxaparin, Beta-Blockers, Nitrates and Clopidogrel. Further line of treatment should be based on magnitude of risk a given patient has. With introduction of intervention for revascularization (Percutaneous Coronary Intervention-PCI or Coronary Artery Bypass Graft Surgery-CABG), a debate started about what is the optimal treatment strategy for such Patient- early invasive or early conservative where invasive approach is reserved for patients with recurrent severe angina or those with high-risk stress test. Several randomized trials have assessed the relative merits of these two strategies. Most of the recent trials have suggested reduction in rates of MI and / of death at 6 – 12 months post event if early invasive strategy is accepted, particularly in intermediate to high risk patient subsets. Low risk patients do equally well with either strategy. The answer to the question 'how early' is not very clear; but available data suggests it to be within first 48 hours of presentation.

Variety of new medicines have found very important place in management of UA / NSTEMI. Glycoprotein IIb / IIIa receptor inhibitors have been studied extensively and have showed significant reduction in death, MI or severe refractory ischemia in short term and death or reMI in intermediate / long term. Though there are some differences; all three IV antiplatelet agents- abciximab, eptifibatid, tirofiban-have shown benefits in patients managed either with medicines only or by early intervention. In addition it has been seen that earlier initiation of this agent leads to more benefits. Use of this agent has received class I recommendation in patients undergoing cardiac catheterization and PCI; while use of eptifibatid or tirofiban is class IIa recommendation in high risk patients kept on medicines only. Direct thrombin inhibitors are another new class of drugs under research.

In addition to this, lifestyle changes and pharmacological interventions to modify variety of CAD risk factors like diabetes, high blood pressure, high lipids, tobacco use etc. is must. Proper use of cardiac rehabilitation programme is very rewarding.

## **Renovascular Hypertension**

*Dr. Ashish Parikh, Delaware, U.S.A*  
*Abstract not received*

## **Acute Coronary Syndrome**

*Prof. Yoseph Rozenman, Tel-Aviv, Israel*  
*Abstract not received*

## **Geriatric (Elderly) Heart Disease**

*Dr. Shmuel Banai, Tel-Aviv, Israel*  
*Abstract not received*

## **Cardiogenic Shock**

*Dr. Hemang Baxi, Ahmedabad, India*  
*Abstract not received*

## **Ischemic Stroke**

*Dr. Ashish Parikh, Delaware, U.S.A*  
*Abstract not received*

## **“Take home message” from recent trials of dyslipedemia**

*Dr. Milan Chag, Ahmedabad, India*  
*Abstract not received*

**Evening Session : 4.45 PM to 6.00 PM**

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**Review of more interesting ECGs, Arrhythmias, Chest X-Rays,  
Echos from Real World By The International Faculty.....An  
Experience of Lifetime.**

*Prof. Shumel Banai, Tel-Aviv, Israel wProf. Marisa Di Donato, Firenze, Italy wDr. Raj Makkar, Los Angeles, U.S.A.wDr. Anuja Nair, Cleveland, U.S.A.wDr. Ashish Parikh, Delaware, U.S.A.wProf. Yoseph Rozenman, Tel-Aviv, Israel wDr. Bharat Shah, California, U.S.A.wProf. Finn Waagstein, Sweden*

*Abstract not received*

# POSTER PRESENTATION

## ABSTRACT: 01

**AUTHORS:** *Dr Rajendra M Gadhavi*

### **Background**

With increase in the number of elderly in India hypertension is likely to emerge as an important public health problem. Rapid urbanization, adaptation of western lifestyle & lack of awareness have exacerbated the situation.

Objective

The study was done to know the prevalence of hypertension and its risk factors in urban government officials.

### **Study design**

Cross sectional study

### **Setting**

Sachivalaya of Gandhinagar city, Gujarat, India.

### **Sample size**

775

### **Mean Age of Study Population**

46.3 years

### **Method**

A pre-tested semi structured proforma administered to the participants. Blood pressure was measured by adopting standard procedure & by standard instruments in sitting posture for three times & average was taken as a reading.

### **Results**

Prevalence of hypertension was highest (47.4%) in class - I cadre and lowest (32.4%) in lower most cadres. Overall prevalence rate of hypertension among study population was 35.5%. The prevalence of hypertension was 35.3% among those having family history of hypertension and 21% among those doesn't have family history of it & association was found statistically highly significant ( $P=0.00002$ ). Association between prevalence of hypertension and BMI was statistically significant ( $P=0.0000$ ) along with association with WHR ( $P=0.0002$ ) The Prevalence of hypertension was 48.1% in diabetic compare to 30.7% in non diabetic and the association between hypertension and diabetes has been found statistically highly significant ( $P=0.003$ ). Out of those having hypertension 35.6% were known cases and 64.4% were newly diagnosed during the study.

### **Conclusion:**

Overall prevalence of diabetes is high among the study population. Positive family history, Diabetes, BMI, WHR and sedentary life style were contributing factor for it.

### **Key words**

Yoga.

## ABSTRACT: 02

### COMPARATIVE EVALUATION OF ATENOLOL AND METOPROLOL ON CARDIOVASCULAR COMPLICATIONS ASSOCIATED WITH STZ-INDUCED TYPE 1 DIABETES IN RATS.

**Shraddha V. Bhadada** and Dr. R. K. Goyal.  
L.M. College of Pharmacy, Ahmedabad.

#### ABSTRACT

**Objective :** Recently, various studies have indicated that lipophilic beta blockers reduce the coronary mortality in diabetics, however, systematic studies have not been reported. In the present study we have compared the effects of chronic treatment with metoprolol and atenolol on cardiovascular complications in STZ-diabetic rats.

**Materials and methods :** Sprague-Dawley rats were made diabetic with streptozotocin (60mg/kg, iv, single dose). Blood samples were analyzed for biochemical parameters by diagnostic kits. Heart rate and blood pressure were measured by non-invasive method using Harvard BP monitor. Histological study of the left ventricular tissue was done.

**Results :** Injection of STZ produced hyperglycemia, hypoinsulinaemia, hyperlipidaemia, increased blood pressure, cardiac hypertrophy, increase in force of contraction, reduction in heart rate and structural alterations in cardiac tissues. Metoprolol and atenolol effectively prevented the development of hypertension in diabetic rats. Metoprolol treatment produced slight but significant reduction in serum glucose levels with elevation in serum insulin levels, while atenolol treatment produced a slight increase in glucose levels but no effect on the insulin levels. Moreover, neither metoprolol nor atenolol treatment reduced the elevated cholesterol levels in diabetic rats. But metoprolol treatment significantly prevented STZ-induced increase in triglyceride levels while atenolol did not have any effect. Metoprolol exhibited a minimal improvement in STZ induced bradycardia whereas atenolol produced a further reduction in heart rate. Metoprolol treatment significantly prevented STZ-induced cardiac hypertrophy and cardiomyopathy while atenolol seems to worsen diabetes induced cardiomyopathy.

**Conclusion :** Our data indicate that metoprolol have some beneficial effects with respect to cardiovascular complications associated with diabetes mellitus over atenolol. It appears to delay the progression the congestive heart failure in diabetic condition possibly by preventing development of cardiomyopathy and hypertrophy.

## ABSTRACT: 03

Title: Utility of bedside echocardiography for diagnosis of suspected pulmonary embolism in a rural set up ICU.

Authors: Dr. Mannari J\*, Dr. Modi A. \*\*, Dr. Tanna M.\*\*\*, Dr. Poptani V.\*\*\*, Dr. Bhanushali J.\*\*\*  
Professor and Head , \*\* Assistant Professor,\*\*\*Medicine Residents

Institution: Dept of Medicine, H.M. Patel Centre for Medical Care and Education

**Abstract:** Current management strategies of acute major pulmonary embolism are largely dependent on degree of hemodynamic instability at presentation. In presence of severe hemodynamic

compromise, physicians rely on the finding of bedside echocardiography and proceed for thrombolytic treatment without seeking further diagnostic certainty in nuclear imaging or angiographic studies. This is especially true for our rural based teaching hospital. We present a case of young married female without any significant past history or risk factors for pulmonary thromboembolism, referred to our hospital with acute onset dyspnea and worsening levels of consciousness. Her physical examination revealed cold clammy extremities, weak pulses, profound hypotension, respiratory distress, normal S1, loud P 2 at pulmonary area, Gr-3/6 systolic murmur at tricuspid area. Electrocardiography revealed sinus tachycardia with RV strain. Bedside echocardiography revealed normal left ventricular systolic function, with moderate to severe right ventricular dysfunction with gross dilatation, paradoxical movement of interventricular septum, severe TR, PAP of 70 mmHg, a hyper echoic mass (clot) of about 2 cm in diameter freely moving in RV cavity. D-dimer assay done >10,000IU CT chest showed embolus in lower lobe branch of left pulmonary artery with infarct associated with bilateral pleural effusion, enlarged RA and RV with hypodense area probably clot in RV. Patient was given ventilatory support, thrombolytic and supportive therapy. She improved with given treatment and discharged on oral anticoagulants.

This case report attempts to reiterate the utility of bedside echocardiography in diagnosing pulmonary embolism. Quick bedside echocardiography clearly displays pathognomonic findings of massive pulmonary embolism. In addition, competing diagnoses (such as myocardial infarction, dissection of aorta and severe acute aortic insufficiency) can be ruled out. Access to such a widely performed test enables physicians to promptly implement therapy without delay.

## **ABSTRACT: 04**

STUDY OF THE RELATIONSHIP BETWEEN BMI & BLOOD PRESSURE IN THE MEDICAL STUDENTS OF KESAR SAL MEDICAL COLLEGE & RESEARCH INSTITUTE, AHMEDABAD.

\* MR.RAVI B. THAKER, \* MS. RUSHIKA B. PATEL, FORAM J. THAKKAR,

\*\* DR. ANIL PANDEY,

\*\*\*DR. K.V.BHATT, DR. S.M. JOSHI (DEAN)

\* 2<sup>nd</sup> Yr. MBBS student, \*\* Asstt. Prof., \*\*\* Prof.& Head; Dept. of Physiology  
Kesar SAL Medical College & Research Institute, Ahmedabad.

### **Background:**

Obesity represents an independent predictor of cardiovascular disease (Framingham Heart study). The majority of patients with high blood pressure are overweight, and hypertension is about six times more common in obese than it is in lean subjects. Weight gain in young people is an important risk factor for the subsequent development of hypertension. All overweight and obese are considered at risk for developing comorbidities such as hypertension, Diabetes, CAD etc.

### **Aim:**

The present study aims to investigate the relationship between BMI & Blood Pressure in the Medical Students of Kesar SAL Medical College & Research Institute, Ahmedabad.

### **Objectives:**

- To establish relationship between BMI & Blood Pressure.
- To impart awareness amongst the students about comorbidities of overweight and obesity.

### **Methods:**

The study was conducted in December 2005 at Kesar SAL Medical College & Research

Institute, Ahmedabad, consisting of 93 students (65 male & 28 female) of 17 – 21years of age. Purpose of the study was explained to the students and verbal consent was obtained from the students. Prior permission was taken from the head of the institution. The anthropometric measurement like Height, weight were taken by standard techniques thereafter BMI was calculated. Blood Pressure was measured by using Auscultatory Riva-Roci method in supine position. Overweight is defined as BMI of 25 to 29.9 Kg/m<sup>2</sup> and Obesity as BMI e" 30 Kg/m<sup>2</sup>. In view of gender differences in autonomic regulation, data of male and female subjects were analyzed separately. Statistical analysis was performed using SPSS package. Data are expressed as Mean value ± Standard deviation.

### Results:

	Age(yrs)	BMI (Kg/m <sup>2</sup> )	Heart rate/min.	*SBP(mmHg)	BP(mmHg)
Male (n=65)	18.4 ± 1.7	24.7 ± 3.8	88.4 ± 12.6	122.4 ± 13.5	86.8 ± 9.6
Female (n=28)	18.6 ± 1.9	23.1 ± 3.2	92.8 ± 13.3	114.4 ± 16.4	77.8 ± 7.6
					*p<0.005

Both SBP & DBP were highest in the overweight and least in the underweight in the study population (p < 0.005). When we compare the data between male and female of the comparable age and BMI, the SBP and DBP were significantly lower in the female compared to male (p<0.05) but heart was higher in female, however it was not significant (p > 0.05).

### Conclusion:

Our result suggests that there are gender differences between BMI & Blood Pressure indices and also in the underweight and overweight as a whole in the study population. These observed differences may be due to differences in autonomic function and or energy metabolism.

## ABSTRACT: 05

UNDERSTANDING CARDIOVASCULAR RESPONSES TO "STATIC EXERCISE" IN THE STUDENTS OF KESAR SAL MEDICAL COLLEGE & RESEARCH INSTITUTE, AHMEDABAD.

\*Ms.Ritika Jagtyani, \*Ms. Latika Mehta, \*Ms. Simplel Patadia, \*\*Dr. Anil Pandey, \*\*Miss Lopa Vaidya, Dr. S.M. Joshi (Dean)

\* 2<sup>nd</sup> Yr. MBBS student, \*\* Asstt. Prof.; Dept. of Physiology, Kesar SAL Medical College & Research Institute, Ahmedabad.

### Introduction:

Exercise, the most common physiologic stress, elicits cardiovascular abnormalities not present at rest and can be used to assess functions of the cardiovascular system. The acute cardiovascular responses to isometric exercise differ importantly from those to isotonic exercise. With isometric exercise a discrete muscle group is called upon to sustain a muscular contraction without performing external work. The O<sub>2</sub> requirements of the contracting muscle are not easily met by an increase in regional blood flow. The changes in Heart rate; Blood Pressure and perceived level of exertion provide data for quantitative estimation of cardiovascular conditioning.

### Aims:

To determine the influence of Static exercise on Heart rate (HR) and Blood Pressure indices.

### Methods:

The study was conducted in December 2005 at Kesar SAL Medical College & Research Institute, Ahmedabad, consisting of 93 students (65 male & 28 female) of 17 – 21years of age. Purpose of

the study was explained to the students and verbal consent was obtained from the students. Prior permission was taken from the head of the institution. The anthropometric measurement like Height, weight were taken by standard techniques thereafter BMI was calculated. Blood Pressure was measured by using Auscultatory Riva-Roci method in supine position. Students were instructed to perform two minutes of 33% of maximum voluntary contraction (MVC) by static handgrip using Hand-Dynamometer instrument. Heart rate & Blood Pressure were recorded before, during and after the Static exercise. Statistical analysis was performed using SPSS package. Data are expressed as Mean value  $\pm$  Standard deviation.

### **Results:**

There was significant increase in HR (from  $90.44 \pm 12.9$  beats/ min. to  $110.6 \pm 14.6$ ;  $p < 0.05$ ) & Mean Arterial Pressure (MAP) (from  $98.6 \pm 9.9$  to  $114.4 \pm 16.4$ ;  $p < 0.05$ ) after 2 minutes of 33% of MVC. When we analyze the data separately for male and female, no significant difference was observed.

### **Conclusion:**

These data suggests that the normal cardiovascular responses to static contraction in the upper limb may be due to combination of the cardiovascular responses to static contraction and muscle chemoreflex produced by these contractions.

### **Discussion:**

Local vasodilatation is limited by a mechanical compression of the resistance vessels caused by sustained muscular contraction so that blood flow to contracting muscle actually falls. The combination of limited blood flow and increased metabolic demand appears to evoke a locally mediated pressure response that probably represents an important adaptation to maintain regional perfusion.

## **ABSTRACT: 06**

TYPES OF OBESITY AND ITS IMPACT ON LIPID PROFILE IN THE STUDENTS OF KESAR SAL MEDICAL COLLEGE & RESEARCH INSTITUTE AHMEDABAD.

\*Ms.Roosha K. Parikh, Sumble Siddiqui, \* Mr. Neelay P. Jain, \* Mitul M. Shah, \*\* Dr. Anil Pandey, \*\*\*Dr. (Mrs.) Nirupama Dixit, # Dr. Bharti Dalal

\* 1<sup>st</sup> Yr.MBBS student, \*\* Asstt.Prof., \*\*\* Prof. Dept. of Physiology; # Dept. of Pathology Kesar SAL Medical College & Research Institute, Ahmedabad.

### **Background:**

The overweight and obesity is a complex multifactorial chronic disorder that develops from an interaction between genotype and the environment. Multiple evidences support the relation between blood cholesterol and CHD risk even in the absence of other risk factors. The overall obesity (based on BMI) is an established determinant of dyslipidemia but there are ample evidences to suggest that central obesity (based on waist circumference and waist-hip ratio) is equally or even more deleterious atherogenic factor.

### **Aims & Objectives:**

- To correlate between BMI, WHR & Lipid Profile.
- To impart awareness amongst the students about comorbidities of overweight/ obesities and benefits of weight reduction.

### **Methods:**

93 students (65 male & 28 female) from Kesar SAL Medical College & Research Institute, Ahmedabad of 17 – 21 years of age were participated in the study in the December 2005 of which only 34 were included in the study (based on BMI & WHR). An equal number of the students with comparable age were selected as control. Purpose of the study was explained to the students and verbal consent was obtained from the students. The anthropometric measurement like Height, weight, waist circumference (minimum circumference between the costal margin & Iliac crest, measured in horizontal plane, with the subject standing) and hip circumference (Max. circumference in the horizontal plane, measured over the buttock) were taken, thereafter BMI and Waist hip ratio(WHR) were calculated. Heart rate & Blood Pressure were measured by standard techniques. Blood samples were collected after 12 hours of overnight fasting. Statistical analysis was performed using SPSS package. Data are expressed as Mean value  $\pm$  Standard deviation.

### **Observation & Results:**

A positive & significant correlation was observed between BMI (cut off point 25 Kg/m<sup>2</sup>) & Lipid Profile (Total Cholesterol mg%, HDL mg%, LDL mg% & Triglycerides mg% were 196.4  $\pm$  20.4, 43.5  $\pm$  12.2, 102.5  $\pm$  11.5 & 134.4  $\pm$  34.4 respectively in the obese/ overweight group compare to control where it was 166.6  $\pm$  16.4, 56.5  $\pm$  12.2, 112.5  $\pm$  12.5 & 114.4  $\pm$  34.4 respectively p <0.05 for Total Chol., HDL Chol. and LDL Chol.). Almost similar results were seen when we compared the data based on WHR or waist circumference alone but there were more decrease in HDL Cholesterol associated with increased LDL Cholesterol found.

### **Conclusion & Recommendations:**

- These data suggest that there is a positive correlation between obesity (central & overall) and Lipid Profile however Waist circumference & WHR is a better indicator for cardiovascular comorbidities.
- Positive attempts should be taken to improve the lipid profile through the use of Aerobic exercise, Yoga, Pranayam & dietary modification.

## **ABSTRACT: 07**

### **EVALUATION OF LEFT VENTRICULAR PERFORMANCE IN THE ELDERLY**

**\*Dr. Anil Kumar Pandey,** \*# Dr. Asim Das, # # Dr. Amalkumar Bhattacharya,  
\* Dept. Of Physiology; Kesar SAL Medical College & Research Institute, Ahmedabad,  
# Baroda Medical College; # # Dept. Of Medicine, M.P. Shah Medical College, Jamnagar

### **Introduction:**

The ability of the cardiac muscle to develop tension is well maintained with aging and such observations predict normal global left ventricular function at rest. But there is a striking decrease in the response to the stimulation of the beta- sympathetic receptors of cardiovascular tissues. This age associated decrease in beta- sympathetic response manifest itself in decreased inotropic response of cardiac muscle, decreased arterial vasodilating response, & decrease heart rate, or chronotropic response. Prevention of cardiovascular disease in the elderly would decrease premature mortality, decrease chronic disability and improve quality of life.

### **Aims & Objectives:**

To determine the impact of aging on left ventricular function in the study population. The main objective of study in this group is: To utilize the information so obtained in the study population to reduce the morbidity and mortality.

### **Materials and Methods:**

The subjects were randomly selected from the patients attending out patient department of Medicine at New Civil Hospital & Govt. Medical College, Surat. Based on the age the subjects were divided into two study groups > 60 years (61-85) and < 60 years (30-60). Subject to purposefulness of the study deletion criteria were applied for exclusion e.g. Obesity based on body-mass index and Waist hip ratio. SBP, DBP and Pulse were recorded in both the groups.

M-mode Echocardiogram was performed for determination of left ventricular internal dimensions. LVMI, RWT, EF%, FS%, and ESS was there-on calculated using ASE convention while E/A ratio was obtained by flow wave velocity using Doppler echo as an indicator of diastolic function.

### **Observation & Results:**

The mean age in the elderly group (n = 32) was  $63.67 \pm 6.34$  and in the control group (n = 47) was  $46.73 \pm 9.34$ . The mean BMI, Pulse, SBP & DBP were similar in reference to comparable age and accessibility.

Post adjustment of the BMI and blood pressure, the various parameters of left ventricular functions namely LVMI, RWT were increased in the elderly group compared to the control. EF% & FS% (contractility Indices) were better in the control group; however it was within the normal limit in the elderly group too. E/A ratio (indicator of Diastolic function) was significantly lower in the elderly group compared to control group (p Value <0.05). ESS (indicator of Afterload) was significantly higher in the elderly group compared to control group (p Value < 0.05).

### **Conclusions:**

Left ventricular function significantly and negatively correlates with age. These measurements should be taken early to plan methods for reduction of afterload and improvement of the contractile state of the left ventricle.

### **Key words & Abbreviations:**

LVMI = Left Ventricular Mass Index, EF % = percent Ejection Fraction, FS % = percent Fractional Shortening, ESS = End Systolic stress, RWT = Relative Wall Thickness, E/A ratio = Ratio of Early & Late Filling Velocity, ASE = American Society of Echocardiography.

### **Keywords and Abbreviations:**

Pulmonary Function Test; Obesity;

BMI = body mass index; WHR = Waist-Hip ratio **Keywords and Abbreviations:** Pulmonary Function Test; Obesity;

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BMI = body mass index; WHR = Waist-Hip ratio **Key Words & Abbreviations**

## International Faculty



### **Banai, Shmuel , MD**

Director, Interventional Cardiology, The Tel Aviv Souraski Medical Center, Tel Aviv, Israel. Studied Medicine in the Hebrew University Hadassah Medical School, Jerusalem, Israel. The Mount Sinai Hospital, New York, NY, USA - elective studies in Cardiology and Pulmonology. Post Doctoral Basic Research in Cardiology at National Institute of Health, National Heart, Lung and Blood Institute, Cardiology Branch, Section of Experimental Physiology and Pharmacology, Bethesda, Maryland, USA. Won many prizes and citations like The Henry Neufeld Prize for the most original research work, Citation by the Faculty of Medicine, The Hebrew University - Hadassah Medical School for excellence in teaching. Worked on many committees like Special Medical Investigation Committee, Ministry of Health, Jerusalem, 2004, Scientific advisory committee, Enterprises, new technologies and developments in Cardiology, Special Medical Investigation Committee, Ministry of Health, Jerusalem, January 2005, Special Medical Investigation Committee, Ministry of Health, Jerusalem, April 2005 as Chairman.



### **Donato, Marisa Di, MD**

Professor in cardiology, University of Florence  
 Director of cardiovascular research center, Policlinico san donato, Italy  
 Cardiology trainee in 1977 from University of Parma, Italy. Associate Professor in Cardiology, University of Florence from 1984. Certification in Medical Decision Making and Clinical Teaching from Stanford University. **Main activity:** Invasive Cardiology from 1975 to 2001. Responsible for the teaching programs in Cardiology for medical students and for the training of Invasive Cardiology fellows. Consultant Cardiologist for Clinical Research at the Cardiothoracic Center of Monaco. Main scientific fields of interest: Physiopathology of the heart, coronary artery disease, heart failure, hemodynamics, ventricular function, left ventricle remodeling and left ventricle reconstructive surgery. Author of near 150 publications, most of them international Journals of Medicine. Speaker at several national and international meetings. Principal investigator for many clinical trials. Chairman of the European STICH Trial Committee from 2004.

### **Bharat Harivadan Shah, MD., FACC, FASE**

Did graduation in medicine from M. S. University, Baroda Medical College, Baroda, India.  
 In 1980-1981 did Internship at Louis A. Weiss Memorial Hospital, Affiliated with University of Illinois. Did Internal Medicine Residency at Christ Hospital, Rush University, Affiliated with Chicago. Did Fellowship - Cardiology at Christ Hospital, Rush University, Affiliated with Chicago. Was Chief of Cardiology at V. A. Hospital, Fort Wayne, Indiana. Is affiliated to Providence Saint Joseph Medical Center, Burbank, California, Glendale Adventist Medical Center, Glendale, California and Verdugo Hills Hospital, Glendale, California



### **Makkar, Rajendra, MD**

Co-Director, Cardiovascular Intervention Center, Cedars-Sinai Medical Center  
 Co-Director of the Interventional Cardiology Research Program, Cedars-Sinai Medical Center. Principal investigator of several research protocols in the Cardiovascular Intervention Center. He has established a program for treating patients with hypertrophic cardiomyopathy using percutaneous transluminal septal myocardial ablation. Dr. Makkar is currently studying the application of stem cells in treating myocardial infarction in an experimental setting. A skilled interventional cardiologist, Dr. Makkar has extensive experience with the development and the clinical application of devices like coronary stents and intravascular brachytherapy. He has received two American Heart Association grants (a postdoctorate fellowship and a Grant-in-Aid) and has been a co-investigator on a National Institutes of Health (NIH) grant. Dr. Makkar has published in several peer-reviewed journals, including Circulation, Journal of the American College of Cardiology, Journal of the American Medical Association and Lasers in Surgery and Medicine. Completed training at Cedars-Sinai Medical Center & UCLA School of Medicine.



### **Nair, Anuja, PhD**

Senior Scientist, Volcano Corporation, Rancho Cordova, CA. Adjunct Staff, Department of Biomedical Engineering, Cleveland Clinic, Former Research Associate, CCF, Cleveland, OH. Research interests in medical signal processing, analysis, ultrasonic tissue characterization and visualization. Currently managing and developing software solutions for characterizing atherosclerotic plaque components in real-time. Doctor of Philosophy, Biomedical Engineering (January 2003). Case Western Reserve University, Cleveland, Ohio. Master of Science, Biomedical Engineering, (August 2000, GPA: 3.86). Case Western Reserve University, Cleveland, Ohio. Bachelor of Science in Engineering, Biomedical Engineering. Emphasis on Biosystems Engineering & Cellular-Molecular Bioengineering (Honors-Magna Cum Laude). Arizona State University Many honours and awards during academic career, Numerous research publications



### **Parikh, Ashish MD, FACC**

Interventional Cardiology & Peripheral Vascular Fellowship The Willam Dorros Isadore Feuer. Interventional Cardiovascular Disease Foundation Milwaukee, WI. Clinical Cardiology Fellowship at Cedars-Sinai Medical Centre at Los Angeles, California. Co-Director ACLS Training at Cedars-Sinai Medical Centre at Los Angeles, California. Internal Medicine Residency at Saint Vincent Hospital at Worcester, MA. House Officer Cardiovascular Surgery at Medical Centre of Delaware Newark, DE. Nephrology Fellowship at V.S. General Hospital, India. Has won many awards a) National Painting Award 1st 1970. b) National Scholarship India 1975 c) Dasha Khadayta Science Achievement Award 1978 & 1980 d) Young Investigator Award-AACIO on the eve of AHA 1976. Work published in peer reviewed journals & presented at numerous meetings. Member of top professional organizations.



### **Yoseph Rozenman MSc, MD, FACC**

Studied Mathematics, Physics and Medicine at the Hebrew University. Later took qualifications in Medicine in the USA. Did specialisation in cardiology both in Israel and the USA.

Extensive teaching experience in Physics, Medicine and Cardiology in the Hebrew University, Israel and Harvard University, Boston, USA. Director of the E. Wolfson Med. Ctr., Holon, Israel, Heart Institute.

In his three decade career like De-Shalit prize in Physics, Sushchik prize in Surgery and many research grants from many prestigious groups and associations like the ACC, AHA, ESC, AMA, Israeli Medical Association and Heart Society and so on at teacher



Torbjorn Holm).  
 in Medicine year

Wallenberg Laboratory, Sahlgrenska University Hospital, Sweden  
 is working in Department of Cardiology, Sahlgrenska university hospital, Goteborg, Sweden. He is an authority investigator to discover the potential benefit of beta-adrenergic-blocking agents in congestive heart failure. His work on the beta-blockers, despite skepticism from the medical community, led to a landmark mortality trial. His recent breakthrough in heart failure therapeutics. Opponent and peer reviewer of scientific theses. Faculty member Groningen Holland University 1995. Faculty opponent dissertation Faculty of medicine Aarhus University (Hans Erik Bötter) Faculty opponent dissertation Faculty of Medicine Oslo University Norway 2001 (M.D. Torbjorn Holm) and Silver Medal, European Society of Cardiology 2001. Cowinner of King Faisal International Prize in Clinical Cardiology 2002



## The Heart Care Clinic

Care Cardiovascular Consultants

201, Balleshwar Avenue, Opp Rajpath Club, S. G. Road, Bodakdev, Ahmedabad-380054,

Phones : 079 26873101, 26871564, 26873224, 26873325, Fax : 079 26872620,

Mobiles : 98250 66664, 98250 66668, Email : [drkeyurparikh@gmail.com](mailto:drkeyurparikh@gmail.com)

For any further information please call

Mrs. **Komal Shah - 9377959025**