CIMS-CON 2012

January 6-8, 2012

Education For Innovation

8th Annual Scientific Symposium, 17th Year of Academics

Abstract Book

Organized by

CIMS®
Care Institute of Medical Sciences

The Heart Care Clinic
Clinical Care Consultants

Local Organizing Committee

Dr. Keyur Parikh
Conference Chairman

Dr. Milan Chag
Chairman, Scientific Committee

Conference Directors & Co-Ordinators

Dr. Anish Chandarana

Dr. Ajay Naik

Dr. Satya Gupta

Dr. Joyal Shah

Dr. Gunvant Patel

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Dr. Dhaval Naik

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Dr. Niren Bhavsar

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Dr. Kashyap Sheth

Dr. Ravi Singhvie

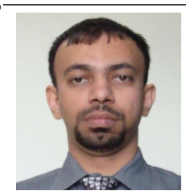
Dr. Amit Chitaliya

Dr. Nitesh Shah

Dr. Amit Patel

Dr. Bhagyesh Shah

Dr. Vipul Thakkar



Dr. Sanjay Shah

International Faculty

Prof. Rafael Beyar
Israel

Dr. Bhavin Dalal
USA

Prof. Uri Elkayam
USA

Dr. Mukesh Hariawala
USA

Dr. Ashit Jain
USA

Dr. Vinit Lal
USA

Prof. J. L. Mehta
USA

Dr. Krishnamoorthy
Niduvaje
Singapore

Dr. Manish Parikh
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Prof. C. Thomas
Peter
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Prof. Guy Heyndrickx
Robert
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Dr. M. Chandra Sekar
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Dr. Samin Sharma
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Dr. Charles
Simonton
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Dr. Jashvantlal
Thakkar
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Dr. Dharmapuri
Vidyasagar
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Dr. Keyur Bhalavat


Dr. Abdul Ghafur


Dr. Manoj Ghoda


Dr. Navin Goyal


Dr. Ashit Hegde


Dr. Rajesh Hydrabadi


Dr. Uday Jadhav


Dr. Sandeep Jain


Dr. Subir Jhaveri


Dr. Mahendra Joshi


Dr. Pradip Joshi


Dr. Manthan Kansara


Prof. S. K. Kaushik


Dr. Hema Kulkarni


Dr. Subodh Kumar


Dr. Ashwin Mani


Prof. Dileep Mavalankar


Dr. Shalmi Mehta


Dr. Sunil Mehta


Dr. Mamta Muranjan


Dr. Winston Noronha


Dr. Sapan Pandya


Dr. Amola Patel


Dr. Baldev Prajapati


Dr. Manohar Santwani


Dr. Bansi Saboo


Dr. Raju C. Shah


Dr. Ateet Sharma


Dr. Mihir Tanna


Dr. Parimal Tripathi


Dr. Varsha Tripathi


Dr. Sharad Vyas

Dr. Chirag Amin
Dr. Vivek Arya
Dr. T. Ayyapan
Dr. Eva Bhagat
Dr. N. Bhattacharya
Dr. Mahadev Desai
Dr. Parindra Desai
Dr. Urman Dhruv
Dr. Batuk Diyora

Dr. Mahesh Gupta
Dr. Prakash Jiandani
Dr. Milan Jolapara
Dr. Dhiren Joshi
Dr. Pranav Joshi
Dr. Vishal Kacchhi
Dr. Jayul Kamdar
Dr. Kavita Kamineni
Dr. Tiven Marwah

Dr. Nilay Mehta
Dr. Biplab Mishra
Dr. Darshana Naik
Dr. Pinki Naik
Dr. Mukul Oza
Dr. Ashok Parekh
Mr. Parth Parikh
Dr. Roosha Parikh
Mr. Shivam Parikh

Dr. Apurva Patel
Dr. Atul Patel
Dr. Kaushik Patel
Dr. Mayur Patel
Dr. Nayna Patel
Dr. Sanjeev Phatak
Dr. Ashwin Sanghvi
Dr. Amar Shah
Dr. Jyotin Shah

Dr. Maulik Shah
Dr. Navneet Shah
Dr. Parag Shah
Dr. Raj Shah
Dr. Jayshree Sheth
Dr. M. J. Sonagara
Dr. Rajesh Teli
Dr. Mayank Thakker
Dr. Harsh Toshniwal

Dr. Hiren Trivedi
Dr. Vivek Uppal
Dr. S. N. Vani
Dr. Vijayasekaran
Dr. Manoj Vithalani

Day-1, January 6, 2012, Friday, Venue : Tagore Hall

08.15 AM	The Year in Cardiology : An update for physicians (How doctors should change) - ***Dr. Keyur Parikh***
08.30 AM	"Cloud Computing" - we all should learn (KBC/KBI) ***- Dr. Keyur Parikh /Dr. Apurva Patel /Dr. Roosha Parikh /Mr. Shivam Parikh/Mr. Parth Parikh***
09.10 AM	Arterial Hypertension-Diabetes-Heart Failure-Renal Nerves and Renal Denervation - ***Dr. Keyur Parikh***
09.30 AM	A-Z Trials of 2011 in Cardiology - Part 1 - ***Dr. Milan Chag***
09.55 AM	**PL-1 :** Acute Myocardial Infarction: From Pathophysiology to Therapeutic Targets - ***Prof. Guy Heyndrickx Robert***
10.15 AM	**PL-2 :** Antiplatelet Therapy in ACS: Emerging Evidence - ***Prof. Lars Wallentin***
10.35 AM	**Tea Break**
	CAD in special populations : Challenges & Management
10.45 AM	CAD in very elderly - ***Dr. Urmil Shah***
11.00 AM	CAD in very young - ***Dr. Joyal Shah***
11.15 AM	CAD in women - ***Dr. Anish Chandarana***
11.30 AM	Non-invasive tools : CACS - ***Dr. Satya Gupta***
11.45 AM	Non-invasive tools : C-IMT - ***Dr. Hemang Baxi***
12.00 PM	**Panel Discussion**
	Arteries & Valves
12.10 PM	TAVI : A Reality - ***Dr. Milan Chag***
12.25 PM	Fully Bioabsorbable Vascular Scaffolds and Vascular Reparative Therapy: The Future is Now! ***- Dr. Charles Simonton***
12.40 PM	Coronary Stents : present and future - ***Prof. Rafael Beyar***
01.05 PM	**Lunch**
	How to manage ?
02.05 PM	Pulmonary embolism - Update in diagnosis, risk satisfaction and management - ***Dr. Urmil Shah***
02.20 PM	Vit - D -Deficiency : How to identify underestimated Risk Factor ? - ***Dr. Anish Chandarana***
02.35 PM	Pulmonary HT : How to manage in 2012 ? - ***Dr. Hemang Baxi***
02.50 PM	SCD: Who are at Risk ? How to detect & manage ? - ***Dr. Ajay Naik***
03.05 PM	HT & DM : Deadly duo: How to optimize treatment ? - ***Dr. Satya Gupta***
03.20 PM	Revascularization strategies for CAD : PCI or CABG or just medicines - ***Dr. Vinit Lal***
03.35 PM	**Panel Discussion**
03.45 PM	**Tea Break**
04.00 PM	**PL-3 :** High Cardiac Risk Pregnancies: natural history and management - ***Prof. Uri Elkayam***
04.20 PM	**PL-4 :** Cross-talk between renin-angiotensin system and dyslipidemia - ***Prof. J. L. Mehta***
04.50 PM	Cardiovascular Robotics – Present and Future (In appreciation of 3 Nobel Prize Winner from Rambam Health Center/Technion) - ***Prof. Rafael Beyar***
	Debate - 1
	Alcohol is good for health !
05.10 PM	Of course ! - ***Dr. Anish Chandarana***
05.20 PM	No evidence ! - ***Dr. Urmil Shah***
2+2	Rebuttal
	Debate - 2
	Carotid Artery Stenosis
05.39 PM	Surgery is gold standards ! - ***Dr. Dhaval Naik***
05.49 PM	PCI is the way to go ! - ***Dr. Ashit Jain***
2+2	Rebuttal
	Debate - 3
	Reduction in CV-Mortality
06.03 PM	Life style modification is enough - ***Dr. Uday Jadhav***
06.13 PM	No, one needs intervention + statin ! - ***Dr. Bimal Shah***
2+2	Rebuttal

Satellite Session - January 6, 2012, Friday (Time : 8.00 pm to 10.00 pm), Venue : Hotel Royal Orchid Central & Hotel Park Plaza

A. Cardiac Pharmacology - I (Venue : Hotel Park Plaza)	
08.00 PM	Improving Outcomes with anti-thrombotic therapy in ACS - ***Prof. Lars Wallentin***
08.20 PM	Pharmacology in Acute Myocardial Infarction - ***Dr. Manish Parikh***
08.40 PM	Treatment of Low HDL - ***Dr. Uday Jadhav***
09.00 PM	Beta blockers: Where are we now ? For HTN - For HF - ***Dr. Milan Chag***
09.20 PM	10 things physician should remember when prescribing drugs - ***Dr. M. Chandra Sekar***
09.40 PM	**Audience - Panel Discussion**
B. Cardiac Pharmacology - II (Hotel Park Plaza)	
08.00 PM	Valvular heart disease native & prosthetic : Anticoagulation - ***Prof. Uri Elkayam***
08.20 PM	Aspirin for the Prevention of Cardiovascular Events in Patients Without Clinical Cardiovascular Disease: The Final Word - ***Dr. Vinit Lal***
08.40 PM	How will I choose my anti-hypertensives ? - ***Prof. J. L. Mehta***
09.00 PM	So many Diuretics - How to use them ? - ***Dr. Joyal Shah***
09.20 PM	ACE, ARB and Renin Inhibitor in Diabetics - ***Dr. Uday Jadhav***
09.40 PM	**Audience - Panel Discussion**
C. Imaging / Diagnostics / Therapeutics (Hotel Park Plaza)	
08.00 PM	Vulnerable plaque : imaging & management - ***Prof. Guy Heyndrickx Robert***
08.20 PM	When will I avoid DES ? - ***Prof. Rafael Beyar***
08.40 PM	Adult CHD diagnosis, challenges & management - ***Dr. Kashyap Sheth***
09.00 PM	End Stage Heart Disease - ***Dr. Mukesh Hariawala***
09.20 PM	Diastolic dysfunction : Echo assessment - ***Prof. S. K. Kaushik***
09.40 PM	**Audience - Panel Discussion**
D. Peripheral Vascular Disease (PVD) (Hotel Orchid)	
08.00 PM	An Innovative confluence : Hypertension, Diabetes, Heart failure : What to do? - ***Dr. Keyur Parikh***
08.20 PM	Save the lower extremities- update in newer modalities of treatment? - ***Dr. Ashit Jain***
08.40 PM	Acute Stroke Management : Thrombolytic & PCI - ***Dr. Ashit Jain***
09.00 PM	2011-2012 Guidelines for Evaluation and Management Peripheral Arterial Disease - ***Dr. Vinit Lal***
09.20 PM	Varicose Veins : prevention, indication and management - ***Dr. Rajesh Hydrabadi***
09.40 PM	**Audience - Panel Discussion**
E. Cardiology Guidelines (Hotel Orchid)	
08.00 PM	Guidelines in Management of AF - ***Dr. Bimal Shah***
08.20 PM	Guidelines in Management of HTN - ***Dr. Urmil Shah***
08.40 PM	Guidelines in Management of Acute Myocardial Infarction - ***Prof. Guy Heyndrickx Robert***
09.00 PM	Guidelines in Management of Valvular Disease : aortic & mitral - ***Prof. Uri Elkayam***
09.20 PM	Guidelines for primary and secondary prevention for development of CAD - ***Dr. Vinit Lal***
09.40 PM	**Audience - Panel Discussion**
F. Year in Cardiology / Cardiac Surgery - What Physicians Should Know (With Real Life Cases) (Hotel Orchid)	
08.00 PM	The Year in Cardiac Surgery - ***Dr. Dipesh Shah***
08.15 PM	The Year in Arrhythmias - ***Dr. Ajay Naik***
08.30 PM	The Year in Echocardiography - ***Dr. Satya Gupta***
08.45 PM	The Year in Interventional Cardiology - ***Dr. Keyur Parikh***
09.00 PM	The Year in anti thrombotic therapy and newer insights from ACC-ESC 2011 - ***Prof. Lars Wallentin***
09.20 PM	The Year in Coronary Artery Disease - ***Dr. Anish Chandarana***
09.40 PM	**Audience - Panel Discussion**

Day-2, January 7, 2012, Saturday, Venue : Tagore Hall

07.30 AM	Interactive ECGs - ***Dr. Hema Kulkarni/Dr. Ajay Naik***
09.00 AM	**Panel Discussion**
	Plenary Lectures
09.20 AM	Role of anti thrombotic therapy in NSTEMI and STEMI - ***Prof. Lars Wallentin***
09.35 AM	Diastolic Heart Failure - ***Prof. Uri Elkayam***
09.50 AM	**PL-5 :** Stem Cells in Cardiology - where are we now? - ***Prof. Rafael Beyar***
10.10 AM	**Panel Discussion**
10.20 AM	**Tea Break**
	CVD : Recent concepts and future
10.30 AM	A-Z Trials of 2011 in Cardiology - Part 2 - ***Dr. Milan Chag***
10.50 AM	**PL-6 :** Carotid Stenting for standard surgical risk patients: CREST data and the latest interpretation - ***Dr. Charles Simonton***
11.10 AM	Introduction to 3-C Oration - ***Dr. Keyur Parikh/Dr. Milan Chag***
11.15 AM	3-C Oration : Update in mechanism and treatment of ACS : 2012 & Beyond - ***Dr. Samin Sharma***
	Heart Diseases - Management : Present & Future
11.35 AM	**PL-7 :** Acute Heart Failure : management - present & future - ***Dr. Bimal Shah***
11.55 AM	MICS & Hybrid : We are there ! : management - present & future - ***Dr. Dhiren Shah***
12.10 PM	Burden of IHD in India - preventive management & future - ***Dr. Hema Kulkarni***
12.25 PM	**Panel Discussion**
12.35 PM	**Lunch**
01.35 PM	Update on vulnerable plaque: therapeutic options - ***Prof. Guy Heyndrickx Robert***
01.55 PM	Treatment of chronic stable systolic heart failure : from diuretics to VADs - ***Dr. Vinit Lal***
02.10 PM	**Panel Discussion**
	"Real Life Cases" (Live Cases from the Cath Lab/OT or Pre-recorded live cases) With Physician Interaction and Management Pathway
02.25 PM	**Clinical Case-1 :** A 68-year-old Man with left main undergoes PCI of the unprotected left main coronary artery - ***Dr. Samin Sharma / Dr. Keyur Parikh***
02.40 PM	**Clinical Case-2 :** A patient with 60 % lesion in LAD, after successful thrombolysis - ***Prof. Guy Heyndrickx Robert /Dr. Joyal Shah***
02.55 PM	**Clinical Case-3 :** A patient with non-healing ulcer in leg - ***Dr. Vinit Lal***
03.10 PM	**Clinical Case-4 :** A patient with severe Heart Failure and Coronary Artery Disease - ***Dr. Manish Parikh/Dr. Hemang Baxi***
03.25 PM	**Clinical Case-5 :** A patient with severe triple vessel disease - ***Prof. Rafael Beyar/Dr. Anish Chandarana***
03.40 PM	**Clinical Case-6 :** A patient with paravalvular leak closure - ***Dr. Milan Chag***
03.55 PM	**Clinical Case-7 :** A Real life case of bioabsorbable stent - A glimpse in future - ***Dr. Charles Simonton /Dr. Urmil Shah***
04.10 PM	**Clinical Case-8 :** Mitral Regurgitation : innovative approach - ***Dr. Dhiren Shah/Dr. Dhaval Naik***
04.25 PM	**Clinical Case-9 :** Radio Frequency segmental Ablation using VNUS Closure FAST catheter/Generator for Primary Varicose Veins - ***Dr. Milan Chag/Dr. Rajesh Hydrabadi***
	Evening Plenary Lectures
04.40 PM	**PL-8 :** Management and Public Health in Medical Practice : A Primer of Doctor - ***Prof. Dileep Mavalankar***
05.00 PM	How to live life ? Lessons to learn (Inspirational Oration for All) - ***Prof. C. Thomas Peter/Dr. Keyur Parikh***
05.20 PM	**Conclusion**

CIMS-CON Young Interventional Course - January 7-8, 2012, Saturday-Sunday, Venue : CIMS Hospital & Tagore Hall

07.45 AM	Selection of guiding catheters, guide wires and balloons - ***Prof. Guy Heyndrickx Robert / Dr. Keyur Parikh***
08.15 AM	Approach to Bifurcations - ***Dr. Samin Sharma /Dr. Keyur Parikh***
08.45 AM	- PCI for STEMI: Guidelines & Strategies - Pharmacoinvasive strategies (facilitated, rescue and transfer PCI) in STEMI - Shock ***- Dr. Manish Parikh /Dr. Hemang Baxi***
09.30 AM	Structural Heart Disease : Basics tips & tricks - ***Dr. Milan Chag***
10.00 AM	Extraction and Filter Devices - ***Dr. Ashit Jain / Dr. Urmil Shah***
10.30 AM	**Panel Discussion**
10.45 AM	**Tea Break**
11.00 AM	Radial Artery Access : technique, results and complications - ***Dr. Satya Gupta /Dr. Joyal Shah***
11.30 AM	Rationale for functional assessment of lesions in patients with MVD & Primer on Coronary Physiology for interventional cardiologists - ***Dr. Anish Chandarana /Prof. Guy Heyndrickx Robert***
12.00 PM	Stent Thrombosis: mechanisms, predictors and treatment - ***Dr. Vinit Lal***
12.15 PM	Chronic Total Occlusion (CTO): Tips for the beginner's - ***Prof. Rafael Beyar***
12.45 PM	Career Development for the "New" Interventionalist - Ten things to do in your first five years to grow your career - ***Dr. Manish Parikh***
01.00 PM	**Lunch**
"Real Life Cases" (Live Cases from the Cath Lab/OT or Pre-recorded live cases) With Physician Interaction and Management Pathway	
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04.25 PM	**Clinical Case-9 :** Radio Frequency segmental Ablation using VNUS Closure FAST catheter/Generator for Primary Varicose Veins - ***Dr. Milan Chag/Dr. Rajesh Hydrabadi***

Certification Courses - Day-3, January 8, 2012, Sunday

(A) Clinical Cardiology - Venue : The Grand Bhagwati

08.45 AM	Management of the hospitalized patient with heart failure **- *Prof. Uri Elkayam***
09.00 AM	Challenges in hypertension management **- *Prof. J. L. Mehta***
09.15 AM	Clinical Care : Dyslipidemia **- *Dr. Uday Jadhav***
09.30 AM	HDL raising :In search of the holy grail **- *Dr. Uday Jadhav***
09.45 AM	Clinical decision making : today and tomorrow **- *Dr. Manish Parikh***
10.00 AM	Clinical Care : ACS **- *Dr. Keyur Parikh***
10.20 AM	Clinical Care : Hypertension **- *Dr. Ravi Singhvie***
10.40 AM	X-Ray chest : A forgotten art - revisited **- *Dr. Milan Chag***
11.00 AM	**Tea Break**
11.20 AM	Challenges in CAD in India **- *Dr. Hema Kulkarni***
11.40 AM	Clinical Care : Syncope **- *Dr. Ajay Naik***
12.00 PM	Constrictive pericarditis versus restrictive cardiomyopathy - diagnostic work up **- *Prof. Uri Elkayam***
12.15 PM	Natriuretic peptides-In HF and ACS.....useful or an extra burden? **- *Dr. Bimal Shah***
12.30 PM	10 points to remember on management of cardiovascular diseases during pregnancy **- *Prof. Uri Elkayam***
12.45 PM	Stem Cells, Angiogenesis and Myogenesis : New evolving cellular therapies in cardiovascular medicine ***- Dr. Mukesh Hariawala***
01.00 PM	Comparison of early surgery versus conventional treatment in asymptomatic severe mitral regurgitation ***- Dr. Dhiren Shah***
01.15 PM	**Lunch**
02.15 PM	Pregnancy & heart disease : Which drugs to avoid? **- *Prof. Uri Elkayam***
02.30 PM	Clinical guidelines in management of HF **- *Dr. Ajay Naik***
02.45 PM	Interactive ECGs with masters **- *Prof. S. K. Kaushik /Dr. Ajay Naik***
03.45 PM	Interactive Echo : Live Cases ***- Dr. Uday Jadhav / Dr. Hemang Baxi / Dr. Anish Chandarana / Dr. Urmil Shah/ Dr. Satya Gupta/ Dr. Joyal Shah/ Dr. Mihir Tanna/ Dr. Ravi Singhvie/***

Certification Courses - Day-3, January 8, 2012, Sunday

(B) Internal Medicine - Venue : The Grand Bhagwati

08.45 AM	Career development for "Senior Physicians" 10 things to do ! **- *Dr. Manish Parikh***
09.00 AM	Role of Prasugrel & Ticagrelor in ACS & PCI **- *Dr. Jashvantlal Thakkar***
09.15 AM	Medical management of chronic respiratory failure **- *Dr. Jyotin Shah***
09.30 AM	Investigation of a case of generalised bodyache **- *Dr. Mahadev Desai***
09.45 AM	Bronchial asthma, bronchitis and asthmatic bronchitis are they same or different? in what context? **- *Dr. Nitesh Shah***
10.00 AM	**Panel Discussion**
10.10 AM	Stress the stress! **- *Dr. Dhiren Joshi***
10.25 AM	Culture and sensitivity : who is benefited? **- *Dr. Harsh Toshniwal***
10.40 AM	Common ailments due to the luxuries we use! **- *Dr. Pradip Joshi***
10.55 AM	Reading the CBC report **- *TBA***
11.10 AM	Dengue Fever **- *Dr. Manohar A. Santwani***
11.25 AM	**Panel Discussion**
11.35 AM	Dermatological manifestations of febrile illness **- *Dr. Sunil Mehta***
11.50 AM	Investigations- do they help or confuse the clinician? **- *Dr. Rajesh Teli***
12.05 PM	Pre Diabetic and Pre Hypertension **- *Dr. Urman Dhruv***
12.20 PM	Glycemic control in critically ill patient **- *Dr. Ashok Parekh***
12.35 PM	**Panel Discussion**
12.45 PM	**Lunch**
01.45 PM	Sleep Apnea - Sleep Study **- *Dr. Amit Patel***
02.00 PM	Transient loss of consciousness- Neurologist's perspective **- *Dr. Pranav Joshi***
02.15 PM	Chronic Hepatitis-B approach **- *Dr. Nilay Mehta***
02.30 PM	Medical management of mild CKD- what to do and what not to do? **- *Dr. Manthan Kansara***
02.45 PM	**Panel Discussion**
02.55 PM	Investigation and management of a case of chronic headache **- *Dr. Parindra Desai***
03.10 PM	GI Bleed - A physician's perspective **- *Dr. Manoj Ghoda***
03.25 PM	PCT, CRP and overall approach to a patient with sepsis: Do's and Don'ts **- *Dr. Atul Patel***
03.40 PM	Management of RA **- *Dr. Sapan Pandya***
03.55 PM	**Panel Discussion**
04.05 PM	Thyriod function tests **- *Dr. Tiven Marwah***
04.20 PM	Male climacteric(Andropause)- does it exist? **- *Dr. Parag Shah***
04.35 PM	Panel Discussion : What is comprehensive management of D.M.? FBS, PPBS, HBA1C or CMBG? **Panelist: *Dr. Sanjeev Phatak, Dr. Navneet Shah, Dr. Mukul Oza, Dr. M. J. Sonagara, Dr. Mayur Patel***
05.05 PM	Panel Discussion : 1) Pioglitazone controversy 2) Are all "Gliptines" same? **Panelist: *Dr. Vivek Arya, Dr. Mayank Thakker, Dr. Bansi Saboo, Dr. Manoj Vithalani***

Certification Courses - Day-3, January 8, 2012, Sunday

(C) Trauma Care - Venue : CIMS Hospital

09.00 AM	Developing a trauma center: an organizational concept - ***Dr. Biplab Mishra***
09.20 AM	ATLS guidelines for trauma management - ***Dr. Subodh Kumar***
09.40 AM	Hemorrhage control in trauma - ***Dr. Winston Noronha***
10.00 AM	Non-operative management of solid organ injury - ***Dr. Kaushik Patel***
10.20 AM	Damage control in trauma surgery - ***Dr. Sanjay Shah***
10.40 AM	**Panel Discussion**
11.00 AM	**Tea Break**
11.20 AM	Management of chest injuries - ***Dr. Sandeep Jain***
11.40 AM	Perils & pitfalls of shock in trauma - ***Dr. Ashit Hegde***
12.00 PM	Disaster preparedness and management - ***Dr. Sharad Vyas***
12.20 PM	Interventional radiology in trauma - ***Dr. Milan Jolapara***
12.40 PM	Medico-legal aspects in trauma - ***Dr. Biplab Mishra***
01.00 PM	**Panel Discussion**
01.15 PM	**Lunch**
02.00 PM	Critical care in trauma - ***Dr. Prakash Jiandani***
02.20 PM	Management of head injury - ***Dr. Batuk Diyora***
02.40 PM	Compartment syndrome in trauma - ***Dr. Navin Goyal***
03.00 PM	Management of pelvis fracture - ***Dr. Ateet Sharma***
03.20 PM	Management of Maxillofacial injury - ***Dr. T. Ayyapan***
03.40 PM	Early management of spinal Injury - ***Dr. Subir Jhaveri***
04.00 PM	Management of gunshot wounds & blast injury - ***Dr. Subodh Kumar***
04.20 PM	**Panel Discussion**

Certification Courses - Day-3, January 8, 2012, Sunday

(D) Critical Care / Pulmonary - Venue : The Grand Bhagwati

09.00 AM	Number Game:"Wean your patient from mechanical ventilation" - ***Dr. Niren Bhavsar***
09.15 AM	Albumin - consensus & controversies - ***Dr. Vipul Thakkar***
09.30 AM	Sedation in ICU - ***Dr. Hiren Dholakia***
09.45 AM	Looking beyond skin? Offence vs defence - ***Dr. Bhagyesh Shah***
10.00 AM	Role of parental nutrition in ICU - ***TBA***
10.15 AM	Glycemic Control in critically ill: How much tight or loose is fair? - ***Dr. Prakash Jiandani***
10.30 AM	**Tea Break**
10.45 AM	Sepsis Update 2011 - Any Change in Practice - ***Dr. Ashit Hegde***
11.00 AM	Medical Issues in TBI Patients - ***Dr. Kavita Kamineni***
11.15 AM	RRT in ICU - Which? When? How long? - ***Dr. Ashwin Mani***
11.30 AM	VAP Bundles. preventive strategies- are we following? - ***Dr. Nitesh Shah***
11.45 AM	**Panel Discussion**
12.00 PM	Hemoptysis - ***Dr. Amit Patel***
12.15 PM	Updates on Sleep Medicine - ***Dr. Bhavin Dalal***
12.30 PM	Fungi - She gives me fever.... - ***Dr. Abdul Ghafur***
12.45 PM	**Panel Discussion**
01.00 PM	**Lunch**
02.00 PM	Tropic Thunder - Tropical Fever - ***Dr. Ashwin Mani***
02.15 PM	Management of Acute Heart Failure - ***Dr. Ajay Naik***
02.30 PM	**Panel Discussion :** Cost effectiveness in ICU - ***All faculty***
02.45 PM	**Panel Discussion :** DNR / DNE - Consent Practice & Law Status - ***All faculty***

Certification Courses - Day-3, January 8, 2012, Sunday

(E) Neonatal & Pediatric Critical Care - Venue : The Grand Bhagwati

08.30 AM	WELCOME NOTE - ***Dr. Kashyap Sheth/Dr. Amit Chitaliya/Dr. Ashwin Sanghvi***
	Critical Care Symposia - I
08.45 AM	Does Diabetic Ketoacidosis still require hectic calculations ?? Recent guidelines - ***Dr. Shalmi Mehta***
09.00 AM	Febrile Neutropenia - is it so simple? - ***Dr. Eva Bhagat***
09.15 AM	Interesting puzzles in field of Pediatric Neurology - ***Dr. Varsha Tripathi***
	Essential Pediatrics Symposia
09.35 AM	Neonatal and infantile metabolic disorders- CATCH ME IF YOU CAN ! - ***Dr. Mamta Muranjan***
09.55 AM	Recognition & stabilization of critically ill child - ***Dr. Baldev Prajapati***
10.10 AM	**Panel Discussion:** Multidisciplinary approach to pediatric critical care in today's era: Where do we stand? **Panel Moderator:** ***Dr. Maulik Shah*** **Panelist:** ***Dr. Mamta Muranjan, Dr. Darshana Naik, Dr. Krishnamoorthy Nijuvade, Dr. Parimal Tripathi, Dr. Raj Shah***
10.20 AM	**Tea Break**
	Critical Care Symposia - II
10.35 AM	Role of bedside sonography of skull : an important NICU tool of current era ... - ***Dr. Krishnamoorthy Niduvaje***
10.55 AM	Approach to child with shock: Stating 2011 status - ***Dr. Raju C. Shah***
11.15 AM	**Panel Discussion:** Alas ! Many fluids in bag - which one to use ? Right selection of fluid in right time **Panel Moderator:** ***Dr. Vivek Uppal*** **Panelist:** ***Dr. Raju C. Shah, Dr. Hiren Trivedi, Dr. Krishnamoorthy Nijuvade, Dr. Amit Chitaliya, Dr. Baldev Prajapati***
	Cardiorespiratory Symposia
11.25 AM	Approach to case of asthma in under 5 year age group - ***Dr. Vijayasekaran***
11.45 AM	Myths and facts about congenital heart disease - ***Dr. Milan Chag***
12.05 PM	**Panel Discussion:** Breath in- Breath out - Chest disease are our daily practice but many question still- let's ask experts ! **Panel Moderator:** ***Dr. Vishal Kacchhi*** **Panelist :** ***Dr. Vijayasekaran , Dr. Shaunak Shah, Dr. Nitesh Shah, Dr. Amar Shah, Dr. Kashyap Sheth, Dr. Ajay Naik***
	Neonatal Symposia
12.20 PM	Outcome in very Low Birth Weight Babies : past, present & future - ***Dr. Krishnamoorthy Niduvaje***
12.40 PM	Respiratory distress syndrome : to tube or not to tube? - ***Dr. Amit Chitaliya***
01.00 PM	**Lunch**
	Need of the hour
01.45 PM	Medico legal aspects while practicing critical care - ***Dr. Mahendra Joshi***
	Perinatology Symposia
02.00 PM	Role of fetal echocardiography - worth peeping in future of forthcoming life : Message on basis of outcomes - ***Dr. Kashyap Sheth***
02.20 PM	Changing need of obstetrician - IVF to surrogacy - A bird's view upon perinatology aspects - ***Dr. Himanshu Bavishi***
02.40 PM	Recent advance in developing perinatology: A combined approach from obstetrician to neonatologist - ***Dr. Amit Chitaliya***
03.00 PM	Glimpse of recent neonatal resuscitation protocol - ***Dr. S. N. Vani***
03.15 PM	Surfactant Therapy - past, present and future - ***Dr. Dharampuri Vidyasagar***
03.35 PM	Early stimulation therapy for newborn! Need of an hour....are we ready for it?? - ***Dr. Amola Patel***
03.50 PM	**Panel Discussion:** Decision making during high risk deliveries **Panel Moderator:** ***Dr. Jayshree Sheth*** **Panelist :** ***Dr. Mahesh Gupta, Dr. Nayna Patel, Dr. Chirag Amin, Dr. N. Bhattacharya, Dr. Dharampuri Vidyasagar, Dr. Krishnamoorthy Niduvaje***
	HANDS ON Workshops : Time :- 04.15 PM Onwards
	Dr. Amit Chitaliya, Dr. Vijayasekaran, Dr. Kashyap Sheth, Dr. Jayul Kamdar, Dr. Niren Bhavsar
Table-1	Role of Bronchoscopy in PICU - ***Dr. Amit Chitaliya, Dr. Vijayasekaran***
Table-2	Bedside intercostal drain placement - Hands on Course : on Mannequin - ***Dr. Jayul Kamdar***
Table-3	Bedside tools of cardiology - let us read ECG - X RAY and learn defibrillation - ***Dr. Kashyap Sheth, Dr. Joyal Shah***
Table-4	Invasive monitoring and procedure in ICU - hey! we only have to do it - Peritoneal dialysis , CVP line , PICC Line & arterial line insertion - ***Dr. Amit Chitaliya, Dr. Niren Bhavsar, Dr. Keyur Bhalavat***

Year in Cardiology: An Update for Physician (How Doctors should Change)

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Mr. Parth Parikh, 3rd Year Medical Student

"Cloud Computing" - we all should learn (KBC/KBI)

Dr. Keyur Parikh, Dr. Apurva Patel, Dr. Roosha Parikh, Mr. Shivam Parikh, Mr. Parth Parikh
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Communications strategies and technology continue to change at an increasingly rapid rate affecting all aspects of society. One only needs to reflect on the global penetration of cell phones to bring that point home. Recent data indicate that traditional phones in the home are an increasingly endangered species, having been replaced by the cell phone!

Likewise, PDAs and tablets are replacing computers not only because of their portability, but also because of the many applications they offer for multitasking and overall rapid communication using one of several techniques (phone call, e-mail, text, and so on).

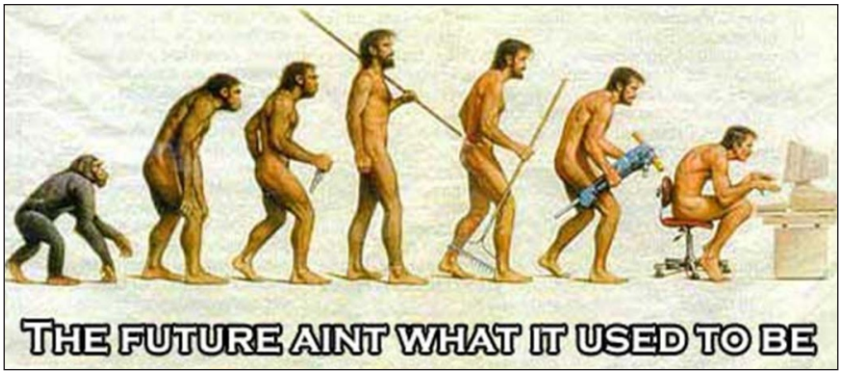
Changes in communication technology and their rapid and widespread adoption have profound implications for the transmission of medical information. An increasingly large number of platforms are now available, forming a "cloud" of new technology.

Cloud computing provides applications, databases, file servers, e-mail,and so on via a computer network, so that desktops have minimal software requirements and instead act primarily as display units.The continued evolution of this cloud of new technologies will permeate medical education. Medical meetings, such as live face-to-face programs and annual scientific sessions, have been the most common means of educating physicians in recent years and have several important functions.

The exchange of information, comments and criticisms can be performed in real time, providing agility in science disclosure. Healthcare professionals make use of information everyday and it is possible that the iPad having access to many Apps like Human Body 3D

Anatomy, Medical Lab Test, and ECG Guide can help streamline processes, as well as provide doctors and other healthcare professionals with the tools they need to diagnose and treat disease. The trend for the future of documents, both from professionals or enterprises, is the "cloud computing", in which all documents will be developed and updated with the use of various equipments: computer, palm, net book, iPad, without software installed on the computer. Clinical information technologies now sporadically available will soon be in routine clinical use, bringing many changes to healthcare. For example, the next generation Internet; real-time clinical decision support systems; off-line, population-based systems; large, integrated, individual patient-level phenotypic and genotypic databases with intelligent data mining

capabilities; wireless, invasive and non-invasive physiologic monitoring devices, have the potential to impact significantly the future healthcare delivery system.



The iPad is a line of tablet computers designed, developed and marketed by Apple Inc., primarily as a platform for audio-visual media including books, periodicals, movies, music, games, and web content. The iPad was introduced on January 27, 2010 by Apple's then-CEO Steve Jobs.The iPad runs the same operating system as the iPod Touch and iPhone—and can run its own applications as well as iPhone applications. The talk will cover various areas where future will take us.

- Doubling time for computer power is 18 months.Millions of chips will be scattered into our world
- The computer will be everywhere, and nowhere.

Arterial Hypertension–Diabetes–Heart Failure–Renal Nerves and Renal Denervation

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Dr. Roosha Parikh
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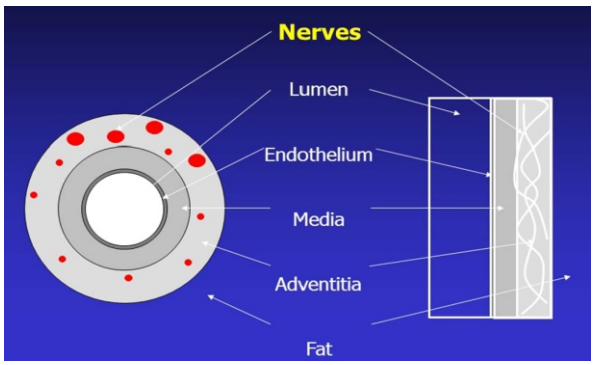
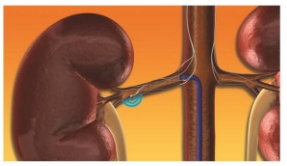
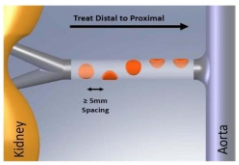
Resistant hypertension is defined as failure to achieve goal blood pressure (BP) when a patient adheres to the maximum tolerated doses of 3 antihypertensive drugs including a diuretic Single largest contributor to death worldwide. Every 20/10 mmHg increase in BP correlates with a doubling of 10-year cardiovascular mortality. Dramatically increases risk of stroke, heart attack, heart failure, & kidney failure. Only half of all treated hypertensives are controlled to established BP targets



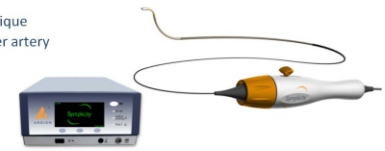
The method of action of RDN (Renal Denervation) therapy is supported by surgical hypertension treatments, called nonselective surgical sympathectomy, which were highly invasive and involved open surgery to sever the sympathetic nerves leading to the kidneys.





This surgery was shown to effectively lower blood pressure yet caused significant side effects. The technique was discontinued as antihypertensive drugs became more affordable and accessible. The Simplicity catheter delivers RF waves to 4–6 locations in each of the two renal arteries, aiming to disrupt the nerves and lower BP

- Standard interventional technique
- 4-6 two-minute treatments per artery
- Proprietary RF Generator
  - Automated
  - Low-power
  - Built-in safety algorithms



The Various studies will be analyzed and discussed in the presentation. Newer applications of the Renal Denervation technology will be discussed.

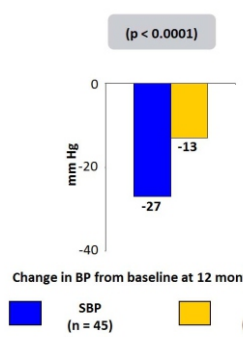
Proven Clinical Outcomes after Renal Denervation:

Renal denervation has been shown to:

- Reduce systemic sympathetic activation
- Reduce congestion (fluid overload) and CHF
- Induce LVH regression & ventricular remodeling
- Improve renal function
- Decrease arterial stiffness
- Reduce arrhythmias
- Reduce hypertension
- Reduce Glucose Tolerance
- Reduce Insulin resistance
- Improve Obstructive Sleep Apnea

SYMPLICITY I

Trial design: Patients with refractory hypertension underwent radiofrequency ablation (RFA) of the renal sympathetic nervous system. BP response at 12 months was monitored.



Results

- Mean procedure time: 38 minutes; complications noted in 2 patients
- Significant reduction in systolic (SBP) and diastolic blood pressure (DBP) at 12 months, as compared with baseline (27 and 13 mm)
- Overall response rate: 89%

Conclusions

- First-in-man trial demonstrates significant reduction in SBP and DBP with RFA of renal sympathetic nervous system in patients with refractory hypertension
- Further randomized data are awaited

Presented by Dr. Henry Krum at TCT 2009

A-Z Trials of 2011 in Cardiology- Part- 1

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There are many trials of importance, but I am presenting a few landmark trials which can change our day to day clinical practice or help us to understand the disease process:

(1) Eplerenone in Patients with Systolic Heart Failure and Mild Symptoms : EMPHASIS HF Study: (Faiez Zannad et al. N Engl J Med 2011;364:11-21)

Background

Mineralocorticoid antagonists improve survival among patients with chronic, severe systolic heart failure and heart failure after myocardial infarction. We evaluated the effects of eplerenone in patients with chronic systolic heart failure and mild symptoms.

Methods

In this randomized, double-blind trial, we randomly assigned 2737 patients with New York Heart Association class II heart failure and an ejection fraction of no more than 35% to receive eplerenone (up to 50 mg daily) or placebo, in addition to recommended therapy. The primary outcome was a composite of death from cardiovascular causes or hospitalization for heart failure.

Results

The trial was stopped prematurely, according to prespecified rules, after a median follow-up period of 21 months. The primary outcome occurred in 18.3% of patients in the eplerenone group as compared with 25.9% in the placebo group (hazard ratio, 0.63; 95% confidence interval [CI], 0.54 to 0.74; P<0.001). A total of 12.5% of

patients receiving eplerenone and 15.5% of those receiving placebo died (hazard ratio, 0.76; 95% CI, 0.62 to 0.93; P = 0.008); 10.8% and 13.5%, respectively, died of cardiovascular causes (hazard ratio, 0.76; 95% CI, 0.61 to 0.94; P = 0.01). Hospitalizations for heart failure and for any cause were also reduced with eplerenone. A serum potassium level exceeding 5.5 mmol per liter occurred in 11.8% of patients in the eplerenone group and 7.2% of those in the placebo group (P<0.001).

Conclusions

Eplerenone, as compared with placebo, reduced both the risk of death and the risk of hospitalization among patients with systolic heart failure and mild symptoms.

(2) Safety and efficacy of dalcetrapib on atherosclerotic disease using novel non-invasive multimodality imaging: a randomised clinical trial: dal-PLAQUE trial: (Zahi A Fayad et al. Lancet 2011; 378: 1547–59)

Background

Dalcetrapib modulates cholesteryl ester transfer protein (CETP) activity to raise high-density lipoprotein cholesterol (HDL-C). After the failure of torcetrapib it was unknown if HDL produced by interaction with CETP had pro-atherogenic or pro-infl ammatory properties. dal-PLAQUE is the fi rst multicentre study using novel non-invasive multimodality imaging to assess structural and infl ammatory indices of atherosclerosis as primary endpoints.

Methods

In this phase 2b, double-blind, multicentre trial, patients (aged 18–75 years) with, or with high risk of, coronary heart disease were randomly assigned (1:1) to dalcetrapib 600 mg/day or placebo for 24 months. Randomisation was done with a computer-generated randomisation code and was stratifi ed by centre. Patients and investigators were masked to treatment. Coprimary endpoints were MRI-assessed indices (total vessel area, wall area, wall thickness, and normalised wall index [average carotid]) after 24 months and 1^{8}F-fl uorodeoxyglucose (1^{8}F-FDG) PET/CT assessment of arterial infl ammation within an index vessel (right carotid, left carotid, or ascending thoracic aorta) after 6 months, with no-harm boundaries established before unblinding of the trial. Analysis was by intention to treat.

Findings

189 patients were screened and 130 randomly assigned to placebo (66 patients) or dalcetrapib (64 patients). For the coprimary MRI and PET/CT endpoints, CIs were below the no-harm boundary or the adverse change was numerically lower in the dalcetrapib group than in the placebo group. MRI-derived change in total vessel area was reduced in patients given dalcetrapib compared with those given placebo after 24 months; absolute change from baseline relative to placebo was –4 • 01 mm2 (90% CI –7 • 23 to –0 • 80; nominal p=0 • 04). The PET/CT measure of index vessel most-diseased-segment target-to-background ratio (TBR) was not diff erent between groups, but carotid artery analysis showed a 7% reduction in most-diseased-segment TBR in the dalcetrapib group compared with the placebo group (–7 • 3 [90% CI –13 • 5 to –0 • 8]; nominal p=0 • 07). Dalcetrapib did not increase offi ce blood pressure and the frequency of adverse events was similar between groups.

Interpretation

Dalcetrapib showed no evidence of a pathological effect related to the arterial wall over 24 months. Moreover, this trial suggests possible beneficial vascular effects of dalcetrapib, including the reduction in total vessel enlargement over 24 months, but long-term safety and clinical outcomes efficacy of dalcetrapib need to be analyzed.

(3) A Prospective Natural-History Study of Coronary Atherosclerosis: PROSPECT study. (Gregg Stone et al. N Engl J Med 2011;364:226-35)

Background

Atherosclerotic plaques that lead to acute coronary syndromes often occur at sites of angiographically mild coronary-artery stenosis. Lesion-related risk factors for such events are poorly understood.

Methods

In a prospective study, 697 patients with acute coronary syndromes underwent three-vessel coronary angiography and gray-scale and radiofrequency intravascular ultrasonographic imaging after percutaneous coronary intervention. Subsequent major adverse cardiovascular events (death from cardiac causes, cardiac arrest, myocardial infarction, or rehospitalization due to unstable or progressive angina) were adjudicated to be related to either originally treated (culprit) lesions or untreated (nonculprit) lesions. The median follow-up period was 3.4 years.

Results

The 3-year cumulative rate of major adverse cardiovascular events was 20.4%. Events were adjudicated to be related to culprit lesions in 12.9% of patients and to non-culprit lesions in 11.6%. Most non-culprit lesions responsible for follow-up events were angiographically mild at baseline (mean [±SD] diameter stenosis, 32.3±20.6%). However, on multivariate analysis, non-culprit lesions associated with recurrent events were more likely than those not associated with recurrent events to be characterized by a plaque burden of 70% or greater (hazard ratio, 5.03; 95% confidence interval [CI], 2.51 to 10.11; P<0.001) or a minimal luminal area of 4.0 mm2 or less (hazard ratio, 3.21; 95% CI, 1.61 to 6.42; P = 0.001) or to be classified on the basis of radiofrequency intravascular ultrasonography as thin-cap fibroatheromas (hazard ratio, 3.35; 95% CI, 1.77 to 6.36; P<0.001).

Conclusions

In patients who presented with an acute coronary syndrome and underwent percutaneous coronary intervention, major adverse cardiovascular events occurring during follow-up were equally attributable to recurrence at the site of culprit lesions and to nonculprit lesions. Although nonculprit lesions that were responsible for unanticipated events were frequently angiographically mild, most were thin-cap fibroatheromas or were characterized by a large plaque burden, a small luminal area, or some combination of these characteristics, as determined by gray-scale and radiofrequency intravascular ultrasonography.

(4) Rivaroxaban in Patients with a Recent Acute Coronary Syndrome: ATLAS ACS 2–TIMI 51 Investigators.(Jessica L. Mega et al. N Engl J Med 2011, Nov 13.)

Background

Acute coronary syndromes arise from coronary atherosclerosis with superimposed thrombosis. Since factor Xa plays a central role in thrombosis, the inhibition of factorXa with low-dose rivaroxaban might improve cardiovascular outcomes in patients with a recent acute coronary syndrome.

Methods

In this double-blind, placebo-controlled trial, we randomly assigned 15,526 patients with a recent acute coronary syndrome to receive twice-daily doses of either 2.5 mg or 5 mg of rivaroxaban or placebo for a mean of 13 months and up to 31 months. The primary efficacy end point was a composite of death from cardiovascular causes, myocardial infarction, or stroke.

Results

Rivaroxaban significantly reduced the primary efficacy end point, as compared with placebo, with respective rates of 8.9% and 10.7% (hazard ratio in the rivaroxaban group, 0.84; 95% confidence interval [CI], 0.74 to 0.96; P = 0.008), with significant improvement for both the twice-daily 2.5-mg dose (9.1% vs. 10.7%, P = 0.02) and the twice-daily 5-mg dose (8.8% vs. 10.7%, P = 0.03). The twice-daily 2.5-mg dose of rivaroxaban reduced the rates of death from cardiovascular causes (2.7% vs. 4.1%, P = 0.002) and from any cause (2.9% vs. 4.5%, P = 0.002), a survival benefit that was not seen with the twice-daily 5-mg dose. As compared with placebo, rivaroxaban increased the rates of major bleeding not related to coronary-artery bypass grafting (2.1% vs. 0.6%, P<0.001) and intracranial hemorrhage (0.6% vs. 0.2%, P = 0.009), without a significant increase in fatal bleeding (0.3% vs. 0.2%, P = 0.66) or other adverse events. The twice-daily 2.5-mg dose resulted in fewer fatal bleeding events than the twice-daily 5-mg dose (0.1% vs. 0.4%, P = 0.04).

Conclusions

In patients with a recent acute coronary syndrome, rivaroxaban reduced the risk of the composite end point of death from cardiovascular causes, myocardial infarction, or stroke. Rivaroxaban increased the risk of major bleeding and intracranial hemorrhage but not the risk of fatal bleeding.

(5) Effect of Two Intensive Statin Regimens on Progression of Coronary Disease. SATURN. (Stephen J. Nicholls et al. N Engl J Med 2011, Nov 15)

Background

Statins reduce adverse cardiovascular outcomes and slow the progression of coronary atherosclerosis in proportion to their ability to reduce low-density lipoprotein (LDL) cholesterol. However, few studies have either assessed the ability of intensive statin treatments to achieve disease regression or compared alternative approaches to maximal statin administration.

Methods

We performed serial intravascular ultrasonography in 1039 patients with coronary disease, at baseline and after 104 weeks of treatment with either atorvastatin, 80 mg daily, or rosuvastatin, 40 mg daily, to compare the effect of these two intensive statin regimens on the progression of coronary atherosclerosis, as well as to assess their safety and side-effect profiles.

Results

After 104 weeks of therapy, the rosuvastatin group had lower levels of LDL cholesterol than the atorvastatin group (62.6 vs. 70.2 mg per deciliter [1.62 vs. 1.82 mmol per liter], P<0.001), and higher levels of high-density lipoprotein (HDL) cholesterol (50.4 vs. 48.6 mg per deciliter [1.30 vs. 1.26 mmol per liter], P = 0.01). The primary efficacy end point, percent atheroma volume (PAV), decreased by 0.99% (95% confidence interval [CI], −1.19 to −0.63) with atorvastatin and by 1.22% (95% CI, −1.52 to −0.90) with rosuvastatin (P = 0.17). The effect on the secondary efficacy end point, normalized total atheroma volume (TAV), was more favorable with rosuvastatin than with atorvastatin: −6.39 mm3 (95% CI, −7.52 to −5.12), as compared with −4.42 mm3 (95% CI, −5.98 to −3.26) (P = 0.01). Both agents induced regression in the majority of patients: 63.2% with atorvastatin and 68.5% with rosuvastatin for PAV (P = 0.07) and 64.7% and 71.3%, respectively, for TAV (P = 0.02). Both agents had acceptable side-effect profiles, with a low incidence of laboratory abnormalities and cardiovascular events.

Conclusions

Maximal doses of rosuvastatin and atorvastatin resulted in significant regression of coronary atherosclerosis. Despite the lower level of LDL cholesterol and the higher level of HDL cholesterol achieved with rosuvastatin, a similar degree of regression of PAV was observed in the two treatment groups.

PL-1: Acute Myocardial Infarction: From Pathophysiology to Therapeutic Targets

Prof. Guy R Heyndrickx, MD, PhD, Cardiovascular Center Aalst, Belgium

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Acute myocardial infarction occurs most often as a result of a thrombotic complication at the site of aruptured vulnerable plaque. Myocardial ischemia, if permanent, will evolve towards lethal myocytenecrosis and irreversible scar formation with permanent loss in LV function as a result. Reperfusion, if timely, will restore coronary blood flow and tissue oxygenation thereby halting and avertingongoing ischemic injury and limiting infarct size but at the same time induce some degree of reperfusion injury which may in part offset the gain of re-oxygenation.

The degree of ischemic injury will depend on the extent and duration of the ischemia. Mitochondrial permeability transition pores (mPTP) have recently been identified as a major cause for cell death and reperfusion injury.A sudden release of reactive oxygen species and calcium overloadat the time of maximal hyperemiaduring reperfusion results in opening of the mitochondrial permeability transition pores(mPTP) which are closed during ischemia.

On opening of mPTP,uncoupling of oxidative phosphorylation, ATP depletion, mitochondrial swelling and necrotic cell death are observed. Multiple interventions at the time of reperfusion aiming at reducing reperfusion injury have been studied without a great deal of success. Recently, ischemic post conditioning induced by intermittent coronary occlusions during the first minute of reperfusion has been shown to significantly reduce infarct size, both experimentally as well as in patients.Controlled, slow reperfusion as opposed to abrupt reperfusionwill delay the recovery of intracellular acidosis byinhibiting the sodium-hydrogen pump which will protect the mPTP from opening thereby protecting the mitochondria and the myocytes from lethal injury. In other words, post conditioning delays Ca++ induced mPTP opening. Several additional intracellular pathwaystriggered by adenosine receptors have been described involved in post-conditioning.

PL- 2: Antiplatelet Therapy in ACS: Emerging Evidence

Prof. Lars Wallentin, Uppsala Clinical Research Centre, Uppsala University, Sweden

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Acute coronary syndrome (ACS) remains one of the most common reasons for acute hospital care and mortality in developed countries, and its prevalence is rising in developing countries. Over the last 20 years, the rapid development of new and effective treatments has reduced short-term mortality by half and led to an increase in long-term survival in patients with ACS.

Current management of ACS includes risk stratification by clinical findings and the use of ECG and biochemical markers. It is now recommended that all patients with an established diagnosis of ACS receive immediate antithrombotic treatment with dual platelet inhibition (aspirin and a P2Y12 inhibitor) plus intravenous or subcutaneous anticoagulation. In addition, most patients also receive beta-blockers, statins and, frequently, angiotensin-converting enzyme (ACE) inhibitors. The majority of patients hospitalized for ACS are rapidly admitted to a catheterization laboratory for identification of the culprit lesion, followed by balloon dilatation and stenting if feasible. At discharge it is generally recommended that patients receive long-term secondary prevention with a combination of aspirin, beta-blockers, statins, ACE inhibitors, and P2Y12 inhibitors for at least a year. Despite these measures, however, there is still a 10% risk of death, re-infarction or stroke during the year following discharge. This increase in risk varies among patient populations, with the highest risk seen in older patients and those with diabetes mellitus, previous myocardial infarction, cardiac or renal dysfunction, manifestations of atherosclerotic disease, or multi-vessel coronary artery disease. If current therapeutic approaches for ACS are to be improved, greater focus will need to be placed on these high-risk groups.

Platelet inhibition is since around 20 years a mainstay in the prevention of myocardial infarction and death in patients with acute coronary syndrome. Aspirin therapy yields consistent inhibition of platelet thromboxane A2 release. However, inhibiting this pathway only modestly attenuates platelet activation without any influence e.g. on ADP induced platelet activation. Aspirin treatment provides a relative reduction of the risk of myocardial infarction and death by 30 – 50 % as compared to placebo in patients with acute coronary syndrome. However, aspirin alone has no convincing effect on prevention of stent thrombosis. Therefore also other pathways needs to be inhibited in the highly prothrombotic of acute coronary syndrome. The P2Y12 receptor plays a major role in the ADP mediated amplification of platelet response regardless of the stimulus. Clopidogrel and prasugrel are thienopyridine prodrugs acting on this receptor by almost identical active metabolites, which irreversibly bind to the receptor. Slow and variable active metabolite generation leads to clopidogrel having a slow onset of action and wide inter-individual variability in the pharmacodynamic response.(5) However, as compared to aspirin alone, clopidogrel provides around a relative 20% reduction in myocardial infarction and also a substantial reduction in stent

thrombosis in patients with acute coronay syndrome as shown in the CURE trial. The thienopyridine prasugrel has a more efficient active metabolite generation than clopidogrel with a rapid onset of action, more pronounced platelet inhibition and no clinically important variability in response. These features therefore lead to a further around 20% reduction in myocardial infarction and around halving of the risk of stent thrombosis in the TRITON-TIMI 38 trial randomizing patients with acute coronary syndrome to prasugrel versus clopidogrel in association with a planned PCI procedure. However, both the addition of clopidogrel to aspirin and the use of prasugrel instead of clopidogrel were associated with a significant increase in major bleeding.

Ticagrelor, the first reversibly binding oral P2Y12 receptor antagonist, does not require metabolic activation, has a rapid onset of action, and can disassociate from the receptor, permitting restoration of platelet function without the need for production of new platelets. In pharmacodynamic studies, ticagrelor have demonstrated greater, more rapid, and more consistent ADP induced platelet inhibition as compared to clopidogrel and more rapid offset of action following cessation of therapy. In the PLATO Study 18 624 patients with acute coronary syndrome were randomized within 24 hours after symptom onset to ticagrelor versus clopidogrel. The results showed a 16% relative reduction of the composite of CV death, myocardial infarction and stroke, a 22 % reduction in total mortality and also a reduction in stent thrombosis. Ticagrelor was not associated with any increase in overall bleeding but during long-term treatment there were more non-procedural bleeding with ticagrelor.

Other targets for platelet inhibition are also under investigation e.g. the protease-activated receptor 1 (PAR1). Preclinical and phase II studies suggested that consistent and high levels of PAR1 inhibition have a beneficial antithrombotic effect with minimal increase in bleeding. Recently the Phase III placebo controlled study of the selective PAR1 inhibitor vorapaxar in 12,944 patients with acute coronary syndromes (TRACER) treated with aspirin and clopidogrel was presented. The study did not meet its primary objective to show superiority concerning the composite of CV-death, myocardial infarction, stroke and urgent revascularization. However, there was a significant reduction in myocardial infarction and stent thrombosis. Disappointingly the treatment was associated with a significant 35% increase in major bleeding and a 3-4-fold increase in intracranial hemorrhage.

In conclusion, several new alternatives providing more rapid and consistent platelet inhibition than clopidogrel are currently tested and introduced treatment of patients with acute coronary syndrome. Some of these new treatments seem to provide additional benefits to the patients without undue increments in the risk of bleeding if used for the appropriate patients.

CAD IN VERY YOUNG

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Coronary artery disease (CAD) is acts like a tsumai because an otherwise healthy person may die or become disabled without warning. When The affected individual is under the age of 40, the tragic consequences for loved ones are particularly catastrophic and unexpected.

Majority of CAD population in India present with malignant form and significantly affecting the young ones i.e. less than 40 years of age. The increase of CAD among Indians is observed throughout the country, as well as among Indian immigrants in different parts of the world. CAD epidemic in India has entered into an epidemiological transition phase. At present 25 per cent death among Indians are attributable to CAD. With the present trend, mortality from CAD will increase by 103 per cent in male and 90 per cent in female from 1985 to 2015. By 2015, CAD will account for 34 per cent all male death and 32 per cent all female death in India.

Combination of abdominal obesity, impaired glucose tolerance, Hyperinsulinaemia, raised Triglyceride, decreased HDL, hypertension known as Syndrome X constitutes a potential risk for CAD among Indians. Emerging Risk Factors: Newer risk factors constituted by a complex interaction due to Genetic predisposition and environment. Genetic predisposition with various Gene Polymorphism, ACE gene, Elevated Lp(a), deranged Endothelial Nitric Acid Synthase (ENOS), elevated level of GP-III, etc, are associated with higher CAD. Other non-lipid risk factors are Hyper-homocysteinemia, elevated Fibrinogen level, infection, inflammation etc. Changing life-style, urbanisation, rural immigration to city life, lack of Physical activity etc are some of the potential risk factors for recent increase of CAD among Indians. Lp(a) seems to be an independent risk factor for premature CAD, specially with high level of LDL-C. Indians have higher (, 30mg/dl) levels of Lp(a) compared to Europeans and Chinese . In young patients with a single culprit lesion, and among most women (especially those with dissection or coronary spasm), a plaque rupture on a previously nonsignificant vulnerable plaque is usually the mechanism of acute presentation. Such cases are likely related to acute physical and/or emotional stress, resulting in enhanced coronary shear forces. If these patients do not alter their lifestyles, CAD progression at an earlier age than usual will result, but it may be avoided by following established preventive measures. This group has a substantial vasospastic component superimposed on a genetic predisposition to vulnerable plaque.

Conversely few subsets are composed of those with diabetes and others who present with established multivessel disease (including those related to lipid abnormalities).These patients are most likely to have rapid progression of a more typical form of CAD. They will have only short-term benefit from revascularization, unless very aggressive control over risk factors is strictly adhered to.The interaction between a genetic propensity to form vulnerable plaque combined with acute stress and/or an active infectious/inflammatory process needs further study.

It is prudent to categorize the pathophysiology of disease in this population for better understanding of CAD.

CAD in Women

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Despite numerous studies on women's cardiac health within last 20 years, the number of female deaths caused by cardiovascular disease keeps on rising and remains the leading cause of death in women in most areas of the world. Pathology, pathophysiology and disease presentation in women are different than those in men. This leads to difficulties concerning diagnosis, treatment and outcome of female population. Overall women have lower rates of anatomical coronary artery disease (CAD) but more symptoms, ischemia and adverse outcomes. Women have much higher prevalence of endothelial dysfunction and heart failure symptoms with preserved systolic function. Novel risk factors like inflammatory markers and reproductive hormones as well as noninvasive imaging and functional capacity measurements can help risk stratification in women, in addition to existing methods. Risk for women with obstructive CAD is increased compared with men still women are less likely to receive guideline-indicated therapies. While for women with evidence of ischemia but no obstructive CAD, antianginal/ antiischemic therapies can improve symptoms, endothelial function and quality of life; trials evaluating impact on adverse outcomes are needed. Future investigations and research should identify tailored diagnostic and therapeutic strategies to optimize outcomes for women.

Non-invasive tools: CACS

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Coronary artery disease is unfortunately is a leading cause of morbidity and mortality in the world. There are a number of ways to predict a patient's risk of a cardiac event, the most well known being the Framingham Risk Score. Unfortunately such scores do not identify all "at risk" patients. The provision of an additional non-invasive method of determining coronary disease at an early stage may help us combat some of these problems.

Coronary artery disease results from the development of atherosclerotic plaques. These plaques consist of abnormal macrophages that accumulate lipids and abnormalities of the endothelium. These plaques can become calcified - which is a common finding. Cardiac risk factors and insulin resistance lead to progression of coronary artery calcification - this may help in deciding who should be investigated with this methodology.

The CACS is a measure of the calcium in the the coronary arteries. The calcium is detected by computerized tomographyscanning. The presence of calcium in the coronary tree is diagnostic of the presence of coronary atherosclerosis. The CACS is measured using CT scanning. This is non-invasive and can be of two

types: Electron Beam CT scan (EBCT) and Multidetector CT scan (MDCT). EBCT is the commonest technology currently used to measure CACS.The amount of calcium detected in the coronary arteries is converted to a calcium score which correlates with the severity of the blockage. The results are reported on a numerical scale. The numerical scale is continuous.

Coronary Artery Calcium Score (CACS)

Coronary Artery Calcium Score (CACS) method of reporting results and their interpretation	
CAC	Interpretation
0	Negative test - i.e. significant obstructive luminal disease highly unlikely
>100	High calcium score - i.e. above average risk of a cardiac event in the next five years
>1000	Very high calcium score - i.e. high risk of a cardiac event

So who should be considered for CAC scans

At present there are no guidelines available however, with more and more studies being performed on its usefulness the following groups are probably most likely to benefit:

- Asymptomatic middle aged men and women (men from 45 years of age and women from 55 years of age).
- Presence of risk factor
- Presence of a strong family history of premature coronary artery disease.

Non-invasive tools: C-IMT

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Carotid intima media thickness (CIMT) is basically noninvasive (B-sound Ultrasonography) test which visualizes carotid intima and is the only test which gives objective evidence of atherosclerosis. It is mainly indicated for intermediate risk group of patients so as to identify high risk patients, so as aggressive preventive treatment strategies can be applied in this patient and reduce CV events.

"Transcatheter Aortic Valve Replacement- A Reality"

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Severe aortic stenosis (AS) is a rapidly progressive disease, once patient becomes symptomatic. Average survival is 5 years after onset of angina, 3 years after onset of syncope and 2 years after onset of heart failure. Surgical treatment is the standard approach if patient has severe AS.

Many patients with severe aortic stenosis and coexisting conditions are not candidates for surgical replacement of the aortic valve. Recently, transcatheter aortic-valve implantation (TAVI) has been suggested as a less invasive treatment for high-risk patients with aortic stenosis.

PARTNER was the randomized trial to assess the safety and effectivenessof TAVI compared with standard therapy, inpatients with severe aortic stenosis and cardiacsymptoms, who cannot undergo surgery("inoperable"), using rigorous evidence-basedclinical trial methodologies. It clearly showed that in such high risk group, compared to medical therapy with or without simple balloon aortic valvuloplasty, TAVI reduced all cause mortality by 49% and risk of mortality or hospitalization by 54%. Among survivors at 1 year, the rate of cardiac symptoms (New York Heart Association class III or IV) was lower among patients who had undergone TAVI than among those who had received standard therapy (25.2% vs. 58.0%, P<0.001). At 30 days, TAVI, as compared with standard therapy, was associated with a higher incidence of major strokes (5.0% vs. 1.1%, P = 0.06) and major vascular complications (16.2% vs.1.1%, P<0.001).

Results for the high risk subgroup of patients in the PARTNER trial who were still candidates for surgical valve replacement and who were randomly assigned to undergo either transcatheter or surgical replacement of the aortic valve are also show TAVI to be non-inferior to surgical AVR.Both TAVR and AVR were associated with important but different peri-procedural hazards:Major strokes at 30 days (3.8 vs. 2.1%, p=0.20) and one year (5.1% vs. 2.4%, p=0.07) and major vascular complications were more frequent with TAVR (11.0% vs. 3.2%, p<0.001). Major bleeding (9.3% vs. 19.5%, p<0.001) and new onset atrial fibrillation (8.6% vs. 16.0%, p<0.001) were more frequent with AVR.

TAVR is already the standard-of-care for inoperable patients with severe aortic stenosis. These results indicate that TAVR is an acceptable alternative to AVR in selected high-risk operable patients.Next Clinical Target will be: aortic and mitral valve failure, Lower risk AS patients (? intermediate risk), Mixed AS and CAD patients, Asymptomatic severe AS, Low flow - low gradient AS (impedance mismatch), Aortic regurgitation.

Coronary Stents: Present and Future

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Since coronary stents were first introduced in 1977, progress in this field has been outstanding, with the vast majority of current coronary interventions involving stents. The motivation to introduce coronary stents was both to prevent acute occlusion during balloon angioplasty and to prevent acute recoil, one of the major factors contributing to overall restenosis. While stents achieved both goals from the start, they also led to two new problems: stent thrombosis due to exposed metal struts, and excessive proliferative response due to increased vessel injury, leading to restenosis rates of up to 30%. Stent thrombosis was minimized by high-pressure stent expansion leading to complete wall apposition of the stent struts and the use of appropriate antiplatelet drugs. Restenosis was reduced dramatically with the introduction of drug eluting stents, whichdecreased late loss by 70-90% as compared to late loss when using bare metal stents. This led to an intense research and development effort focused on drug elution. While the first stents used durable polymers that eluteddrugs by diffusion, other solutions included degradable polymers to retain the drugs on the metal surface of the stent.

Research has been directed toward fully absorbable polymer stents for over a decade. However, only recently have drug-loaded degradable polymersbeen suggested as a possible solution to the exaggerative proliferative response observed following previous absorbable stent implantation attempts. The appeal of a stent that will fully resolve in 6-12 months following implantation is clear; however, the new absorbable stents must compete against the high standard set by current and future drug eluting stents. In the future, there may be a variable solution for each patient. It is my proposal that the majority of patients will still be treated with drug-eluting stents. However, a certain proportion of patients will enjoy bare metal stents with enhanced surface properties; some patients may also benefit from absorbable stents. Beyond biology, stent mechanics have improved tremendously over the years;today's stents are much more flexible and can reach high, tortuous, and distal locations. Advances in material sciences will enable manufacture of stents with much thinner struts, a lower insertion profile, higher vessel conformability, and the ability to deal with ostial and bifurcation locations. The role of self-expanding stents for those applications will be tested against the balloon expandable technology that is the only clinical solution for coronary applications today.

In summary, technological, biomedical, and molecular science, provide interventional cardiologists with one of the most robust method for revascularization, a toolthat will remain for decades to come.

Vitamin-D Deficiency: How to Identify Underestimated Risk Factor?

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For years, it has been recognized that vitamin D is a hormone of critical importance to calcium homeostasis and bone health. Vitamin D deficiency is a widely prevalent condition, present in approximately 30 to 50% of general population in many countries of world. Indoor lifestyles and sun avoidance campaigns have decreased sun exposure and contribute heavily towards this high prevalence. A growing body of data suggests that low 25-hydroxyvitamin D levels may adversely affect cardiovascular health. Vitamin D deficiency activates rennin- angiotensin- aldosterone system and can predispose to hypertension and left ventricular hypertrophy. Also, hyper parathyroidism caused by vitamin D deficiency, leads to increased insulin resistance and that in turn; is associated with metabolic syndrome, diabetes, hypertension, vascular inflammation and increased cardiovascular risk. Plenty of epidemiologic studies have linked low 25-hydroxyvitamin D levels with multiple coronary risk factors and adverse cardiovascular outcomes. Though vitamin D supplementation is simple, safe and inexpensive; before advocating routine screening for it and supplementing it to those deficient for cardiovascular health, large randomized, controlled trials are needed. Till then its role and recommendations for optimizing musculoskeletal and general health do exist.

Pulmonary HT: How to manage in 2012?

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Pulmonary hypertension (PH) defined as a mean pulmonary arterial (PA) pressure of greater than 25 mm Hg at rest or greater than 30 mm Hg during exercise, is characterized by a progressive and sustained increase in pulmonary vascular resistance that eventually leads to right ventricular (RV) failure. Pulmonary hypertension is a life-threatening condition if untreated; treatment success rates vary based on etiology. Therapy for pulmonary hypertension is targeted at the underlying cause and its effects on the cardiovascular system. Novel therapeutic agents such as prostacyclin and others undergoing clinical trials have led to the possibility of specific therapies for these once untreatable disorders.

HT & DM: Deadly Duo: How to Optimize Treatment?

Managing Hypertension in Diabetic Patients

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Detecting and managing hypertension in people with diabetes is one of the most effective measures to prevent adverse events, and pharmacotherapy is one of the most effective ways to maintain target BP levels in primary care. Increased blood pressure (BP) is a leading risk factor for death and disability, particularly in people with diabetes. Over the past few years, the incidence of hypertension and diabetes in India is increasing rapidly.

Many classes of drugs have been used in numerous trials to treat patients with hypertension. All classes of drugs have been shown to be superior to placebo in terms of reducing morbidity and mortality. Often, numerous agents (three or more) are needed to achieve specific target levels of BP. Use of almost any drug therapy to reduce hypertension in patients with diabetes has been shown to be effective in decreasing cardiovascular risk. Keeping in mind that numerous agents are often required to achieve the target level of BP control, recommending specific agents becomes a not-so-simple task. The literature continues to evolve, and individual patient conditions and preferences also must come into play.

Combinations of 2 drugs

In general, the drugs prescribed in combination should be selected from initial therapy options. A notable exception is the combination of an ACE inhibitor with an ARB, which has more adverse effects than ACE inhibitor therapy alone and has no therapeutic advantage over either drug alone. As such, ACE inhibitor and ARB combination therapy is specifically not recommended unless there is macroalbuminuria or resistant heart failure, for which the combination might have additional advantages. Studies show that people with hypertension at high cardiovascular risk owing to diabetes or other diseases have a greater risk reduction for cardiovascular events when treatment is with an ACE inhibitor plus a CCB rather than an ACE inhibitor plus a diuretic; physicians should consider this combination in high-risk patients.

Combinations of 3 and 4 drugs

In the absence of contraindications, diuretics are generally advised for patients in whom BP is difficult to control; often, higher doses of diuretic medications are required in resistant hypertension. Maintaining a normal serum potassium level is important to minimize the effect of diuretics on blood glucose levels and to maximize cardiovascular event reductions. Regular monitoring of serum potassium is recommended if

spironolactone is prescribed, especially if baseline serum potassium levels are in the high end of the normal range, if glomerular filtration rate is reduced, or if there is concurrent use of other drugs that cause potassium reduction.

Although multiple drugs are required for BP control, extensive lowering of BP in people with diabetes is one of the very few cost–saving medical interventions (ie, the cost of BP lowering is less than the cost of the complications prevented by BP lowering). Furthermore, quality of life can improve with more intensive BP lowering. Referral to another specialist or primary care physician with expertise in lowering BP should be considered if BP cannot be controlled.

While lowering BP by any means will help to reduce cardiovascular morbidity, there is evidence that may help guide the selection of an antihypertensive regimen. The UKPDS showed no significant differences in outcomes for treatment for hypertension using an ACE inhibitor or a β-blocker. In addition, both ACE inhibitors and angiotensin II receptor blockers (ARBs) have been shown to slow the development and progression of diabetic nephropathy. In the Heart Outcomes Prevention Evaluation (HOPE) trial, ACE inhibitors were found to have a favorable effect in reducing cardiovascular morbidity and mortality, whereas recent trials have shown a renal protective benefit from both ACE inhibitors and ARBs. ACE inhibitors and β-blockers seem to be better than dihydropyridine calcium-channel blockers to reduce MI and heart failure. However, trials using dihydropyridine calcium-channel blockers in combination with ACE inhibitors and β-blockers do not appear to show any increased morbidity or mortality in CVD, as has been implicated in the past for dihydropyridine calcium-channel blockers alone. Recently, the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) in high-risk hypertensive patients, including those with diabetes, demonstrated that chlorthalidone, a thiazide-type diuretic, was superior to an ACE inhibitor, lisinopril, in preventing one or more forms of CVD.

Clinical Pearls

1. Clinical trials demonstrate that drug therapy versus placebo will reduce cardiovascular events when treating patients with hypertension and diabetes.
2. A target BP goal of < 130/80 mmHg is recommended.
3. Pharmacological therapy needs to be individualized to fit patients' needs.
4. ACE inhibitors, ARBs, diuretics, and β-blockers have all been documented to be effective pharmacological treatment.
5. Combinations of drugs are often necessary to achieve target levels of BP control.
6. ACE inhibitors and ARBs are agents best suited to retard progression of nephropathy.

Revascularization Strategies for CAD: PCI or CABG or Just Medicines

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Coronary artery disease (CAD) is the single most common cause of death in the developed and developing world. The morbidity, mortality and socio-economic importance of this disease make timely diagnosis and cost effective management of CAD of the utmost importance. The goals of treatment of CAD involve therapies to prevent/minimize ischemia and to improve survival. These goals can be achieved with variety modalities including medical therapy and revascularization with either percutaneous coronary intervention (PCI) or coronary artery bypass graphed surgery (CABG).

The management strategies for the treatment of CAD have evolved over the years. For the treatment of spectrum of ACS (UA/NSTEMI/STEMI), the debate is less controversial with current emphasis more on early invasive therapies. For the treatment of chronic CAD, the recent trend has been more towards conservative measures with more emphasis on aggressive medical treatment. Studies in recent years like COURAGE (2007) have shown that patients on Optimal Medical Therapy (OMT) have better long-term outcomes than patients treated with invasive therapies.The recognition of the importance of OMT is transforming patient management, both in patients undergoing revascularization and in patients being treated conservatively. Optimal Medical Therapy remains the cornerstone of management in all patients with CAD, as it is logical, inexpensive, and very effective in improving long-term outcomes. The challenge is to implement these measures in all patients.

PL-3: High Cardiac Risk Pregnancies: Natural history and management

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3 million women aged 18 to 44 in the United States have heart disease and about 1% of pregnant women have some type of cardiac problems. Most, heart problems in pregnancy include congenital heart disease (40%), arrhythmias (20%), valvular heart disease (15%), cardiomyopathies (8%), disease of the aorta (6%), ischemic heart disease/myocardial infarction (3%) and others. Pregnant women with heart disease are at increased risk for both neonatal and down cardiovascular complications. Most complications related to heart disease in pregnancy are due to the hemodynamic changes, arrhytmogenic effect and the thrombogenic effect of pregnancy as well as the side effects of drugs. The potential complications include the development of congestive heart failure, arrhythmias, thrombo- embolism and mortality. The predictors of cardiac complications during pregnancy include prior cardiac events including heart failure, TIA, stroke or arrhythmias, New York heart association class higher than II, cyanosis, left heart obstruction and systemic ventricular dysfunction. Although most of cardiac conditions, well-tolerated during pregnancy and their potentially fatal conditions which include: complex congenital heart disease, Marfan syndrome with dilated aorta, cardiomyopathies with left ventricular dysfunction, acute myocardial infarction, mechanical prosthetic heart valves and pulmonary hypertension.

PL-4: Cross-talk between renin-angiotensin system and dyslipidemia.

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This lecture is aimed at presenting the synergistic interaction between two systems in the body- the renin-angiotensin system (RAS) and dyslipidemia. Angiotensin II (Ang II) formed as a result of activation of RAS results in stimulation of type 1 receptors. This results in oxidant stress, vasoconstriction, inflammation and platelet aggregation. Recent work shows that oxidized LDL cholesterol (ox-LDL) is much more injurious to the vessel wall components and causation of atherosclerosis than native unmodified LDL. Our studies have shown that ox-LDL levels are increased in the atherosclerotic arteries. It is of note that ox-LDL accumulation results oxidant stress, vasoconstriction, inflammation and platelet aggregation- effects very similar to those of Ang II. Both RAS and dyslipidemia mutually activate each other. Ang II enhances tissue uptake of ox-LDL, and together they cause extensive tissue damage. Animal studies show that RAS inhibitors decrease atherogenesis in dyslipidemic models. Clinical studies also provide evidence that therapy with RAS inhibitors plus anti-lipidemic therapy is better than either agent alone. It is note that conditions that show heightened RAS activity- such as hypertension and diabetes often co-exist with dyslipidemia. As such, all co-morbid conditions should be simultaneously treated with proper understanding of the mechanism/s of disease and pharmacologic properties of the drugs.

Cardiovascular Robotics—Present and Future: In Appreciation of Three Nobel Prize Winners from Rambam Health Center/Technion-IIT

Prof. Rafael Beyar, MD, DSc

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Since the early days of invasive and interventional cardiology, there has been a strong effort toward development of new devices and therapeutic modalities, typically performed manually by the operator, and under direct visual guidance of the angiography system. Multiple modalities have revolutionized our diagnostic and therapeutic environment with newer three dimensional and intravascular imaging modalities, bare metal and drug eluting stents, trans-catheter valves and valve repair mechanisms, therapeutic devices for structural heart disease and congenital malformations, and electrophysiological interventions.

Despite these great advances in technology, the catheterization laboratory environment, in which the operator stands next to the patient, exposed to radiation and subjected to inconvenience, has evolved only to a limited extent since the early days of catheterization. Today, a few new devices are under evaluation for robotic manipulation of catheters. The Stereotaxis navigation system (Stereotaxis, St. Louis, Missouri) moves the tips of flexible catheters and wires via huge magnets that surround the body. Magnetecs (Los Angeles, California) offersa similar approach; however, electromagnetic fields are the driving force for catheter deflection. The Hansen navigation system (Mountain View, California) uses mechanical modules to manipulate and deflect catheters,primarily for electrophysiology. The CorindusCorPath Robotic Catheterization System (Natick, Massachusetts) uses mechanical modules to advance and rotate coronary wires, balloons, and stents. This latter system is designed to complete the entire procedure from a "cockpit" that includes all needed controllers for the procedure. With the initial feasibility and safety being demonstrated in small trials (1, 2), a large clinical trial is currently under way, scheduled for completion by the end of 2011.

All these devices allow for remote control of catheterization procedures either for electrophysiological procedures or for cardiac catheterization. Such devices allow for semiautomatic maneuvers, quality assurance, and computer guidance under a precise imaging system. The future environment in the catheterization laboratory will be influenced by such systems, andmay lead to improved precision, ergonomics, and safety in the catheterization laboratory.

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Improving Outcomes with anti-thrombotic therapy in ACS

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Current management of ACS includes risk stratification by clinical findings and the use of ECG and biochemical markers. The most important clinical risk factors are age, diabetes mellitus and previous manifestations of vascular disease i.e. previous myocardial infarction, stroke or peripheral arterial disease. Also laboratory investigations at entry contribute to risk stratification i.e. signs of ischemia (ST-deviation), myocardial injury (troponin), myocardial dysfunction (NTproBNP), myocardial stress (GDF-15) and renal dysfunction (creatinine clearance). All patients with ACS receive immediate antithrombotic treatment with dual platelet inhibition (aspirin and a P2Y12 inhibitor) plus intravenous or subcutaneous anticoagulation. Patients at intermediate to high risk are rapidly sent for early coronary procedures while lower risk patients derive no benefit from early intervention. At discharge patients will receive long-term secondary prevention with a combination of aspirin, beta-blockers, statins, ACE inhibitors, and P2Y12 inhibitors for at least a year..

Platelet inhibition is since around 20 years a mainstay in the prevention of myocardial infarction and death in ACS. Aspirin treatment inhibits the platelet thromboxane A2 release, which modestly attenuates platelet activation. Aspirin treatment provides a relative reduction of the risk of myocardial infarction and death by 30 – 50 % compared to placebo in patients with acute coronary syndrome. However, aspirin alone has no convincing effect on prevention of stent thrombosis. The P2Y12 receptor plays a major role in the ADP mediated amplification of platelet response regardless of the stimulus. Clopidogrel and Prasugrel are thienopyridine prodrugs acting on this receptor by almost identical active metabolites, which irreversibly bind to the receptor. Slow and variable active metabolite generation leads to clopidogrel having a slow onset of action and wide inter-individual variability in the pharmacodynamic response. As compared to aspirin alone, clopidogrel provides around a relative 20% reduction in myocardial infarction and also a substantial reduction in stent thrombosis in patients with acute coronary syndrome as shown in the CURE trial. The thienopyridine prasugrel has a more efficient active metabolite generation than clopidogrel with a rapid onset of action, more pronounced platelet inhibition and no clinically important variability in response. These features therefore lead to a further around 20% reduction in myocardial infarction and around halving of the risk of stent thrombosis in the TRITON-TIMI 38 trial. However, both the addition of clopidogrel to aspirin and the use of prasugrel instead of clopidogrel are associated with a significant increase in major bleeding. Ticagrelor, the first reversibly binding oral P2Y12 receptor antagonist, provides more rapid, and more consistent ADP induced platelet inhibition than clopidogrel and more rapid offset of action following cessation of therapy. In the PLATO Study ticagrelor as compared to clopidogrel provided a 16% relative reduction of the composite of CV death, myocardial infarction and stroke, a 22 % reduction in total mortality and also a reduction in stent thrombosis without any increase in overall bleeding but still with a rise in non-procedural bleeding with during long-term treatment. Over the last years the addition of anticoagulation with factor-Xa inhibitors have been tested. The addition of low doses of rivaroxaban lowed the risk of CV-death, myocardial infarction and stroke by 16% although at a substantial 3 – 5 times increase in major bleeding. Therefore it seems unlikely that this triple combination will be brought into routine care.

In conclusion, several new management strategies are currently introduced in the routine care of patients with acute coronary syndrome. These new treatments seem to provide additional benefits to the patients without undue increments in the risk of bleeding if used for the appropriate patients. Still an improved tailoring of the treatment and the testing of new combinations is needed in order to offer the best balance between benefits and risks for the individual patient.

Pharmacology in Acute Myocardial Infarction

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The objectives of this lecture will be geared towards an overview of the recent trials and updated guidelines with respect to pharmacology in the therapy of acute myocardial infarction. We will be gearing the discussion on the entire spectrum of acute coronary syndromes from unstable angina, NSTEMI to STEMI. The pharmacology considerations will be focused on the updated guidelines with respect to anti-thrombins, and antiplatelet therapies in the treatment of acute myocardial infarction and overview of mechanisms of action, kinetics, dosing, indications and contraindications will be reviewed for the audience.

Ten Things a Physician Should Know Before Prescribing Drugs.

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While the contribution of modern medicine to increased longevity is widely acknowledged, there is much less awareness about the morbidity and fatality caused by improper drug use. In the US, where prescription drug use is more strictly regulated and enforced (compared to India), over nineteen thousand deaths were attributed to medications in 2009. As a physician's primary objective in writing a prescription is to improve a patient's condition and get the best possible outcome, it is essential that adoctor's prescription contain all the correct information: the right medicine, right patient, right amount, right time of delivery and the right method of delivery.Error in any of these steps will result in an unfavorable outcome. In this presentation, I will present various typesof prescription errors that can completely derail a physician's intended outcome and some straightforward solutions for resolving this problems.I will also discuss how the newly graduating Pharm D (clinical pharmacist) in India can play a role in improving patient care in this country's less regulated medical environment.

Valvular Heart Disease, Native and Prosthetic: Anticoagulation

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The selection of prostatic heart valves in women in their childbearing age is still problematic, because the ideal valve is not available. Bio- prosthetic valves provide the advantage of having lower incidence of Thrombo- embolism and therefore no need for anticoagulation. At the same time they have inferior hemodynamic profile and limited durability when compared to mechanical valves. Mechanical valves have superior hemodynamics in general and excellent durability but are associated with higher incidence of thromboembolism embolism and therefore require mandatory anticoagution. The selection of a prosthetic valve in young women should therefore be dependent on the availability of an excellent system for administration and monitoring of anticoagulation, patient's preference and any other reasons for anticoagulation such as atrial fibrillation. In addition the dose of warfarin required to achieve effective anticoagulation may play a role too. The selection of a bioprostatic valve should take into consideration hemodynamic performance, implantability and durability.

Warfarin has been shown to be effective in prevention of thromboembolic complications in pregnancy however it crosses the placental barrier and may be associated with warfarin embryopathy and increase incidence of fetal loss and bleeding complications. There has been increasing interest and experience with the use of low molecular weight heparin in women with prosthetic valves in pregnancy. Isolated reports however, suggest that the use of heparin even in a therapeutic those may still be associated with thromboembolic complications suggesting the need for close monitoring of the level of anticoagulation. Our suggested regimen anticoagulation of pregnant women with prosthetic about is shown in the table.

Recommended Approach for Anticoagulation in Women with MPHV during Pregnancy	
Higher Risk	**Lower Risk**
Old generation MPHV in the mitral position, MPHV in the tricuspid position, atrial fibrillation, history of TE on heparin. Warfarin (INR 2.5-3.5) for 35-36 weeks followed by IV or SQ UFH (mid interval APTT > 2.5) to parturition + ASA 80-100 mg/day. **OR** LMWH SQ Q 12 hours (trough anti Xa ⩾ 0.7 IU/ml, peak anti Xa <1.5 IU/ml0 or UFH SQ Q 12 hours or IV (mid interval APTT >2.5) for 12 weeks, followed by warfarin (INR 2.5-3.5) to 35-36 weeks then UFH IV or SQ (mid interval APTT >2.5) to parturition + ASA 80-100 mg/day.	New generation MPHV in the mitral position and an MPHV in the aortic position. LMWH SQ Q12 hours (trough anti Xa ⩾0.6 IU/ml, peak anti Xa > 1.5 IU/ml) to 35-36 weeks then UFH IV or SQ (mid interval APTT > 2.0) to parturition. **OR** LMWH SQ Q12 hours (trough anti Xa ⩾0.6 IU/ml, peak anti Xa < 1.5 IU/ml) or UFH SQ q k12 hours or IV (mid interval APTT > 2.0) for 12 weeks followed by warfarin (INR 2.5-3.0) until 35-36 weeks than UFH IV or SQ (APTT >2.0) to parturition.

Aspirin for the Prevention of Cardiovascular Events in Patients Without Clinical Cardiovascular Disease: The Final Word

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Cardiovascular diseases (CVD), which include coronary heart disease (CHD), cerebrovascular disease, and peripheral artery disease, are by far the leading cause of death in most developed countries. It accounts for almost one million deaths annually in the United States alone.Aspirin for secondary prevention of CVD after MI, TIA, CVA, stable angina, and CABG is routinely used to reduce risks of MI, stroke, and vascular death. Evidence from basic research, clinical investigations, observational epidemiologic studies, and multiple randomized clinical trials has provided strong support for the net benefits of Aspirin in reducing CVD events in most patient subgroups

Increased risk of bleeding, chiefly from the gastro intestinal tract and very rarely from intracranial vessels has raised the question of using aspirin for primary prevention of CVD. In a 2006 systematic review of 22 randomized trials of low-dose Aspirin (75 to 325 mg/day) for adverse effects it was found that Aspirin increased the risk of any major bleeding, major GI bleeding, and intracranial bleeding 1.7 to 2.1 times compared to placebo. The absolute annual increase in risk was 1.3 per 1000 patients for GI bleeding and 3 per 10,000 patients for intracranial bleeding. There was no significant difference in risk of bleeding at doses of greater than 162 to 325 mg/day compared with 75 to 162 mg/day.

Based on evidence from 9 large-scale primary prevention trials of men and women without established CVD Aspirin produces a statistically significant and clinically important reduction in the risk of a first MI. Aspirin has not been shown to lead a statistically significant reduction in stroke or cardiovascular death. Any decision to use Aspirin for primary prevention should be an individual clinical judgment that weighs the absolute benefit in reducing the risk of first MI against the absolute risk of major bleeding with long-term administration.

How will I choose my Anti-Hypertensives?

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Hypertension is a very common disorder that often co-exists with other morbid conditions such as metabolic syndrome and diabetes, and if not properly controlled leads to end-organ damage- such as renal insufficiency and heart failure. Fortunately, there are a large number of anti-hypertensive drugs available to the clinician, and all drugs are almost equally efficacious. Some principle of therapy, however, must be kept in mind:

1. Drug adherence is a major issue and poor adherence is due to high cost, multi-dosing and side-effects.
2. Patients with high BP can be started with combinations drugs right from the start.
3. Side effects can be minimized by using combinations of drugs, each in small dose.
4. Most anti-hypertensive drugs now are available in generic form and once a day dose.
5. Drug therapy may be individualized: heart failure patients be given BB, ACEI, ARBs, and diuretics; patients with CAD be given BB and CCBs, patients with DM be given ACEI or ARBs or CCBs, and patients with renal disease be given ACEI or ARBs.
6. Life-style changes can potently reduce BP and should be part of overall therapeutic plan.

So many diuretics- How to use them.

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In recent era heart disease is knocking doors of majority of the families. Disease ranges from hypertension to ishcmeic heart disease to heart faliure . These disease pattern is again complicated by other co morbidities like diabetese, kidney disease or drug interactions and side effects.

Diuretics play a pivotal role in management of this disease population. It is a main stay in treatement of hypertenison and heart failure. Multicenter trials are there to prove its class I role.
Total body water is approximately 60% of body weight. It is distributae in to two compartments. Intracellular (1/3 of TBW) and extracellular (2/3 of TBW).Extracellular fluids again is distributed into plama and interstitial compartments.

Diuretics works on the principle of fluid balance and primararily acts via kidneys. Various diuretics acts from proximal tubule to distal connecting tubules. There effects on disease part and metabolic parameters also vary. Each diuretic has specific indications and trials also aprove this.

Diuretics may be detrimental derive from the many studies that consistently demonstrate that patients receiving higher doses have worse outcomes). The hypothesis that diuretics (especially furosemide) cause deterioration is reasonable, as they cause neurohormonal activation, which, in turn, could exacerbate heart failure. Furthermore, diuretics can worsen renal function, and,
With worsening renal function being prognostic, the connection between diuretics and outcomes seems logical. With publication of a study showing that furosemide increases cardiac dilatation in an animal model of heart failure, the conclusion that the use of diuretics should be minimized was obvious.

It is also not clear that declining renal function caused by diuresis engenders poorer outcomes. Clinicians often limit diuresis because of worsening renal function, but a recent study suggests that hemoconcentration is associated with improved outcome, even though it is also associated with increased serum creatinine. Similarly, DOSE (Diuretic Optimization Strategies Evaluation) showed more diuresis and higher serum creatinine with higher diuretic doses, but no evidence that this had adverse consequences.

A clinician shout be aware of neurohumoral effects of diuretcs and also Diuretic resistance, which poses a major clinical challenge. Various regimes have been stated to over come this resistance.

Thus diuretics though a potential tool is not always free from of non faliure of treatment.

ACE, ARB and Renin Inhibitor in Diabetes

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ARBs have established a record of BP lowering efficacy and placebo-like safety. Recent outcome studies suggest that ARBs have beneficial effects beyond those expected from BP lowering alone. They appear more effective in preventing stroke than β blockers, as shown with losartan based treatment in patients with left ventricular hypertrophy, and also seem to be more stroke-protective than conventional therapy in the elderly. ARBs may be more effective than β blockers in regressing left ventricular hypertro-phy. There is also evidence that they prevent renal deterioration in diabetic and nondiabetic nephropathy and recent data with valsartan support the notion of a beneficial effect on the risk of new-onset diabetes in high-risk patients. However, in this trial (VALUE), myocardial infarctions were fewer with the comparator medication, amlodipine. This was possibly due to a greater BP decrease with the calcium channel blocker (CCB) in the early months of the trial.

The cumulative evidence from these trials strongly supports the view that, in hypertensive patients, combination therapy with CCB/ACE-I or CCB/ARB is likely to be associated with better cardiovascular outcomes, including myocardial infarction and stroke, than regimens containing beta-blockers and thiazide diuretics and those CCB/ACE-I combinations are preferable to diuretic/ACE-I combinations on major cardiovascular endpoints. Added to this should be the cost-effectiveness analysis from the NICE Guidelines which clearly demonstrates that CCBs and ACE-Is or ARBs are more cost-effective treatment choices than beta-blockers or thiazide diuretics

Generally, similar endpoint reductions have been demonstrated with ACE-Inhibitors and ARBs, although there is a suggestion that ACE-Inhibitors may be slightly more cardioprotective and those ARBs may confer some advantages in stroke prevention

For the treatment of hypertension per se, dual RAAS blockade, in general, is not recommended. In the ONTARGET study, there were more adverse events with a combination of telmisartan and ramipril than with individual agents and cardiovascular endpoints, despite a small additional blood pressure reduction, were not improved compared with monotherapy. Thus, there is little if any reason to combine an ARB with an ACE-Inhibitor for the treatment of hypertension. However, as blockade of the renin–angiotensin cascade by either an ACE-Inhibitor or an ARB increases plasma renin activity, the argument has been put forward that the addition of a DRI could have additional benefits

US Food and Drug Administration this week approved an antihypertensive medication that combines hydrochlorothiazide with valsartan, an angiotensin receptor blocker (ARB), and amlodipine, a calcium-channel blocker (CCB) The approval isn't for first-line hypertensive therapy. Instead, the drug is approved for patients who are unable to obtain adequate blood-pressure control with dual therapy that includes CCBs, ARBs, and diuretics.

Vulnerable Plaque: Imaging and Management

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Intravascular tomographic visualization by means of IVUS and OCT has helped overcome the limitations of standard coronary angiography which are well known to all angiographers. In addition to interrogating the lumen area (% diameter (D) stenosis; minimal luminal D, reference D, cross sectional area,) IVUS and OCT enable the assessment of the vessel wall architecture (plaque atheroma, plaque burden, remodeling). IVUS uses pulsed ultrasound while OCT is the optical analogue using electromagnetic waves from a light source to create the image.Gray scale IVUS based on the echogenicity provides a limited insight into atheroma composition. Most image artifacts, especially NURD and ring down artifact are specific to the construct of the system. Post processing of the radiofrequency IVUS signal, with frequency/power information rather than signal amplitude, allows for better plaque composition determination and is therefore called 'Virtual histology ' (VH). The accuracy to predictfibrous tissue has been shown to be 80%, for lipidic tissue 81%, for calcium 90% and for lipid core 86%. One of the major advantagesof OCT is the ability to provide images with a level of resolution (range 10-40µm) ten-fold higher than with conventional IVUS. This however, comes at the expense of a limited penetration depth into the vessel wall of approximately 1.0-1.5 mm due to the attenuation of light in the tissue and the need for a blood free environment.It follows that OCT can provide unprecedented structural and compositional information about atheroma plaques and in particular about culprit plaques in acute coronary syndromes. Plaque composition and especially fibrous cap and even the detection of macrophages within the plaque are within the realm of new OCT developments. Current application of OCT is directed towards plaque characterization according to clinical presentation and cardiovascular risk factors.

Acute Stroke Management: Thrombolytic & PCI

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Management of patients with stroke has revolutionized over the past five years. The innovations have occurred in three area: formation of stroke centers, better imaging techniques, better tools to revascularize cerebral circulation. We will discuss changes that have occurred in all three areas of stroke management.

2011-2012 Guidelines for Evaluation and Management of Peripheral Arterial Disease

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Peripheral arterial disease (PAD) of the lower extremities is a common cause of impaired ambulation and a leading cause of lower extremity wounds and amputation. PAD is also associated with generalized atherosclerosis elsewhere in the body. Patients with PAD, therefore, are at markedly increased risk of cardiovascular and cerebrovascular events and mortality. A majority of cases of PAD go undetected in routine clinical practice. Thus there is a considerable interest in detection of PAD through routine screening. ACC/AHA came out with guidelines to screen patients for PAD in 2005. In 2011, they updated the guidelines to include more patients for screening of PAD. It should be however noted that the American College of Physicians (ACP) and U.S Prevented Services Task Force (USPSTF) recommend against routine screening for PAD. The Trans-Atlantic Inter-Society Consensus (TASC) Document on management of PAD was first published in 2000 and updated in 2007 (TASC II). This represents a consensus of sixteen medical and surgical specialty societies. The rational for screening to detect patients with asymptomatic PAD is based upon two objectives: 1) to identify early PAD and intervene to prevent progression and complications related directly to PAD 2) to identify patients at high risk for coronary heart disease or other cardiovascular diseases to aggressively treat risk factors and prevent events related to CVD. Ankle-Brachial Index (ABI) measurement is a simple, accurate and relatively inexpensive test and is the primary tool for screening for PAD.

Varicose Veins: Prevention, indication and management

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Exciting New Development In Treatment Of Varicose Veins With Multiple Options Available With Focus On Radio Frequency And Thermal Ablation Of Varicose Veins.

Guidelines in Management of Atrial Fibrillation

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This lecture will focus on the current ACC/AHA/ESC guidelines on the management of atrial fibrillation. We will briefly review the current state of the literature on the pathogenesis of atrial fibrillation. We will review the current guidelines and review the most recent updates to the evidence regarding treatment for atrial fibrillation (pharmacologic and procedural) and new therapies for stroke prevention.

Guidelines in Management of Acute Myocardial Infarction

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ESC guidelines for treating patients with acute ST elevation myocardial infarction (STEMI) are based on currently available evidencewith the aim of assisting physicians in selecting the best management strategiestaking into account the impact on outcome, as well as the risk/benefit ratio of specific diagnostic or therapeutic means. Very early reperfusion of the occluded infarct related artery is the mainstay in the treatment of patients with acute STEMI. Pre-hospital (ambulance) clinical and ECG diagnosis is critical for reducing delays between onset of symptoms and reperfusion. Primary PCI with stenting is the best perfusion treatment to save lives and should be performed within 120 min after ECG diagnosis. If PCI cannot be performed within this time, fibrinolytic therapy should be started as soon as possible. With no contra-indications all patient should receive aspirin (ASA), a thienopyridine (clopidogrel or prasugrelas well as heparin or bivalirudinpriorto primary PCI or LMWH or UFH in case of fibrinolysis. In case of failed fibrinolytic therapy, rescue PCI is indicated in the presence of large infarct size within 12h after onset of symptoms. After successful fibrinolysis transfer to a center for interventional cardiology between 3 to 24hrs after the start of fibrinolytic therapy is indicated. Oral ACE inhibitors, beta-blockers and statins should be started if no contra-indications. After discharge all patient should be treated with ASA, thienopyridine, beta-blockers and statins and in case of LV dysfunction with ACE- inhibitor (or ARB). Major gaps in evidence relates to the role of PCI in STEMI patients presenting later than 12h after onset of symptoms. It is unknown whether pre-hospital fibrinolysis during transport to a PCI center is beneficial if primary PCI cannot be performed within the recommended time window. In patients needing triple anticoagulation, the optimal duration of these antithrombotic regimen, is unknown.

Guidelines in the management of valvular heart disease: Aortic and mitral

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Echocardiographic evaluation should be performed in any young woman who has valvular heart disease, even in the absence of symptoms. The management of the valve disease should, whenever possible, be discussed before the onset of pregnancy, particularly in cases of mitral stenosis <1.5 cm2 suitable for percutaneous mitral valvotomy and in cases of aortic stenosis<1.0 cm2. Close follow-up is mandatory after the beginning of the second trimester. In cases of poor functional tolerance, medical treatment should include beta-blockers in severe mitral stenosis, vasodilators in regurgitant valve disease and diuretics. Percutaneous mitral valvotomy is indicated during pregnancy only if the patient remains symptomatic despite medical therapy.Open heart surgery should be performed only when the mother's life is threatened and if viable the fetus should be delivered beforehand. In a pregnant patient with a mechanical prosthesis, the choice of anticoagulant therapy during the first trimester should take into account the greater thromboembolic risk with heparin and the risk of embryopathy with vitamin K antagonists. The use of vitamin K antagonists during the first trimester is the safest regimen for the mother. Delivery should if possible be planned and its modality discussed in close collaboration with the obstetricians and anaesthetists.

Guidelines for Primary and Secondary Prevention for Development of CAD

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Cardiovascular diseases (CVD), which include coronary heart disease (CHD), stroke, and peripheral artery disease, is far and away the leading cause of death. In the U.S, since 1975, CVD mortality has decreased by 24 to 28% overall. It is estimated that nearly half of the decline is due to earlier diagnosis and more aggressive treatment particularly with medical therapies of life saving benefit. The remaining half of the decline in CVD mortality is related to favorable changes in risk factors such as the decline in cigarette smoking and aggressive management of blood pressure and lipids. The majority of known risk factors for CVD are modifiable by preventive measures including lifestyle changes and drug therapies of proven benefit. In the INTERHEART study across 52 countries representing every continent, 9 modifiable risk factors accounted for 90% risk of first myocardial infarction. These associations were noted in men and women, old and young, and in all of the regions in the world. For primary prevention, the modifiable risk factors such as diet, smoking, HTN, dyslipidemia, physical inactivity, obesity, diabetes, and alcohol use should be considered in all patients. In addition several large-scale primary prevention trials and their meta-analyses demonstrate that aspirin reduces the risk of first MI in apparently healthy individuals at moderate to high risk.

In patients with established CVD, there is a very high risk of subsequent cardiovascular events, including MI, stroke, and death from CVD. Identification and treatment of the established risk factors is the cornerstone of treatment. In addition, adjunctive drug therapies of proven benefit include aspirin, statins, beta-blockers, and ACEI or ARB is equally important in the treatment. In the most recent ACC/ AHA guidelines, there is a strong recommendation to include cardiac rehabilitation as part of the secondary prevention strategy.

The Year in Cardiac Surgery

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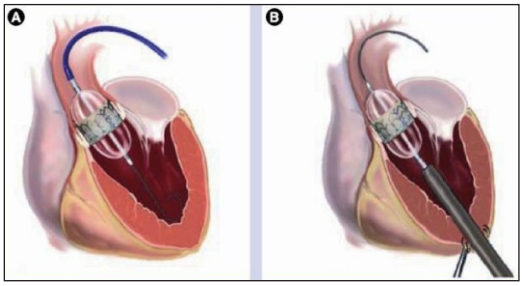
With the Innovations in technology and modifications of existing techniques Cardiac surgery continues to evolve. The recent advances have resulted in increased durability and the improvement in outcomes. The surgical treatments are now available for cardiac diseases treated medically for years. Cardiac surgery now serves to improve the quality of life in patients thought to be terminal and treated only for palliation.



Robotic surgery and anastomotic devices have further revolutionized the concept of minimally invasive cardiac surgery. Gene therapy and stem cell transplantation for neoangiogenesis are also being studied and applied. Atrial fibrillation is now routinely performed with various less invasive approaches and different energy sources. More and more mitral valve repairs and replacement, atrial septal defect closures are being done through the minimally invasive technique via a mini left thoracotomy. With the evolution of the technology the VADS are getting smaller and smaller which allows Stage III-B and stage IV heart failure patients being treated with ventricular assist devices as destination therapy. The surgical management of heart failure is rapidly advancing with the options of passive and active constraint, ventricular restoration and biventricular pacing through placement of left lateral free wall leads. Transapical transcatheter aortic valve implantation has recently become a suitable technique for high-risk and elderly patients with severe aortic stenosis. Hybrid procedures for coronary revascularization and ablative treatment for AF are in vogue and on the rise.

All the eligible patients should be offered with the option of Cardiac surgery when possible so that they can get best available care.

The Year in Interventional Cardiology

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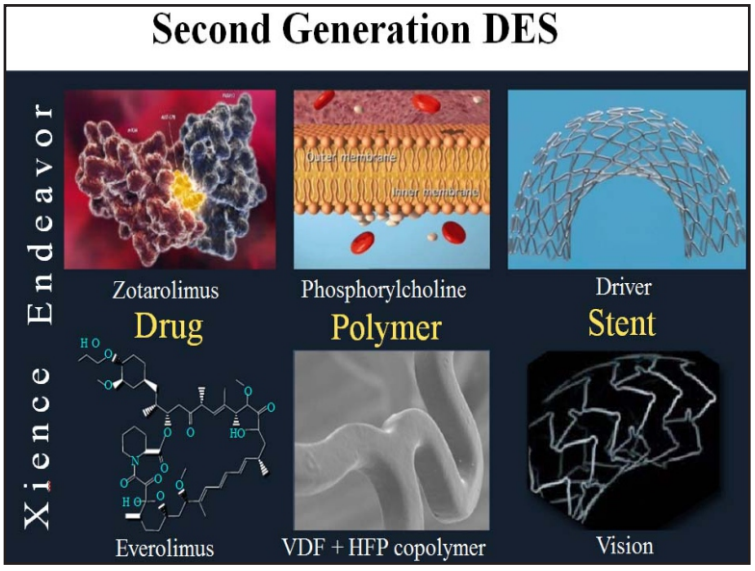
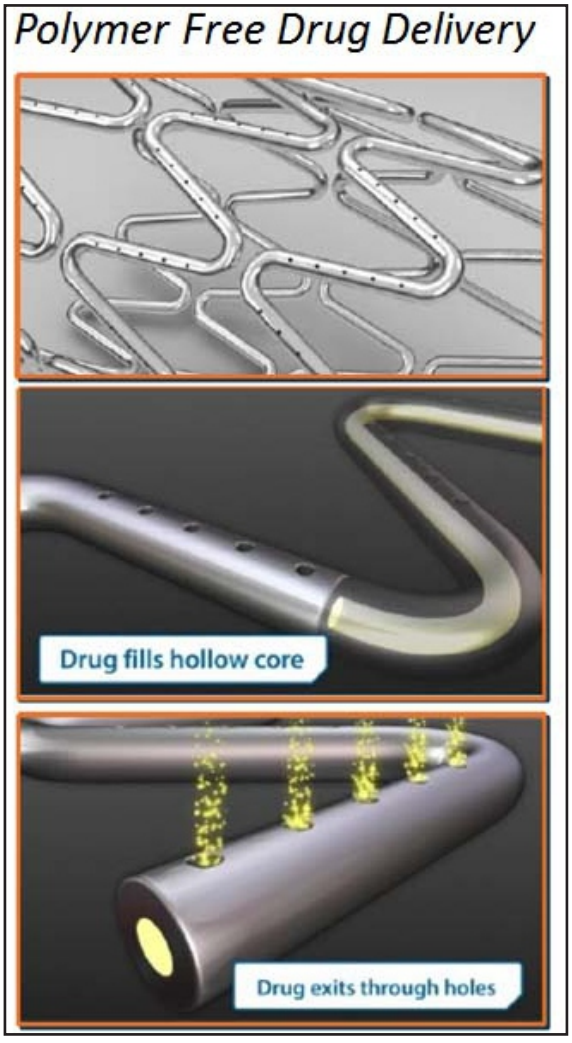
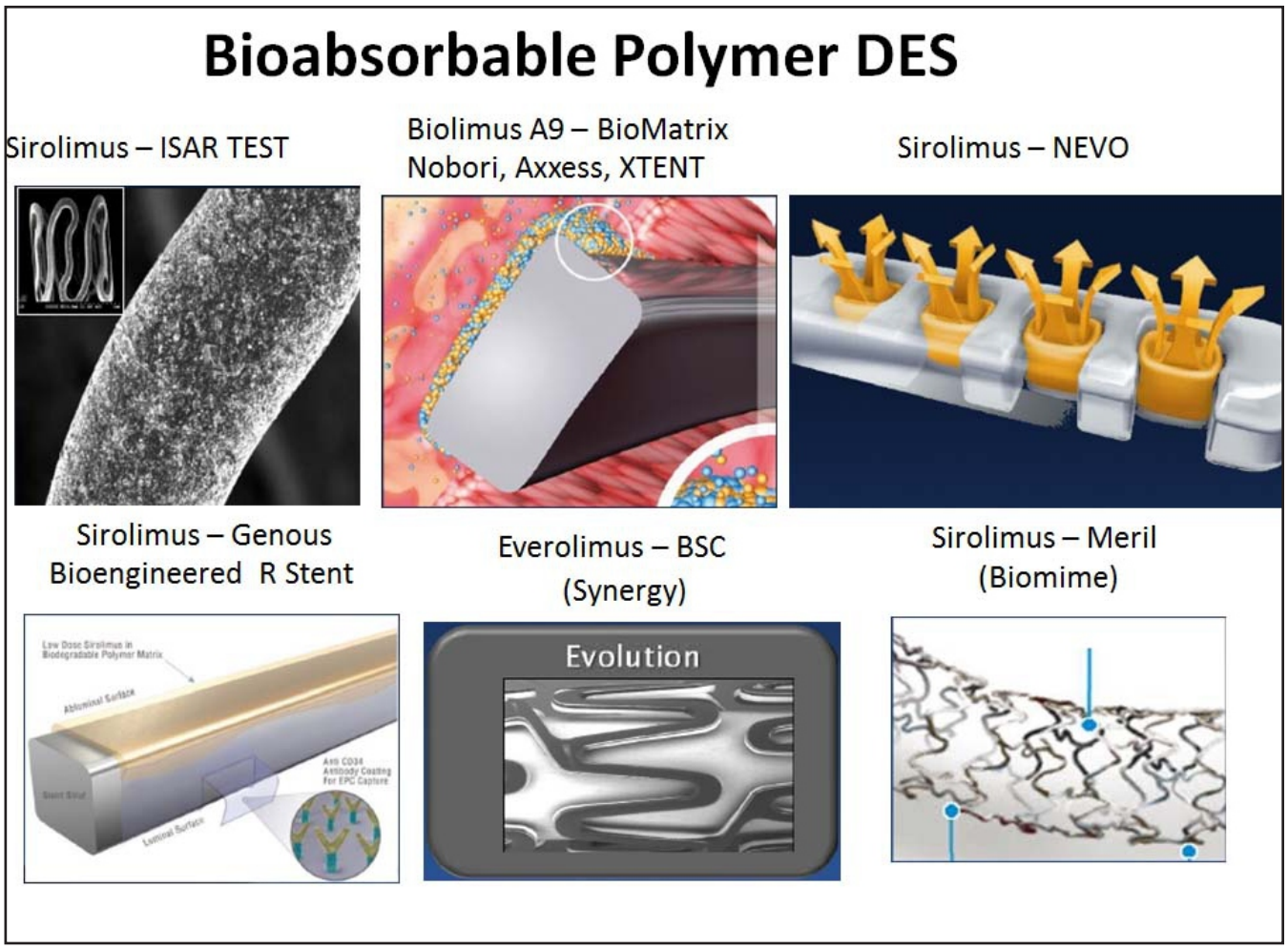
Aim of this lecture is to provide recent advances in the field of interventional cardiology. In recent years, a reduction in mortality following acute myocardial infarction (MI) has been observed due to increasing use of primary percutaneous coronary intervention, shorter door-to-balloon and door-to- needle delays, and specific use of pharmacological treatment following MI. Second-generation drug eluting stents (DES) including zotarolimus-eluting stent and everolimus-eluting stent were developed to reduce restenosis, besides being more deliverable and reduce stent thrombosis compared with first-generation DES. The

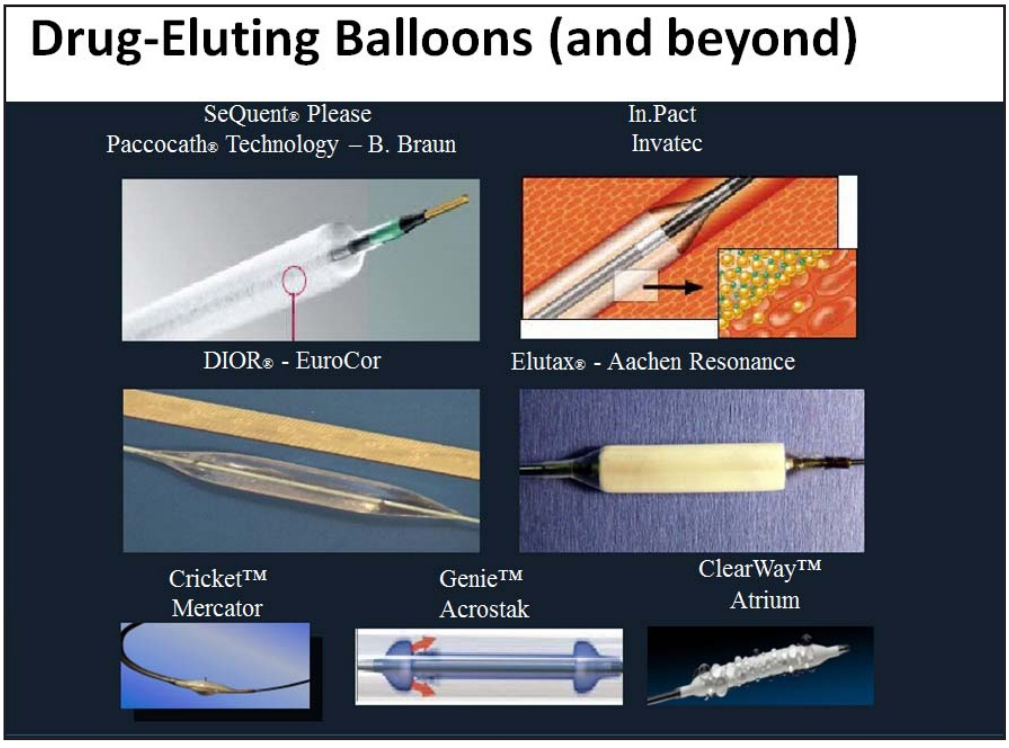




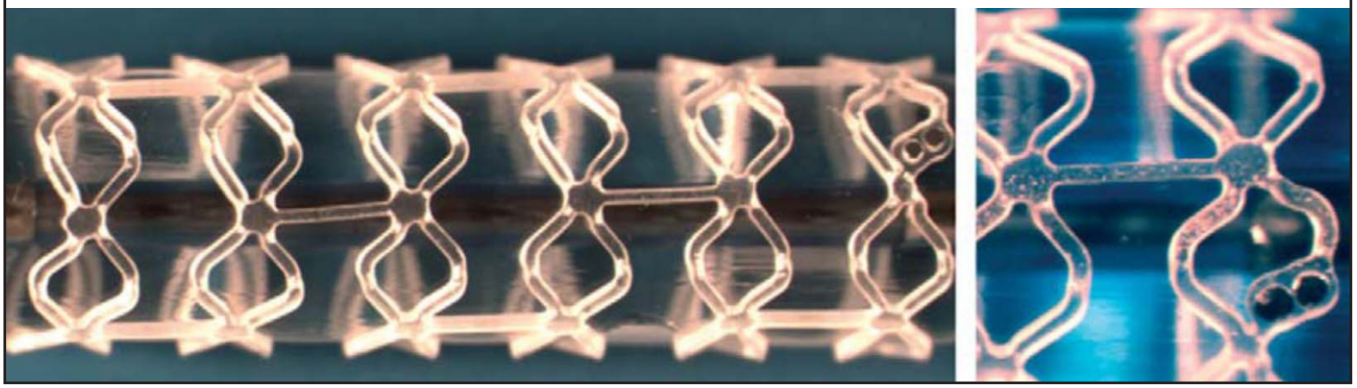
future of DES includes second generation durable polymers, bioabsorbable polymers, polymer free drug delivery, bioabsorbable stents, and drug eluting balloons. Another modus to treat stroke is use of carotid artery stenting in acute atherosclerotic extracranial internal carotid artery occlusion with severe stroke symptoms is feasible, safe and useful



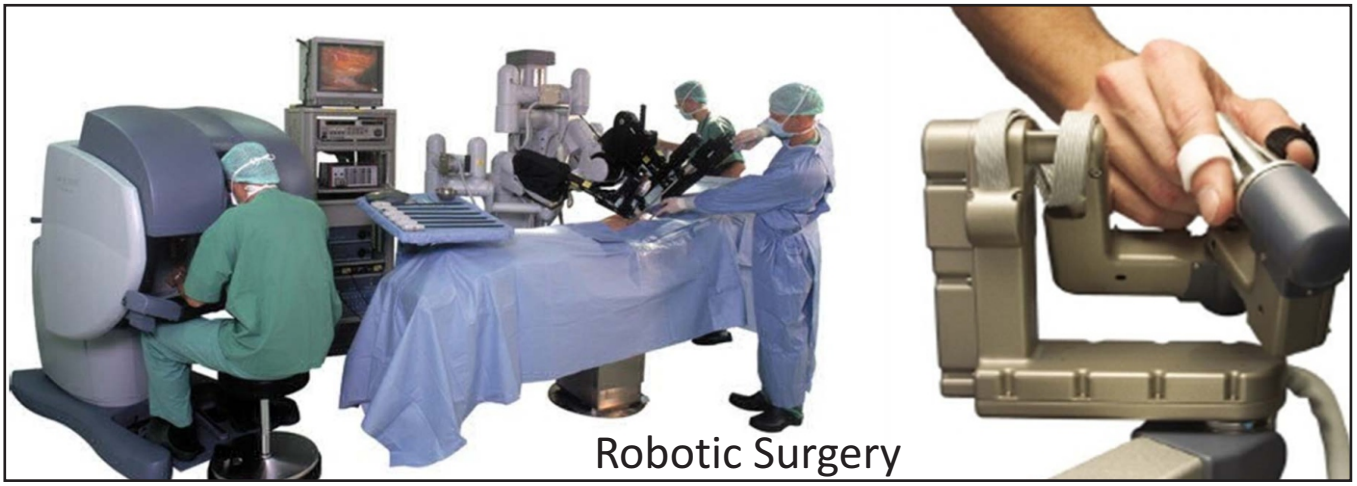
within the first 6 hours after symptom onset. Future of valves extends to transcatheter aortic valve implantation as an emerging alternative to standard aortic valve replacement in patients with symptomatic aortic stenosis considered to be at high or prohibitive operative risk. In hypertensive patients, renal denervation has showed great promise as a safe and effective therapeutic technique and, potentially, for other

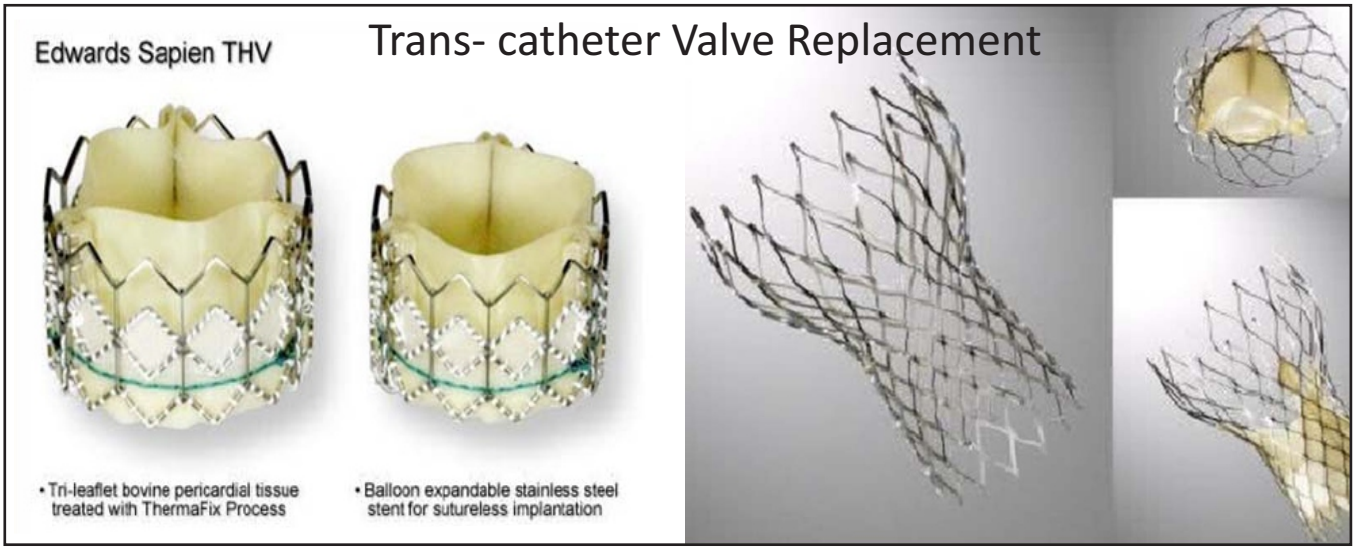


Bioresorbable vascular scaffold



diseases thought to be associated with renal sympathetic hyperactivity. The above perspectives will provide a broad overview of the field for general cardiologists, as well as a framework for more detailed study for those with a specific interest in interventional cardiology.





The Year in anti-thrombotic therapy and newer insights from ACC-ESC 2011

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Ticagrelor, the first reversibly binding oral P2Y12 receptor antagonist, does not require metabolic activation, has a rapid onset of action, and can disassociate from the receptor, permitting restoration of platelet function without the need for production of new platelets. In pharmacodynamic studies, ticagrelor have demonstrated greater, more rapid, and more consistent ADP induced platelet inhibition as compared to clopidogrel and more rapid offset of action following cessation of therapy.

In the PLATO Study 18 624 patients with acute coronary syndrome were randomized within 24 hours after symptom onset to ticagrelor at an 180 mg loading dose followed by 90 mg b.i.d. versus clopidogrel 300 – 600 mg loading dose followed by 75 mg o.d. for 6 – 12 (median 9 months). The main results, presented in 2009, showed a 16% relative reduction of the primary composite of CV death, myocardial infarction and stroke including a significant reduction in cardiovascular mortality, myocardial infarction and stent thrombosis and a 22 % reduction in total mortality. The ticagrelor treatment was not associated with any increase in overall bleeding but during long-term treatment there was a 31 % relative increase in non-procedural bleeding.

Over the following years a series of additional results from the PLATO trial have been presented and published showing consistent effects of ticagrelor versus clopidogrel in ACS patients

- planned for invasive treatment
- planned for non-invasive treatment
- with ST-elevation acute coronary syndrome
- with non-ST-elevation acute coronary syndrome
- with previous stroke
- with peripheral arterial disease
- with diabetes mellitus
- with high age
- with female gender
- with renal dysfunction
- with side effects of dyspnoea
- with CABG within 7 days of cessation of treatment
- with concomitant proton pump inhibitor treatment
- Regardless of Cyp2C19 polymorphisms influencing clopidogrel metabolism.

The PLATO trial included a pre-planned evaluation of quality of life and health economy. Ticagrelor was associated with an increase in quantity of life at an unchanged quality as compared with clopidogrel. The consumption of health care resources was reduced with ticagrelor because of fewer cardiovascular events requiring hospital care. As compared to clopidogrel used at the cost of a generic drug, ticagrelor still turned out to be cost-effective with an estimated cost of around 2,500€ per life year or quality adjusted life year gained.

In conclusion, ticagrelor is a new and cost-effective treatment reducing the risk of recurrences and improving survival that can be applied to a broad population patients admitted to hospital because of ST-elevation or non-ST-elevation acute coronary syndrome.

Role of antithrombotic therapy in NSTEMI and STEMI

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Antithrombotic treatment with platelet inhibition and anticoagulation is since around 25 years routine treatment in ST-elevation (STEMI) and non-ST-elevation myocardial infarction (NSTEMI). Antiplatelet treatment with aspirin treatment provides a relative reduction of the risk of myocardial infarction and death by 30 – 50 % compared to placebo in patients with both NSTEMI and STEMI. Adding the irreversible P2Y12 receptor inhibiting thienopyridines clopidogrel or prasugrel provide further reduction in myocardial infarction and stent thrombosis in patients with NSTEMI or STEMI with an increased effect at more intense platelet inhibition. However, both treatments are associated with an increase in major bleeding, which also is related to the intensity of platelet inhibition. Ticagrelor, the first reversibly binding oral P2Y12 receptor antagonist, provides more rapid, and more consistent ADP induced platelet inhibition than clopidogrel and more rapid offset of action as compared to the thienopyridines. In the PLATO Study ticagrelor, as compared to clopidogrel, provided consistent benefits in patients with both NSTEMI and STEMI with a 16% relative reduction of the composite of CV death, myocardial infarction and stroke, a 22 % reduction in total mortality and also a reduction in stent thrombosis without any increase in overall bleeding but still with a 31 % rise in non-procedural bleeding with during long-term treatment. Therefore dual antiplatelet treatment with aspirin and a P2Y12 inhibitor from the acute phase up to one year after the acute event is currently routine care in patients with both NSTEMI and STEMI. Further platelet inactivation by intravenous glycoprotein IIb/IIIa inhibition might be considered at PCI procedures in the acute phase of both NSTEMI and STEMI in order to reduce the risk of peri-procedural complications.

Short-term anticoagulant treatment with unfractionated heparin or lmw heparin in association with the index event has also been established over the last 20 years. Lmw heparin have been shown to be more convenient and either non-inferior or superior to unfractionated heparin concerning the risk of ischemic events and bleeding both in NSTEMI and STEMI. For patients managed non-invasively the use of subcutaneous factor-Xa inhibition with fondaparinux has been shown non-inferior to lmw heparin concerning ischemic events and associated with a reduced the risk of bleeding and thereby also improved long-term survival. For patients managed invasively with early PCI procedures in NSTEMI or STEMI an intravenous infusion of the direct thrombin inhibitor bivalirudin has been found as efficacious as the combination of unfractionated heparin and a glycoprotein IIb/IIIa inhibitor. The bivalirudin treatment is though associated with a lower risk of major bleeding and thereby with an improved long-term outcome. The continuation of long-term anticoagulation with oral factor-Xa inhibition in addition to dual anti-

platelet treatment has recently been tested and found associated with a reduction in ischemic events, but also associated with a clinically concerning increase in the rate of major including intracranial bleeding.

In conclusion, the combination of antiplatelet and anticoagulant treatment is currently the recommended treatment in the acute phase both at NSTEMI and STEMI. For long-term treatment still the use of dual antiplatelet treatment with aspirin and P2Y12-receptor inhibition is still the routine treatment to reduce the risk of recurrent ischemic events and improve survival. The use of long-term anticoagulation in addition to anti-platelet treatment needs further evaluation in order to find a combination with an acceptable bleeding risk.

Diastolic Heart Failure

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Diastolic heart failure of heart failure with preserved ejection fraction is a clinical syndrome characterized by the symptoms and signs of heart failure, a preserved ejection fraction (> 50%), and abnormal diastolic function. Approximately 50% of patients presenting with heart failure symptoms have been shown to have preserved LV ejection fraction. This syndrome is more commonly seen in women, older individuals, and in patients with past or current history of him hypertension. The clinical presentation of patience with diastolic heart failure is usually the same as in patients with systolic heart failure. However, these two conditions have substantially different phenotypes and pathophysiology. Mortality associated with diastolic heart failure is only slightly lower than that seen with systolic heart failure but it is often not related to heart failure causes. Multiple clinical trials in heart failure secondary to diastolic dysfunction including the use of ACE and angiotensin receptor blockers have failed to show substantial effect on outcome. The guidelines recommendations for the management of diastolic heart failure are based mainly on clinical information and not on clinical trials. These recommendations include control of blood pressure, control of tachycardia in patients with atrial fibrillation, the use of diuretics to lower blood volume, revascularization when ischemia is thoughts to be causing diastolic dysfunction, and restoration of sinus rhythm in symptomatic patients with atrial fibrillation. Recent studies have demonstrated a potential beneficial effect of the phosphodiesterase 5 inhibitors sildenaphil citrate in the reduction of pulmonary hypertension and LV filling pressure in patients with diastolic heart failure. In addition a favorable effect of exercise training in this patient population has been demonstrated.

PL-6 : Stem Cells in Cardiology—where are we now?

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Human embryonic stem cells (HESC) were introduced to the medical scientific community with Thompson et al.'s (1) groundbreaking paper in 1998,showing to the world for the first time the ability to grow these cells in vitro. These cells lines, which can be grown perpetually, have been demonstrated to turn into many cell types under certain conditions. Kehat et al. (2) were the first to show differentiations of these cells to cardiomyocytes, and described their cellular and electrophysiological characteristics. Further studies have shown that these cells can integrate with the myocardium and function as pacemakers (3);when injected in large numbers to acutely infarcted tissue, they can enhance ventricular function in the rat model. However, despite enormous advances in basic research, and the applicationsfor patients, this technology has been hampered by ethical and religious issues due to use of HESC produced under research protocol from an 8-day embryo, and by biological issues such as the need to solve the immune response to those cells before they can be used in patients. A recent study using HESC in neurology by Geronwas recently stopped prematurely, without demonstrating any benefit to four patients.

Recently, a new approach using genetic manipulation of adult fibroblasts to generate induced pluripotent cells (HIPC) was described by Takahashi and Yamanaka (4). With this technology, skin biopsy can be used to obtain cells that are then turned into HIPCs of the same patient. The Technion laboratories have shown that such cells, when generated from patients with genetic diseases, can be used to study the mechanisms of those diseases, and can be a strong tool to predict individual response to drugs in certain conditions. The ability of these cells to differentiate into cardiac cells can also be used as a method to generate auto-transplanted cells from the same patients. While this presents an extremely appealing solution, problems regarding safe gene transfer technology and the potential of those cells to function in vivo need to be evaluated before clinical testing can become feasible.

Many other approaches for injecting various types of stem cells derived from the same patient have been clinically evaluated. Many small studies have been published, but the proof of any such approach may only be possible when a large randomized study becomes possible. Stem cell research is more exciting today than a few years ago, with new directions and approaches requiring extensive basic and applied research on a worldwide basis.

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PL-8 : Acute Heart Failure Management: Present and Future

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We will highlight the current state of recommendations for the management of acute decompensated heart failure. Recent trial and therapies will be reviewed and their role in the management of acute heart failure. We will also examine ongoing trials of new interventions and exciting compounds in the early clinical phase.

Update on Vulnerable Plaque: Therapeutic Options

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Acute coronary syndromes (ACS) are due to destabilization of the atheroma plaque secondary to plaque rupture, fissure or erosion. In an effort to ascertain the prevalence and clinical significance of non-flow limiting lesionsdefined as vulnerable plaque, the Prospect trial was launched whereby in 697 patients with ACS, treated with primary PCI, the non- culprit vessels were studied withcoronary angiography, IVUS-VH andpalpography. Subsequent MACE during FU (20.4% over 3 years)were equally attributable to recurrence at the site of culprit lesions and to non-culprit lesions (NCL). NCL, responsible for unanticipated events (11.6% pts) were angiographically mild and most were TCFA with large plaque burden and small luminal area(26/51). Specificity of plaque characterization to predict MACEis insufficient since only 26 out of 595 TCFA were sites of recurrent events.Potential therapeutic options to treat vulnerable plaquesare: plaque sealing, pharmacologic reduction in lipid core and pharmacologic inhibitionof plaque neovascularization. 1) The effects of plaque sealing were studied in the Secritt trial were thepreliminary results showed that plaque sealing with a dedicated stent was feasible and associated after 6 months follow up with a positive remodeling. 2) The potential for pharmacological reduction in lipid core was evaluated in the IBIS II trial where it was shown that Darapladip, a potent and selective Lp-PLA2 inhibitor, haltsthe progression of necrotic core after 1 year. 3) Based on the observation that plaque formation is associated with increased neovascularization, experimental studies in cholesterol fed rabbits have shown that non-invasive molecular imaging using paramagnetic nanoparticles targeted to integrins are capable of imagingthe diffuse proliferating neo-vasculature associated with atherosclerotic plaque formation. Deliveringthe anti-angiogenic agent (fumagillin),using the same nanoparticles,has been shown to inhibit proliferation of vascular smooth muscle. Further studies will be needed to prove that by inhibiting neovascularization, plaque stability is enhanced and clinical outcome is improved.

MICS and Hybrid Surgery – A Revolution in Making

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Can you imagine a heart surgery which involves only a short 3-4 inch incision in the side of chest and which is also fast and pain free with quick recovery from the hospital within 3-4 days?

This type of heart surgery is possible nowadays using advanced key hole (called minimal access technique in medical terminology) and hybrid cardiac surgery which also avoids the usage of heart lung machine.

The treatment of heart diseases has seen many major advances over the last decade in so many ways, that now major heart surgeries like bypass surgery, angioplasty, closure of holes in children and valve surgeries have become very easy, safe with predictable outcomes.

One of the problems of traditional heart surgery was that - the heart was approached by cutting open the sternal bone. Hence the incision (called sternotomy) would be long and extends from near the neck to the upper abdomen. This creates many problems for the individual like cosmetic i.e. affects the physical appearance of chest (which is of special significance to women).

Also the long scar creates psychological problems like loss of confidence and also mild chronic nerve related pain. Also this long scar takes a long time to heal.

Use of heart lung machine in heart surgery is also associated with many problems like bleeding during heart operations, prolonged recovery time from the ICU etc.

Cardiologists and cardiac surgeons have been working together to overcome these two main problems associated with traditional heart surgery i.e. use of heart lung machine and need for sternotomy and long incisions. This has led to the introduction of “key hole and hybrid heart surgeries”.

Comparison of Early Surgery versus Conventional Treatment in Asymptomatic Severe Mitral Regurgitation

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Optimal timing for surgical intervention in asymptomatic patients with severe mitral regurgitation (MR) remains unclear.

The 2006 American College of Cardiology/American Heart Association guidelines include “prophylactic” mitral valve repair as a Class IIa indication for surgical intervention in asymptomatic patients with severe mitral regurgitation. The recommendation is supported by data published by the Mayo Clinic[1] that suggest excess mortality in unoperated patients with severe mitral regurgitation and no 'conventional' indication for intervention (symptoms, left ventricular systolic dysfunction, substantial dilation, pulmonary arterial hypertension, or new-onset atrial fibrillation). In contrast, literature from Vienna, Austria[2] suggests no excess mortality associated with an approach of watchful waiting.

As the mortality associated with mitral valve surgery has certainly declined over the past few decades, there has been increased interest in optimal timing for surgery in patients who suffer from isolated mitral valve regurgitation. While the natural progression of asymptomatic MR varies amongst patients, it appears that with increasing age both a reduction in left ventricular compliance and degeneration of already diseased leaflets results in increased complications, including the development of congestive heart failure and pulmonary hypertension.

When comparing the early surgical intervention group with the conventional treatment group, over a ¼ of the patients ended up eventually meeting criteria for surgical intervention and their complication risk was higher than in the early surgical intervention group. Given the increased risk of cardiac surgery in older patients, one could postulate that this would be an argument for earlier intervention

It should be recognized that the American College of Cardiology/American Heart Association guidelines do suggest that early mitral valve repair at experienced surgical centers should be recommended in patients with asymptomatic MR.

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Treatment of Chronic Stable Systolic Heart Failure: From Diuretics to VADs

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Heart failure (HF) is a common clinical syndrome representing the end-stage of a number of different cardiac diseases. It can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood. Heart failure can occur by two mechanisms: systolic dysfunction (HFrEF, Heart failure with reduced Ejection Fraction) or diastolic dysfunction (HFpEF, Heart Failure with preserved Ejection Fraction). The management of HF varies between decompensated or compensated state of the patient. Several major societies have extensive guidelines for the treatment of HF such as those by ACC/AHA, Canadian Cardiovascular Society, the European Society of Cardiology (ESC), and the Heart Failure Society of America (HFSA). The management begins an accurate assessment of the etiology and severity of the disease. Lifestyle modification, avoidance of drugs that may contribute to HF, treatment of the underline cause of HF, pharmacologic therapy to relieve symptoms, slow the progression of HF and improving patients survival, and device therapy like Implantable Cardioverter- Defibrillator (ICD), Cardiac Resynchronization Therapy (CRT), and Ventricular Assist Devices (VAD) are part of the treatment protocol. Pharmacologic therapy to improve the symptoms can be achieved by diuretics, digoxin, beta-blockers, ACEI, and ARBs. Prolongation of patient survival has been documented with ACEI, beta-blockers, ARBs, hydralazine/nitrates, and aldosterone antagonists. The majority of patients with HF due to systolic dysfunction (HFrEF) respond well to the above therapy. However, some patients with refractory HF do not improve or experience rapid reoccurrence of symptoms. Specialized strategies are needed for these patients which includes continuous IV inotropic therapy, CRT, VADs, or eventual cardiac transplantation.

Clinical Case-1: Discussion

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The patient is a 70-year-old woman with type 2 diabetes mellitus. She is 5' 2" and weighs 74 kg, has a sedentary lifestyle. She has no cardiac history. In the last 6 months, the patient experienced increasing shortness of breath and recently complained of dyspepsia. She stayed home from work the previous 2 days due to "flu". Patient presented in the emergency department with mid-sternal chest fullness. Her heart rate was 90 beats per minute; blood pressure 145/96 mm Hg, and hemoglobin 12.2 g/dL. Creatine kinase was normal. Cardiac markers as measured in the afternoon were positive for troponin. An electrocardiogram (ECG) showed left ventricular hypertrophy with a lateral ST depression. Recent laboratory results are suggestive of hyperlipidemia (LDL-C: 120 mg/dL; triglycerides: 276 mg/dL; HDL-C: 31 mg/dL). Other laboratory results indicated a serum creatinine level of 1.4 mg/dL and A1c was 8.1%. Medications included aspirin 325 mg/day, Ramipril 5 mg/day, and metformin 500 mg twice daily. Her physician had prescribed omeprazole 1 month ago for dyspepsia.

The questions within the case are designed to upgrade the current knowledge. The correct answers based on evidence-based information will support the most appropriate choice.

Cardiac markers: Positive for troponin and creatine kinase was normal.

- **Diabetes is estimated to account for what percentage of additional deaths from coronary artery disease (CAD) in 2000 compared with 1980?**
  1. 1.7%
  2. 36%
  3. 0.62%
  4. 9.8%
- **Platelet hyperreactivity and hyper aggregability in DM are related to a decrease in which of the following?**
  1. Prostacyclin and nitric oxide (NO) production
  2. Thromboxane A2 (TXA2) synthesis
  3. Expression of adhesion molecules (eg, glycoprotein [GP] IIb/IIIa)
  4. Arachidonic acid metabolism

- **When should catheterization/angiography be performed in this patient?**
  1. ⩽12-24 hours of admission
  2. Between 24 and 48 hours of admission
  3. >48 hours of admission
  4. Catheterization/angiography is not indicated
- **Based on current evidence-based guideline recommendations, what is the most appropriate antiplatelet therapy on admission in this patient?**
  1. Aspirin alone
  2. Aspirin + clopidogrel
  3. Aspirin + prasugrel
  4. Aspirin + prasugrel + eptifibatide
- **Does the evidence support the benefit of an invasive approach in this female patient?**
  1. No, only men benefit
  2. No, only cardiac marker negative-women benefit
  3. Yes, men and cardiac marker positive-women benefit
  4. No. The appropriate treatment approach is undetermined in women
- **Based on the Synergy Between Percutaneous Intervention With Low Taxus and Cardiac Surgery (SYNTAX) score(0-22) for this 70-year old woman with osteal left main disease and positive troponins, what is an appropriate treatment strategy?**
  1. Revascularization with PCI only
  2. Revascularization with CABG only
  3. Revascularization with PCI or CABG
  4. Medical therapy
- **Patient opted for PCI.**
- **If the patient had not been pre-treated, what would be the most effective thienopyridine loading and maintenance dose to add to aspirin for PCI?**
  1. Clopidogrel 900 mg + 75 mg daily
  2. Clopidogrel 300 mg + 75 mg daily
  3. Clopidogrel 600 mg + 75 mg daily
  4. Prasugrel 60 mg + 10 mg daily
- **Is long-term dual antiplatelet therapy (DAPT) indicated in patients with NTSE ACS who undergo PCI?**
  1. No, only aspirin
  2. Yes, with aspirin plus clopidogrel only
  3. Yes, with aspirin plus prasugrel only
  4. Yes, with aspirin plus clopidogrel or prasugrel

- **What is the most appropriate dosing strategy for aspirin and clopidogrel in patients with NTSE ACS scheduled for CABG?**
  1. Aspirin 325 mg + clopidogrel 75 mg
  2. Aspirin 75-325 mg and discontinue clopidogrel ⩾5 days prior to CABG
  3. Aspirin 75 mg + clopidogrel 600 mg
  4. Aspirin 325 mg and discontinue clopidogrel immediately prior to CABG
- **Is long-term DAPT (1 Year) indicated in patients with NTSE ACS who undergo CABG?**
  1. No, only with aspirin
  2. Yes, with aspirin and clopidogrel only
  3. Yes, with aspirin and prasugrel only
  4. Yes, with aspirin and clopidogrel or prasugrel
- **What population has the highest frequency of the CYP2C19*2 variant allele, which is associated with poor metabolism of clopidogrel?**
  1. Blacks > Asians > whites
  2. Whites > blacks > Asians
  3. Blacks > whites > Asians
  4. Asians > blacks > whites
- **Compared with non-carriers, are patients with ACS who are carriers of the CYP2C19 reduced-function allele at significantly higher risk for cardiovascular events on long-term DAPT?**
  1. No
  2. Yes, with prasugrel but not clopidogrel
  3. Yes, with clopidogrel but not prasugrel
  4. Yes, with both clopidogrel and prasugrel
- **What were the bleeding results of TRITON-TIMI 38 in patients with and without DM?**
  1. Rates of TIMI major bleeding were significantly higher in patients without DM treated with prasugrel vs clopidogrel
  2. Rates of TIMI major bleeding were significantly higher in patients treated with prasugrel vs clopidogrel irrespective of diabetic status
  3. Rates of TIMI major bleeding were significantly higher in patients with DM treated with prasugrel vs clopidogrel
  4. Rates of TIMI major bleeding were significantly higher in patients with DM treated with clopidogrel vs prasugrel
- **Should omeprazole be discontinued in this patient?**
  1. Yes
  2. No
  3. No, but separate prasugrel and omeprazole dosing by ⩾6 hours
  4. No, but increase prasugrel dose

Selection of Guiding Catheters, Guide Wires and Balloons

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Technical knowledge and understanding the behavior howcatheters, guide wires and balloons performtogether with operator experience will enhance the success rate of PCI,especially in difficult cases.Guiding catheters (GC)do function as a conduit for transport of wires and devices, support for device delivery against resistance, contrast injection and recording arterial pressure. Size of GC will depend on the size of the access vessel (radial versus femoral) type of procedure (simple versus complex, need for special devices such as rotablator, Tornus). Selection of catheter curve will be guided by the presence of dilated ascending aorta (Xray), elongated aorta (Xray), presence of aortic valve disease and/or history of arterial hypertension, all factors often present in elderly patients.Guide wires look like simple tools but have in fact a complex structure. They are used to steer through the vessel in order to access the index lesion (steerability, flexibility & visibility) and to cross the lesion (visibility &crossability). In addition they need to support and facilitate delivery of any interventional device required to treat a specific anatomy or to resolve any potential complication (support). Special cases have dedicated wires such are CTO procedures.Balloons come in different materials: compliantsemi-compliant, non-compliant (highpressure). Special balloons such as cutting balloon and CTO balloons are available for dedicated procedures.

PCI for STEMI: Guidelines and strategies

Pharmacoinvasive Strategies (Facilitated, Rescue and Transfer PCI) in STEMI

Shock

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The objectives of the lecture will be an in-depth dive into the Guidelines and strategies of PCI for STEMI and the number of recent trials and consensus documents governing all aspects of the diagnosis, management and therapeutic options currently available in 2012. In addition, all of the most recent trials and meta-analysis with respect to facilitated, rescue and transfer PCI in the treatment and triage of STEMI will be reviewed with special attention to the current needs of practicing physicians in India. Finally the remainder of the discussion will be based on the current guidelines and review of the shock trial and accepted practices both with ESC and ACC/AHA focus.

Radial Artery Access: Technique, Results and Complications:

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Femoral artery route has been an traditional way of doing and cardiac angioram. Its still a gold standard to many intrventional cardiologist. Radial route came into practice widely in this decade especially in last five years. It has changed vastly the acceptance of angiography and has helped a lot in patient compliance. Right radial aretery is the one which is used in majority of cases. Radial access is like that of femoral access though with different hard ware. In experienced hand access in single try is > 95%. Various anomalies of radial artery and aorta do make sometimes this procedure lengthy and complex.

These incluseds abberancy, tortuosity, loops, spasms. Calcifications and leusoria. With good knowledge of hardware and experience, one can easily handle such hurdles. Complications do occur but is less than 1%. Which includes pain, swelling, hematoma, bleeding and loss of radial pulsations. Last one carries a bit higher incidence. Many of the centers are now shifting towards radial route as their first choice in interventions. How evere it should not be made a religion. Pateint safety and operator ease are of prime importance.

Rationale for Functional Assessment of Lesions in Patients with MVD & primer on coronary physiology for interventional cardiologists

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PCI, when introduced in 1977, was based on the concept that balloon dilatation of a flow limiting coronary stenosis should alleviate symptoms in patients with 1vessel disease (VD). Stimulated by the initial results and with improvement in the procedural technique as well as in the catheter material, the field of indication soon was extended to patients with 2 and 3VD. While a direct link between a flow limiting lesion and ischemia was apparent in symptomatic patients with single VD, this link was more elusive and difficult to establish for each lesion separately in patients with MVD due to the limitations of coronary angiography as well as myocardial perfusion imaging. Fractional Flow Reserve (FFR) measurement has been shown to be superior in sensitivity and specificity compared to angiography and even myocardial perfusion imaging in differentiating flow-limiting from non flow-limiting lesions. The FAME trial was initiated to answer the question whether PCI guided by FFR measurement (cut off value: 0.80) was better than angiographic guided PCI. Routine measurements of FFR during PCI significantly reduced the rate of the primary composite endpoint of death, myocardial infarction and repeat revascularization at 1 year (18.3% vs 13.2%) and at the same time reduced the number of stents used(2.7±1.2 vs1.9±1.3)while resulting in a similar if not improved functional result. The FAME trial provides strong evidence that coronary angiography and clinical data alone are not sufficient for decision making about the appropriateness of revascularization of individual lesions in patients with MVD

Stent Thrombosis: Mechanisms, Predictors, and Treatment

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Coronary artery stents, particularly drug-eluting stents (DES), are used in the majority of patients who undergo percutaneous coronary intervention (PCI). Stent thrombosis (ST) is an uncommon but serious complication of stents that almost always present as death or a large non-fatal myocardial infarction, usually with ST elevation. Stent thrombosis can occur acutely (within 24 hours), sub-acutely (within 30 days), or as late as one year (late) or more (very late) after stent placement. Stent thrombosis within the first year occurs with equal frequency in patients with bare metal stents (BMS) or DES as long as the patients with DES are treated with dual antiplatelet therapy (DAT) for the recommended duration. The period of risk requiring DAT is longer with DES due to delay in neointimalcoverage. The risk factors for development of ST fall into broad categories of procedural, patient, lesion, and stent characteristics as well as the cessation of antiplatelet therapy. These risk factors predispose the patient to ST by one or more different mechanisms. The single most important predictor of early and late ST is the absence of Clopidogrel therapy at the time of the event. Therefore the key to prevention of ST is DAT for as long as possible. The management of ST is the same as treatment of ACS. Emergent revascularization by PCI is the treatment of choice and is possible in over 90% of cases. Urgent CABG should be strongly considered in the remaining patients.

Management of the hospitalized patient with heart failure

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There are a significant number of patients hospitalized for heart failure in the United States and the entire world. There are number of reasons for this phenomenon which include the aging of the population as well as improvement in outcome and prolongation of survival in patients with various cardiovascular conditions including with heart failure. A number of registers and clinical trials performed in the United States and Europe in the last several years have provided information about the characteristics of patients hospitalized for the indication of heart failure. The average age in the United States has been 75 years, more than 50% are women and half of the patients have preserved ejection fraction. This patient population has multiple co morbidities including coronary artery disease, history of hypertension, diabetes, atrial fibrillation, and renal abnormalities. Decompensated heart failure syndromes are associated with substantial in-hospital mortality, a very high rate of hospital readmissions, and high. The great majority of the patients hospitalized for heart failure have symptoms related to volume overload. The diagnosis is mostly based on clinical presentation and the use of BNP can significantly add to the diagnosis of this condition. The management includes the use of positive pressure ventilation, diuretics, ultra filtration, vasodilators and inotropic drugs. The appropriate use of these medications as well as their potential complications will be discussed.

Challenges in Hypertension management

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Hypertension is a very common disorder that often co-exists with other morbid conditions such as metabolic syndrome and diabetes, and if not properly controlled leads to end-organ damage- such as renal insufficiency and heart failure. Despite the availability of a large number of anti-hypertensive drugs, it is difficult to control BP in a significant number of patients. Some principle of therapy in these patients:

- Resistant hypertension is lack of BP control despite use of 3 drugs
- Drug adherence is a major cause of resistant hypertension, and poor adherence is due to high cost, multi-dosing and side-effects. Improve adherence by simplifying drug regimen and involving family members..
- Patients with resistant hypertension- advanced age, females, co-existint DM, CKD and LVH
- Keep in mind "white coat hypertension".
- Ambulatory BP monitoring can identify patients with poor-prognosis- patients with wide pulse pressure and "non-dippers".
- Pseudoresistance- non-adherence, inadequate use of NSAIDs, excessive caffeine and smoking, use of OTC drugs and oral-contraceptives. .
- Common causes of true resistant hypertension- obesity, endocrinopathies (thyroid disorders, pheochromcytoma, hyperaldosteronism).
- Renal artery stenosis- results of treatment- not very impressive.
- Life-style changes can potently reduce BP and should be part of overall therapeutic plan.

Clinical Decision Making: today and tomorrow

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A discussion regarding the landscape of medicine and how it will impact our future and the future of mankind.

Challenges of CAD in India

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Challenges of CAD in India are mainly related to root causes that are behavioral, societal & environmental. Lack of infrastructure in rural areas forces people to migrate to cities. Haphazard industrialization & migration has lead to aggressive marketing of unhealthy food products causing dramatic shift in diet & day to day living behavior in individuals, families & communities. Adverse lifestyle & socioeconomic factors are leading to high intake of salt, fast foods containing trans-fats and sedentary life styles. At the other end of the spectrum, in spite of rising per capita income & GDP, more than 60% live under poverty line leading to overcrowding, infections, maternal malnutrition and detrimental early life changes. Economically deprived are most affected due to extreme mental stress causing early CAD.

Prevention of risk factors causing CAD (Premordial prevention) is the main challenge & is a key to solve this problem. Risk factors lead to higher risk of CAD without threshold effect. Majority of events occur with moderate elevation of many risk factors than marked elevation of a single risk factor. Once CAD sets in, drug & interventional therapy is prohibitively expensive and at best palliative, hence the importance of timely preventive interventions. As a nation what is needed is to evolve strategies & policy measures at grass root level that makes healthy food items like vegetables, fruit, whole grains, nuts & fish easily available at affordable prices. Town planners need to think about open spaces for people to undertake daily physical exercise. Labeling of industrial formulation of food products & proper framework for tobacco control strategies including taxation must be worked out. These interventions will have strong, sustained & long term impact on preventing CAD in India. Affordable medical insurance will help those who suffer from CAD. United Nations High Level Meeting held in Sept. 2011 & First Global Ministerial meeting held in April 2011 emphasize the importance of monitoring & surveillance of risk factors and will decide regarding primary preventive action & intervention. Both meetings also emphasized the fact that resources for surveillance, prevention & treatment are severely limited in developing countries. The discussion also brought out the fact that population directed interventions have highest favorable health impact. It was worked out that expense for prevention of NCD per person per year in India would be $ 1.52 which is much lower than the expenditure involved in treating the disease.

The rise & fall of CAD mortality in the western world in the latter half of 20th century correlated directly with changes in lifestyle in the society rather than changed in genetic pool. Therefore India should consider taking help from the world Heart Federation representing 198 societies of Cardiology & Heart Federations worldwide with strong support from ACC/AHA & ESC, whose expertise & experience in prevention and treatment of CVDs are substantial to advocate and assist with the implementation of strategies & initiatives that will lessen the burden of CAD in India.

Constrictive Pericarditis versus Restrictive Cardiomyopathy-Diagnostic Work Up

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The differentiation between constrictive pericarditis and restrictive cardiomyopathy is clinically challenging even after careful physical examination and extensive tests because of the similarities in clinical presentation and hemodynamic data. The incidence of constrictive pericarditis is increasing and recent data from the Mayo Clinic demonstrated that the 25% of the cases are due to cardiac surgery, 22% are idiopatic, 20% secondary to pericarditis, 11% secondary to radiation, 6% to infection, 5% to college vascular disease and 11% secondary to other causes. In some patients with acute constrictive pericarditis the symptoms and constrictive physiology features resolve with medical therapy alone while the majority of patients require surgical treatment. The clinical presentation of constrictive pericarditis includes elevated JVP, peripheral edema, hepatomegaly, pericardial knock, ascites, Kusmaul's sign, and pulsus paradoxus. Clinical symptoms include CHF in most patients, chest pain, G.I. symptoms, Tamponade, arrhythmias, and liver disease. The diagnoses of constrictive pericarditis and a differentiation from restrictive cardiomyopathy often requires use of multiple diagnostic modalities including chest x-ray, CT, echocardiography, MRI, constrictive pericarditis cardiac catheterization, and even surgical exploration. This presentation will focus on the appropriate hemodynamic evaluation of the patient suspected of having constrictive pericarditis.

Natriuretic Peptides in HF and ACS - Useful or Extra Burden

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The purpose of this talk will be to review the role of natriuretic peptides in normal and abnormal physiologic process. We will review the application of these biomarkers in heart failure and acute coronary syndromes and their evidence to guideline clinical decision-making.

10 points to Remember on Management of Cardiovascular Diseases during Pregnancy

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- Most women with heart disease can become pregnant.
- The management of a pregnant patient with heart disease should ideally start before pregnancy.
- The diagnosis and management of heart disease in pregnancy should also take into account fetal safety
- Drugs, including safe drugs, should be used in pregnancy only if absolutely indicated and in the smallest dose effective.
- A key to good outcome in women with severe heart disease is close follow up.
- Normal physiological changes of pregnancy can either mimic or obscure signs and symptoms of CV disease.
- Women with Eisenmenger syndrome, severe pulmonary arterial hypertension, severe LV dysfunction, Marfan syndrome with dilated aorta, valvular or congenital heart disease with reduced functional capacity (NYHA class $\geqslant$ III) should not become pregnant.
- Shortness of breath early after the delivery – think peripartum Cardiomyopathy.
- Acute myocardial infarction in pregnancy predominantly due to coronary atherosclerotic disease during early part of pregnancy and coronary dissection during late pregnancy and early post partum period.
- Most women with heart disease can deliver vaginally.

Pregnancy and heart disease: which drugs to avoid?

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Table: Drug Safety during pregnancy and lactation

Drug	Risk Category	Information in human	Potential Complications	Safety for breast feeding
Drug	Risk Category	Information in human	Potential Complication	Safety for breast feeding
Furosemide	C	Limited	Hypotension and decreased uterine perfusion	Compatible
Intravenous nitroglycerin	B	Modest	Hypotension and decreased uterine perfusion	Unknown
Intravenous nitroprusside	C	Limited	Thiocyanate toxicity	Unknown
Nesiritide	N/A	None	Hypotension and decreased uterine perfusion. Effect on the fetus unknown	Unknown
Dopamine	C	Limited	Unknown	Unknown
Dobutamine	B	Limited	Unknown	Unknown
Milrinone	C	Limited	Unknown	Unknown
ACE inhibitors/ Angiotensin receptor blockers	C	Limited	Renal insufficiency, oligohydramnios, IUGR, prematurity, bony malformation, limb contractures, PDA, pulmonary hypoplasia, RDS, hypotension, anemia and neonatal death.	compatible
Carvedilol	C	Not available	Unknown beta 2 receptor blocking may cause premature uterine contractions	Unknown
Bisoprolol	C	Not available	Unknown	Unknown
Metoprolol succinate	C	Not available	Unknown	Unknown
Metoprolol Tartrate	C	Modest	Relatively safe	Compatible monitoring of infants for sings of beta blockade recommended
Digoxin	C	Modest used for both maternal and fetal indications	None reported	Compatible
Spironolactone	C	Limited	Possible antiandrogenic effect and feminization	Compatible
Warfarin	D	Modest	Teratogenic effect in 1st trimester (Warfarin embryopathy), increased maternal and fetal bleeding.	Compatible
Heparins	C	Extensive	Do not cross the placenta	Compatible

IUGR-Intrauterine growth retardation, PDA- Patient Ductus Arteriosus, RDS-Respiratory Distress Syndrome

Sleep Apnea – Sleep study

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For centuries it was thought that snoring is a sign of deep and good sleep, but it is a myth. Actually snoring is a sign of a serious disease called Sleep apnea syndrome. As the evolution occurred, science progressed and along with it lot of gadgets invented which made lifestyle of human being sedentary. It also brought lot of lifestyle disorderes like HT, CAD, DM, CV stroke etc. Sleep apnea is also one such lifestyle disorder. Incidence of sleep apnea is very high in obese and morbidly obese persons. According to one study, every 5th person in US is suffering from sleep apnea. Incidence in India is also very high and increasing very rapidly. Though majority of persons are obese, it can affect nonobese persons also. Pathophysiology of sleep apnea is very complex but in simplified way it occurs due to narrowing and collapse of upper airway leading to complete cessation of breathing and apnea. Because of this repeated episodes of apneas leading hypoxia and arousals, patients of sleep apnea does not get quality sleep. This leads to symptoms of daytime hypersomnolance, loss of memory, irritability and depression.

If sleep apnea is not diagnosed and treated early, it increases the risk of HT, CAD, DM and C-V stroke by manifold. It can be diagnosed very easily by a test called polysomnography or sleep study. Here patient is admitted overnight and various parameters are recorded like EEG, SpO2, snoring, apneas, thoracic and abdominal movements, nasal flow etc. Based on this whether pt is having sleep apnea or not is decided and severity is also decided. Once sleep apnea is diagnosed, it can be very easily treated by an instrument called CPAP. It is called as Continuous Positive Airway Pressure. It pumps air by pressure through nose and does not allow airway to collapse and prevents apneas. This has to be used every night by patient. It has got efficacy as high as 95%. Other modalities of treatment like Surgery (UPPP etc.) and Oral appliances are also available but it is not as effective as CPAP therapy.

Management of RA

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Rheumatoid arthritis is the commonest cause of inflammatory polyarthritis involving small and large joints. The newer criteria do not require a strict symmetrical distribution and lay more emphasis on number of joints, small joints and serology. Anti CCP antibody (Cyclic Cittrulinated Peptide) is the most specific test for RA with a specificity from 95-98% in various series. Differential diagnosis of polyarthritis of few weeks duration includes viral arthritides especially Chikungunya infection, other viruses like rubella, parvovirus, connective tissue diseases like lupus, scleroderma, infections like leprosy and tuberculosis and early vasculitis cases and in a child, acute leukemia.

Assessment of disease activity in rheumatoid arthritis has gone more objective now with the use of indices like the Disease Activity Score (DAS 28) using ESR or CRP and similar other indices like the CDAI, SDAI etc. These are derived from SJC (swollen joint count), TJC (tender joint count), physicians and patients global assessment and acute phase reactants. Long term control of RA is better if treatment decisions are based on such objective monitoring. The goals of treatment are symptom relief, disease modification and prevention of structural progression (radiologic) and complications. Ultimate targets are remission and cure.

Methotrexate still remains the anchor drug at doses higher than before (upto 20-25mg weekly). Leflunomide is another option. Combination DMARDs control the disease quicker and better than monotherapy. Sulfasalasine and hydroxychloroquine are used in combination with MTX. Corticosteroids are disease modifying especially if used early in disease – are known to prevent erosions. Use of cytotoxics like cyclophosphamide and asathioprine are limited to extraarticular complications of RA.

Biologics are the newer compounds that have brought revolution to the treatment of RA. Their advantages are quick onset of action, better control, prevention of erosions and use in MTX refractory patients of RA. Disadvantages are cost, tuberculosis and fungal infections and theoretical risk of malignancy after long term use.

Small molecules like the JAK and Syk inhibitors are the latest compounds in clinical trials in RA which have the advantage of being oral compounds. With such an orchestra of different treatment arms, each better than the other, remission and even cure should not be difficult in the near future especially if used early on in RA.

Hemorrhage Control In Trauma

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Hemorrhage is the second leading cause of Mortality and Mortality in Trauma. Bleeding can be External or Internal. External sources including wounds on Scalp and Extremities bleed profusely at times and losses are often underestimated. Various Hemostatic agents have been tried out and their role is discussed.

Internal Bleeding can be from Thorax, Abdomen, Pelvis or Long Bone Fractures. Hemorrhage may not be the only cause of Shock as Cardiac Tamponade, Spinal shock etc can cause shock without major bleeding. Treatment is directed towards Resuscitation- both at the site and in the Hospital, followed by control of bleeding. Control is by Pharmacological, Interventional and Surgical methods.

Pharmacological methods include Tranexamic acid and Recombinant Factor VIIa Interventional methods are becoming more important in controlling bleeding in Pelvis and Solid Organs. FAST is a quick method of assessing Abdominal Bleed

CT scan was used only in Hemodynamically stable patients, but is now being used to assess site of Bleeding prior to Angiography.

Surgery consists of repair of bleeding tissue, vascular repair and excision of bleeding organs. Pelvic bleeds are controlled by use of Fixator, Preperitoneal pelvic packing and Angioembolisation. Stabilisation plays an important part in controlling bleed from fractures.

In severe bleeding, Massive Transfusion is needed and its pros and cons are discussed. The role of Damage Control Surgery is also highlighted. Diagnostic Laparoscopy is of limited use as far as patients with bleeding are concerned.

Title: Damage Control in Trauma Surgery

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To define, it is reemergence of the surgical "Bail Out" procedure. It is an abbreviated surgery and restoration of near normal physiology, in a staged approach to life-threatening injury.

Some of the polytrauma patients develop what is known as the 'lethal triad' --coagulopathy, metabolic acidosis, and hypothermia. This is a life-threatening condition which significantly contributes to increased mortality. In such patients if surgeon goes for definitive repairrequiring longer operative time, the mortality rate is very high.

In phase I of Damage control surgery (DCS),an abbreviated laparotomy is done only to control hemorrhage & contamination rather than definitive repair.In Phase II, then patient goes in the intensive care unit for optimization of the hemodynamics. Once the patient is hemodynamicallystable (about 24-48 hours), patient again goes for definitive surgery (phase III) for injured organs as well as definitive closure of abdomen if possible.The survival rate is shown quite high and statistically significant in various published series though with increased complications.

Management of Chest Injuries

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Chest injuries are a major cause of preventable deaths due to trauma. High index of suspicion and consideration for mechanism of injury can prevent missed injuries. Anatomically it is important to remember that any injury below nipple level on chest can be associated with abdominal injury.

Patients of thoracic trauma suffer from difficulty in ventilation which is affected by inadequate oxygen exchange due to parenchymal lung injury, changes in intrathoracic pressure, pump failure due to myocardial injury and splinting of chest wall due to pain. There may be further a problem of hypovolemic shock due to blood loss.

Thoracic injuries are clubbed as "Deadly Dozen". Six immediate life threatening injuries can be detected during the primary survey (lethal six) while other six are detectable during the tertiary survey (hidden six). The lethal six are:

- Airway obstruction
- Tension pneumothorax
- Massive hemothorax
- Open pneumothorax
- Flail chest.
- Pericardial tamponade

Most of these injuries can be managed by general practitioners without the need of specialist surgeon. Management of majority of these injuries includes conservative measures like oxygen supplementation, good analgesia and chest physiotherapy along with insertion of a chest tube.

The hidden six are Ruptured aortic arch, Tracheobronchial tree injury, Myocardial injuries, Diaphragmatic tear, Esophageal injury and Lung contusion. These are a major cause of morbidity and fatal if missed.

Interventional Radiology In Trauma

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With the technical advances and the increasing availability of sophisticated imaging equipment, techniques, and protocols, and with continually evolving transcatheter endovascular therapies, interventional radiology has become an integral part of clinical management of trauma patients. Endovascular techniques represent an attractive option for both stabilizing and definitively treating patients who have sustained significant trauma, with resultant vascular injury. This presentation is an overview of the various endovascular treatments that may be required in the management of patients with vascular trauma, based on the different anatomical regions and organs.

Management of pelvis fracture

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The Pelvis has a unique place in our body because it connects the axial skeleton to the lower limbs.Fractures of Pelvis have the highest mortality rates of all Skeletal injuries.These injuries are caused by High Velocity Trauma.The surgical anatomy is worth understanding to know the relation between the mechanism of injury and the type of fractures.These fractures require a high degree of suspicion for diagnosis.The treating surgeon should be familiar with the primary resuscitative measures while treating pelvic fractures before shifting the patient to a tertiary care centre,as these fractures have associated injuries and require a team approach for comprehensive management.The tertiary care trauma centres should also have a algorithm in place for management of such fractures.

Early Management Of Spinal Injuries

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Introduction

Along with significant infrastructure development including smoother and better roads, we have seen an influx of high speed cars on our otherwise potholed roads. Added to this, is a low tolerance for driving rules, creating high speed impacts. Despite numerous attempts by the authorities to institute seat-belts and helmets, offenders take pride in flouting these norms. All this has led to higher incidence of spinal injuries, either isolated or as part of poly-trauma.

Primary management at the site of injury

Paramedical personnel need to be trained to apply a cervical immobilising collar, regardless of the injury, and all patients to be shifted on to the spine board. Even the people at large need to be made aware of the potential harm caused by inadvertent manipulation of the injured spine. Maintenance of the arterial BP, is of prime importance in preserving the blood flow to the spinal cord and thus prevent further damage.

Management in the era

All patients with suspected spinal trauma need to be transported on a spine board or a hard stretcher only. After primary assessment, a detailed neurological evaluation and documentation needs to be done. In order to palpate the entire spine, the patient can be log-rolled and a DRE can also be done at the same time. C-spine clearance is a must. Basic radiological evaluation should be done in the ER only, and if necessary a CT scan as well. If the patient doesnot pass urine voluntarily, catheterise the patient.

Pharmacological management of the injured spinal cord

Controversy exists regarding the role of MP in the acutely injured spinal cord. As per the NASCIS III protocol, MP should be given to all patients with acutely injured spinal cord, arriving within 8 hours of the injury, in order to prevent secondary damage by inflammatory mediators. Many other drugs have shown promise, but are as yet inconclusive. All spinal cord injured patients have loss of sympathetic tone following the injury, and need pharmacological support to maintain their BP. Consistent maintenance of MAP above 85

mm Hg is vital to the survival of the residual neurons. DVT prophylaxis needs to be instituted in these patients, by way of LMWH or calf-pumps.

Nursing care of the sci patient

These patients develop pressure sores within no time, and hence, log rolling on an hourly basis must be instituted from the time of arrival of the patient in the ER. Early spinal stabilisation helps prevent secondary injury to the spinal cord, and facilitates nursing care. Skin and back care, along with chest and four limb physiotherapy must be instituted on a twice daily basis. Maintaining electrolytes and appropriate nutrition is a must for the overall positive outcome of these unfortunate patients.

Conclusion

If appropriate and timely care is taken, a significant number of these spinal injured patients go on to have a meaningful life. Our goal should be to preserve whatever residual function the patient has, and make sure he becomes a useful member of the society.

A Number Game: " Wean Your Patient from Mechanical Ventilation"

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Weaning or discontinuation of mechanical ventilator is a process to discontinue any patient kept on mechanical ventilation due to respiratory failure. It is actually a milestone in any patient's recovery in the ICU. Patients spend average 40% of total duration of mechanical ventilation in discontinuation process. There are some issues to be discussed to make this process the most successful. First, physician need to identify the potential of the particular patient before initiating the process. Second, attempt to determine which parameters to be assessed for determining success or failure of the process. Third, what are the parameters which guide to discontinue the mechanical ventilation and remove the artificial airway? Forth, what if we fails, how to reassess and which factors are to be looked for. And finally we will define strategy for long term management.

Albumin: Consensus and Controversies

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- Intravascular fluid therapy is a common critical care intervention. However, optimal type of resuscitation fluid, crystalline or colloid remains controversial and unanswered. More specifically expensive colloid like albumin is used for various therapeutic indications or physiological goals. Despite many theoretical-physiological effects because of its oncotic and non-oncotic effects, there has been little quality evidence to support its widespread clinical use.

- Systemic reviews have led to conflicting result regarding safety and efficacy of albumin. Contradictory to Chochrane Injuries Group's result which challenged SAFE trial provided strong evidence that 4% albumin is as safe as saline. Subgroup analysis of albumin treated group revealed a trend towards decreased mortality in patient with septic shock and trend towards increased mortality in trauma patient.

- There is no evidence from RCTs that resuscitation with colloid reduces the risk of death, compared to resuscitation with crystalloids in patient with hypovolemia, burns, trauma or hypoproteinemia.

- However there is an RCT showing that albumin administration may improve organ function in hypoalbuminemic critically ill patients. It results in less positive fluid balance and better tolerance to enteral feeding.

- There are limited data supporting use of albumin in specific patient populations including dialysis related hypotension following large volume paracentesis in diuretic refractory ascites, to prevent renal failure in SBP associated with cirrhosis, to improve oxygenation in ARDS (with hypoalbuminemia) in combination with furosemide.

- At last, importantly, results of subgroup analysis, higher (than 4%) albumin concentration and other synthetic colloids require vigorous evaluation in clinical trials.

Sedation in ICU

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Abstract: Sedation is an integral part of management of a ventilated and critically ill patient. Fine balancing between under sedation and over sedation required to be done. Each icu must have protocols or guidelines.This lecture discusses patient factors in the icu for which sedation and analgesia are needed, Monitoring different parameters for the level of sedation. Uses of different drugs along with it's side effects. Delirium: causes, patient effects and management of patient with delirium. This lecture discusses few studies regarding different agents, combination of agents and monitoring protocols. It also discusses about newer agents like paracetamol and dexmedetomidine. It focuses briefly on special classes like geriatric, females, paediatrics, critically ill, renal compromised are naming few.

Conclusion: Appropriate sedation, proper monitoring, correct agent or combination of agents and proper outcome are the keys to formation of sedation protocols in icu

Hemoptysis

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Hemoptysis is a presentation of many lung diseases. As the burden of Tuberculosis is very high in India, there are lot of patients presenting with hemoptysis. In a patient with hemoptysis, if it is not controlled timely, patients will die within minutes. Still there is lack of awareness regarding different modalities of treatment at the physician level. While managing a case of hemoptysis it is very important to determine the cause of hemoptysis as well as the source of bleeding ie. which lobe or segment. Bronchoscopy is very helpful diagnostic tool in determining the source as well as the cause of bleeding. Though it should be remembered that it dous not have any therapeutic role. If hemoptysis is persistent and does not stop with conservative treatment then one should always go for definitive treatment to stop hempotysis. There are two options for definitive treatment. First is Bronchial artery embolization and another is resection of the lobe or segment particularly if the disease is localized. Bronchial artery embolization is preferred if the disease is bilateral. Bronchial artery embolization is routinely done now a days and it gives success rate up to 95-99% in different studies. Though there is a chance of recurrance it is a life saving procedure and should always consider this option rather than playing waiting game.

Updates on Sleep Medicine

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Obstructive sleep apnea (OSA) is one of the most important sleep disorders recognized in last 2 decades in field of sleep medicine. Typically it was considered to be disease of obese people and in India it was grossly ignored as obesity was not that prevalent. Now as obesity is increasing OSA recognition needs attention. After one the large Chinese study it is recognized that OSA can also happen in slim people due to abnormal upper airway. Even though OSA is common why it does needs work up and treatment! In last few years we have recognized that patients with OSA are at increased risk for several cardiovascular complications like HTN, atherosclerosis, stroke, atrial fibrillation etc. Recently OSA has been linked to increase mortality. If we treat OSA can we decrease the risk of these complications? In this lecture we will discuss about cardiovascular complications of OSA and effect of treating OSA on those complications.

Interesting Puzzles in Field of Pediatric Neurology

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A CONVULSING CHILD, STATUS EPILEPTICUS AND NON CONVULSIVE STATUS

CONVULSION OR SEIZURE

A seizure or convulsion is defined as paroxysmal, involuntary, excessive gray matter discharges which clinically manifest as intermittent, paroxysmal, stereotyped disturbance of consciousness, motor function, behaviour, perception and sensation, autonomic function. These disturbances may occur singly or in combination.

A CONVULSING CHILD IN OPD

- Recovery position, Roll the child on to one side with upper knee bent and head lowered
- Maintain clear airway

Before / No IV Access you can give

	Route of administration	Paediatric dose
Diazepam - Intramuscular Diazepam has erratic absorption and is not recommended	Rectal	0.2 to 0.5mg /kg
Midazolam - The action starts in 2-5 minutes and the Mean peak plasma concentration at 15 minutes.	Intranasal or Buccal	0.2 to 0.4 mg/kg
Midazolam - as effective as IV diazepam	Intramuscular	0.4mg / kg

STATUS EPILEPTICUS

(SE) presents in a multitude of forms, dependent on etiology and patient age (myoclonic, tonic, subtle, tonic-clonic, absence, complex partial etc.). Generalized, tonic-clonic SE is the most common form of SE.

Conventional "textbook" definition of status epilepticus:

- Single seizure > 20 minutes
- Series of seizures >20 minutes without full recovery

Why 20 minutes?

Animal experiments in the 1970s and 1980s had shown that neuronal injury could be demonstrated after 30 min of seizure activity, even while maintaining respiration and circulation.

If appropriate therapy is delayed, SE can cause permanent neurologic sequelae or death thus any child who presents actively convulsing should be assumed to have SE.

The longer SE persists,

- the lower is the likelihood of spontaneous cessation
- the harder it is to control
- the higher is the risk of morbidity and mortality

Treatment for most seizures needs to be instituted after 5 minutes of seizure activity

Common causes:

- Febrile status epilepticus
- Sudden reduction of AEDs
- Acute cerebral trauma
- Idiopathic epilepsy
- Bacterial meningitis
- Encephalopathy/Encephalitis
- Poisoning
- Others: Vascular, Tumour, Drugs

Treatment

- Site I/V line & take blood for relevant investigations
  - Glucose
  - Ca / Phosphate/ Mg/ Na
  - CBC, Urea & electrolyte, Arterial blood gas
  - Blood culture
  - LFTs, ammonia
  - Anticonvulsant levels

Treatment of stage 1 SE

	Route of administration	Paediatric dose
Diazepam	IV	0.25 – 0.5mg / kg
Lorazepam	IV	0.1 mg / kg
Midazolam	IV	0.2 mg / kg as bolus

Treatment of Stage 2 SE

	Route of administration	Paediatric dose
Phenytoin	IV infusion (not exc 50 mg / min)	20 mg / kg at 25 mg / min
Fosphenytoin	IV infusion (not exc 100 PE / min)	15-20 PE/ kg
Phenobarbital	IV bolus (not exc 100mg/min)	15-20 mg / kg
Valproate	IV bolus + maintenance	20 mg/kg bolus followed by 10 mg / kg 8 hourly
Levesam (Levetiracitam)	IV bolus + maintenance	20 mg/kg bolus followed by 10-20 mg/kg/dose 8- 12 hourly

- IV valproate may be a good alternative - one very recently published RCT has shown that IV valproate is as effective as IV phenytoin
- recent uncontrolled evidence that IV levetiracetam may also be good alternative treatment

Treatment of Refractory (stage 3) CSE – Intubate and ventilate

- 31-44% of patients with status & mortality of 16-23%
- Continuous or at least intermittent/repeated EEG is essential

Midazolam	0.2-0.4mg/kg bolus followed by infusion of 0.1-1.0mg/kg/hr
Thiopentone	Pentobarbital infusion 1-5 mg/kg/hr after bolus of 10 (5 – 15) mg/kg
Propofol	Propofol infusion 5-10 mg/kg/hr after bolus of 2 mg/kg
Phenobarbital	10-20mg/kg bolus at 25mg/min followed by an infusion of 0.5-1mg/kg/hr increasing to 1-3 mg/kg/hr
Ketamine	1-5mg/kg bolus followed by 1-5mg/kg/hr

- the length of time for anaesthetic treatment required is different depending upon the cause

NON- CONVULSIVE SE?

If child does not begin to respond to painful stimuli within 20 - 30 minutes after tonic - clonic SE, suspect non - convulsive SE & ask for urgent EEG

- Up to 20% of children with SE have non - convulsive SE after tonic - clonic SE
- Common in children with viral encephalitis
- A child with non- convulsive status should be treated like convulsive SE

Role of Bedside Sonography of Skull: An Important NICU Tool of Current Era...

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Cranial ultrasonography (CUS), an easy-to-use imaging modality at the bedside, has become a standard of care for preterm very-low-birth-weight (VLBW) infants in most neonatal intensive care units in the developed countries. The advantages of this imaging tool are its noninvasiveness, absence of ionizing radiation and its ability to diagnose intraventricular haemorrhage (IVH) and ventriculomegaly (VM) with high degree of sensitivity and specificity. Repeated CUS examinations, especially in the presence of severe grades of IVH or posthemorrhagic VM, help in predicting outcome and clinical decision making in the preterm infant. CUS is also useful to diagnose cystic variety of periventricular leucomalacia (PVL), the presence of which strongly predicts adverse neurodevelopmental outcomes.

As most cases of IVH, VM and PVL in the preterm neonates are asymptomatic, these lesions can only be diagnosed by routine screening CUS examinations. In most neonatal units, all VLBW (<1500g) infants or those born at <32 weeks of gestation are candidates for screening CUS. In the presence of other perinatal risk factors such as monochorionic twin gestation, abruptio placentae or perinatal asphyxia, larger and more mature babies also need to be screened for abnormalities on CUS.

Major IVHs usually occur during the first few days after birth; hence the first CUS is usually done within 72 hours of life. Subsequent examinations would depend on the birth weight, gestational age and neonatal course of the infant. At the very least, a repeat CUS should be done prior to discharge from the neonatal unit as cystic PVL, porencephalic cysts and ventriculomegaly may take a few weeks to evolve.

Conclusion: Bedside CUS is a useful tool in the NICU that assists in the management of preterm VLBW infants. Clinicians and neonatologists caring for preterm infants should familiarize with usage of ultrasound machine at the bedside and be able to diagnose common neuropathological conditions in this vulnerable population.

Outcome in Very Low Birth Weight Babies: Past, Present and Future

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Preterm birth, especially before a gestational age of 32 weeks, has profound impact on public health and education worldwide. At present preterm infants who are of very-low-birth-weight (<1500g) represent approximately 2% of all live births and utilise considerable amount of manpower and resources in neonatal intensive care units. With advances in perinatal and neonatal care survival of very-low-birth-weight (VLBW) infants has increased significantly over the last two decades in the developed countries. In Singapore, the current overall survival rate in VLBW infants is more than 90% and the limit of viability appears to between 23-24 weeks of gestation. However these survivors, due to various complications related to prematurity, are at an increased risk of long-term adverse neurodevelopment outcomes. For example, cerebral palsy rates among VLBW survivors range between 8-10% in most reviews. Follow-up studies have also revealed high rates of neurodevelopment disabilities among those born close to the limit of viability.

Several risk factors have been associated with adverse long-term outcome in this population such as maternal chorioamnionitis, lower gestational age, and extremely low birth weight, neonatal morbidities such as intraventricular hemorrhage (IVH), sepsis, necrotizing enterocolitis (NEC), bronchopulmonary dysplasia (BPD), and major abnormalities on cranial ultrasound at the time of discharge. Some of these risk factors such as late-onset sepsis, NEC and severe BPD are modifiable and low incidence of these neonatal morbidities may result in improved long-term outcomes.

Conclusion: Resuscitation policies for extremely preterm infants should be based not only on the survival but also on the long-term neurodevelopment outcomes especially among those born close to the limit of viability.

Respiratory distress syndrome: to tube or not to tube?

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The use of mechanical ventilation in premature infants with respiratory distress syndrome (RDS) and respiratory failure often results in barotrauma, volutrauma and chronic lung disease (CLD). Research indicates that early surfactant therapy and initiation of nasal continuous positive airway pressure (CPAP) for these infants significantly reduces the need for mechanical ventilation and the incidence of CLD. Clinical trials indicate that optimal management of neonatal RDS could be improved by early surfactant treatment followed immediately by extubation and stabilization on CPAP. Evidence suggests a synergistic effect between early surfactant administration (within 2 h of birth) and rapid extubation to nasal CPAP with a significant reduction in the need for mechanical ventilation and its associated morbidities.

Early surfactant replacement therapy with extubation to NCPAP compared with later, selective surfactant replacement and continued mechanical ventilation with extubation from low ventilator support is associated with a reduced need for mechanical ventilation and increased utilization of exogenous surfactant therapy. All these modalities are ultimately is towards goal of less and less mortality, morbidity and less days on oxygen , i.e. prevention of chronic lung disease.

Considerable effort has been devoted to the development of strategies to reduce the incidence of bronchopulmonary dysplasia (BPD), including use of medications, nutritional therapies, and respiratory care practices. Unfortunately, most of these strategies have not been successful. To date, the only two treatments developed specifically to prevent BPD whose efficacy is supported by evidence from randomized, controlled trials are the Parentral administration of vitamin A and corticosteroids. Two other therapies, the use of caffeine for the treatment of apnoea of prematurity and aggressive phototherapy for the treatment of hyperbilirubinemia, were evaluated for the improvement of other outcomes and found to reduce BPD. Cohort studies suggest that the use of continuous positive airway pressure as a strategy for avoiding mechanical ventilation might also be beneficial. Other therapies reduce lung injury in animal models but do not appear to reduce BPD in humans. The benefits of the efficacious therapies have been modest, with an absolute risk reduction in the 7-11% range (2). Further preventive strategies are needed to reduce the burden of this disease.

Medico legal Aspects while Practicing Critical Care

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While sitting as a member in Consumer Commission, I have noticed that the doctors do not know what amounts to "Medical Negligence"! I have delivered many lectures on this subject before various medical associations and at every place I have also noticed it!

- Aetiology of medical negligence complaint.
- Medical Negligence.
- Common allegations.
- Common Defences

Role of Fetal Echocardiography: Worth peeping in future of forthcoming life : Message on basis of Outcome

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The field of pediatric cardiology is ever expanding. Early diagnosis and timely intervention, whether medical, surgical or catheter based, provide good long term outcome for many patients suffering from significant congenital heart diseases (CHD). Currently, the morbidity and mortality after many neonatal and infant cardiac interventions is very low with good long term survival. So naturally the focus of professionals involved in pediatric cardiac care has changed to provide earliest diagnosis of structural cardiac anomaly and plan the management. What else can be early if we can do these anomalies in fetal life by fetal echocardiography!! Fetal echocardiography has become an important tool not only in preventive aspect of CHD management, but also helps us in prospectively planning about management of unborn child with heart problem.

We will be discussing several issues related to fetal echocardiography like timing, limitations, estimating risk in fetal life and planning delivery in fetus with cardiac problems.

Surfactant Therapy - Past, present and future

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Respiratory Distress Syndrome is the major cause of deaths in premature babies. Thus there is a great need for decreasing mortality and morbidity from RDS.. Over the last 3-4 decades great many advances have taken place in the management of babies with RDS. Successful treatment of respiratory distress syndrome with Constant Positive Airway pressure (CPAP) was first described by George Gregory in 1971 .. Then the first report by Fujiwara et al in 1980 of the successful surfactant replacement therapy in newborns with respiratory distress syndrome opened up new hope of further reducing both mortality and morbidity from respiratory distress syndrome. Numerous randomized clinical trials of surfactant replacement therapies using different forms of surfactants around the world have established the efficacy of surfactant replacement therapy in reducing mortality and morbidity in RDS . Surfactant replacement therapy has been used both as prophylactic and rescue therapy. Prophylactic therapy has been shown to have lessened the lung injury and chronic lung disease. Based on the findings of numerous recent studies it is found that the treatment of RDS in the delivery room can be achieved by using CPAP as the first option and then use of surfactant in those infants who do not get better on CPAP or need intubation for mechanical ventilation. Other options include administration of surfactant followed by application of CPAP. Discussion will include various options including the benefits of CPAP and ,nCPAP and Surfactant therapy as the strategies of treatment in RDS.

Early Stimulation Therapy for a Newborn! Need of an Hour... Are We Ready For It??

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The newborn infant in the neonatal intensive care unit (NICU) is cared for with highly advanced medical technology- reducing mortality, but the incidence of disability and neurodevelopmental problems among survivors remains high and problematic. This highlights the need for a follow up care that would ensure systemic monitoring of general health and neurodevelopmental outcome. The term 'early intervention' designates educational and neuroprotection strategies aimed at enhancing brain development. Early interventional strategies seek to take advantage of cerebral plasticity. Neuroprotection, a term initially used to characterize substances capable of preventing cell death, now encompasses all interventions that promote normal development and prevent disabilities, including organisational, therapeutic and environment-modifying measures, such as early stimulation programs. The programs target the child and the parents both. A basic knowledge about neurodevelopmental assessment and follow up of high risk newborn is essential for any practicing neonatologist and paediatrician. All health facilities caring for sick neonates must have a follow up programme. It requires establishment of multidisciplinary team. The follow up protocol should include assessment of growth, nutrition, development, vision, hearing and neurological status. F Timely specific intervention must be ensured after detection of deviation of neurodevelopment from normal.

First step in high risk clinic is assigning level of neurodevelopmental follow up based on risk severity. Assign the baby to the highest level indicated by risk.

Mild risk for NDD	**Moderate risk**	**High risk**
Prenatal risk factors	Abnormal fetal growth	Fetal distress
>-37 weeks	33-36	<33 weeks
>2500 grams	1500-2500	<1500 grams
Booked pregnancy/intramural baby	Suboptimal perinatal care	Suboptimal transport
Completed course of ANS	Incomplete course of ANS	No ANS
No need for resuscitation	Need for resuscitation at birth	APGAR <3 at 5min. Encephalopathy Multiorgan injury
Levene grade 1	Levene grade 2	Levene grade 3
Not required ventilation	Uncomplicated course of ventilation	Ventilation > 7 days
No shock	shock	Refractory shock
Transient hypoglycemia	Hypoglycemia, bl.sugar <25mg/dl, >3 days	Symptomatic hypoglycemia
Suspect sepsis	Sepsis	Meningitis
NNJ needing phototherapy	NNJ needing exchange transfusion	Kernicterus
NICU admission	Complex course	CLD
Preterm, IVH gr 1 or2	IVH > gr 2 on USG	Ventriculomegaly and/or cystic PVL, hydrocephalus
Normal neurologic exam. At birth	Severe/prolonged encephalopathy	Abnormal neurological exam. At discharge
Good home environment	Suboptimal home environment	Parent concern for NDD

Newborns at low risk requires follow up in clinic by their pediatrician or parents. Newborns at moderate risk of NDD requires screening atleast once in 1st & 2nd six months and then once between ages 1&2, 2&3, and 4&5 by developmental paediatrician. If assessment is normal – reassess on f/up, if inconclusive then watchful wait and reassess but start early stimulation and if it is abnormal then detailed developmental assessment and developmental therapy is advised.

Newborns at high risk of NDD should be advised for intervention with serial comprehensive care at discharge.

Algorithm for follow up of at risk neonates
At risk neonate

Start early stimulation

Predischarge
Assign level of follow up

- Medical exam; nutrition and growth ass.
- Neurological examination – neurobehaviour
- Neurosonogram
- ROP screening
- Hearing screening
- Thyroid screen
- Assessment of parents- parent education, DOC

Follow up

- Medical , nutrition, growth assessment
- Neurological examination
- Developmental tests
- Functional assessment
- Behaviour, cognition, psycho- educational

Timely specific intervention

Updates on Sleep Medicine

Dr. Bhavin Dalal, MBBS, MD, DNB

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Obstructive sleep apnea (OSA) is one of the most important sleep disorders recognized in last 2 decades in field of sleep medicine. Typically it was considered to be disease of obese people and in India it was grossly ignored as obesity was not that prevalent. Now as obesity is increasing OSA recognition needs attention. After one the large Chinese study it is recognized that OSA can also happen in slim people due to abnormal upper airway. Even though OSA is common why it does needs work up and treatment! In last few years we have recognized that patients with OSA are at increased risk for several cardiovascular complications like HTN, atherosclerosis, stroke, atrial fibrillation etc. Recently OSA has been linked to increase mortality. If we treat OSA can we decrease the risk of these complications? In this lecture we will discuss about cardiovascular complications of OSA and effect of treating OSA on those complications.

Guidelines in Management of Atrial Fibrillation

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This lecture will focus on the current ACC/AHA/ESC guidelines on the management of atrial fibrillation. We will briefly review the current state of the literature on the pathogenesis of atrial fibrillation. We will review the current guidelines and review the most recent updates to the evidence regarding treatment for atrial fibrillation (pharmacologic and procedural) and new therapies for stroke prevention.

Aspirin for the Prevention of Cardiovascular Events in Patients Without Clinical Cardiovascular Disease: The Final Word

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Cardiovascular diseases (CVD), which include coronary heart disease (CHD), cerebrovascular disease, and peripheral artery disease, is by far the leading cause of death in most developed countries. It accounts for almost one million deaths annually in the United States alone..Aspirin for secondary prevention of CVD after MI, TIA, CVA, stable angina, and CABG is routinely used to reduce risks of MI, stroke, and vascular death. Evidence from basic research, clinical investigations, observational epidemiologic studies, and multiple randomized clinical trials has provided strong support for the net benefits of Aspirin in reducing CVD events in most patient subgroups

Increased risk of bleeding, chiefly from the gastro intestinal tract and very rarely from intracranial vessels has raised the question of using aspirin for primary prevention of CVD. In a 2006 systematic review of 22 randomized trials of low-dose Aspirin (75 to 325 mg/day) for adverse effects it was found that Aspirin increased the risk of any major bleeding, major GI bleeding, and intracranial bleeding 1.7 to 2.1 times compared to placebo. The absolute annual increase in risk was 1.3 per 1000 patients for GI bleeding and 3 per 10,000 patients for intracranial bleeding. There was no significant difference in risk of bleeding at doses of greater than 162 to 325 mg/day compared with 75 to 162 mg/day.

Based on evidence from 9 large-scale primary prevention trials of men and women without established CVD Aspirin produces a statistically significant and clinically important reduction in the risk of a first MI. Aspirin has not been shown to lead a statistically significant reduction in stroke or cardiovascular death. Any decision to use Aspirin for primary prevention should be an individual clinical judgment that weighs the absolute benefit in reducing the risk of first MI against the absolute risk of major bleeding with long-term administration.

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