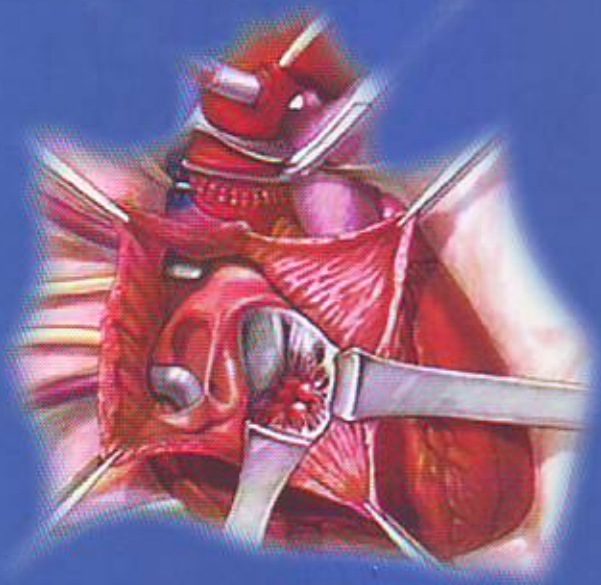
Synopsis

CARDIAC SURGERY

1st Edition



N.A. KAMRUL AHSAN

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Cardiac Surgery : *Morphology, Diagnosis, Indication, Natural History, Technigues and Results*

Edited by :

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Preface

There is hardly any alternative to vivid knowledge of the subject besides rigorous clinical and laboratory investigations, meticulous patient care, disciplined training of young surgeons to improve cardiac surgical care. Attainment of a consistant & integrated knowledge of cardiac surgery is obviously time consuming and laborious. Similarly recapitulation of knowledge is not always easy and attainable in large reference textbooks.

With this recognition we decided to prepare a comprehensive text that entails the current knowledge and practice of prevalent cardiac surgical problems in our background, as a ready reference to the big volume textbooks.

We'll be happy should this would be of help of practicing & young surgeons who attained knowledge of the subject. We hope the synopsis will prove of help to a diverse readership particularly to postgraduate students.

Fundamentals of text writing elaborating details of sign &symptoms, clinical examinations have been avoided. We think reader will take it easily.

Illustrations & drawings have been taken from different books,approval could not be obtained for which we apologize to authors.

We are not beyond any mistakes in spite of our all effort. Any suggestions, constructive criticism is welcome to improve our next update edition.

We are grateful to our colleagues & faculty member of National Institute of Cardiovascular Diseases and ours students for constant inspiration to make the job done during the past years.

We thank our sponsors & publishers to help us in propagating scientific knowledge

February – 2008
Dhaka-Bangladesh

N.A.KAMRUL AHSAN
FACS

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WHAT'S NEW IN CARDIAC SURGERY

Major development

Concentrated : > 2004

- CAD
- CHD
- Valvular Diseases
- Transplantation
- Assist Devices
- Surgery in arrythmias
- Thoracic aorta
- Robotic tech.for MIS

...ARE THE AREAS OF RECENT MAJOR DEVELOPMENT

OPCAB

- Extensively studied with result of sophisticated one.
- Appoximately 20% CABG –OPCAB in 2004
- Some centres 100% of procedures ...Others virtually not at all.
- Assesing results randomized prospective studies- authors suggest that it is time for surgical community
- For specific recommendation- either embracing OPCAB,a way to improve quality care or if against, to stop procedure.
- Most surgeons belive that particular situation fovour one over other & CLINICAL JUDGEMENT STILL BE REQUIRED

Metaanalysis : Randomized trials

- Used composite end point-Death,Stroke or Myocardial Infraction
- Showed trend toward reduction in risk of end points.
- This analysis includes matched 9 studies for total randomized 558 to CABG & 532 to OPCAB

Neurological Complications still vexing problem

*Several studies examined:103 OPCAB without side clamp & 57 side clamp Found lower incidence (0% vs 5.3%)

HEPARIN COATED CIRCUIT

- Ovurum & collegues used routinely in 2500 cases with low heparin(ACT>250 sec),minimal transfusion ,early extubation & rapid recovery
- Authors obtained out standing reslut (Storke 0.8%,deficit 0.6%, MI 1.1%)

5 YRS RANDOMIZED TRIAL

comparing RA graft,freeRIMA&SVG

(Single centre trial)

*RA & freeRIMA compared in<70yrs

*RA & SVG compared in >70yrs (outcomes)

- Cardiac event-free survival & graft patency not significant. 5yrs interim result did not support hypothesis:

RA superior in terms of patency/events than free RIMA or SVG

- Volume & Mortality to examine generally accepted inverse relationship:

*Concluded- Low risk pt. benefit significantly more than high risk in high vol.centres

(Suggested the study needs to be repeated)

Drug-eluting stent

- Generally available late spring 2003 in States
- Studied potential impact on referral
- Reviewed angio & medical records of 196 pts referred for CABG

Hypothesis :Drug-eluting stent & near zero restenosis rate

Found:A total 154pts(79%) still are surgical candidate after these stenting. 21% need not CABG.

- Pts with chronic total occlusion,Left main stenosis & a need for valve surgery still considered best treated by operation
- Percteneous revascularization indicated in diffuse narrowing, restenotic lession & small coronary arteries

Ultimate effect of drug eluting stents may be detemined in next few years

ASPIRIN THERAPY

70 CENTRES IN 17 COUNTRIES CONTRIBUTE

* Found striking difference with / without aspirin with in 24 H of CABG

* Mortality : 1.3%vs 4%

* Less incidence of MI,Stroke,renal failure,bowel infraction

- No increase of haemorrhage gastritis or healing problem

Congenital Problem

- Hypoplastic Lt.Ht. Syndrome:
- Experience of Palliative surgery
- Evoluted manmagement protocol

Traditional Syst-Pul shunt causes over circulation, early collapse & death due to low distolic pr.

*RV-PA shunt is advocated

*4-5mmPTEF used in 1st stage

*2nd stage- Bi-directional Glenn procedure applied

*Mean distolic is heigher than others shunt

*Long term durability yet to study

- Mayo clinic reported >1000m casesfor various diseases including TOF-PA(Mortality decreased to 3.7%)
- Valve conduit needs re- operation but quality of life is excellent

Alternate searches for conduit

- Free style Porcine aortic root (Stent less, Medtronic)
- Bovine Jugular conduit with valve (Medtronic)

Muscular VSD

- With new devices used Self expandible double disk introduced through a sheath in sternotomy / subxiphoid incission under echo window

Double Switch Procedure

- Restoration LV to systemic circulation in Corrected TGA or Discordant was attempted with Excellent early result

Valvular Diseases

- Valve sparing aortic root replacement : is subject of report

SEVERAL WORKERS REPORTED...

- Stentlees Composite Graft for replacemnet of:

  *Aortic root

  *Aorta

With excellent haemodynamic result.

Problem of

Prosthesis-Patient mismatch

- Studied Relationship between Prosthesis size adjusted for patient size & long term survival
- Study concluded –there is no significant co-relation between small prosthesis-patient size in long term survival
- Study includes >1000, 19mm or small size prosthesis
- Study concluded –there is no significant co-relation between small prosthesis-patient size in long term survival
- Study includes >1000, 19mm or small size prosthesis

This study is contrary to:

- General Impression – Prosthesis size & sustained elevated gradient trnslate to decreased long term survival
- Concludes that with available prosthesis few patient should require root enlargement with attendent complexity

Transplantation / VAD

- LVADs developed as 'bridge to transplatation'
- FDA approved "HEART MATE"(Thoratec corp.) for use as a "destination therapy" not candidate for transplant.

Workers presented VAD 3rd INDICATION- "Bridge to recovery"

- they say initiating Reverse remodeling & hypertropy. LVADs helps to restore excellant function after removal of LVAD

Developing VADs

- DeBakey VAD(MicroMed Inc)
- Jarvik 2000(Jarvik Heart Inc)

Are on evaluation for their unique miniaturization & ease implantation

DOMINO HEARTS

- Donar heart of living donars who were themself undergoing heart-lung transplant (Domino)-had not ben subjected to deleterious effect of brain stem death showed better freedom from CAD at 1, 5, 10yrs followup (99%,83%,77%)
- 97 recipient were studied angiographycally

Experience: Supported the hypothesis that brain stem death is a factor for CAD in transplants

Arrythmias

- New classification is forwarded:
- Continuous,
- Intemittant- With foci in PV that needs trigger
- For continous AF –ablation & atrial reduction are reported.

Radiofrequency ablation,cryoablation,microwave ablation,focus ultrasound ablation & laser light ablation were described last years with fairly constant sucess during surgical procedures

Thoracic Aorta

- Workers postulated:
- Complete extended graft replacement is safe,may cofer benit to Type A dissection
- Study showed not the superiority of Total arch replacement
- It did show-Graft arch replacement can be done with reasonable safety.

STENT-GRAFTING

- First medium-term result of Descending aorta presented by Demers & co-workers at Stanford
- 1992-97 1st generation stent grafts used in 103 pts with Thoracic aortic areurysm
- 62 cases were unfit for conventional surgery
- Follow up was 100% complete at 4.5 yrs
- Hospital mortality 9%.Survival 78%vs31%(Good vs Poor candidate) at 8 yrs.
- Concluded that survival after stent repair acceptable in good candidates .But considered bleak in inoperable cohort.

Robotic Surgery

- Sophisticated visualization, multiarticulated miniatured instruments used in different procedure (children/adult)
- Workers opined that robotic surgery,viewed by many as marketing tool,too expensive & cumbersome for Use by majority surgeons.
- Development should be limited to commited telerobotic surgeon for development & validation

CONCLUSSION

- In 2003 cardiac surgery celebrated 50thaniversary since inception exponential progress has continued to present day
- Along with many other technologies not even imaginable at that time
- Provides a stimulating look at the continuing creativity of today's Cardiac Surgeons

AORTIC ANEURYSM / DISSECTING ANEURYSM

True aortic aneurysm is a permanent localized dilatation of the aorta50% or greater than normalcontained by the all natural wall layers

Etiology:

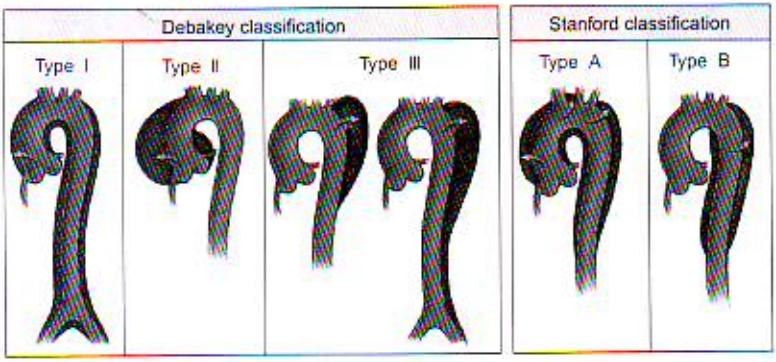
1. Infection
2. False aneurysm
3. Medial Necrosis
4. Atherosclerosis
5. Dissection
6. Trauma

DISSECTING ANEURYSM

Starts as a break/tear in inner layer & extends either direction

- Mechanism: Hydrostatic force of pulsatile blood in the tissue plan
- Tear may be located at any level
- Most common:Ascending,Transverse & descending aorta
- Frequently progressive
- Complications depend on the site & extension
- Ascendings are life threatening for frequent rupture than other sites
- Dissecting may cause only pain & eventually quiscent- Called Ch.dissection
- May remain for years with pressure symptoms

CLASSIFICATIOS :



INVSETIGATIONS

- X-ray- Thorax are most evident:
  Presents as mediastinal widening
  Ascending & Transverse>Middle & Rt.upper mediastinum
  Descending > Lt.mid line / Shadow
- Aortography – Most precise
- CT Angio-Noninvasive with limitation regaqrdig collaterals,run-off

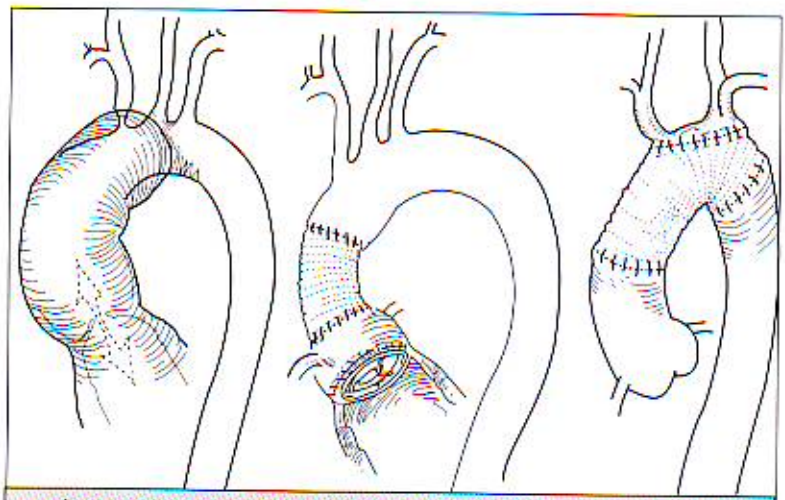
Treatment:

Way of treatment depends on location & cause of - Aneurysm,Ascending Aorta:

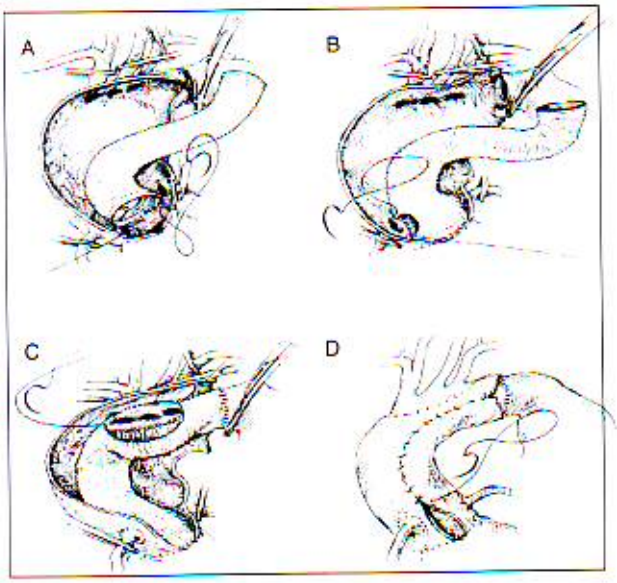
- All cases best managed by operation
- Urgent when symptoms/associated with AR./rupture
- Under femoral CPB
- Technique depends on type/nature lession

Procedural technique differs-

Aortorahpy
Patch aortoplasty
Segmental replacement
Graft-valve composite replacement



Aneurysm of Ascending with Root of Aorta & Valve replacement



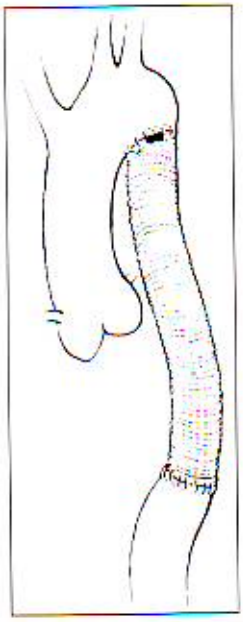
Transverse Aorta -

- Involvement is variable
- Technique also varies

  Temporary occlusion of aorta/vessel

  Coronary/Cerebral flow to maintain

  Graft replacement necessary

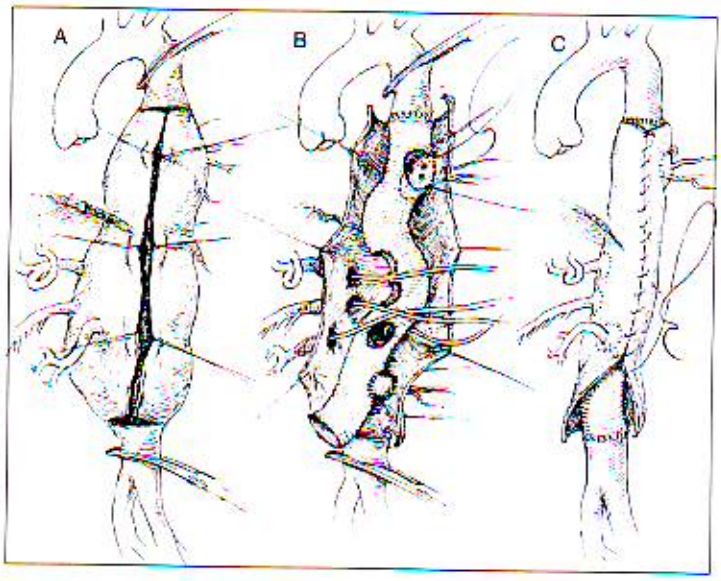


Descending Aorta

- Frequently associated with emergency
- May be acute/chronic
- Starts as tear in any part
- Associated with occlusion of branches with ischemia
- May dissect through the outer layer, RUPTURE

TECHNIQUE

- Replacement with tubular graft – should include origin of tear
- Flow to be directed to true lumen

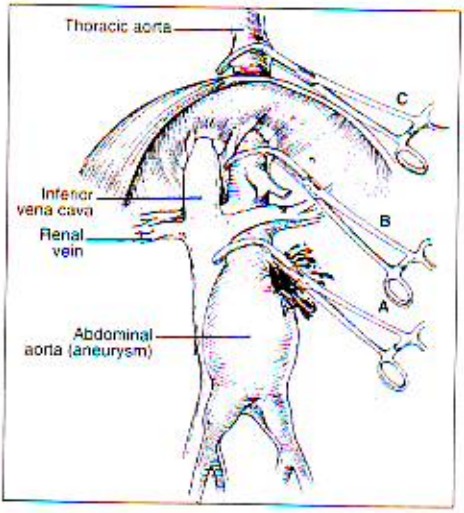


Rupture aneurysm,Abdominal:

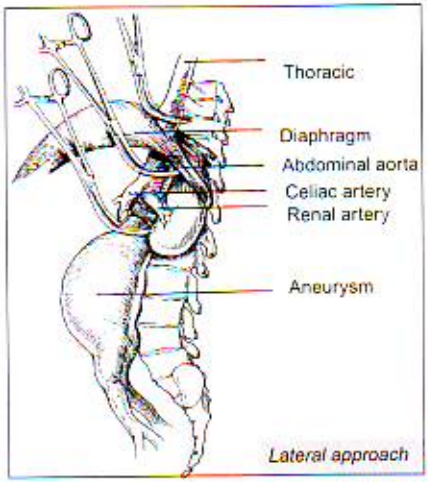
Leaking -Initial stage sealed by clot,should get surgical attention to prevent frank episode

Frunk rupture -Catastrophic event Diagnosis based on clinical examination

-Pulsatile mass with generalized excrutiating pain is diagnostic



Operative technique : Ruptured aneurysm



RESULT OF OPERATIONS IN DISSECTION

Hospital mortality for various types has been 9-33%

Modes of death : With present experience brain injury,Malperfusion syndrome now are the main factors to death

Other factos:

Acuity of dissection
Location of dissection
Comorbidity
Procedural risk factors- Inclusion of arch,use of composite graft
Post operative risk factors-Cardiac dysfunction,neurological & renal problems requiring dialysis are powerful risk factors

DISEASES THAT ARE AMENABLE TO SURGERY

ANEURYSM

Congenital /Developmental
  Marfan Syndrome,Ehlers-Danlos Syndrome
Degenerative
  Cystic Medial
  Non specific(atherosclerotic)
Chronic post traumatic
  Blunt trauma
  Penetrating trauma

Inflammatory

Takayasu,Behcet,KwasakiDiseases,Gaint cell arteritis

Infected

Bacterial ,Fungal,.Spirochetal,Viral

Mechanical

Post stenotic,asscociated AVfistulae

Anastomotic

Post arteriotomy

FALSE ANEURYSM

CHRONIC AORTIC DISSECTION

Type A

Type B

PENETRATING ATHEROSCLEROTIC ULCER

INTRAMURAL HAEMATOMA

DIFFUSE ATHEROSCLEROTIC DISEASE

Natural History :

Less information is available due to lac of widely available exact diagnostic tools.

Medical treatment is of limited value

RESULT OF OPERATIONS (Aneurysm)

Early: Ascending Aorta: Mortality in heterogenous patients is 0-9%

Aortic arch: Heigher ,6-20% related to complexity of the procedure

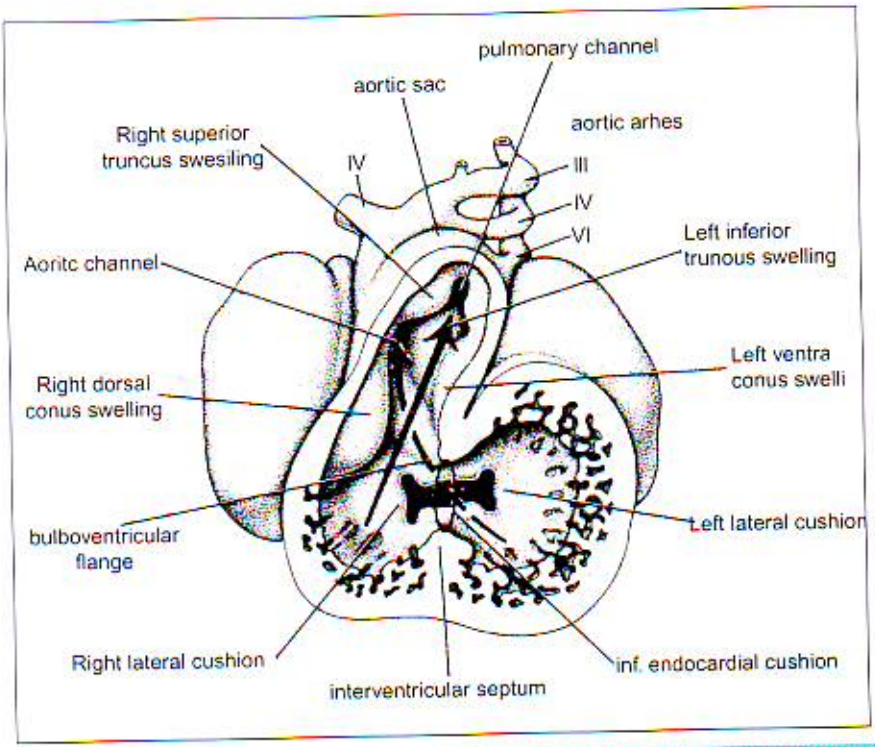
Descending : 5-10%

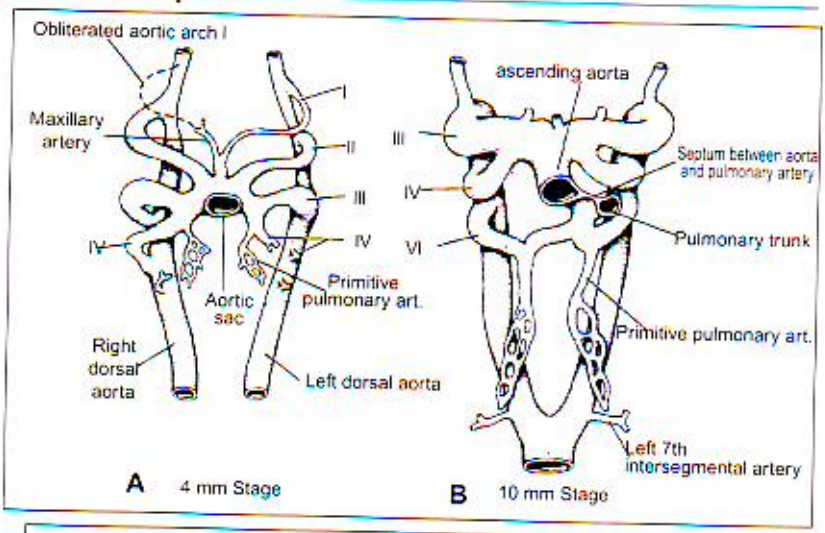
Thoraco abdominal : With experience approximately 3-15%

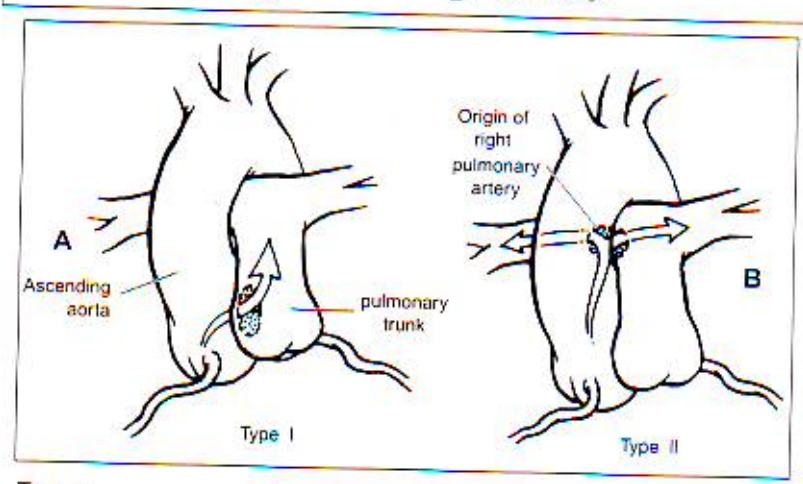
AORTOPULMONARY WINDOW (APW)

DEVELOPMENT

- Rt superior,Lt inferior truncal chusions forms proximal A-P septum (cephalic)
- Rt grows distally to the left & Lt to the right
- Thus a spiral septum is(APS) formed
- IV(4th) aortic arch continuous with aortic channel& VI with pulmonary
- Distal part arises from the wall between IV & VI arch
- At the same time rt conus & Lt conus swelling develops grows distally to unite truncal septum
- Proximally they unite to forms membranous septum along with inferior atrioventricular cushion
- Abnormal migration of VI arch may take place







Types

- Type I –Distal incomplete fusion
- Type II – Incomplete at down stream
- Type III - Severe unequal septation along with dislocation of Rt. sixth arch(abnormal origin of RPA)

Morphology

- Usually large,oval
- At Left lateral wall of aorta
- Inferior to origin of RPA,close to left coronary artery
- May also into RPA

Associated |Anomalies

- PDA
- Coarctation
- VSD
- TOF
- TGA
- Abnormal CA origin

Diagnosis

- Infant with growth failure
- Simlar to Large VSD
- Marked cardiomegalyVariable intensity of systolic murmur
- In some continuous when smaller & pulmonary hypertension is less
- Echo can diagnose definitely
- Aortography shows early filling of P.Trunk.identify other anomalies

Surgery

- Evaluation & confermation
- Under CPB
- Trough Aorta / PT
- Patch closure
- Modification necessary when associated with abnormal origin of RCA
- Hospital mortality is very low with good survival

ARRYTHMIAS : SURGICAL MANAGEMENT

INTRODUCTION

- **Sinus Node Dysfunction:**
  Aging
  Amyloid deposition
  Direct Damage

- **Damage Sinus-AV node pathway**
  Congenital absence
  Surgical Procedures-Atrial Switch,Fontan
  Post-Op fibrosis

Parmanent Pacing

At the end of Surgery – Parmanant electrodes are placed(caped) & brough to chest wall/abdomen subcyteneous. If block persists the Generator placed or transvenous approach may be done

Tachycardia

- Nodal reentry & Tachycardia
- Ectopic Atrial Tachycardia

Intervention

❖ Developed devices of Pacing & AICDs(automatic implantable cardioverter -defibrillator) are available.
❖ Most of these arrythmias can be delt with pharmacheutical measures or PCIs WPW syndromes
❖ Characteristically allows paroxysmal tachycardia by atriventricular reentry
❖ Pt with otherwise normal heart,normal contraction and normal life expectancy
❖ Sudden death only in small portion with associated atrial fibrillation(paroxismal) Intervention
❖ Symptomatic with acessory pathways are indicated for cath.ablation

- Pt with WPW undergoing for cardiac surgery should be preoperatively ablated
- When PCI not available surgical approach (endo/epicardial) to be done to avoid life threatening fibrillation

Atrial Fibrillation & Flutter

- Most common arrythmia diagnosed
- Associated with thromboembolism,Stroke, Pulmonary embolism for slow & stagnation
- Chronic fibrillation leads to cellular changes & fibrosis of atrial myocardium
- Tachycardia-induced cardiomyopathy is end-stage complication

Treatment :

2 APPROACHES:

- CONTROL OF VENTRICULAR RATE & ANTICOAGULATION
- RESTORING SINUS RYTHM

Control Ventricular rate

- Drugs-Digoxin,ß-blocker
- If not control within 48h anticoagulation should started
- Aspirin has little effect
- Warfarin protects well

Restoring Sinus Rhythm

- Cardioversion
- Ablation A-V conduction – CHB & PPM
- Intracoronary Ethanol ablation is alternative to RF current
- Surgical separation of conduction pathway(Maze IIIProcedure)-Prevent reentry & Separation of specific route
- Modified Radiofrequency Maze III with Uni/Bi polar cryoablation
- PCIs based on pulmonary orifices only , not often successful & when applied to LA may be dangerous
- Yet accepted in the interest of avoiding operation
- Syptoms interfares normal life spite maximal medication is indication of surgery
- Contraindicated with severe LV dysfunction

Post Surgery AF / Flutter

- Flutter / Fibrillation frequent (20-40%) after Cardiac Surgery
- Appears in 3 days or later after CABG
- Cause not known.Probably due to inotrops,change in cellular electrical property,alteration of neurohormonal &Electrolyte balance
- Age with atrial myocardial changes, ß blocker withdrwal ,HTN are factors Treatmaent Strategy
- As self limited , directed towards control of ventricular rate
- First line of drug ß blockers
- Scientific data does not supports digoxin
- Ca+ blockers not more effective than ß blockers
- Anticoagulation if not controlled in 48 h & Cardioversion anticipated
- Cardioversion is immidiate if instability present

Other METHODs

- Overdrive Pacing - Bipolar electrodes at Atria,Ventricle(set at 400-800pm >Gradual reduction)
- Atrial defibrillation -Defibrillation electrodes during operation at Rt. & Lt atria..Monophasic shock (2-10J) are delivered (Syncronus, Epicardial atrial defibrillator)

VENTRICULAR :

Tachy / Fibrillation

- Uncommon in absence of MI
- Commonly with large area/ aneurysm
- Result of Macro/Micro reentry circuit or may be with normal / abnormal automaticity
- Usually with low EF (.30) capare to .50 of CABG candidates

Treatment:

Continued developing:

- Aneurysmectomy & isolation of focus with CABG
- Excission of the scar after mapping
- Monomorphic –Cryoablation
- AICD

Other Situations

- RV dysplasia(arrythmogenic)- Simple Ventriculotomy
- LV Dysplasia(arrythmogenic), Rare – Mapping & ***ENCICLING ENDOCARDIAL VENTRICULOTOMY***
- Focus/Substrate is subendocarial/LV septum
- Near margin of infract/Aneurysm
- Areas of mixed cell & fibrosis causes slow conduction or block respectively
- Necessary to initiate tachy.
- Reentrant circuit provided by viable myscle bundle in infracted area

Other Causes

- Diseases causing - Hypertrophy.Interstitial fibrosis,Degenerative changes,Myocarditis-Myocardium can act as substrate for arrythmias

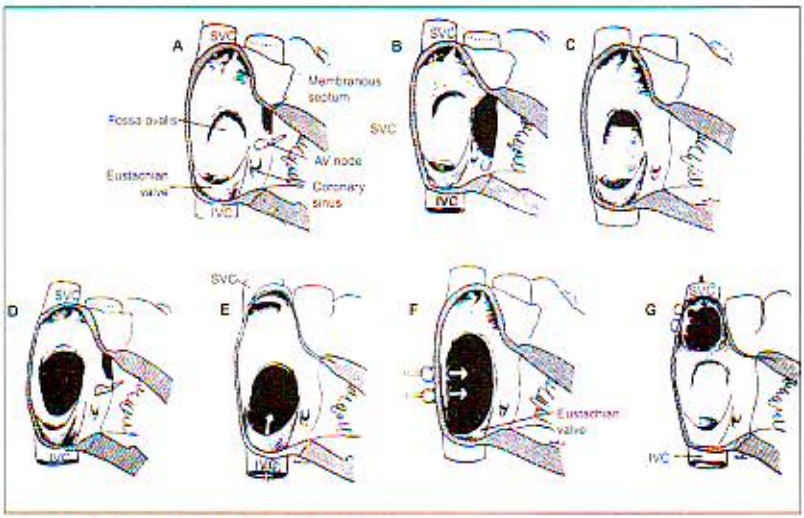
ATRIAL SEPTAL DEFECT : (ASD)

Defination

A hole of variable size in the atrial septum

Morphology

- The atrial septum may have defect at any location
- Most common is fossa ovalis defect (also Secondum Defect)
- The floor of the fossa may be fenestrated
- When all of the fossa ovalis tissue (septum primum remnant) is absent ASD becomes confluent with orific of IVC
- A defect in the posterior inferior part of the septum is posterior defect & right pulmonary vein opens into the defect
- Frequently true anomalous venous connection (APVC) may be present
- When the defect lies beneath the orifice of SVC,usually associated with APVC that terminmates to SVC / at the junction. SVC may override the defct to tha left atrium (SINUS VENOSUS SYNDROME)



- Coronary Sinus Defects are part of unroofed coronary sinus syndrome.When complete unroof, CS is a hole between left & right atrium
- Confluence of the defects(Fossa ovalis & abscence of posterior limbus) make large ASDs
- Ostium primum defects results when AV septum is absent(also called AV Canal defect,AVSD)

Anomaolus Pulmonary Venous connection in ASDs (PAPVC)

1) Sinus Venosus Syndrome – In superior Vena Caval ASD the right upper & middle lobe vein drains to SVC /at junction. May also connect to LA
2) RSPV to Superior Vena Cava
3) RSPV to RA
4) RSPV to IVC

Clinical Features

- Depends on the size of L~R shunt
- When Qp/Qs < 1.5~1.8 there is no symptom
- When Qp/Qs >1.8 symptoms appear
- Features are often atypical
- Symptoms of effort breathlessness to Heart Failure at different age are found

Diagnosis

- Splitting heart sound is unrelated to Qp/Qs
- X-ray chest reflects large Qp/Qs
- P.trunk shadow is enlarged & right ,Left pulmonary arteries are enlarged to the periphery of the field
- Vascular markings are encreased/plethora
- Transverse arch shadow is small

ECG :

• Incomplete RBBB & left axis deviation may be present(AVSD)
Echo: Is diagnostic

Cath & Cine

- When diagnosis of an atypical uncomplecated ASD in children,adolescent & young adult is done, Cath is not necessary
- Cath & cine angiography are necessary in infants(associated lession) & in many adults(Pulmonary hypertension & Mitral morphology) and also in case of suggested PAPVC
- If desturation (<97%) - Cath is indicated
- CAG is also suggested in older than 40s

Inoperability:

- Reliable criteria of inoperability is absolute value of pulmonary vascular resistance (Rp) normalized to body surface area,claculated with maesures O2 uptake than asumed
- Qp/Qs are less discreminating
- Precise criteria yet have not been established like VSD
- Using VSD criteria Rp >8u/m2 is contraindication for complete closure(Isoprotenol,100% O2 administration are indicated to see reversibility)
- When vascular reactivity to O2 & others (Rp falls,SaO2 increases),operation is recomanded (Rp < 7u/m2)
- High Rp will accelerate PVD & less tolerable than ASD
- In isolated PAPVD, Qp/Qs <1.8 is contraindication of operation

Operations

1)Patch closure under CPB

2)Surgery of anomalous connection with or without ASD(RPV to RA,RPV to IVC,LPV to Inniminate Vn,Bilateral PAVCs)

3) In Scimitar Syndrome (PAPVC to IVC with hypoplasia Lung) additional lobectomy/ Pneumonectomy with ligation of arterial supply

ATRIOVENTRICULAR SEPTAL DEFECT (AVSD)

DEFINATION

Characterized by abscence of septal tissue,above & below the normal level of AV valves including the AV septum, in hearts with two ventricles

MORPHOLOGY

- Deficiency/Abscence of AV Septum results Ostium Primum above AV valve
- Deficiency of basal inlet ventricular septum below AV valves-varies in size

INSPITE OF THESE DEFICIENIES ASD/VSD MAY NOT BE PRESENT DEPENDING ON AV ATTACHMENT

MORPHOLOGY

- 5 OR MORE LEAFLETS ARE PRESENT
- VARIABLE COMPLETENESS OF COMISSURES
- LSL,LIL VARIES IN SIZE & DEGREE OF BRIDGING ACROSS IVS
- MAY BE 2 AV ORIFICES

SPECTRUM

- One end- Ostium Primun(Partial AV Canal):Interatrial but no interventricular LSL,LIL
- Extreme End- Large Interatrial,Interventricular defect & common AV valve orifice

Down Syndrome is common in IVS defect

Older terms are useful:

- Partial AV Septal Defect
- Complete AV Septal defect
- Atrial Septal Defeciency & Interatrial communication: Partial AV Septal

Defect

Defeciency of AV septum :Ostium Primum ASD

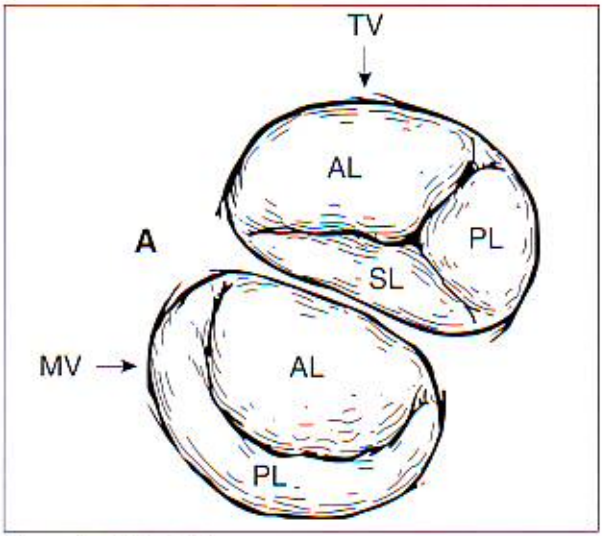
- ❖ Bounded by inferiorly displace AV valves
- ❖ Above by Crescent shaped septum terminating to AV anulus
- ❖ Distance between cresent and AV valves is variable-detemine the size of ASD
- ❖ Fossa Ovalis/Fossa Ovalis ASD may be present
- ❖ Atrial Septal Defeciency & Interatrialcommunication: COMMON ATRIUM
- ◆ Defeciency in anterior limbus (may be with AV septal defect)
- ◆ Entire limb may be absent along with AV septum –COMMON ATRIUM
- ❖ *Atrial Septal Defeciency & Interatrialcommunication:*

Abscence of Interatrial connection

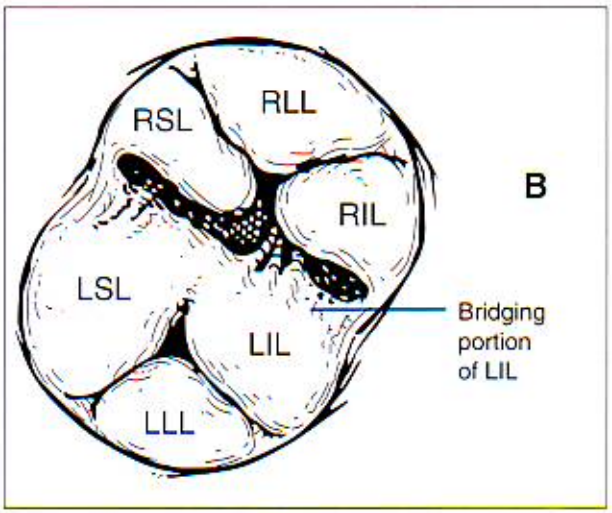
- ◆ Rare variant
- ◆ Associated with basal inlet ventricular defect
- ◆ Rt AV valve with chodal attachment across VSD(Straddling)
- ❖ Ventricular septal deficiency & Interventricular communication
- ◆ Partial AV septal defect: Deficiency inlet septum with no interventricular communication.LSL,LIL are attached to the short septume
- ◆ Complete AV canal defect: Large deficiency creates communication. LSL,LIL are separated
- ◆ Ventricular septal deficiency & Interventricular communication
- ◆ AV Valves:

Displaced towards the apex due to abscence of basal septum.Normal 'wedge' of Aortic valve between AV valves is lost > diagnostic criteria in imaging

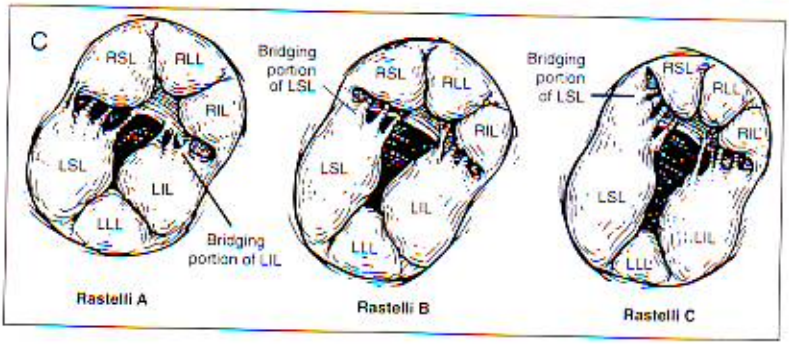
AV Valves : Surgical Orientation



Normal : MV & TV



Partial Defect:LSL LIL LLL Of MV –joined togather at the crest resembles mitral leaflet with a cleft.Some times chordae passes to vent.septum



Complete Defect: Similar but LSL LIL not connected. LIL-bridges little(Gr 1-2 accross),

LSL:

Not bridging or slight (Gr.0-1) >Rastelli type A.

Bridging Moderate (Gr.2-3) >Rastelli type B.

Bridging Marked (Gr 4-5) > Rastelli type C

LV outflow

- Charcteristically narrow in all AVSD
- LVOT obstraction rarely occurs
- Descreate sub-aortic stenosis/ heaped up AV valve develop post operative obstraction(over looked preoperatively)

Associated annomalies:

PDA, TOF, DORV, TGA.

PLSVC.

Unroof CS

PULMONARY VASCULAR DISEASES

- In Partial AVSD,ASDs uncommon
- In Complete AVSD,Large VSDs early & progressive
- More evident when associated with Down Syndrome

Clinical Features

- L-R shunt unless Pul VD/ PS

- In large VSD /Compl AVSD shunt large ,PVD rapid
- Pt with Par AVSD & no/mild AV regurge presents in 1st decade , identical with ASDs
- Pt. with CAVSD present 1st year of life
- In Intermediate gr.presentation depend on size of VSD & A-V regurgitation

Other investigations :

CxR.ECG.Echo

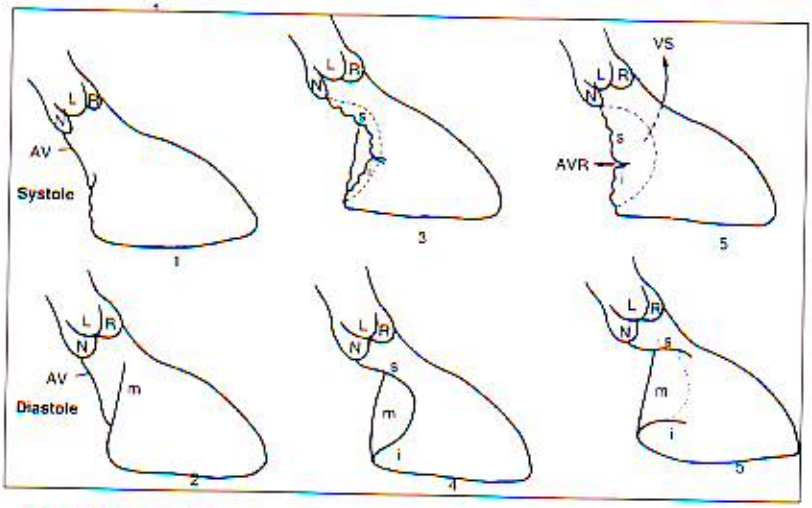
Limitation of echo – to identify double orifice AV orifice , Asses PVR

Car. Cath

- Shunt,PVR,SVR,Flow,RVPr,LVPr to be calculated – when major annomlies co-exxists or operability questioned due to PVR
- Angiocardiography- delenates morphologc features
- Whatever be technique:
- Relative size of two ventricles & AV orifices must be determined
- Severe hypoplasia makes anatomical correction difficult

Special Situation

- LVOT obstraction-particularly in Rastelli A
- More evident postopertively
- Due to VSd patch pullin LT AV apically & anteriorly
- Initial Septal repair must place Lt Av valve in correct position- to prevent AV regurge(Cehalad displacement) & LVOT obst(apical displacement)



Surgical Technique

Priniple:

1. Closing ASD
2. Closing VSD
3. Avoid damage to Coductive tissue
4. Create competent,nonstenotic AV valves

Technique:

❖ 2 patch tech ❖ Single patch tech

Varies but if used properly resluts good

Result

❖ Hospital mortality 3%(UAB) -14%(Paediatric Cardiac Consortium,US)

Mode of death:

Early for Acute HF due to -MS / MVR (Lt AV), Pulmonary dysfunction

Late for subacute HF due to failure of Repaired MV

Natural History

❖ PAVSD ,Mild MVR,No major anomally >similar to ASDs .Develop PVR in small gr.

❖ PAVSD with moderate to severe MVR symptomatic in infancy & die in 1st decade

❖ CAVSD unfavourable ,without operation die by 2 years

AWAKE CORONARY ARTERY BYPASS (ACAB)

- ARE YOU NUTS?
- THIS PHYSICIAN PROBABLY GONE CRAZY!
- This was the initial reaction of Physician community 1998 when ACAB first introduced by Karagoz
- Now this has emerged as important tool in various cardiac centres

Rationale

- Performing OPCAB (Bypass) under epidural anaesthesia obviously decreasing invasiveness
- High epidural anaesthesia at T3-4 achieves somatosensory and motor block in the chest
- Need of perioperative monitoring a direct cosequence of GA rather underlying disease

Elemination of GA in CABG enables early recovery & mobilization with out imposing health risk

- Some Pt. of ACAB could be dischaged from hospital on the afternoon of operation & this MAY ULTIMATELY LEAD THE WAY TOWARDS 'Ambulatory CABG' –a dream of todays surgeons

Benefits: Intra /Post operative

- Intraoperative benefit largely –Sympatholysis (neuroaxial blockade)
- Post op- Profound analgesia
- Several reports-reduced stress response / sympathoamines
- 4 decades research failed to find anaesthetic technic to attenuates stress & resultant surgical sequelae
- Epidural anaesthesia prevents surges of stress hormones
- Sympathetic fibres T1-T5 innervates myocardium& coronary-have role in flow
- Decreased prevalence arrythmias / HR observed in TEA during manipulation

- ❖ Cannulating T2-T3 continous infusion of ropivocaine (0.5%) + sufentanil (1.66_g/ml)
- ❖ Ensures sensory block from neck to abdomen with arms
- ❖ Excellent postop recovery
- ❖ TEA reduces hemodynamic compromise as a result of narcotic medication before intubation is some patients

Technics: Developed

- ❖ To avoid extracorporeal blood contact
- ❖ To avoid mechanical ventilation / paralysing agents
- ❖ To reduces intraoperative stress and postoperative pain (TEA).

- ◆ This allows Awake coronary artery bypass graft surgery (ACAB) avoiding the drawbacks of mechanical ventilation and general anesthesia particularly in high-risk patients
- ◆ As an addition to MIDCAB / OPCAB
- ◆ Patients with certain risk profiles, including chronic obstructive pulmonary disease, coagulation disorders, and aberrant neurological conditions, get benefit from operations without cardiopulmonary bypass.

ACAB was introduced in January 2006 at Natinal Institute of Cardiovascular Diseases.Dhaka (NICVD) & 37 cases were recorded from the registry of up to Dec 2006.

Patients include were:

- *Significant (>70%) lesion of LAD, diagonal,Cx branches,or the right coronary artery (RCA) with good Pts. compliance & good target vessels*
- *Absence of recent antithrombotic / fibronolytic therapy*
- *Presence of comorbidity did not affect patient selection*

DEMOGRAPHIC DATA

• *No.of Patients*	*37*
• *Age*	*55.3±26*
• *Gender ratio(M:F)*	*12:1*
• *Body surface area*	*1.5±0.3*
• *LV EF*	*40±26.6*
• *Previous MI*	*09 (24%)*
• *COPD*	*00*
• *Renal Disease*	*06 (16.2%)*
• *Dialysis*	*00*
• *Peripheral Vasular Disease*	*08 (21.6%)*
• *Diabetes mellitus*	*10 (27.2%)*
• *High Risk Pts.*	*13 (35%)*

All data were reviewed prospectively.

Operative techniques for ACAB

◆ *Complete median sternotomy :*

The chest was opened with an standard pneumatic saw and particularly careful LIMA dissection was necessary to avoid pneumothorax in the spontaneously breathing patient.

After dissection of the LIMA in conventional technique without opening the pleural cavity, the pericardium was opened.

With the aid of mechanical stabilization anastomoses were performed by standard beating-heart bypass technique(OPCAB).

A wide,pledget-armed,U-shaped suture, which was placed at the acute margin of the heart and pulled toward the patient's left shoulder to expose the inferior surface of the heart .

◆ *Left anterior mini-thoracotomy / antro-lateral thoracotomy:*

Left thoracotomy was made through a incision in left 4th intercostal space.LIMA was harvested. Pericardiotomy was done vertically and parallel to the phrenic nerve.This procedure was used when only the left sided grafts were implanted

Anesthesia for ACAB:

❖ High TEA was used. The maximum permissible block level was C6, which was monitored by a possible development of Horner syndrome.

- Antiplatelet therapy was stopped 5 days before surgery in all cases.
- In the operating room an infusion of 0.5% bupivacaine, with 2%lignocaine and fentanyl into the epidural space was started. Thus sensory block was achieved between the neck and the abdomen,including both arms.
- Thoracic epidural catheter was used for not only intraoperative but also postoperative pain management for 3 days.
- Depending on pain perception additional analgesics were used.

OPERATIVE DATA

• *Total Cases (n=) :*	*37*
• *MIDCAB*	*12*
• *OPCAB*	*25*
• *Procedure time (hr):*	*04.46 ± 0.06*
• *Anastomoses:*	
Single Vessel	*05 (13.6%)*
2 vessels	*13 (35.1%)*
3 vessels	*17 (45.9%)*
4 vessels	*02 (5.4%)*
• *Conversion*	*02 (5%)*
• *Extubation in OT*	*01 (2.7%)*
• *Chest tube drainage*	*200 + 70*
• *ICU stay (d)*	*1.5 ± 0.5*
• *Hospital stay (d)*	*6.5 ± 02*
• *Need for analgesia in 3d*	*02 (5%)*
• *Post-Op Compl. (Soft tissue infec)*	*03 (8.1%)*
• *Anastomosis (No.)*	*2. 43 per pt.*
• *IMA*	*37(100%)*
• *RSVG*	*53(58%)*
• *Total Graft*	*90*

RESULTS

- 5 patients underwent single-vessel CABG, 13 patients underwent double-vessel cabg,and 17 triple & 2 pts underwent 4 vessel CABG.
- 2 patients in this series required secondary intubation one after completion of internal thoracic artery harvesting because of arrythmia (n=1) and another for uncontrolled pneumothorax (n=1), which was extubated in OT room after operation.
- Mortality of this early report is nil
- No important post operative complications except soft tissue wound infection(3) were noted
- Horner syndrome was observed in no patients.

Discussion

- TEA provides excellent conditions for off-pump / MIDCAB coronary artery bypass surgery by dilating the coronary arteries and the internal thoracic artery and by reducing heart rate and arrhythmias during manipulation of the heart.
- The threshold level of blood pressure remains unknown (usually a BP>70 mmHg we considered mandatory).
- The operative time also seems to be shorter, and this is an important factor for the awake technique.
- Most relevant possible disadvantage for harvesting a second conduit & a second anastomosis for the attachment of free graft to LIMA is the potential for diversion of significant LIMA flow to noncoronary/coronary vascular beds, representing some variant of a steal syndrome.
- We used this technique in 6 cases.
- In others (6) we used descending aorta for proximal anastomoses in MIDCAB
- Revascularization of the circumflex territory in Mid Sternotomy (OPCAB) is in most cases difficult because of hemodynamic impairment associated with exposing the vessel.
- We found use of Starfish/Urchin stabilization along with verticalization of the apex provides an excellent haemodynamic toleranance

- We recommend in single vessel bad proximai disease the H-graft technique for the elderly and high risk patients because it is a fast procedure that avoids intercostal retraction.
- In younger patients for single graft partial lower sternotomy or the rib café-lifting technique should be used to provide patients the benefits of the internal thoracic artery graft without an ugly scar
- ACAB although initially used in highly selected,compliant & mentally stable pts, it can also safely be used in all csaes who are compliant to the procedure.
- This first report of our study demonstrate the feasibility & safety of ACAB as other workers reported.
- In our patients ACAB achieved excellent acceptance, even some pts persued our team for the procedure to avoid general anaesthesia and early post operative recovery.
- Combination of benefits of OPCAB, a small incission, avoidance of GA, Positive pressure ventilation & effective pain management may allow ACAB to compete PCI techniques.
- With futher work & refinement of the procedure, out-patient CABG may become feasible

INITIAL IMPRESSION

- ACAB a promising adjunct to minimally invasive CABG
- May be potential use in hybrid setting
- With times AMBULATORY BYPASS not imposible at all.

C P B - DAMAGES & COMPLICATIONS

INTRODUCTION

- Undesireable response of CPB were evident from the early deays
- Overlooked for new tech excitement.
- Observed : Diffused bleeding / Puffy Operated Pts. / Hyperthermia / Pulmonary dysfunction / Bad cardiac performance
- Still there is paradox

RESPONSEs

- 2 response system activated with foreign surface contact with diversion of blood in CPB
- Specific (Immune)-Slow to develop for few days
- Nonspecific (Inflammatory) - Starts early minutes,also called Systemic Inflammatory Response Syndrome (SIRS), Can be stimulated by other agents
  - Humoral
  - Cellular
  - Metabolic

SIRS

Mediated by -

- Neutrophil activation
- Platelate activation
- Complement
- Kallikrein
- Coagulation ,fibrinolytic & other cascades
- Immune Cytokines(T cell)- depend on duration CPB

Damages

Myocardial:

STUNNING

Systolic & Diastolic dysfunction-Variable /Prolonged period without out necrosis (Myocardial cell survives 20 mins normothermic Ischemia)

CAUSES

Free radicle injury

Ca+ influx

- STUNNED MYOCARDIUM RESPONSE TO INOTROPS
- RESPONSIBLE FOR POST OF LOS
- After short ischemia when normal flow restored contractility still diminished for certain time-Known as 'PERFUSION-CONTRACTILITY MISMATCH'

Hibernation

- Perfusion –contraction match'- Both are low
- A chronic potentially reversible segmental (less often Global) cotractile dysfuction

LV Dysfunction

Systolic Dysfunction:

- Localized / Global Regional motion abnormality in stress
- Results of transient ischemia
- Related to global stunning is increased LVEDV

Diastolic Dysfunction:

- Reduced LV filling rate(peak)
- Increase filling time
- Due to impaired diastolic relaxation of papillary muscle- An active energy dependent process

PREVENTION OF
UNDUE CPB-RESPONSES

- TRUE PREVENTION YET NOT KNOWN
- MOLECULAR BIOLOGY IS HOPEFULL
- PALLIATIVE MEASURES (used as addetives) ONLY AVAILABLE

MANAGEMENT
DURING ISCHEMIA

Circumstance that decreases utilization of O2 consumption lengthens safe ischemic time,includes:

- Immidiate cessation of electro mechanical activity
- Hypothermia

Management

During Reperfusion

- Maintain electro mechanical quiescence during first 3-5 min of reperfusion
- Control PH of reperfusate to prevent myocardial acidosis
- Minimize free radical damage
- Low ionized calcium in reperfusate
- Maintain low perfusion pr. (30mmHg) in 1st 1-2 min of reperfusion
- Maintain adequet reperfusion flow to even distribution
- Control reperfusion pr. until recovery is complete (at 30-75mmHg for 1st 01-02 mins or at pre-op diastolic pr of Pt.,which ever is lower).

CABG : RENAL DYSFUNCTION & DIALYSIS DEPENDENT RENAL FAILURE

Introduction

- Renal dysfunction yet to be a risk factor for death after CABG
- Preoperative elevation of creatinine &BUN are risk factor for death similar to patient who develop renal failure after surgery

Adverse effects of CPB

- Complement activation
- Platelate aggregation & degranulation
- Neutrophil aggregation
- Intravascular haemolysis
- Ischmia secondary to circulatory arrest/low flow perfusion
- Microembolisation
- Abnormal distribution of flow

Evaluation

- A serum creatinine 1.5-2 mg/dl considered as lower limit if insufficiency
- Older pts(>65y) with reduced muscle mass & lower value may also indicative of insufficiency
- When moderate insufficiency is noted priror to CABG through urologic evaluation is considered (If cretinine >2.0mg/dl)
- Mild to Moderate dysfunction – evalution aimed to prevent worsening after surgery.Preoperative hydration is important
- If preexsisting dysfunction deteriorates after exposure to dye or low output or other reasons CABG should be defered until baseline values are returned
- Non oliguric chronic dysfunction if associated with odema & electrolyte imbalance, to be corrected before if time allows

Management : Renal Dysfunction

(Creatinin>1.5 mg/dl)

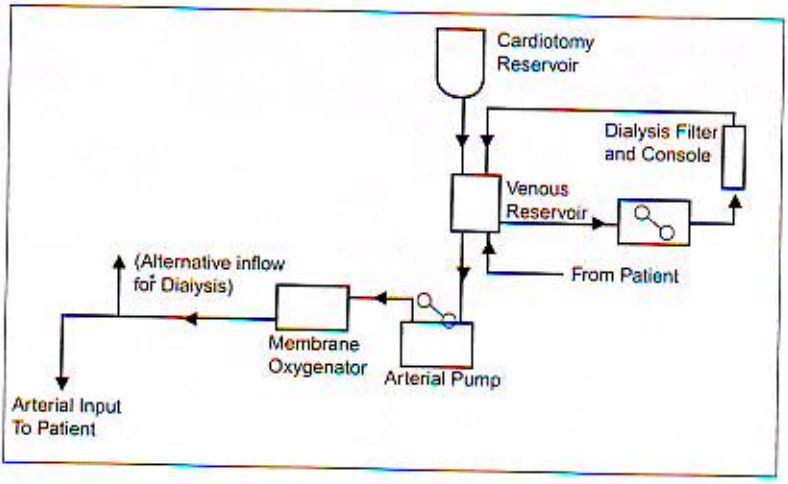
Non oliguric

- Pre op Hydration
- Avoid- ACE inhibitors,K+ sparing diuretics,Keep all medication in renal failure dose
- Dopamin (2.5µg / kg / min shortly after anaesthesia
- After CPB – Mannitol 0.5gm/Kg (0r /and Frusemide,20mg/m2 with incremental dose, if urinary out put falls below accepted level
- Inspite these measures if Output <20ml/min/m2 with good Cardiac output than
- "Renal Cockail" (400 mg Frusemide +100 ml 20% Mannitol at 1mg/kg/hr of Frusemide)
- With these measures even an oliguric can be converted to non-oliguric state
- Management of acute hyperkalemia should be accordingly

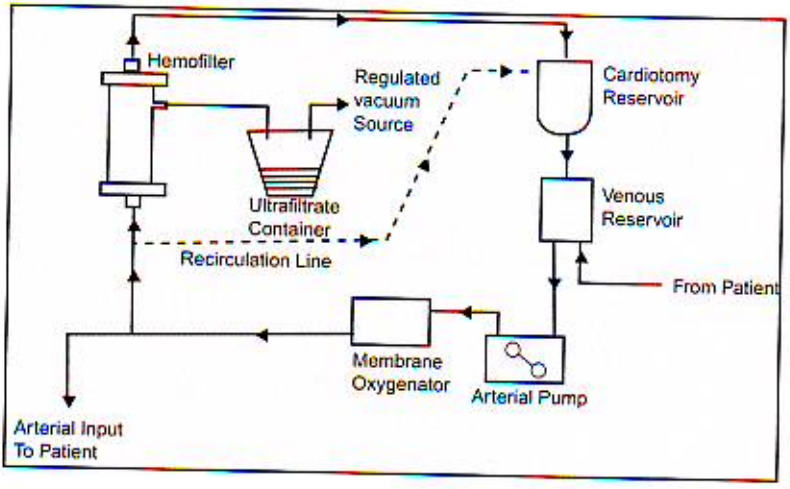
Dialysis Dependent Failure

- Asses method of dialysis
- Prior to surgery & as necessary post operatively
- Final dialysis to be done on the day before surgery
- Goal is to bring electrolytes to normal range & weight
- Site for post op dialysis may be selected before, subclavian (transvenous Catheter)
- All medication /Anaesthetics to be adjusted to dose of renal failure
- Haemofiltration / Intraoprative dialysis is indiated if fluid overload, hyperkalaemia or haemodilution during CPB is observed

Addition of Dialysis to Pump Circuit



Hemofilter to Pump Circuit



Conclusion

- Renal dysfunction is a serious threat to successful CABG despite advbances in this field
- Intraoperative& Post operative renal hypoperfusion is the Key etiology
- Aggressive use of Circulatory assists devices in future are pomising

CEREBRAL & SPINAL PROTECTION IN AORTIC SURGERY

Cerebral Damage : Risk Factors

- Duration of circulatory arrest is a clear detrminants of brain damage
- Cerebral oxygen consumotion in hypothermic CPB at low flow reduced below normal-due to decreased density
- At 37°C it is same as before CPB
- Safe duration is affected by many known& unknown factors
- Bamage is rarelydefuse i nadult & manifested by intellectual/motor deficits
- In neonats &children by seizuress,choreoathetoid movements

Risk factors

- Duration
- Temperature
- Rate of cooling&rewarming
- Flow & distribution
- BP
- Electrical activities
- Management of reperfusion

Evident that circulatory arrest of 60 minor more at 15°–18°C associated with irreversible damage although tolerated by some

Brain Injury: Aortic Surgery

- Temporary dysfunction-Agitation, Disorientation, Psycosis, Chorea, seizure
- Stroke

Cerebral Protection

Hypothermic Circulatory arrest

- Simple & widely used
- Provides bloodless field
- Scrupulous apllication of technique is important to minimize complications(20%)

- For frequent cognitive dysfunction safe duration is closure to 30min
- Adjunct (Retrograde cerebral perfusion) may increse safe duration.

Retograde Brain Perfusion

- Meaningful metabolic benifit is debated.
- Primary role as adjunct
- Bicaval cannulation
- Cooling at 18°C
- Needs 3rd pump
- All retrograde cardioplagia & no clamping Aorta
- Bypass line (3/8) from arterial to SVC
- Venous line to pump is clampedArterial line to Femoral Artery is clamped
- CVP not >30

Antegrade perfusion :

- Low flow hypothermic perfusion reserves PH & energy
- Direct cannulation of brachiocephalic arteries
- Used also retrogrde after arch replacement through a separate 10mm tube
- Flow 800~1200 ml/min at 20°C
- May not be safe more>80 min

Spinal cord Protection

Paraplegis/Paraparesis with/without bladder is immidiate or late result of unprotected cord during surgery for–

Distal arch

Descending aorta

Thoracoabdominal aorta

Risk Factrors

- *Duration of clamping:*
  - No Probability when <15 min
- *Temperature:*
  - Moderate to profound have lower incidence of injury than normothermia

- 20°C allows clamping >100 min
- Irrigation of cold (4°C)saline in epidural space for 24~28 °C CSF is adjunctive

◆ *Level of clamp:*

- Beyond renal arteries (L2) for 60 min incidence is less (0.1%)
- At diaphragm for 60 min (10%)
- Distal to subclavia for 60min incidence 80%
- With increase in distance of 2 clamps number of intercostals exclude from spinal collaterals

Spinal Artery anatomy

◆ Variability of origin of Arteria Rediculari s magna - T9~T12 (75%)

◆ Clamping distal is more safe

Collateral

◆ Disease where good collateral developed incidence is zero (Coarctation) compare to degenerative diseases

◆ Incresed Intraspinal & Decreased aortic presure with clamp reduces colla teral flow

Protection of Cord

◆ Hypothermia & Low room temperature

- Cooling/Heating blanket(30~32°C) with additional cooling of pleural space for 15 min
- It is inadviseable to operate on descending & thoracoabdominal aorta with surface cooling

◆ Perfusion of Distal Aorta

- To maintain 60~70mmHg for spinal flow maintanance

(LA-FA bypass,Aorto-aortic shunt,PartialCPB)

◆ Grafting of Intercostal& Lumber arteries

- Most of the posterior wall to graft to the tube
- Favourably arteries below T6 / T7

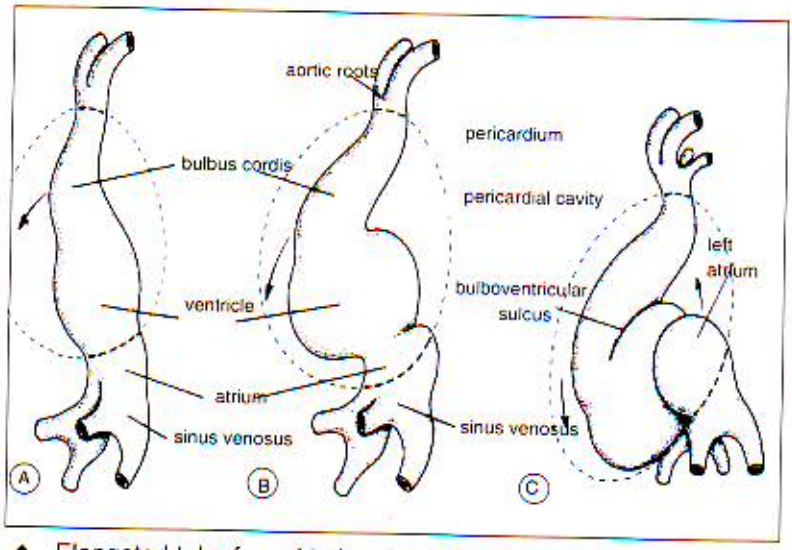
THORACO ABDOMINAL REPLACEMENT

- Combination Femoro-Femoral CPB, hypothermic arrest, grafting of intercostals, additional arterial cannula graft- widely accepted

CONGENITAL HEART DISEASES

DEVELOPMENT

- Developing heart tube in splanchnic mesodermal pericardial cavity attac- hed by dorsal mesocardium
- Forms the bulboventricular portion
- Atria,Sinus venosus remains outside in septum transversum
- Next events – Rapid growth of bulboventricular portion than cavity



- Elongated tube forced to bend in cavity
- Cephalic part bennds ventrally, caudally& to right
- A-V junction comes to left & dorsally
- Atrial still paired & connected to ventricle – A-V Canal
- THE LOOP FORMS-Rt.ventrl,Out flow tract(conus cordis,trancus)& lt. vent
- Traco-conus shifts medially & atrium buldges both sides to Rt. & Lt atria (with oblique conus)

Sinus venosus

- PAIRED HORNS_Lt disapeares to coronary sinus

- Rt horn incorporated to atrium through rt venous valve that gradually incorporated & forms vaves of Infer.vena cava,CS

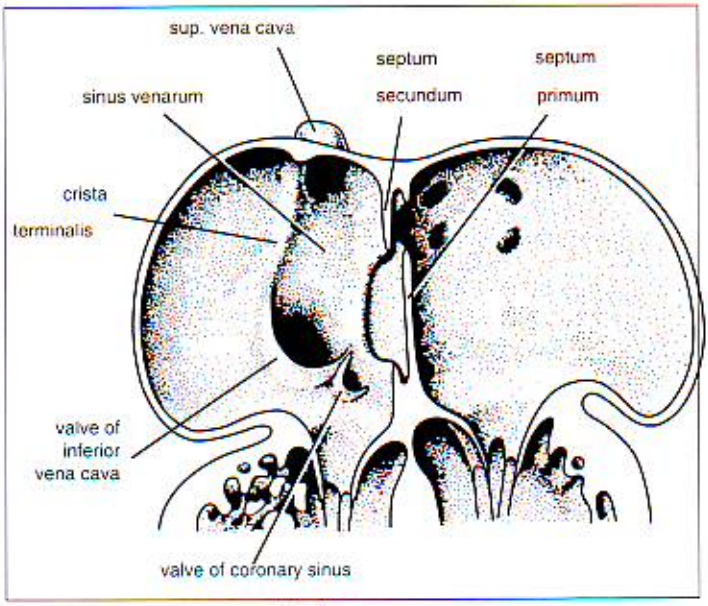


Fig-2

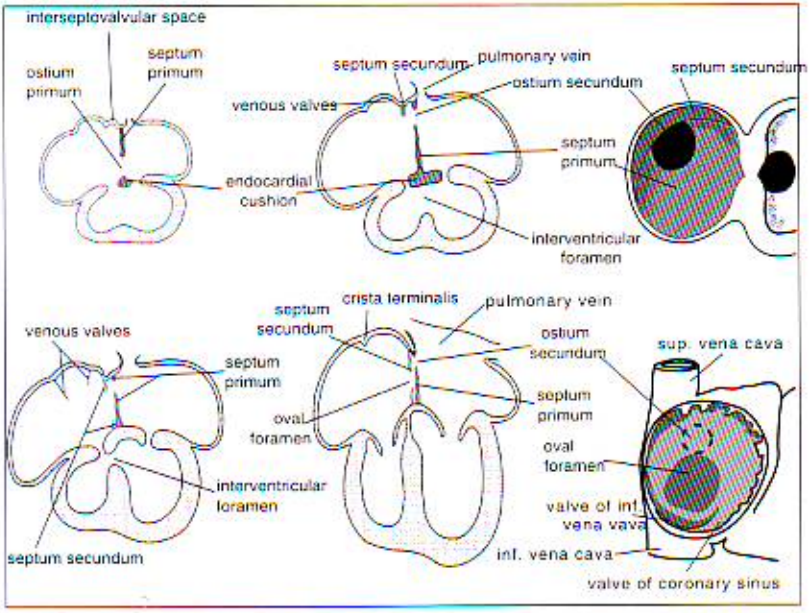


Fig-3

SEPTATION

- Trancus depresses the roof of atrium-forms a crest that forms the septum primum grows to cushions>forms ostium primum
- Primary septum unites with cushion to form septum primum, meanwhile perforation appears > Ostium secundum
- Interseptovalvar space develops septum secundum > grows & overlaps osteum secundum

ENDOCARDIAL CUSHION

- Appears supr,inf boarder of AV canal
- At this stage conus tube separated from ventricle by a flange - bulbov-entricular flange
- At times this comes to mid point of supr. cushion
- With further growth the flange devides canal into rt & lt AV orifices

ABNORMAL AV CANAL

- PERSISTANT AV CANAL
- TRICUSPID ATRESIA
- EBSTEIN'S ANOMALLY

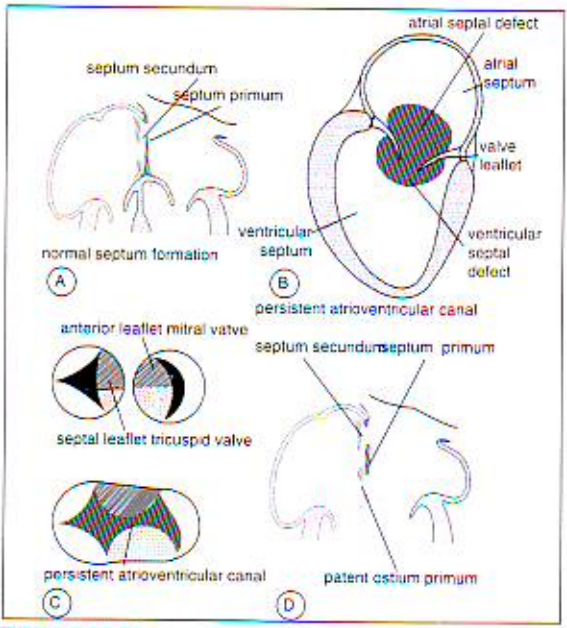


Fig-4

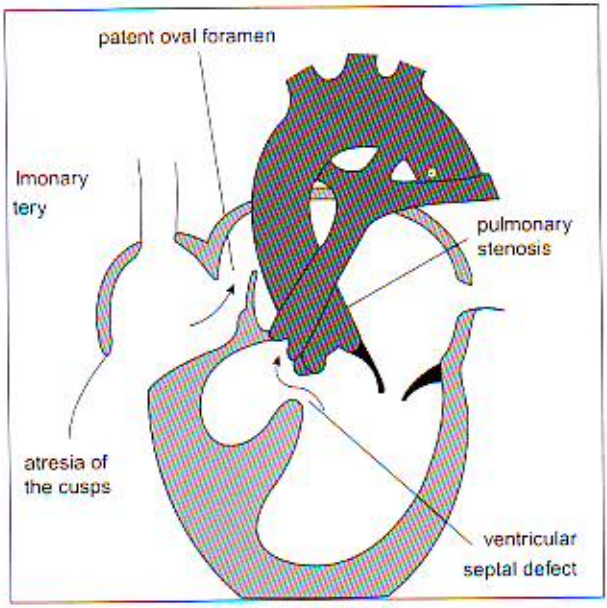


Fig-5 Tricuspid Atresia

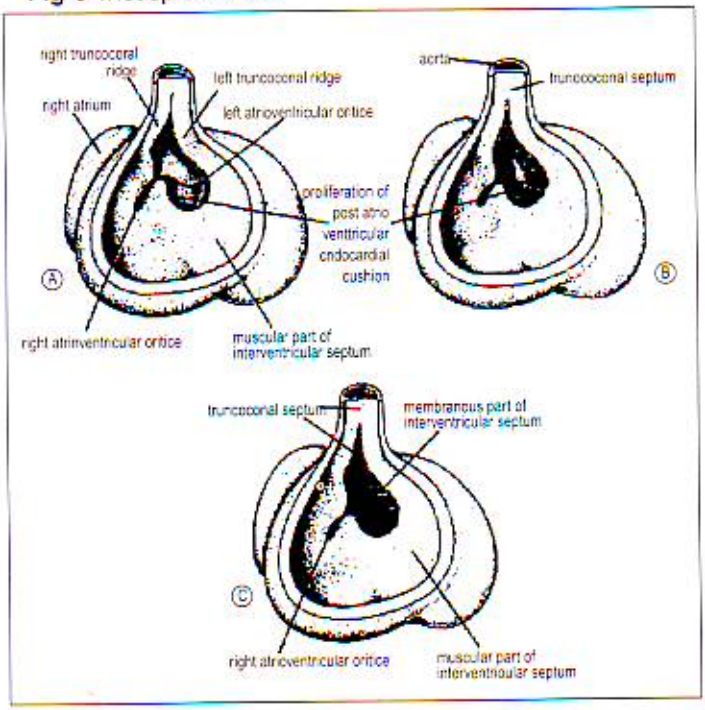


Fig-6

Septation Ventricle

- Dilatation of primary ventricle,diverticulation with trbeculation forms mus -cular septum inferiorly
- Superiorly & posteriorly by apposition of flange

SEPTATION CONUS

- Ridges appear inside truncus >Rt.sup & Lt inferior >devides in two chanal with formation of aorticopulmonary septum.It twists while growind distally.
- Similarly two conus ridges devides the conus >Proximal end of Rt.meets superior boarder of rt.Av orifice > completes Rt.ventricle
- Lt conus swellins extend along rt side if muscular septum>conus septu thus forms devidesn conus antero-lateral & post lat. part
- Post-lat part continuous with Lt.definite ventricle
- This reduces size of inter ventricular foramen
- Further closure done by growth of membrane from cushion to meet abuttind edges of conus septum

Abnormal IVS :

- SUPRA CRYSTAL
- INFRA CRYSTAL
- MASCULAR
- SINGLE VENTRICLE

ABNORMAL
TRANCO-CONUS
TOF/DORV

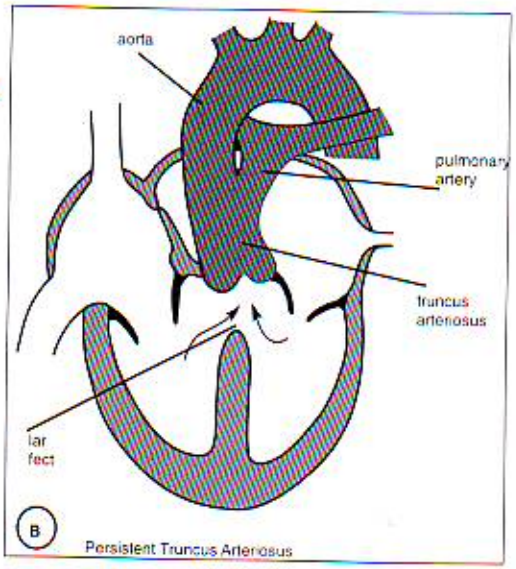


Fig-7

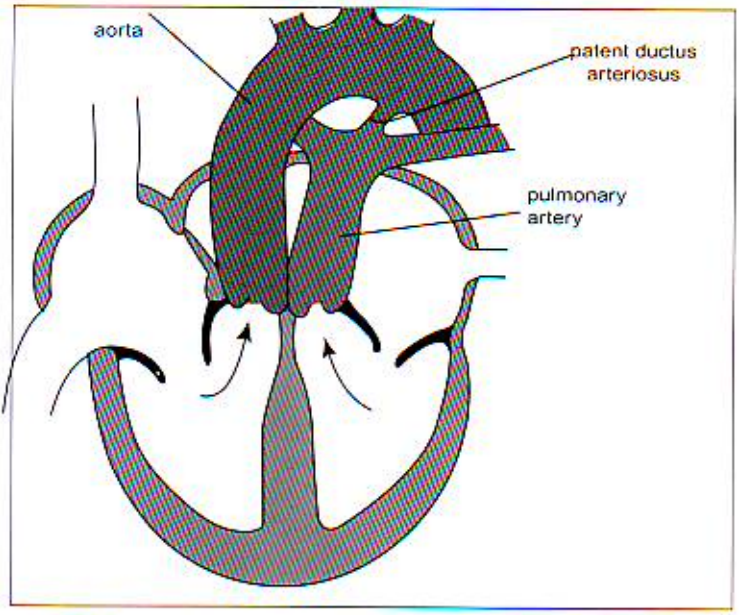


Fig 7 . Transposition of Great Vessels

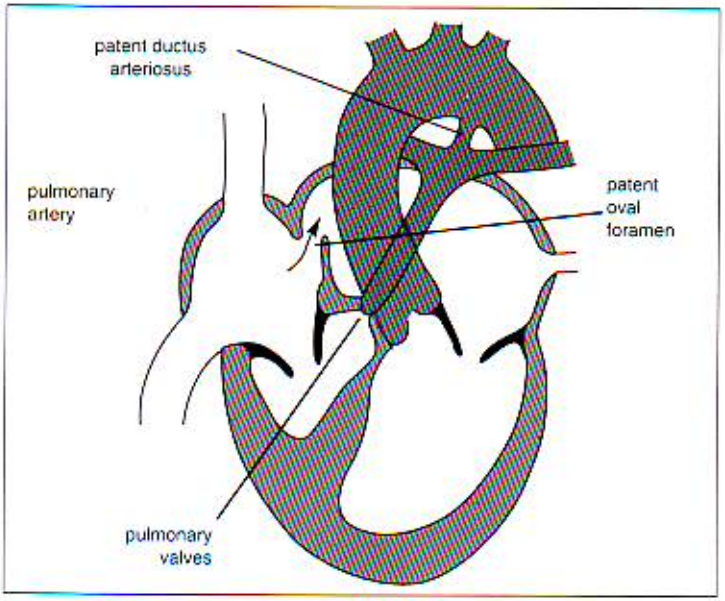


Fig 8 . Persistant Truncus Arteriousus

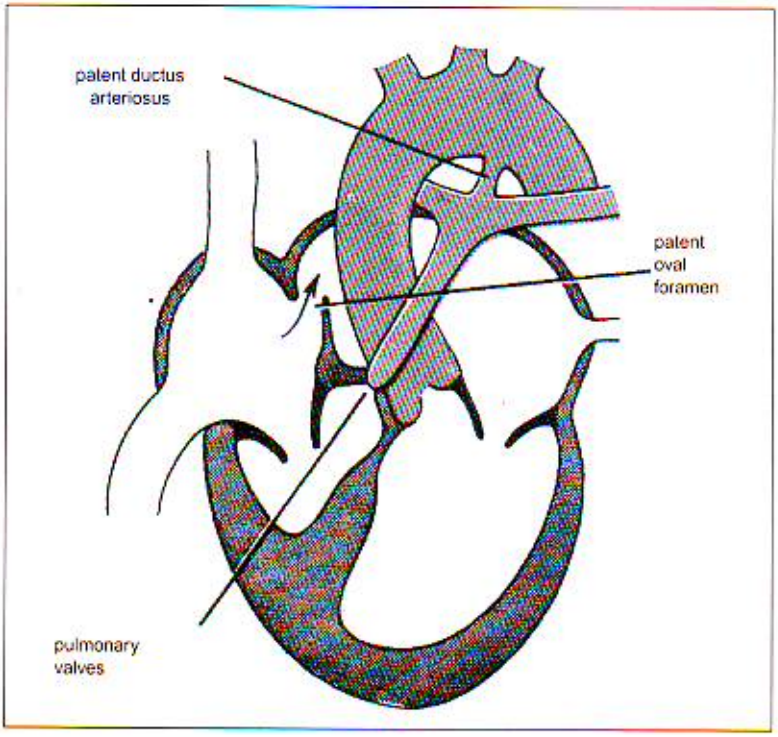
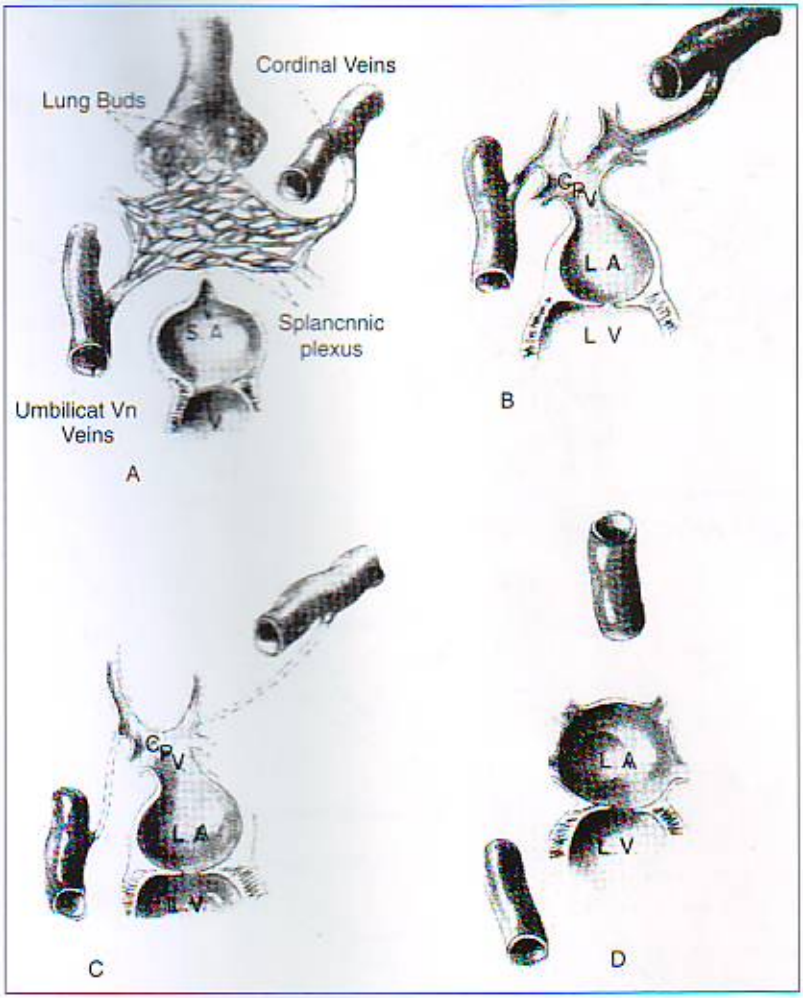


Fig.12 Pulmonary Atresia(Valvular)

PULMONARY VEINS

- Splancnic plexus drains lung bud – shares connection with crdinal, umbilical vn
- Common pulmonary Vn invaginated from LA>joins the spl.plexus
- Pul Vn drains to LA > primitive connections disappear
- with differential growth pul.Vn are incorporated in LA & CPV disappear



◆ ABNORMAL DEVELOPMENTS OF PULMONARY VEIN

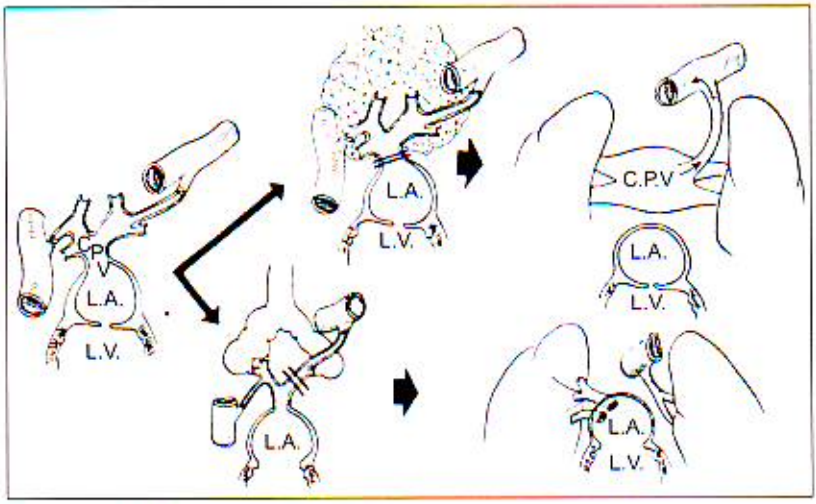


Fig.13 *APVC (upper) I PAPVC (lower)*

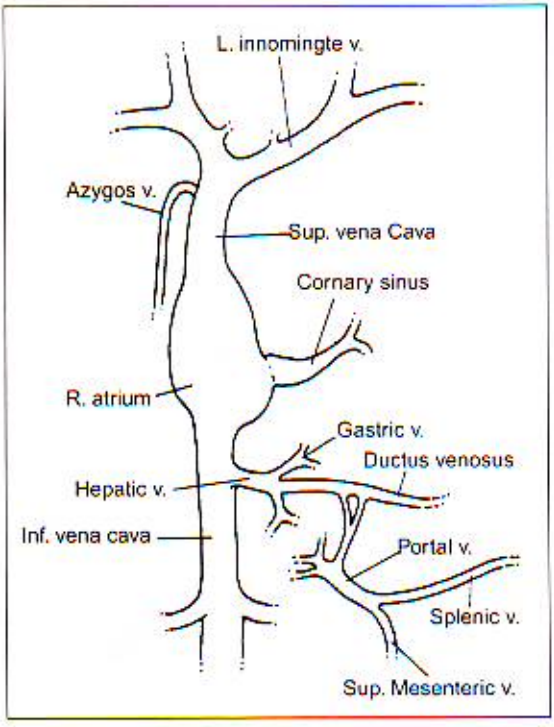


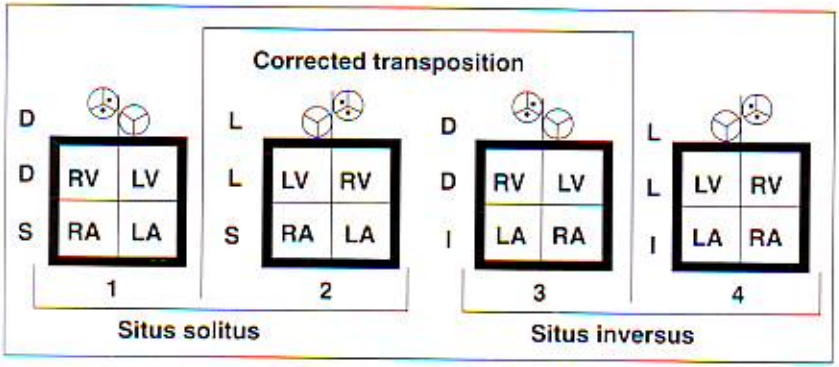
Fig.14. *Routes ogf Drainage of TAPVC*

CONGENITALLY CORRECTED TGA (CCTGA)

Defination

Anomaly with VA (Ventriculoarterial) discordant (TAG) & AV (Atrioventricular) discordant connection. Circulatory Pathway 'in series'

Transposition of the great arteries



MORPHOLOGY

- Usually Atria, Situs Solitus
- Ventricle L-loop (left handedness i.e., when left hand palm placed over RV IVS, index indicates -inlet RV, Fingres-outlet, wrist-apex) : LV to the right & RV to the left. L-malposition of aorta.
- LV slightly posterior & inferior to RV
- When Situs Inversus – Mirror image

Ventricle

- Usually fibrous continuity between MV with PV, Developed Septum seperating TV(left side) & aortic valve
- LVOT below PV lies between > septal leaflet of mitral valve(Rt) & muscular septum (Lt)
- Aorta & Pulmonary trunk are parallel
- Apex formed by RV
- Rotation may be bizarre

Pulmonary Outflow Tract

- Wedged between MV & TV from LV(Rt), more marked than TGA
- PV at right & post to the AV
- Pulmonary outflow tract is potentially restrictive when LVH present (organic 50%)
- PV thickened,abnormal cusps
- PS similar to TOF /TOF withPA

Mitral Valve

- Rotated & in fibrous continuity of PV
- Papillary musle from LV walls
- MV anomaly is 55%

OTHER VALVES

- AORTIC VALVE
- Usually normal
- Over RV infundibulum
- L-Position / D-Position
- Subaortic stenosis is rare

Tricuspid Valve

- Left sided
- Coarse trabeculation
- Structure anomaly always present
- Mostly Ebstein /Dysplasia
- Anterior leaflet not sail like as Ebstein & not dilated

A-V node

- Normal position but not giving 'HIS'
- Distribution over the septum is similar but anterior to VSD

SEPTUM

- Aneurysm of the membranous septum may be present(without VSD)
- May be obstractive

VSD

- Present 80%
- Large,subpulmonary (conoventricular/ membranous)
- PV over rides VSD to arise from RV(lt)
- In 10% patients VSD juxtra arterial (Conal)
- Inlet VSD uncommon as multiple

CORONARY ARTERY

- Rt sided (LCA) supplies Rt sided LV
- RCA supplies RV (lt)
- NCC is anterior,RCA gives LCA
- Single CA from RCC may be present

Other anomalies

- TS
- Ebstein
- Coarctation
- PDA
- Overriding AV

Clinical Criteria

- Some restriction of Pulmonary flow although juxta arterial VSD (in contrast to normal VSD)
- PS is Uncommon that needs shunt in earlylife (30% patients are cyanotic)
- Growth failure,exercise intolerance are in childhood / 2nd decade
- If important PS then mild / moderate cyanosis is present
- TV incompetance complecates features
- CHB (soon after birth) in 10-30% & may be temporary during sternotomy, Cath
- Reversal precordial Q wave pattern in right precordial leads (Q in V2, AVR and QS in V3, avL)

Natural History

- Ventricular function
- Not normal But sufficiently good to maintain normal life

- Survival at 7th, 8th, 9th decades exists though LV starts detrioration in 2nd decade
- CCTGA with other anomalies CI is maintained in exercise
- LV dysfunction is poorly understood
- Pregnancy is also well tolerated

With other aomalies

- 25% at 4th decade develops failure
- Survival rate, unavailable
- If PS with VSD & cyanosis present in early life and similar to TOF. But abscence of muscular componant (Infundibulum) give better performance than TOF
- PS with VSD more likely in situs inversus
- Unless PV is stenotic /movement does not obstruct subvalvular area. Little can be done to narrowing(Only conduit is preferable)
- Even PV gr.50mmHg does not require any thing

INDICATIONS

- CCTGA per se not definite indication of surgery
- Double Switch is left open for Morphologic RV with systemic circulation
- VSD indicates' surgery like other VSDs
- VSD+PS requires conduit:indication & staging like TOF with PA
- TR, MR indication similar Replacement / Repair like other MV, TV Diseases
- Late survival influences indication of all various procedures(20-45%)
- What ever the procedure the patient develop situation only indicates TRANSPLANTATION effective

OPERATIONS

- Closure of VSD
- VSD+PS
- PA- tomy & excission with dialator measurement
- Aneurysmal membrane repaired like VSD with all muscle in tract are cut (not septal)
- Valvotomy may be insufficient for bicuspid valve,supravalvular or sub valvular narrowing
- If Z > -1 than pulmonary trunk may be possible
- No conduit if PLV/ RV = <0.8 after repair

- LV:RV measured next morning,if <0.7 no return to OT for conduit

Use of Valve conduit(Extracardiac)

- Severe PS,Cyanotic or repair may be unsatisfactory
- Transanular patch is option (by some)

Correction of TVR(It)

- Feasible when important incompetance is present

DOUBLE SWITCH PROCEDURE

- For long term fate of morphologic RV & TV in systemic circulation
- Intracrdiac buffle to connect LV to Aorta & a conduit fron RV to PA with mustard /Senning
- Technically,substantially more complex

DOUBLE SWITCH +BDG

(Only the IVC returned is diverted to TV)

RESULT

RV supporting systemic circulation

Early:

- When CCTGA with VSD mortality 5%
- When VSD with PS 10-20%
- When with TR 15-25%
- Long term survival,recent years (10-15%)- 90%
- NO PROSPECTIVE STUDY AVAILABLE FOR RESULT OF LV SUPPORTING SYSTEMIC CIRCULATION (Double Switch)

COR-TRIATRIATUM

EMBRYOGENESIS

- Stenosis of Common Pulmonary Vein (CPV) that fails to be incorporated in LA results the anomally

MORPHOLOGY

- Classically an acessory chamber receives all pulmonary veins & drain to Left Atrium (LA)

Types

Diagnosis

- Majority present sign of pulmonary obstraction in early life
- X-ray shows pulmonary venous engorgement
- Echo: In most cases it is difficult to identify the accessory chamber to CS or PLSVC to CS

Cath:

- Pressure gradient between pulmonary (wedge) & LA is the hallmark
- L-R shunt & Pulmonary hypertension are rule
- Slelective Pulmonry arteriography in venous phase (prolonged transit time)opacifies the chamber &LA

Surgery

- Successful therapy for the anomally
- Open correction under CPB & resection.
- Post operative is good

CORONARY CIRCULATION : MYOCARDIAL PROTECTION

DEVELOPMENT

- Heart developed in splancnic mesoderm
- Primitive heart tube seperated from myoepicardial mentle by cardiac jelly
- Coronary arteries developed from epicardial mentle

FUNCTION OF THE HEART

- Transformation of metabolic energy (O_2 substrate) to mechanical energy (Cir. pressure, Flow)
- Chemo-Mechanical energy transducers > Sarcomares (Functional units)

FLOW

- NORMAL CORONARY FLOW:0.7-0.9 ml/g/min
- O2 extraction 75% at rest,100% at stress
- With adquet perfusion pr. flow is auto- regulated by arteriolar resistance (influenced by metabolic demand)

FOCUS OF ACTION

- MYOCIN CROSS-BRIDGE
- Ca+>Myocin>Hydrolyse>ATP>ADP +Phosphate→Splitting ATP release CHEMICAL ENERGY brings >conformational changes in MYOCIN BRIDGE> Contraction Myocin→Mechanical Changes(Cross-Bridge dynamics)
- Dysfunction of Cross-bridge dynamics is similar(in characteristics -metabolic,energetic & functional) in all Cardiac Disease (Valvular /Ischemic)
- Core dysfunction > S/R
- With critical /significant (75%) stenosis coronary pr. distal to lession falls>blood flow shifted away from edocardium (high intramural pr.)

CAPACITY OF CORONARY BED

- Capacity defined as flow at which coronary pr. equalled systemic pr
- DISEASE FREE PRIMARY CORONARY TRUNK (LAD,Cx,RCA)

>100 ml/min

- Flow depends on arterial pr, cavity pr.& transmural pr
- Ischemic necrosis begains in sub- endocardium
- This ischemic injury progresses exponentially with time
- Explains the importance of time in Cardiac Surgery

SCORING OF PERFUSION

- RESULT OF TREATMENT remains imprecise unless quantification of obstruction,distribution & severity of perfusion is performed
- Simpler Scoring methods acts as accurate guide line to surgeons
- No substitute to see personally by surgeons deciding for/against operation

Scoring in troduced since 1972 (GLH)

- LV free wall divided 3 segments of fixed size, IVS into Ant.& posteror- with a given fixed value of 15
- Myocardial value directly proportional to amount of myocardium perfused by each artery if unobstracted (If 2 atreries are equal size the segmental value is devided to each

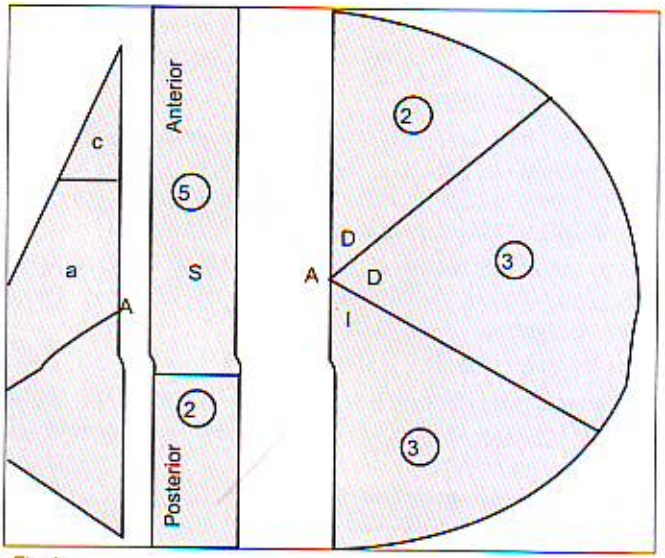


Fig-1

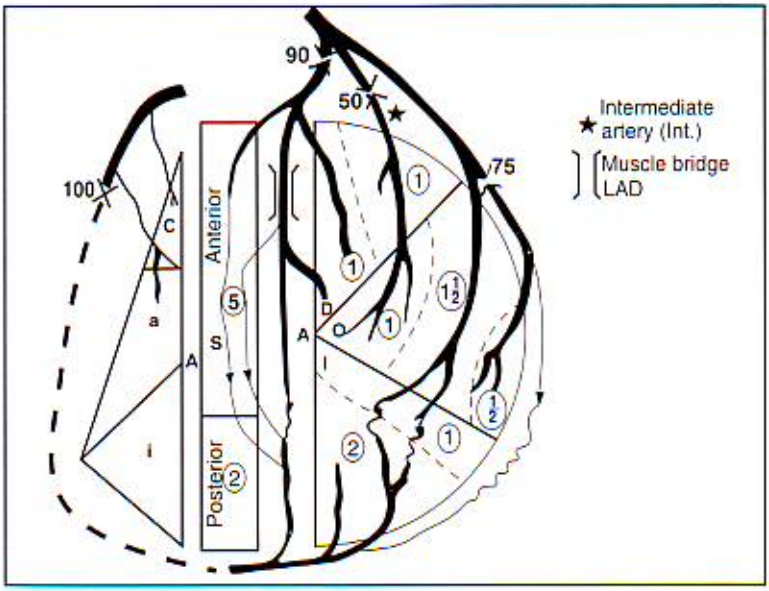


Fig-2 Right Dominant

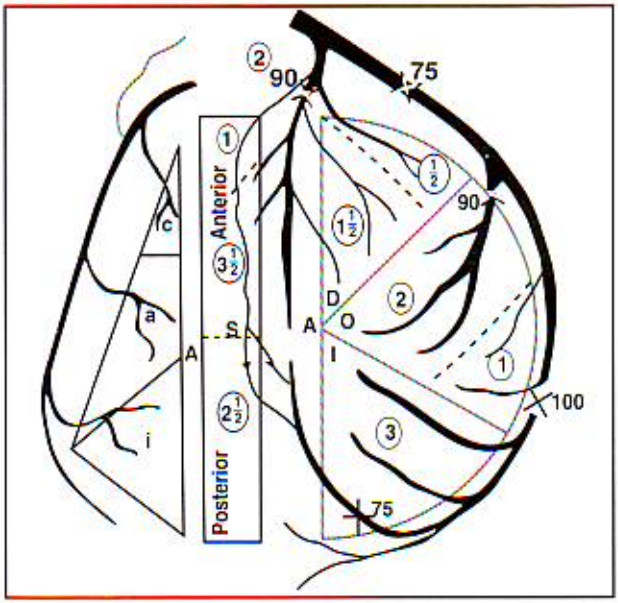


Fig-3 Left Dominant

- 50% diameter (75% cross section)loss-Important
- 67% diameter(90% cross sec.)loss- severe
- Grades:
- A- 100% >grading to the obstruction of the
- B- 67-100% particular branch is computed to
- C- 50-67% the given myocardial value
- D- 33-50%
- When the resultant Myocardial value: < 5 =Single Vessel Disease
- 5 -10= DVD
- 10-15 =TVD

Pt.Specific Prediction & Comparison of outcome in CABG :

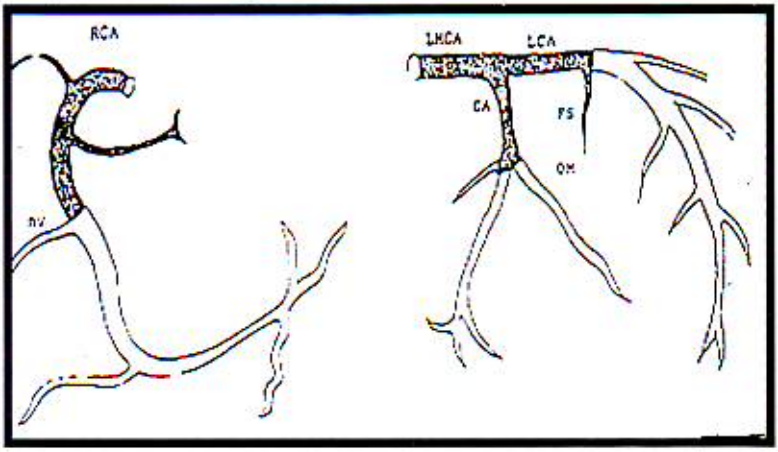
- Indicaton of surgery based on time-related probability of good out-come after operation & Comparison of this outcome with other alternative treatment or no treatment
- Prediction based on:
- 1)Time related probability of freedom unfavourable outcome (death)
- 2) Time related predicted comparative benifit of CABG Vs other options (3) contd....
- In IHD this comparisom is complex for Multivariable factors & treatment Options in IHD
- At unrealistic minimum no. of factors to consider :
- 1. No. of system with important stenosis
- 2. Lt. ventricular function
- 3. Severity of reversible ischemia
- 4. Presence of acute Infarction
- For a realistic prediction Soft ware is necessary

MORPHOLOGICAL SCORING

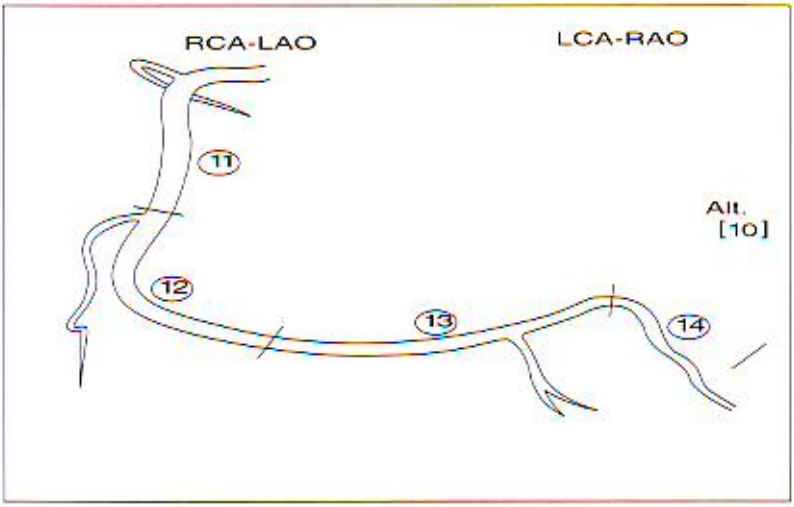
(Extent & Severity)

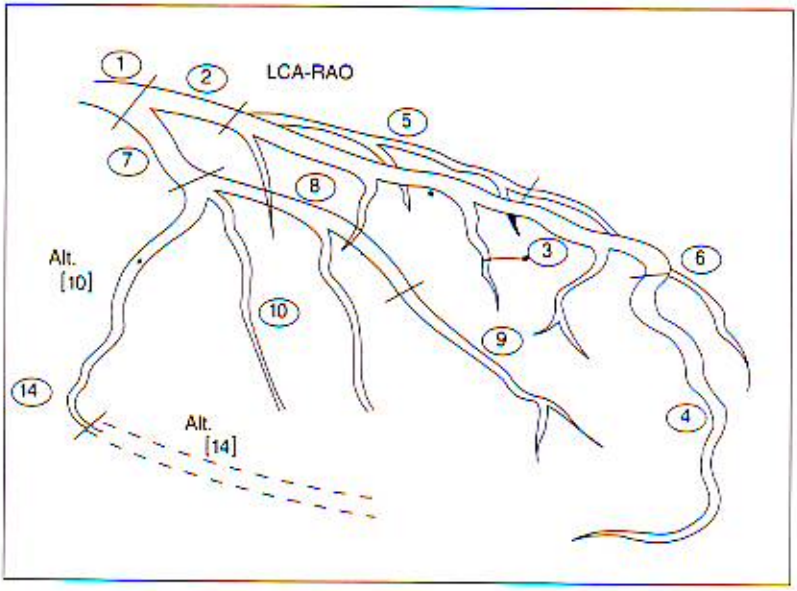
- Grade 1 - <50%
- 2 - 50-69%
- 3 - 70-95%
- 4 - 95-99%
- 5 - 100%

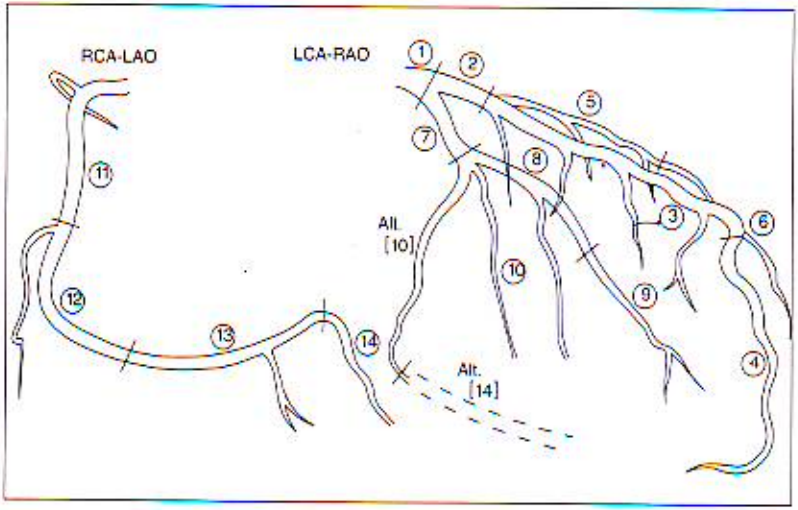
Segmental Coronary Artery: Proximal Lesions



Segmental CAs :




LCA-RAO
1
2
5
7
8
3
6
Alt.
[10]
10
9
14
4
Alt.
[14]


RCA-LAO
LCA-RAO
1
2
5
7
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11
3
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Alt.
[10]
10
12
9
13
14
4
Alt.
[14]

- Diffuse lession > If >705 (i.e. 3-5 Grs) involving:
- 3 of 5 segments of LAD
- 3 ,, 5 ,, Cx
- 2 ,, 4 ,, Cx (non dominant)
- 2 ,, 4 ,, RCA
- INDICATION OF REVASCULARISATION DEPENDS ON ADEQUET DISTAL VESSELS (Size & run-off)

MYOCARDIAL PRESERVATION

- MYOCARDIAL STUNNING :
- Damage from a period of ischemia resulting both systolic & diastolic dysfunction of variable period without necrosis (hrs to days)
- Ischemia leads to necrosis(MI)
- Takes 20 mins
- Some investigastors found 6hrs normothermic ischemia compitable to myocardial cell servival
- STUNNING OCCURS WHEN AFTER A ACUTELY DIMINISHED FLOW, REPERFUSION starts.
- Inspite of normal flow>diminiahed contraction(Perfusion-contratility mismatch)
- Causes : Hypothesis
  - » Diminished cosumption (to protect necrosis)
  - » DENIED AS STUNNED CELL SHOWS HIGH CONSUMPTION
- May be abnormal energy utilization (other than high energy PO4) – Unlikely
- Current hypothesis - Free radicle(neutrophil etc) Experimentaly Super oxide dismutase introduced before ischemia can prevant stunning
- Ca+ influx during reperfusion also responsible -"stone heart"
- HYBERNATION: IS PERFUSION – CONTRACTION MATCH (both are low)
- *>Chronic potentially reversible state of dysfinction*

MYOCARDIAL MANAGEMENT

- Objectives: To limit ischemic injury by some combination of:
- -Hypothermia
- -Electro-mechanical arrest

- -Wash out
- -O2 / substrate enhancement
- -Onchotic / Buffer manipulation

No single method unequivocally best.

FACTORS TO CHOICE

- Technique that influences duration of Cross clamp.
- Sourgeons conviction that injury prevention possible despite complexity of procedure
- Institutional enviornment
- Costs

METHODS

1) Continous Normokalemic perfusion:

- Normothermic – Not ideal,
- -flow distribution abnormal (small heart)
- -collaterals impeded
- -transmural infraction
- Mild to Moderate Hypothermia(25-30 c) – good result

2) Fibrillating Heart perfusion:

- - Normothermia(37°c)-Fibrillation by current
- -Hypothermia(Moderate)-Fibrillation sponteneous/current
- [sub endocardium injury in hypertrophy]

3) Moderate Hypothermia 25-30°c (Intermittant)

- Surgeon works on clamp for 15 mins than release-Does not provide adequet exposure

4) Profound Hypothermia -22°c for 45- 60 min X clamp- May be used in infant surgery

5) Drugmediated protection β blockers, Ca+channal blockers, hypothermia & Intermitant ischemia

6) Cold cardioplegia(multidose)-

- Asanguinous- low K+ for maimtainance & substrate added
- Sanguinous-

Hyperkalemic cold - works well

Bucuckberg formulation(Blood-crystalloid mix + free Ca+& Glucose + Buffer) –as good as blood cardioplegia

7) Blood Cardioplegia (Cold) - less costly, only Blood & K+ (22 mmol/L)

MODE

ANTEGRADE

- 150 ml / min/m^2 - for 3 min (average adult 750 ml)
  [If root pr. < 30 mmHg Rate to increase- Not total dose]
- Re Infusion –after 25 min for 1min(K+ reduced to 10 mmol/L)
- If serum K+ 7-8 mEq /L > Bolus of 400mg/Kg Glucose(50%)+ 0.2U/Kg of Sol.Insulin

RETROGRADE

- Through CS at Pr. <50mmHg

RESULT

- With cold cardioplegia 'safe' duration is not unlimited, Probably 100 min is safe
- Continuous (cold) perfusion(Ante/Retro)-
- Alternative to single dose /Multidose intermitant
- Continous(Warm) perfusion(Ante/Retro)-
- Provides good protection. But some time surgically inconvenient
- 8) Cold cardioplegia with controlled Aortic root perfusion & warm cardio-plegia induction:
- (Minimize reperfusion injury & stunning with better performance. Not widely accepted)
- Circuit: 1.Mini Heat Exchanger
  2.Two pumps
- Technique:
- Warm,hyperkalemic modified blood infusion upto 70 mmHg
- >Total dose 500ml then > normothermic,normokalemic unmodified blood > continued till sinus rythm returns (usually 20min) > Xclamp released after deareation

ANCILLARY PROTECTION

- ANAESTHESIA to CPB starty – Pt at high risk of damage
- Cortrol of HTN
- Care of Oxygen demand
- Manipulation of catocolaemines
- Anxiety

COUNTER PULSATION : IABP

PHYSICOLOGY & MECHANISM

IABP only augments cardiac function by reducing afterload & increasibg diastolic pressure.This is contrary to VADs that can completely replace pumping function of failing heart

Basic Strategy to use devices:

Mostly IABP is firstly used.Can be put in ICU.

If IABP+Pharmacological support fails adequet tissue perfusion– LVAD indicated

INDICATIONS : IABP

1. Post cardiotomy cardiogenic shock (inability to wean from CPB)
2. Cardiogenic shock after AMI,unresponsive to medical therapy
   - Primary myocardial dysfunction
   - VSD
   - MR(papillary rupture)
3. Unstable Angina
   - Pre MI & Post MI
   - Failed angioplasty- travelling to operation
4. Ventricular tachyarrhythmias caused by ischemia
5. Bridge to transplantation
6. High-risk cardiac patients undergoing general surgery
7. Adjunct to mechanical ventricular assistance

CONTRAINDICATION :

1. Aortic Regurgitation
2. Dissection
3. Thoracic aneurysm
4. Peripheral Vascular Disease(severe)
5. Blood dyscarasias
6. Irreversible Brain Injury
7. End stage Ventricular failure

IABP : Inflation & deflation of ballon in synchrony with cardiac cycle can optimize O2 consumption. 3 parameters can be adjusted accordibg to changing requirement of Pt.

Ballon size : 4.5 -12.0 F(Typica adult requires 8.5 -9.0F cath ,40cc ballon)

Synchronization achieved by various trigger modes:

- ECG - Most frequently used
  - HR >150/m decreases IABP efficiency
  - Usefull in arrest
  - Usefull in AF
- Pressure- Incinsistant ECG trigger
  - Usuing electrocautery
  - Requires systolic BP >50 mmHg
- Pacer - For A-V / Ventricular pacing
  - Requires 100% pacing
- Internal – When NO CARDIAC OUTPUT
  - Set rate
  - When BP<50mmHg

(Augmentation should be <1/2)

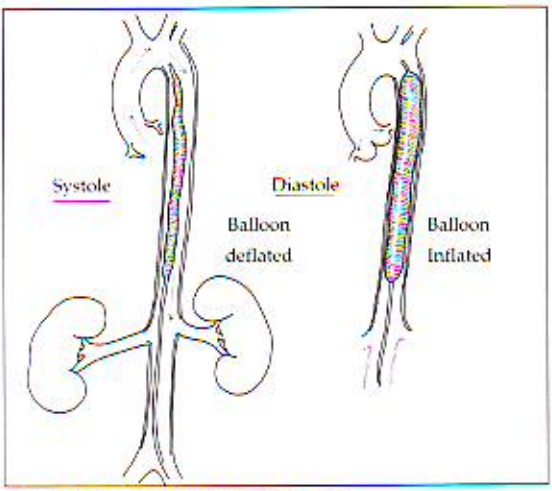


Fig : Correct positioning IABP

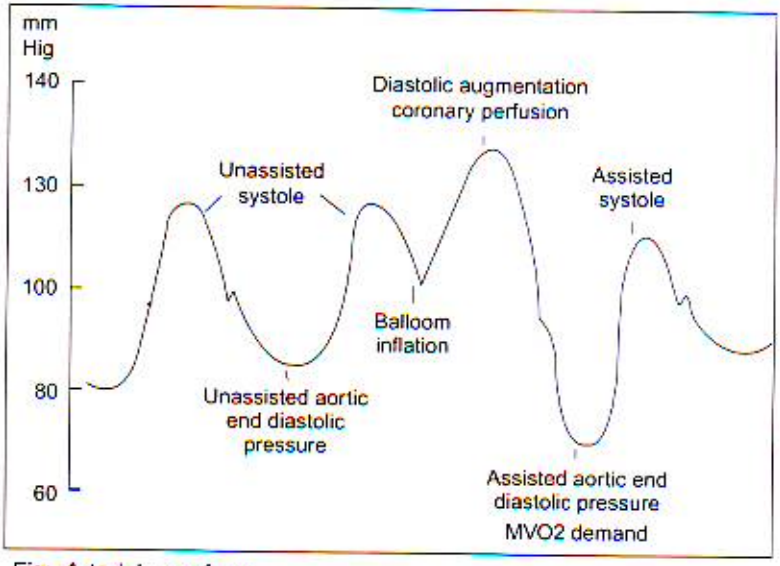


Fig: Arterial waveform

Inflation must occur just after av closure & deflation as AV opens for proper augmentation.Optimum timing can be determined by wave form.The sme wave form & Pt. status reveal TIMING ERRORS.

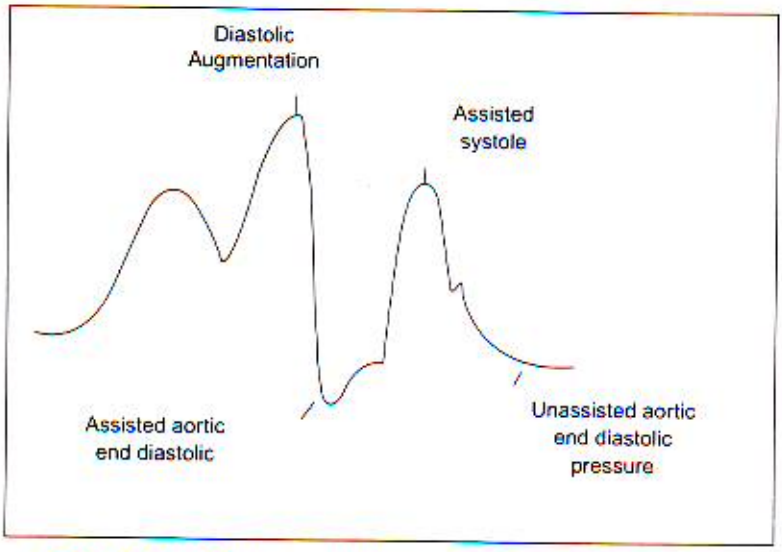


Fig.Early Deflation (Premature deflation in diastole)

Characters:

1. Sharp drop after diasl.augmentation
2. Suboptimal augmentation
3. Assisted EDP equal/greater

Effects:

1. Suboptimal CA perfus.
2. Retrograde CA perfus.
3. Angina
4. Suboptimal afterload reduction

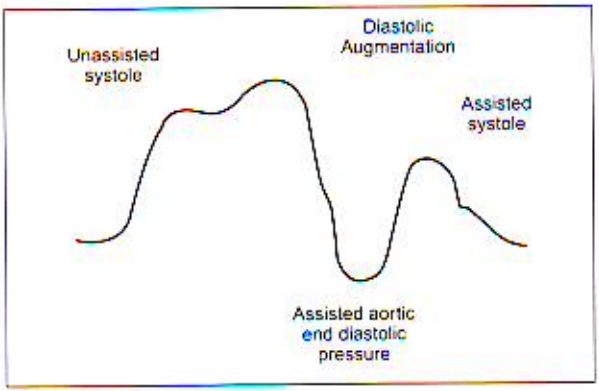


Fig. Early Inflation

Character:

1. Inflation before notch
2. May encroach systole

Effects:

1. Premature closure AV
2. LVEDP / PCWP increases
3. Increased MVO_2 demand

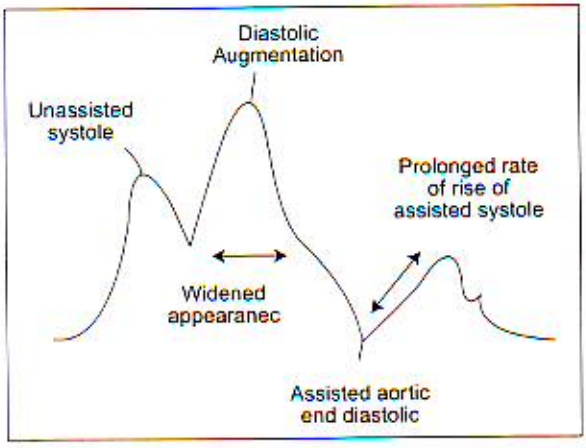


Fig. LATE DEFLATION

Characters :

1. Assisted EDP may be equal essentially absent
2. Diast.augmentation widen

Effects :

1. Afterload reduction
2. Impade LV ejection
3. MVO_2 increases

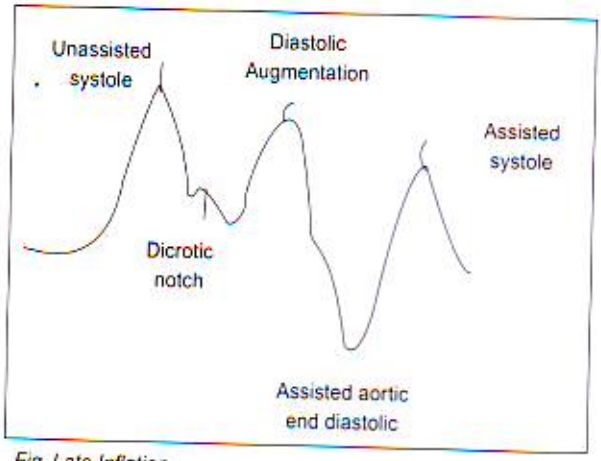


Fig. Late Inflation

Character:

1. Inflation after notch
2. Sub optimal augmentation
3. Abscence of sharp V

Effects:

1.Sub optimal coronary perfusion.

INFLATION FREQUENCY SET UP : By FREQUENCY: HR (1:1 , 2:1)

APPROPRIATE ADJUSTMENT WITH INFLATION /DEFLATION TIMING.

WAVEFORM & PATIENTS STATUS REVEALS CORRECT TIMING

WEANING FROM IABP

PRINCIPLE : To withdraw support incrementally & to asses haemodynamics at each step

- Pt must be stable with minimum inotrops
- CI >2 L/min/m2
- Systolic Pressure >90mmHg
- LA RA pres

sure <20 mmHg

- ❖ HR <100/min
- ❖ Urine >0.5 ml/kg/H

_ Decrease inflation frequency 1:1 to 2:1 to 3:1 at 1-2 hr interval

_ Decrease amount of augmentation to minimum of 50% for prevention of thrombosis

_ When minimum support tolerated for hrs withdraw taking care to purge clot from proximal & distal femoral artery

_ Direct pressure for some minutes(20),apply sand bag for 6hrs, confined to bed for 12 hrs

IABP : COMPLICATION OF

1) Limb Ischemia

Most common (5-19%)

Related to cardiac output, catheter diameter,intimal injury,thrombosis.

2) Perforation –Common in shock,PVD

Sup.Femoral Artery – thrombosis,leg ischemia

Abdominal vessel - Retroperitoneal haemorrhage

3) Incorrect position

Viscral ischemia,Aortic insufficiency

4) Aortic dissection (<5%)-Usually retrograde ;often seals with own

5) Wound complication (1-3%)

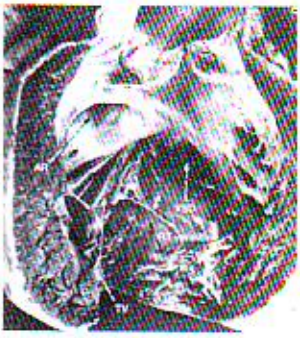
6) Catheter failure (Gas escape)

VADs : When function of heart remain inefficient inspite of ballon,iotrops use of VAD may be considered

- ❖ VADs takes over the function of heart but ballon only optimize cardiac function

DOUBLE OUTLET RIGHT VENTRICLE (DORV)

Defination



- Congenital anomaly in which both great arteries arise wholly or in large part from the right (>50%).

DORV WITH Associated Diseases:

- In association of AV Discordant
- Univentricular AV Connection
- Atrial Isomerism

- Uncommonly Arteries overlies doubly commited Juxtra arterial VSD *(Double outlet both Ventricle)*
- TOF is an entity with variable amount of dextro position of the aorta
- In TOF when the aorta >50% from RV catagorized as TOF WITH DORV (DORV WITH PS)
- Taussig-Bing Heart, PA arises wholely or nearly so from RV with A type of DORV (Equally from the RV,LV or more than 50%,not entirely, from LV is not a type DORV)
- If arises entirely or so from LV -TGA with VSD

Morphology

- Sub aortic
- Large with restrictive flow
- Mostly conoventricular(between 2 limbs)
- May extend to the mitral/tricuspid continuity
- May be with anomalous cordae
- DORV with L –malposition(Lt to PA),VSD Sub aortic/Juxta aortic

Sub Pulmonic

Taussig- Bing Heart is the example

- Infundibular septum is sagital and not a part of IVS
- Doubly Commited VSD
- Uncommom variant

- Abscence of connus
- Anterior & inferior boarder formed by STM & Ant.limb
- Resemble Juxta arterial VSD/TOF with Juxta arterial VSD /Some times DOLV
- Both semilunar valve over RV difficult to deferentiate from overlying LV(DOBV)
- Non Comitted/Remote
- Away from Semilunar valve

Great Arteries

- Rarely both overlies RV (in intact septum / non commited VSD)
- Both overlies whn VSD sub aortic
- Doublycommited VSD or sub pulmonary VSD is overrided by one / both great arteries
- Normal, D-position, L-position, side by side relation are found in DORV
- Doubly commited VSD have normally related great artery
- Cine/Operative assesment of Great arteries position are not acurate often

Pulmonary Stenosis

- Common in subaortic VSDs
- Mostly Infundibular
- All types of PS somilar to TOF are found

Forms of DORV:

- Simple DORV :
- Easily repairable
- Sub aortic VSD
- Aorta to right& Side by side or posterior
- Coronary anatomy is normal

Spectrum : DORV with perimembranous VSD & non commited inlet extension VSD & doubly commited with out let extension VSD.

- Taussig-Bing Heart :
- VSD anterior, superior & subpulmonic
- LMCA anterior to Pulmonary trunk

- PA overlies both ventricle
- Aorta at right & anterior or parallal(1st portion)

Spectrum: DORV with transposition with large VSD or DORV with non commited VSD contd.

- DORV with Doubly commited VSD:
- Uncommon
- Juxta arterial VSD
- DORV with noncommited VSD:
- Away from great arteries
- In the trabecular septum
- Easy to identify
- VSD sub aortic with PS
- Rarely subpulmonic
- May be non commited
- DORV with c-AVSD:
- Sub aortic & large
- Deep but not extended like noncommited
- DORV with Superior-Inferior ventricle :
- VSD perimembranous with inlet extension
- Straddling of TV against VSD
- Hypoplasia of LV

Clinical Feature

- Highly varies like the morphology of the disease. Not clinicaly cyanotic as Qp is high
- Cyanosis determined by smilunar valvea,VSD and position / presence of infundibular
- septum (High sturated LV blood flows to aorta,systemic largly to PA)
- PS associated with severe cyanosis

Pulmonary Vasacular Disease

- Progress is rapid when without PS

- Cardiac Cath / Cine :
- Cath is not routinely necessary in neonats/young infants(<1yr)
- Older Infants/Children (<11yr) may be needed to asses haemod ynamics, Rp,extra/Intra cardiac morphology (Pulmonary vasculature, Aortopulmonary collaterals)

- CINE IS IMPORTANT FOR COMPLEX RELATION SHIP

Natural History

- Simple DORV & Doubly Commited ,non commited VSD have similar course like Large VSD
- Sub Pulmonic VSD (Taussig Bing)- Similar to TGA- Sever PVD at early & poor prognosis
- When associated with PS- Course is similar to TOF

Tecnique of Operations ;

Depends on VSD,Great artery relation ship,Age & Ventricular Morphology:

1) Sub Aortic VSD with Adequet TV to PV distance/ PS – Tunnel Repair
2) Sub pulmonic VSD without PS – Arterial Switch + VSD closure or Tunnel or Atrial switch
3) Doubly Commited VSD – Patch closure(LV to Aorta) by enlarging VSD anteriorly to avoid obstruction
4) Non commited VSD – Buffling VSLecompte Repair D to Aorta(enlarging VSD with / without muscle insertion of straddle TV or FONTAN
5) DORV with pulmonary out flow obstraction (or) VSD + TGA without LV outflow obstraction-Lecompte Repair
6) DORV with AVSD – Repair Septal defect & TOF

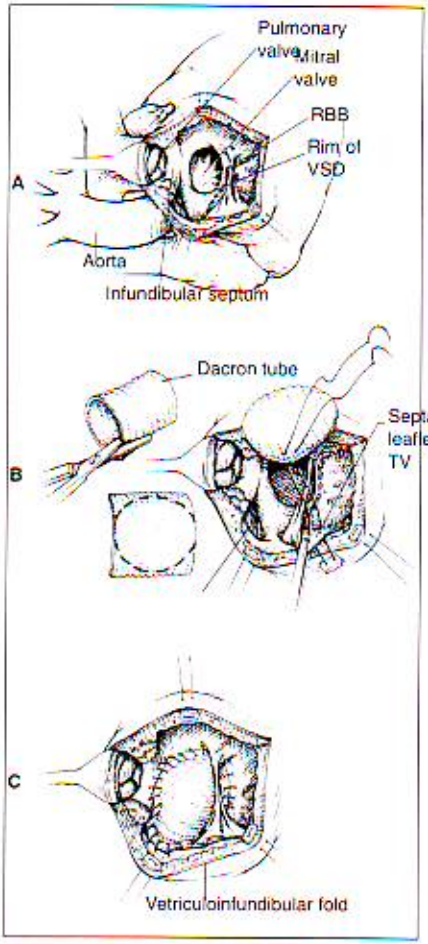


Fig.1: Intraventricular Tunnel Repair of SIMPLE DORV

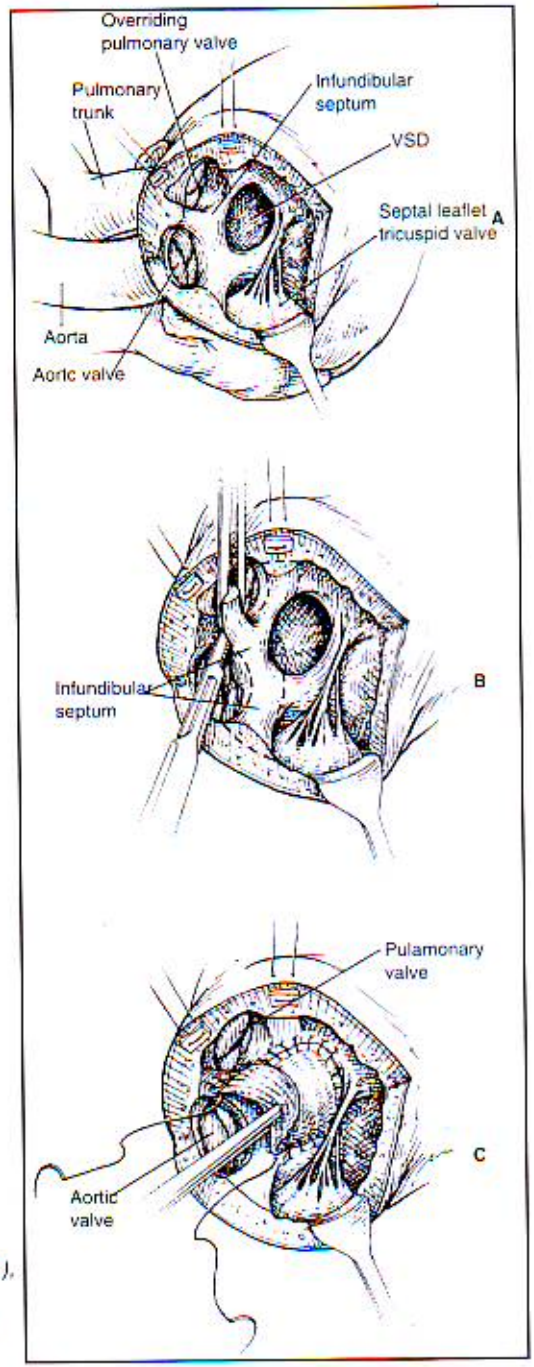


Fig 2: Intra ventricular Repair VSD (Taussig Bing). PA &Aorta side by& with adequet distance of PV&TV

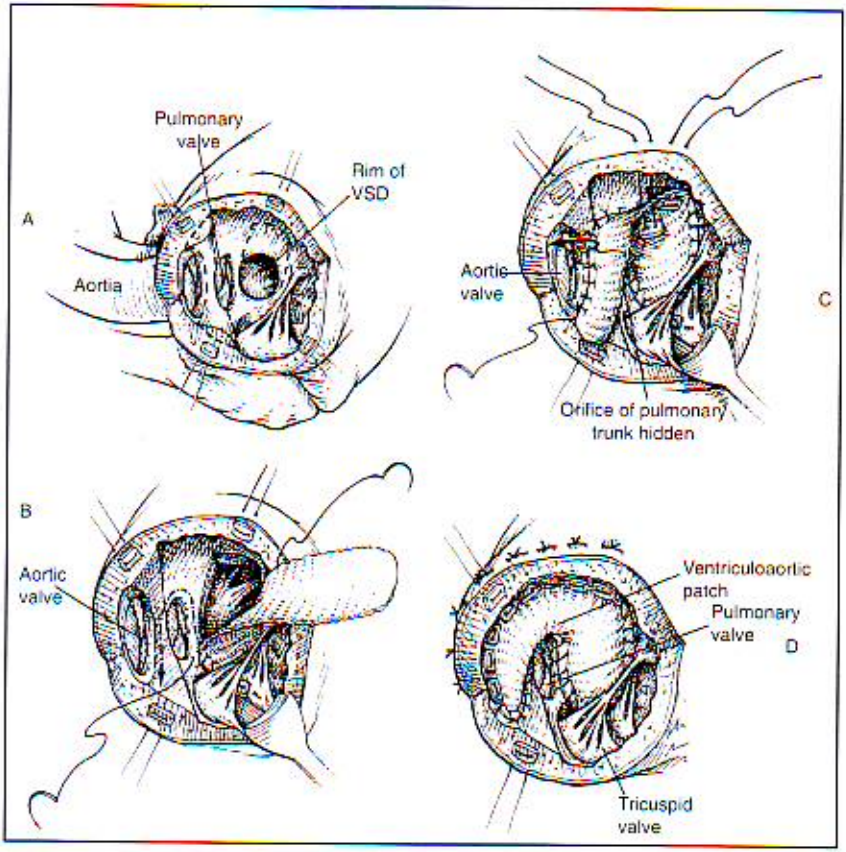


Fig 3 : Intra ventricular Repair VSD (Taussig Bing), with short distance of PV&TV (Patrick-McGoon Method)

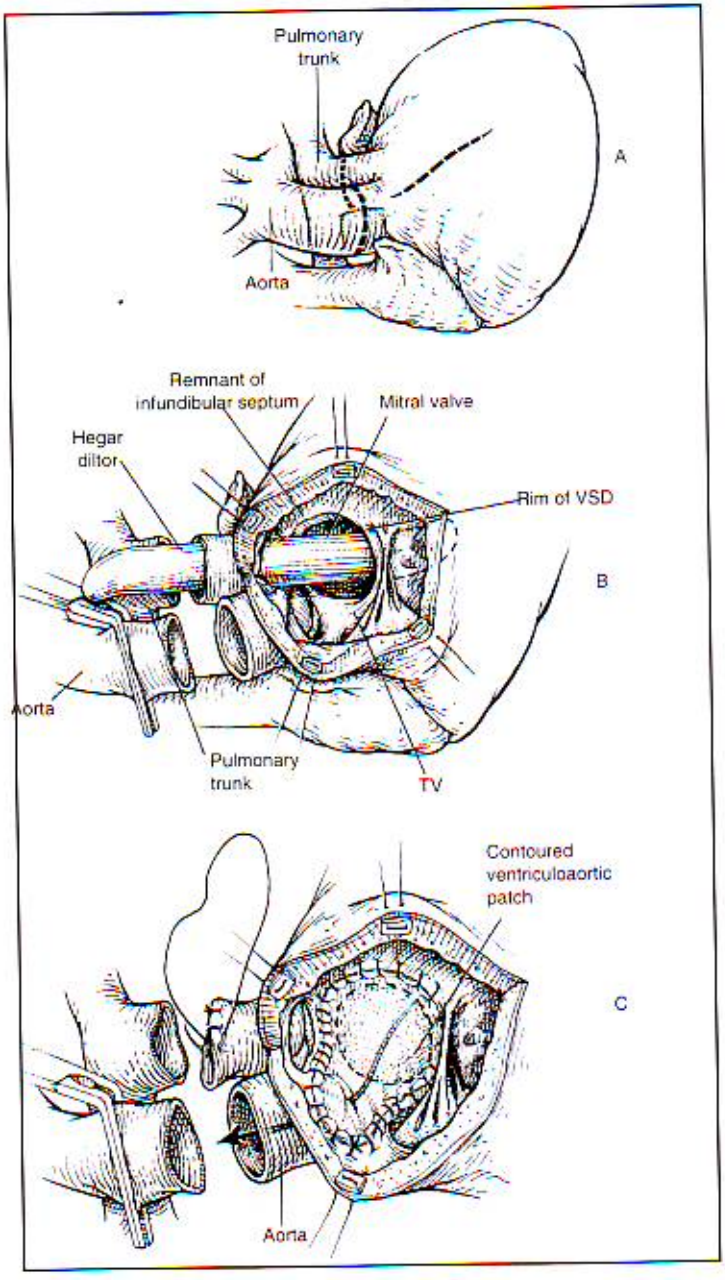


Fig 3 : Lecompte Procedure

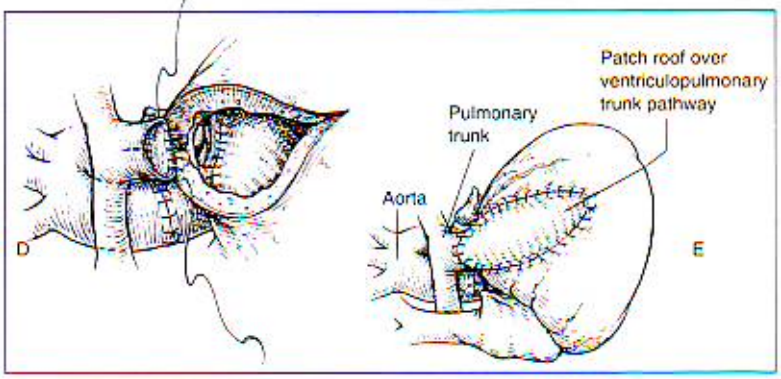
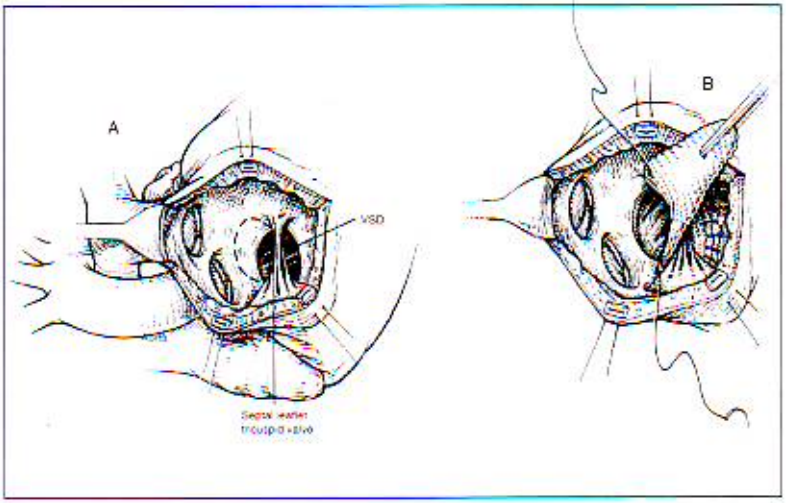
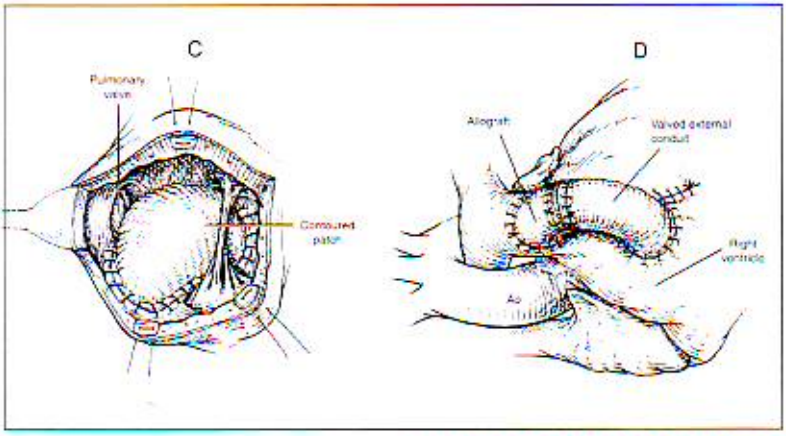


Fig 4 : Repair of Non commited





Result

Hospital Mortality

- Simple DORV – 20% Some reported 15% n=162,personal communication) Over all mortality (4 types of Operations,n=124 by Brown)-4.8%
- Taussig - Bing Heart- 50%
- DORV with Doubly commited VSD – Not determined (small number)
- DORV with Non commited – similar
- FONTAN(in DORV with non commited VSD) - 5~10%
- No data of time related survival is available
- Simple DORV.DORV with Doubly commited with no PS - are favourable types of DORV regarding survival after repair & are not risk factor for death
- Simple tunnel (in DORV +subaortic VSD) have- similar risk like karge VSD
- Complexe Tunnel repair is a risk factor for death(Rastelli) Current ear riks minimizes.Similar to Switch operation
- PS +DORV often needs nonvalved/valved conduit increses mortality
- Result of all complex procedures risk are uncertain
- Modern development probably allows better result , yet to wait for data

Indication of Operations

- 1) Simple DORV with Sub aortic VSD-should be corrected by 6 months.
- Echo suffices diagnosis –

  Tunnel Repair

  If with PS then similar to TOF
- 2) DORV with sub pulmonic VSD (Taussig-Bing) -Arterial Switch Operation (ASW)+ VSD closure in 1st month /as soon

No indication of Tunnel (IVR) Repair unless:

- Subpulmonic stenosis- Tunnel + PA conduit
- (Arterial switch Operatiom(ASW) contraindicated)

- Except ASW all procedures should be delayed until 3~6 yrs. Interim BT Shunt MAY BE NECESSARY

Indications

- 3) DORV with Doubly commited VSD – Similar to simple VSD(Care to enlarge VSD to put non obstractive patch)
- 4) DORV with Non cxommited VSD-
- IVR with enlarging VSD
- when tunnel obstracting - PT conduit / LeCompte
- If tunnel directed to PA-VR with ASW (If no PS & IVR patch obstructing PV)

  Or

  T Banding (Early) than - FONTAN

FORMS OF VSD & PS

Introduction

- Few Pt with Infundibular PS with/without VSD do not meet the criteria of TOF or low–lying Infundibular stenosis (Intraventricular stenosis)
- Comprises mostly Children,young adult not infant

Morphology

Infundibular PS +VSD :

- Stenosis is severe enough for resection
- VSD may be conoventricular,Large or may be small

Valvular PS And VSD

- Isolated valvular PS rerely coexsists with VSD(Alabama Universty experience 8 cases in 17 yrs,1967-83)
- Age is similar to infundibular variety
- Stenosis is anatommic not functional
- VSD may be conoventricular,large or small or muscular
- Combination may merely represent chance co-exsistance of two malformation
- Surgery is electively similar

Risk Factors: Transannular Patch

- Transanular Patch is not per se independent factor for death in early phase. Lately cause of RV dysfunction
- Trans anular patching is risk factor in neonates & young infants unaccompanied by syst-Pulmonary shunt
- RV cavity size reduced
- Die due to hypoxia /RV failure
- TR is additional risk

Combination : Valvular & Infundibular PS /Atresia with VSD

- 10 pts in 17 yrs (UAB) reported

- VSD may be multiple muscular also
- Dextrocardia,D-loop Ventricle may coexsists
- In these unique cases Management must be individualized

VSD transformation to TOF

- Some VSD with Large VSD in infancy develop later infundibular stenosis (5-10%)
- Mild to modearte Stenosis found at surgery for VSD may probably represent these cases
- Stenosis may be severe to define TOF.
- These transformed cases probably born with some anterior displacement of infundibular septum

Surgery

- Similar to Low lying Stenosis
- Infundibular patching/Vertical ventriculotomy are routine
- Important is to identify infundibular stenosis at time of repair otherwise reoperation may be required
- Results are excellent
- (InUAB experience of 17 yrs) out of 48 ,one reported unexplained death

HEART FAILURE

DEFINATION

CLINICAL SYNDROME RESULTING FROM DEFECTIVE CARDIAC FILLING (DIASTOLIC) OR EMPTYING (SYTOLIC) WITH INSUFFICIENT TISSUE METABOLISM

PATHOPHYSIOLOGY

- Evokes compensatory mechanism:

In short term > to restore cardiac function
Over time > develops secondary damage to myocardium> Remodeling > decompensation > death

Neuro humoral

Angiotensin II > Salt retention
Increased adenergic response- with increased density of receptors

- Over time -Phosphorilation Ca receptors
- Reduced uptake Ca+

REMODELLING

> Myocardial Hypertropy & dilatation
> Reorganisation of myocytes
> Ischemia
> Fibrosis>Accelerated HF

Overloading>Re activate embryonic growth factors>leads unnatural muscle growth> Ultimate cell death

CLINICAL FEATURES

- Stage A-Risk factor (HTN,DM,Alcohol,F/H)
- Stage B-Structrual disorder (hypertropy) with out symptoms
- Stage C-Structural + Symptoms
- Stage D-End stage (Frequent hospital/medical)

NYHA classification denotes functional capacity (Stage B,C)

GOAL : TREATMENT

SLOW / REVERSE REMODELLING
TREATMENT

Theraputic:

4 drugs (Diuretics,ACE inh, B-blockers, Digitalis)

> Short term-Inotropes

> Digitalis

Neurohumoral Agents:

Carvedelol

Surgical:

Mitral Valvoplasty

Partial Ventriculectomy

Endoventricular Patch plasty

MECHANICAL CIRCULATORY SUPPORT:

- TEMPORARY - After Surgery
- INTERIM -Replaced by donar Heart / Recovery
- PERMANANT
  - -Implantable
  - - Paracorporeal(Partial implantable)

Power Source:

Electric

Pneumatic

*Pulsatile

*Non pulsatile

HISTORY :

- 1920-Bryncho nenkho first attemted artificial circulation
- 1940-In Russia reported
- 1953- HEART- LUNG MECHINE INTRODUCED
- IABP-Invented in1962
  1968-Clinical introduction
- 1982 –Jarvic – 7(VAD) was introduced(Pneumatic,Parmanant)
- 1990 -VAD USED AS TEMPORARY BRIDGE TO TRANSPLANT
  TEMPORARY VENTRICULAR ASSIST DEVICEs (VADs)

Uses:1)After Surgery
2)Bridge to transplant
3)Bridge to recovery

May be used with IABP

FDA approves 5 devices:

ABIO MED BVS5000	Pneumatic	Temporary
• Thoratec VAD (Rt/Lt)	,,	,,
• Thoratec Heart Mate (IP1000LVAD)	,,	Parmanant
4) Novacor	Electrical	,,
5) Thoratec Heart Mate (TCI LVAD)	,,	,,

All are pulsatile flow

VADs

Parmanant, Paracorporeal

- ❖ Thoratec VAD PNEUMATIC

- ❖ LV / RV or both
- ❖ Untill recovery LV
- ❖ Bridge to transplant

TO PLACE OVER ABDOMINAL WALL

- ❖ Under development

VADs

Parmanant Implantable

- ❖ Pneumatic Thoratec Heart mate IP1000 LVAD
  -long pipe line
  -out side dreiver console
- ❖ Electrical - Thoratec Heart mate TCI SVE LVAD
  - Novocor
  Use: Normal Life style
  Long wait

ICU PROTOCOL

Protocol for Reducing Arterial Blood Pressure and Afterload

Sodium nitroprusside is used intravenously (IV) either continuously / intermittently as required.

Acts directly on arterial and, to a lesser extent, venous smooth muscle, and thus decreases systemic and pulmonary vascular resistance and systemic venous tone. Onset and need of action are immediate.

The dose >> 1 to 10 mg. kg^{-1} min^{-1} (doses larger than this are not used), regulated in most cases to maintain a mean arterial blood pressure 10% above the normal value for the patient's age.

In patients with thick left ventricular walls or coronary artery disease (concern about coronary perfusion pressure) keep mean arterial blood pressure 20% above normal desirable (or ,150 mm Hg in adults)

- **50 or 100 mg of Sodium nitroprusside are dissolved in 150 mL of 5% glucose in water.**

The drug may be administered with a slow-infusion pump or with a servo-pump using a closed-loop system under computer control .

[When nitroprusside is being administered, methods should be available for measuring blood thiocyanate levels, because values of > 10 g dL^{-1} are potential toxic. The toxicity is due largely to the formation of cyanide, the major metabolic product of sodium nitroprusside

Toxicity is manifested by signs of:

Intracellular suppression of oxygen consumption (elevation of mixed venous oxygen levels, narrowing of arterial-venous oxygen difference, and metabolic acidosis)

Anorexia,

Muscular spasms,

Disorientation, and

Convulsions.

***Treatment** : IV infusion over 15 minutes of 150 (mg. kg^{-1} of 25%*

solution of sodium thiosulfate (10 (mg . kg^{-1} min^{-1}).

Nitroglycerin : decreases venous lone but also decreases coronary resistance.

It is therefore particularly useful when myocardial ischemia is present . Dose: of 0.5 to 3.0 mg. kg^{-1} min^{-1} are recommended.

(Nitroglycerin is absorbed into polyvinyl tubing used as IV infusion, and the concentration reaching the patient is less than planed until the tubing becomes saturated.)

Nitroglycerin is not as effective as nitroprusside in lowering arterial pressure.

Phentolamine is another vasodilator used on occasion.

It has an-adrenergic receptor blocking effect, produces direct smooth muscle relaxation, and reduces pulmonary vascular resistance.Its recommended infusion rates are 1.5 to 2 mg. kg^{-1} min^{-1}.

Phenoxybenzamine is a noncompetitive blocker of-receptors with a prolonged (12 to 24 hour) effect and a delayed 20 to 60 minute) onset.

It acts on both arterial and venous vessels and has no important side effects. It is administered IV in a dose of 1 mg Kg^{-1} with the solution diluted in 20 to 50 ml of normal saline solution and infused slowly over about 15 minutes.

(The disadvantaged of phenoxybenzamine is that its-blocking effect is complete for at least 12 hours; thus, the drug is not used until the need for prolonged afterload reduction has been established by the patient's response to a sodium nitroprusside infusion over about 12 hours. Rather than continue infusing sodium nitroprusside at a relatively high rate, phenoxybenzamine may be substituted, and the dose repeated in 12 to 15 hours, when it is clear that its effect is wearing off.)

Mlrinone and amrinone (phosphodiestrease inhibitors) may also produce a salutary vasodilatory effect.

Protocol for Optimizing Preload

To optimize postoperative cardiac performance, atrial pressure may be manipulated, often by infusion of blood or blood substitute.

Elevation of PLA or volume infusion peruse has disadvantages; thus, the target PLA must have an upper limit. One must:

a. Judge or measure cardiac output

b. Decide on the most appropriate PLA (or right atrial pressure PRA)

c. Set an upper PLA (or PRA) limit

d. Design parameters to guarantee attainment of the desired goals particularly adequate filling volume.

e. Limit risk of overinfusion.

In the operating room and intensive care unit, d) and e) are accomplished as follows:

1. A PLA limit is selected to attain adequate cardiac output. (This limit is modified from case to case, and from time to time in any one case is necessary).
2. Blood or a blood substitute is selected and infused to attain the desired PLA limit.

In ICU:

- Maximize hourly infusion as a multiple of hourly chest drainage (e.g. 1x, 2x, or 3x measured chest drainage).
- Measure total infusion, based on body surface area (e.g. 250, 500, or 750 ml. m 2).

The physician's order should have two parts:

1. The PLA limit
2. The parameter by which the infusion to attain that limit is blocked. For example:

PLA = 12 mmHg. arterial blood pressure (PAO)≤120 systolic

PLA = 8 mmHg. infuse 250 mL m^2

PLA = 10 mmHg. infuse to limit of 2x chest drainage

PLA = 18 mmHg. infuse for measured (CI), (2.5 $L.mm^{-1}.m^{-2}$.

PRA = 16 mmHg. infuse to PAO 100 systolic (e.g tetralogy repair)

Protocol for infusion of Inotropic Agents

The rate of infusion is calculated and recorded in micrograms per kilogram body weight per minute (mg. kg $^{-1}$ min^{-1}). Using a microdrope apparatus, the number of drops per minute equals the

number of milliliters per hour. The formula for milliliters per hour (drops per minute) as a function of infusion rate (mg kg $^{-1}$ min^{-1}_ body weight (Kg) and concentration of calecholamine is

ml. h^{-1}(or drops per minute) =

(Infusion rate (mg kg^{-1} min^{-1}). weight (Kg).60

Concentration (mg mL^{-1})

The drug is diluted in 5% glucose and water (in infants less than 13 kg. >>diluted in 10% glucose water).

First, the rate of drug infusion is selected, and then the drip rate. The concentration of drug needed in the solution may be found by giving the equation above..

For each drug, a "standard" rate of infusion based on past experience has been determined. Infusion rates are altered according the hemodynamic state and response of the individual patient to the infusion "Standard" rates of infusion should, however, be exceeded any under special circumstances.

The "standard" rates of infusion are:

Dopamine	10.0 mg. kg^{-1} min^{-1}
Debutamine	10.0 mg. kg^{-1} min^{-1}
Isoprotoreonol	0.05 mg. kg^{-1} min^{-1}
Epinephrine	0.1 mg. kg^{-1} min^{-1}
Norephinephrine	0.1 mg. kg^{-1} min^{-1}

Protocol for Managing of Ventricular Electrical Instability

Early Interventions for Ventricular Electrical Instability

1. Give lidocaine > IV bolus injection (the dose is 1 mg .kg-1 for adults and children, although in adults the usual dose is 50mg) >> if the arrhythmia is premature ventricular contraction (PVC) or ventricular tachycardia (VT) with a good hemodynamic state.

◆ If >> VT and reduction of cardiac output > immediate DC cardioversion (100 and then 200 J).

2. Draw a blood sample for determination of serum K+; when the result its available:

Treat hypokalemia (K^+ concentration < 4.0 mEq.L^{-1}) if present:

a. Administer 5 mEq K+ IV bolus

b. Administer 20 mEq K+ in 50 mL of 5% glucose over hour; then obtain repeat serum K+ level measurement and repeat treatment until serum level is satisfactory (at least 3.5 mEq, L^{-1} and

preferably 4.0 mEq L^{-1}).

c. Double the IV maintenance K^{-1} dose

d. Recheck serum K+ level. If it is <4.0 mEq-L^{-1}, order oral K^+ supplement as 20% KCI, 10 mL twice a day in orange juice (60 mEq approximate daily dose).

3. If the ventricular rate is less than 80 to 90 beats/min. initiate pacing. If basic rhythm is sinus or atrioventricular (AV) junctional> use atrial pacing.

4. When cardiac rhythm is other than sinus or AV junctional or atrial pacing fails to result in 1:1 AV conduction> use ventricular pacing .

5. In the presence of second or third degree AV block, consider AV sequential pacing.

Interventions after Control of the Urgent Situation

1. If the arrhythmia recurs promptly or is not controlled by these simple measures, >>> continuous IV lidocaine infusion in a dose of 20 to 50 mg. kg^{-1} min^{-1}, or 0.02 mg Kg^{-1} min^{-1}.

(To calculate the number of drops per minute of a solution of 2 g of lidocaine in 250 mL of solution.)

Drops per minute =

$$\frac{\text{Dose - weight (kg) x 60}}{4}$$

(The lidocaine level should be measured at 12 hours to prevent anxiety.)

2. Additional measures to control ventricular arrhythmias:

a. Procainamide 15mg kg^{-1} IV over 30 minutes (usually 600 mg IV

can be given rapidly)

i. Maintenance dose 500 mg IV q6h

ii. Observe for hypotension and torsade de pointes

b. Bretylium (a quaternary ammonium compound) 5 to 10 mg.kg^{-1} IV over 1 minute

i. Maintenance dose 5 to 10 mg kg-1 every 6 to 8 hours

c. Amiodarone; initial loading dose 150 mg IV over 10 minutes

i. Maintenance dose 0.5 mg . min^{-1} to approximately 1000 mg-24h^{-1}

Protocol for Acute Digitalization

Digitalizing Dose:

An estimated digitalizing dose of digoxin provides a convenient guideline for the cardiac surgeon. The estimated dose of digoxin, when no digitalis has been given in the past 10 days, may be considered to be 0.9 mg. m^{-2} intravenously and 1.6 mg. m^{-2} orally.

(The digitalizing dose in infants may be considered 50 mg. kg^{-1} intravenously, and the maintenance dose 10 to 15 mg. kg^{-1} day^{-1}.)

Atrial Fibrillation:

When the ventricular rate is at least 110 beats.min^{-1} and no contraindication to digitalis exists, small doses of digoxin are given by the following schedule unit the ventricular rate is 90 to 110 beats. min^{-1}:

1. When the rate is between 110 and 120 beats min^{-1}, give a dose of 0.15 mg intravenously (IV) for adults and (10% of the estimated digitalizing dose for children.)
2. When the rate is 120 to 140 beats. min^{-1}, give an initial dose of 0.2 mg IV for adults and(15% of the estimated digitalizing dose for children.)
3. When the rate exceeds 140 beats . min^{-1}, give an initial dose of 0.25 mg IV for adults and (20% of the estimated digitalizing dose for children.)
4. Usually, several subsequent doses 2 to 3 hours apart are required, and the dose must be reassessed at each interval based on the ventricular rate.

Esmolol: 0.5 mg. kg^{-1} - min^{-1} IV infusion

Adverse effect: hypotension bronchospasm

Propranolol: 0.05 mg kg^{-1} or 5-mg bolus IV

Adverse effect: hypotension

Amiodarone: 150 mg IV over 10 minutes

Adverse effect: Rarely hypotension

- **Unstable hemodynamics**

  DC cardioversion

  Amiodarone

- **Recurrent atrial fibrillation**

  Procainamide 500-1000 mg p.o., 6-8 hours

  Sotalol: Initially 80 mg BID

Anticoagulation

The prevalence of stroke attributable to postoperative atrial fibrillation is unknown. It seems justifiable to initiate anticoagulation of atrial fibrillation persists longer than 48 hours or is recurrent in >> warfarin is recommended.

If elective cardioversion is planned, intravenous heparin is recommended and is prescribed in therapeutic doses.

Protocol for Rapid Atrial Pacing via Atrial Wires

Rapid Atrial Pacing

The technique of rapid atrial pacing is applied to atrial flutter, defined as a general atrial rate of 250 beats min^{-1}, with a constant beat-to-beat cycle length >> can be interrupted by rapid atrial pacing.

(This is rarely possible in atrial flutter-fibrillation, a more rapid type of atrial flutter, with a rate >350 beats min^{-1}, and is not possible in atrial flutter-fibrillation, a more rapid type of atrial flutter, with a rate >350 beats-min^{-1}, and is not possible in atrial fibrillation).

The ECG limb leads are placed on the patient for monitoring, and the two atrial wires are connected to the rapid atrial pacer.

(Because the atrial pacing threshold is usually high during atrial flutter, output is set at 10 to 20 mA.

5. When the ventricular rate is controlled, begin oral maintenance doses of digoxin 6 to12 hours after the last IV dose (the usual oral maintenance dose is 0.25 mg . day^{-1} for adults, for children, see section on sinus rhythm below).

Whe the ventricular rate is not controlled by the time the estimated digitalizing dose has been administered (which

can occur in patients in atrial flutter-fibrillation or in those receiving calecholamines), further digoxin should be given only with caution to guard against the occurrence of digitalis toxicity before rate control is achieved. Additional measures are useful.

Sinus Rhythm:

The digoxin dose cannot be titrated by heart rate, as in atrial fibrillation. Therefore, when digitalization is indicated, one third of the estimated digitalizing dose of digoxin is given, usually IV, and this dose is generally repeated 3 hours later.

Six hours later, a maintenance schedule is begun, usually with one twelfth of the estimated digitalizing dose given twice daily.

(The serum digoxin level is measured daily for 2 days. The appropriate level is 1.5 to 2.0 ^{mg} . ml^{-1}.)

[Alternative Protocols for Acute Management of Postoperative Atrial Fibrillation:

Digoxin has classically been recommended, but it may be slow to act (3 to 8 hours) and relatively ineffective at decreasing heart rate in postoperative patients with increased sympathetic activity. In the elderly or in the presence of compromised renal function, the therapeutic window is narrow and toxicity may occur.]

SITUATONS:

◆ ***Stable haemodynamics adult Patients:***

Intravenous verapmil, diltiazem, esmolol, propanolol, or amiodarone are effective alternative therapies:

Verpamil: 5-10mg IV as bolus; may repeat after 10-15 minutes

Adverse effect: Infrequently hypotension later increase of heart rate.

Diltiazem: 20mg IV over 2 minutes, may repeat x1

Adverse effect: Infrequently hypotension

Bipolar atrial pacing is used because the stimulus artifact then rarely distorts the ECG tracing, and the atrial complex in the ECG is clearly seen so that atrial capture can be verified.)

Techniques:

Ramp technique

Atrial pacing is begun at a rate 10 beats. min^{-1} faster than the atrial flutter rate. The rate of atrial pacing is then gradually increased. When the typical negative atrial complex in lead II changes to a positive atrial complex, indicating capture by pacing, atrial pacing is either abruptly stopped or gradually slowed until the ventricular rare is considered satisfactory.

Constant rate technique

Facing is initiated at a rate 10 beats min^{-1} faster than the spontaneous atrial flutter rate. After pacing at this rate for about 30 seconds, pacing is either abruptly stopped or the pacing rate quickly slowed until the ventricular rate is considered satisfactory. If the maneuvers are unsuccessful in interrupting the atrial flutter, they are repeated with the initial atrial pacing rate increased in increments of 10 beats - min^{-1}.

Continuous Rapid Atrial Pacing

When atrial flutter is interrupted by the procedures just described hut recurs with unacceptable frequency, continuous atrial pacing at 100 to 600 beats min-1 is used. This results in continuing atrial fibrillation with variable AV block. The ventricular rate can then be controlled by digoxin.

When premature atrial beats are continuous or recurrent despite pharmacologic treatment, continuous atrial pacing at about 200 to 230 beats min-1 usually result in their suppression and a 2:1 AV conduction ratio with an acceptable ventricular rate.

Protocol for Control of Hemoconcentration

Causes:

In children,

Excessive capillary leakage

Excessive temperature elevation,

Tansient high cardiac output state, ultimately leading to metabolic acidosis.

If the hemoglobin level is ≥16 g.100 mL^{-1} and there is evidence of plasma leakage from the intravascular space.

1. Give 20 ml. m^{-2} of 25% serum albumin is a syringe slowly (over 5 minutes)>> if left atrial pressure (PLA) ≤ 15 mmHg.

   If PLA ≥ 15mmHg,consider administering furosemide (Lasix) and then albumin.

2. Repeat hemoglobin measurement in 1 hour.

3. If hemoglobin measurement in(step 2) , ≥16 g . 100 mL^{-1}, repeat (steps 1 and 2.)

Protocol for an Intubated Patient

1. The ventilator should be used with its air heating and humidifying devices and the valves for intermittent mandatory ventilation and positive end expiratory pressure (PEEP) functioning.

2. The patient to be well-positioned and well-secured orotracheal tube in place or, in infants and young children (for greater security and comfort) and adults in whom postoperative ventilation for more than 24 hours is likely, a well positioned and well-secured nasotrachcal tube in place.

3. Initially,

   Fractional concentration of oxygen (FiO_2) is set - 0.6,

   Tidal volume (TV) - 12 to 20 mL kg^{-1}, and

   Inermittent Mandatory Ventilation (IMV) at

   12 to 14 breaths - min-1 in adults,

   20 to 25 breaths min-1 in older children,

   30 breaths/min in young children, and

   30 to 40 breaths min-1 in infants.

End-inspiratory pressure should normally be <40 cm H_2O.

In all situations, visual, palpatory, and auscultatory observation of the patient's chest must be used to confirm that TV is adequate for good air movement in and out of the lungs.

These observations must be made whenever the patient becomes restless or agitated or there is any other reason to suspect inadequate gas exchange.

In patients with :

Chronic obstructive lung disease and

Glenn or Fontan operation >> PEEP of 5 to 10 cm H_2O (4 cm H_2O in patients ≤ 4 years old) may be used.

4. When the patient is admitted to the ICU,

Baseline blood gas analysis is obtained and
Ventilatory parameters are adjusted accordingly.

Continuous monitoring of oxygen saturation sould be measured in most patients.

[In adults, additional arterial blood gas analyses are not done routinely thereafter,unless there is a change in the clinical status of the patient.)

In neonates and children, more frequent arterial blood gas analyses are usually necessary.]

5. A supine portable chest radiograph is obtained upon arrival in the intensive care unit and reviewed by a physician for :

Placement of the tip of the endotractical tube;

Presence of pneumothorax,

Atelectasis,

Vascular congestion, or

Gastric distention; and

Size of the mediastinal silhouette.

The chest radiograph is routinely repeated in the first postoperative morning.

6. Turning of the patient and sterile suctioning of the airway are performed each hour to clear retained secretions and minimize atelectasis.

Suctioning is performed after hand bagging with 100% oxygen, hyperventilation for several breaths, and instillation of 1 to 5 ml. or sterile saline solution down the endotracheal tube

Suctioning is followed again by hand bagging with 100% oxygen. The length of the endotracheal tube must be known, so that the suctioning catheter can be passed with certainty beyond the tube into the trachea.

7. In patients without severe preoperative pulmonary dysfunction criteria for extubation include the following:

a. Patient awake and alert, indicating recovery from anesthesia and ability to protect his or her airway.

b. Satisfactory hemadynamic state

c. Absence of important drainage from chest tubes

d. Arterial Po2 ≥ 70 mmHg or SaO2 > 90% to 92% (in the absence of intracardiac right-to-left shunting)

On intermittent mandatory ventilation (IMV) of 6 breaths min^{-1} and Fio2 of 0.40

e. Spontaneous respiratory rate < 25 breaths . min^{-1} in adults,

<40 breaths min^{-1} in young children,<50 breaths.min^{-1} in infants.

f. Absence of increased work of breathing (use of accessory respiratory muscles)

g. Normal PaCO2 and pH (paCO2 may be somewhat elevated with a normal pH, if metabolic alkalosis is present).

Protocol for Oliguria

Defination:

A urine output in the early postoperative period of,

<0.5 to 1.0 mL . kg^{-1} .h^{-1} in infants & children

<0.5 mL - kg^{-1} . h^{-1} (30 to 35 mL . h^{-1}) in adults.

Rationale

To reverse the nearly universal occurrence of fluid retention following CPB.

Treatment:

1. Exclude low cardiac output as the cause of the oliguria.

2. Insert a urinary catheter if not already in place.

3. Administer a diuretic.

a. Furosemide (Lasix) : 1 mg kg^{-1} for infants and children and 20 to 40 mg for adults administered intravenously (IV) as a bolus. Usually, no greater diuretic response is elicited with higher doses. However, doses of up to 180 to 240 mg may be necessary in patients with chronic heart failure, cirrhosis, and the

Give Calcium chloride IV.: 10 mg · kg^{-1} for infants and small children

200 mg · kg^{-1} for adults

[to decrease the cardiovascular effects of hyperkalemia.]

5. If these measures do not result in a potassium level <5.5 mEq · L^{-1}, then Nephrology consultation should be obtained.

Protocol for Seizures in Infants and Children

General Comments

Generalized or focal seizures are an infrequent but potentially serious occurrence following cardiac surgery in infants and children.

Etiologies are :

Metabolic;

Infections;

Cerebral edema,

Embolism, or

Hemorrhage;

Decreased cerebral perfusion.

[In most patients no specific causative factor is identified.]

- Evaluate to identify possible correctable causes and
- Describe an initial treatment regimen
  - *Consult with a neurologist who is knowledgeable about cardiac surgical patients.*

The following generalizations are useful:

1. In infants and small children, whether the seizure is generalized or focal is not helpful diagnostically.
2. Respiratory arrest, discoordinate respiratory activity, or sadden inability to adequately mechanically ventilate can be an indication of seizural activity in infants. Additional evidence of seizures is usually present on detailed evaluation.
3. After initial control of seizures, anticonvulsant therapy should be continued through the recovery period. Decisions regarding long-term therapy are made by the neurologist or pediatric cardiologist before hospital discharge.

nephrotic syndrome.

i. The expected result is at least a doubling of the urine output over 2 to 3 hours.

ii. If the diuresis is inadequate, other diurites may be used in adults.

[Ethacrynic acid (Edecrin): 50 to 100 mg IV in 50 mL of solute over 30 minutes. (Ototoxicity occurs in 2% to 3% of patients).

Bumetanide (Bumex): 0.5 to 2.0 mg IV over 1 to 2 minutes.

Torsemide (Demadex): 10 to 20 mg IV as a bolus.]

iii. A continuous infusion of the diuretics may be safer and more effective in some patients.

[Dose: This is usually given after a bolus, if the diuretic effect is not sustained.

Furosemide is given at an initial infusion rate of 20 mg .h^{-1}, increasing to 40 mg . h^{-1} if necessary

Protocol for a Serum K^+ Level >5.5 mEq · L^{-1}

(in Acute Renal Failure)

1. Give glucose and insulin solution intravenously (IV).

For adults,

20 units of regular insulin

50 mL of 50% dextrose

(Give IV over 10 minutes.)

For children and infants,

0.5 mL of regular insulin per kilogram of body weight

2 mL of 25% dextrose per kilogram

(Give IV over 10 minutes.)

6. Give Sodium bicarbonate IV

For adults, 1 ampule (44 mEq) IV.

For infants and children, 1 mEq · kg^{-1}

4. If Potassium levels exceed 6.5 mEq · L^{-1} and

the patient is not receiving digoxin:

nephrotic syndrome.

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the patient is not receiving digoxin:

4. Most children having a seizure in the early postoperative period will not have a chronic seizure disorder.

5. Choreiform movements are more serious symptoms than are seizures and are more apt to persist.

6. Because of its potential to cause cardiorespiratory depression and its short duration of action, diazepam is best avoided as an anticonvulsant unless the patient is being artificially ventilated.

Initial Evaluation and Treatment

1. At the onset of a seizure, determin

Arterial blood gases and pH;

Serum glucose, calcium, and electrolytes;

Cardiac index;

Body temperature

2. Interventions are made in an attempt to correct:

a pH< 7.25 or > 7.50;

$PaCO_2$ < 25 mmHg;

PaO_2 < 80 mmHg; and

Base deficit > 10 to 15 $mEq.L^{-1}$.

(In some patients, prompt control of seizures will correct low values.)

b. Serum glucose level <40 $mg.dL^{-1}$ in infants and <60 $mg.dL^{-1}$ in order children)

c. Serum calcium level <7 $mg.dL^{-1}$ in infants and <8 $mg.dL^{-1}$ in order children.

d. Serum sodium level <125 $mEq.L^{-1}$. Usual management in this situation is restriction of salt and water intake

e. Cardiac index <2.0 $L.min^{-1}$, m^{-1}

f. Body temperature >38.6oC (>101.5oF).

Initial Anticonvulsant Therapy:

When seizures are first noted, steps are taken to terminate them or, if they are no longer present, to prevent their recurrence while the chemical and other variables are being determined.

1.Give

a. 0.1 to 0.2 mg . kg^{-1} of diazepam IV and

b. 15 mg . kg-1 of phenobarbital IV over 5 to 10 minutes as a loading dose.

2. If seizures are not controlled by these measures, additional doses of diazepam (if the patient is being ventilated) may be used.

(The effect of phenobarbital may not be apparent for several hours, but if problems continue at this stage, consider giving a further 5 mg.kg^{-1})

3. If there has been spontaneous termination of seizure activity and prevention of recurrence is desired, omit step 1a and proceed to step 1b.

4. Continuing major seizures will rarely be a problem. If a loading lose of phenytoin (Dilantin) (20 mg kg^{-1} orally) is given,

(followed by maintenance with 3 to 4 mg.kg-1 day^{-1} given orally.)

5. An alternative (especially when seizures interfere with effective ventilatory suppor) is paralysis with pancuronium.

Maintanance Anticonvulsive Therapy

The administration of phenobarbital (2.5 mg . kg^{-1}, 12 h^{-1}) can be instituted 12 to 24 hours after giving the initial loading dose.

Protocol for Intravenous Fluids

In adults and children (>2 years of age or >13 kg in weight).

1. Day of operation:

a. 500 mL of 5% glucose in water.m^{-2} 24 h^{-1}

b. 10 mEq of K^+ m^{-2} 24 h^{-1}

2. First and second postoperative days:

a. 750 mL of 5% glucose in water.m^{-2} 24 h^{-1}

b. 20 mEq of K^+ m^{-2} 24 h^{-1}

3. Third postoperative day:

a. 750 mL of 5% glucose in water. m^{-2} 24 h^{-1}

b. 250 mEq of 5% glucose in saline solution .m^{-2} 24 h^{-1}

c. 10 mEq of K+ . m^{-2} 24 h^{-1}

or 100 mL of 5% glucose in

one-quarter-strength saline

solution .m^{-2}24 h^{-1}

4. If oral intake has not been established on the third postoperative day, consider gavage feeding or intravenous (IV) hyperalimentation.

In infants and small children (<2 years old or <13 kg in weight).

1. Day of operation:

a. Calculate patient's saline requirement.

i. 250 mL . m^{-2}24 h^{-1} are required

ii. If this is 75 mL or less for the individual patient, use only balanced salt solution for flushing the arterial catheter,

if it is 75 to 150 mL, use balanced salt solution for flushing the arterial and left atrial catheters;

if it is >150 mL, flush the arterial, left atrial, and right atrial. (and, if present, pulmonary artery) catheters with balanced salt solution.

iii. Give no additional sodium-coating fluids if step ii supplies the patient's needs. Otherwise, subtract the amount in step ii from the requirement and give the difference.

b. Calculate the patient's water requirement.

i. 500 mL . m^{-2} 24 h^{-1} of 10% glucose are required.

ii. Subtract 72 mL the number of intracardiac catheters being flushed with 10% glucose in water from the amount in step (1), and order that amount.

c. Give no potassium in the IV fluids.

2. Days thereafter

a. Calculate patient's saline require ment

i. 250 ml.m^{-2} 24 h^{-1} are required

ii Proceed as in Ia

3. If oral intake has not been established on the third postoperative day, consider gavage feeding or IV hyperalimentation

[Note that when medications such as lidocaine, catheholamines, and sodium nitroprusside are administered, the amount of fluid thereby infused must be determined and subtracted from the daily fluid requirement.]

Protocol for Infant Feeding

Infants can rapidly develop a profoundly catabolic state after major surgery. Caloric intake should be raised to adequate levels as soon aspossible after operation.

- When the respiratory assistance via an endotracheal tube continues into the third postoperative day, gavage feeding is begun unless specific contraindications exist.
- In extubated infants, weakness and under development may prevent proper feeding and result in aspirant, intaking intermitant gavage feeding necessary.

The steps are as follows:

1. As a precaution, prepare the endotracheal suction catheter for immediate use.
2. Check to be certain that the masogastric tube is in the stomach, if intermittent gavage is to be used, a feeding catheter is inserted for each feeding and then removed.
   a. Aspirate the tube. If stomach contents are not obtained or if large quantities of air with a little mucus are obtained, the tube is probably in the trachea.
   b. While listening over the stomach with a stethoscope, inject a little air and listen for the typical noise.
   c. Persistent coughing suggests that the tube is in the trachea.
   d. Absence of a normal cry suggests the tube is in the trachea (steps a, b, and d apply to patients without an endotracheal tube).
3. If these checks indicate that the tube is in the stomach and if aspiration does not reveal >10 to 15 mL of fluid in the stomach, then initial feedings can be begun.

These feedings are injected slowly over 2 to 3 minutes or allowed to enter by gravity, preferably with the infant sitting upright.

much larger doses of NaHCO3 are indicated (44 mEq for adults, 1 mEq . kg^{-1} for infants and children)

Protocol for Severe Metabolic Alkalosis

Indication

Severe metabolic alkalosis exists if blood pH is > 7.60 or base excess>5.0 mEq. L^{-1}

Some surgeons use total base excess >50 mEq.

Total base excess (mEq) =

Base excess (mEq.1^{-1}).0.3 (l.kg^{-1}) .body wt. (kg)

Rationale:

Cardiac surgical patients who develop severe metabolic alkalosis are usually slow

to convalesce and resume normal alimentation and are frequently on moderate or large

diuretic programs. This complication may be more common in infants. The condition is associated with a volume-contracted state (dehydration), with potassium and chloride depletion, and, in its more severe form, with hypercapnia. The major complications of severe alkalemia include a leftward shift of the oxyhemoglobin dissociation curve, with attendant tissue hypoxia, peripheral and central chemoreceptor depression, hypoventilation, and hypoxemia; refractory cardiac arrhythmias; excessive myocardial contractility, with attendant increase in oxygen consumption; tetany; and altered calcium metabolism. Mild forms of metabolic alkalosis may be corrected with volume expansion and replacement of potassium and chloride. Severe alkalemia (pH 7.60) mandates aggressive therapy.

The administration of hydrochloric acid corrects metabolic alkalosis directly without dependence on renal or hepatic metabolic function.

(Complications of hemolysis and tissue necrosis are not a problem if central venous administration of dilute hydrochloric acid is used.)

However, because of the respiratory depression (CO_2retention and hypoxia) caused by metabolic alkalosis, the too rapid administration of hydrochloric acid may produce inappropriate hyperventilation and hypocapnia with an intracellular-extracellular hydrogen dysequilibrium.

With the concomitant correction of saline and potassium chloride

Otherwise, the infant is placed on his or her right side, with the head inclined to at least a 15ºangle.

4. The gavage feeding is given every 3 hours on the following schedule:

   a. Give sterile water, 10 to 15 mL

   b. If well tolerated, give 10% dextrose in water, 30 mL

   c. If well tolerated and if the residual is less than 5 mL, giveLanolac 30 mL. and,if well tolerated, full strength Lanolac or in increasing amounts.

5. If needed:

   a. Consider giving (27 calories per ounce) if diarrhea is not present.

   b. Consider continuous drip infusion to avoid a bolus effect (residual fluid in the stomach is aspirated and measured every hours).

Protocol for Metabolic Acidosis

Indication

Metabolic acidosis exists if the base deficit is>2 mEq . L^{-1} and pH is <7.35 or $PaCO_2$ is <30 mmHg.

Rationale

Treatment is directed only at the extracellular fluid, and a conservative dose of $NaHCO_3$ is given, because more can easily be administered if needed.

Extracellular volume - 30% body weight (kg)

Base deficit (mEq . L-1) .0.3.body weight (kg)

= total extracellular base deficit

Treatment

1. Administer $NaHCO_3$ so that the amount of Na^+ (mEq) equals half the total extracellular base deficit.
2. Remeasure the base deficit in 30 to 60 minutes and repeat treatment if indicated.

Note that in acute reduction of cardiac output or cardiac arrest,

deficits, intravenous (IV) infusion of hydrochloric acid is a safe and desirable therapy for severe metabolic alkalosis.

Treatment:

1. Use an internal jugular or right atrial catheter for infusion.
2. Prepare 0.15N hydrochloric acid in sterile water (12.5 mL concentrated hydrochloric acid (36%) diluted to a volume of 1000 mL with sterile water).

   (One liter of 0.15N HCl contains 150 mEq of H+ and 150 mEq of Cl-)
3. Determine the amount of hydrochloric acid required for correction of the metabolic alkalosis. Calculate the chloride deficit and the total base excess by the formulae:

   Cl(deficit = 0.3 body weight (kg) · Cl- concentration (mEq · L^{-1})

   Total base excess (mEq) =0.3 · body weight (kg) · base excess (mEq · L-1).
4. Administer the chloride deficit as 0.15N HCl over 16 to 24 hours.

   The maximum infusion rate should be about 0.2 mEq H+ · kg^{-1} · h^{-1}.

   The infusion continues until the base excess is within an acceptable range, that is, <5 mEq · L^{-1}.

   As a check, total base excess should be reduced to between 0 and about 1 mEq · kg^{-1}.

   (Infusion tubing should be changed every 12 hours and the acid infused from a glass container, because the effect of hydrochloric acid on plastic is uncertain.)
5. Correct the patient's volume deficit based on current and previous body weight by volume expansion with saline solution. Administer the maintenance daily IV fluids and sodium and potassium requirements and replace any prior deficits. Correct potassium deficit and maintain potassium level greater than 3.5 mEq · L^{-1}.
6. Monitor arterial blood gases and electrolytes every 4 to 6 hours and blood urea nitrogen and creatinine levels once or twice daily.

Protocol for Hyperthermia

Indication

Hyperthermia exists if rectal temperature is≥ 38.0°C (≥101°F)

Rationale

Hyperthermia increases metabolic demands and, thus, myocardial oxygen consumption. Severe hyperthermia (central temperatures ≥41.1°C [≥106 °F]) may permanently and severely damage the brain.

Treatment

1. If rectal temperature is ≥38.3°C (≥101°F) give acetaminophen as a rectal suppository every 4 hours. The dose in infants and children is 10 mg · kg^{-1} (rounded to the nearest 30 mg), and in adults, 650 to 1300 mg.
2. Consider using a cooling blanket or cold sponging and a fan or ice bags applied to the body.
3. If rectal temperature is ≥39.4°C (≥103°F)
   a. Insert esophageal temperature probe for continuous monitoring of central temperature and intensify efforts to improve cardiac output. Check for possible transfusion reaction.
   b. If esophageal temperature is ≥39.4°C (≥103°F)
      i. Make preparations for peritoneal dailysis with room temperature or cooled dialysale, to be initiated if simpler measures do not promptly control hyperthermia.
      ii. Give acetaminophen as in step 1.
      iii. Give dexamethasone. 0.25 mg · kg-1IV every 6 hours.
4. Give sodium nitroprusside. 1mg · kg^{-1} · min^{-1}, to increase peripheral heat loss if arterial pressure remains acceptable with this drug; if inotropic agents are necessary, give preference to amrinone and isoproterenol.
5. Abolish muscular heat production, particularly when an infant is restless, by paralyzing with pancuronium (0.1 mg · kg-1).
6. Continue efforts to improve cardiac output.

Protocol for Autotransfusion

1. Several commercial chest drainage systems that accommodate autotransfusion are available.

The two general types are

(1) those that transfuse shed blood directly from the drainage system and

(2) those that contain a drainage receptacle (a collapsible bag) that is manually transferred as an independent infusion setup.

2. A filter is optional.
3. Often there is a threshold for autoinfusion (that is, drainage > 100 mL in adults).
4. A time limit for duration of autotransfusion is set for 4 to 6 hours.
5. Reinfusion of shed blood is governed by:
   a. Amount of drainage
   b. Atrial pressure limits
6. With either system the total volume of chest drainage must be noted. This is a simple arithmetic sum of volume currently occupying the chest drainage reservoir plus the amount of chest drainage autotransfused. This total is noted hourly.
7. Contraindications to autotransfusion include:
   a. Infectious endocarditis or other infection
   b. Exogenous chemicals in the mediastinum
      i. Betadine
      ii. Antibiotics
      iii. Glue
      iv. Other chemicals
   c. Blood dyscarasias

Anticoagulation

The prevalence of stroke attributable to postoperative atrial tibrillation is unknown. It seems justifiable to initiate anticoagulation of atrial fibrillation persists longer than 48 hours or is recurrent in these instances, warfarin is recommended. If elective cardioversion is planned, intravenous heparin is recommended and is prescribed in therapeutic doses.

LEFT VENTRICULAR THROMBUS (LVT)

Etilogy

- Most common factor is IHD
- Acute MI (25-40%) develop LVT at the apex (akynaesia)
- May grow as a protruding mass
- Post MI aneurysm is susceptable for thrombus formation (MURAL), adhe -rent to aneurysmal wall
- Besides LVT may seen in Cardiomyopathoes,injuries,after valvular surgery

Diagnosis

- Invasive ventriculography are less sensitive in aneurysmal LVT
- But LV graphy is Fairly sensitive to identify protrudibg thrombus
- Non invasuve including CT scan are sensitive

Morphology

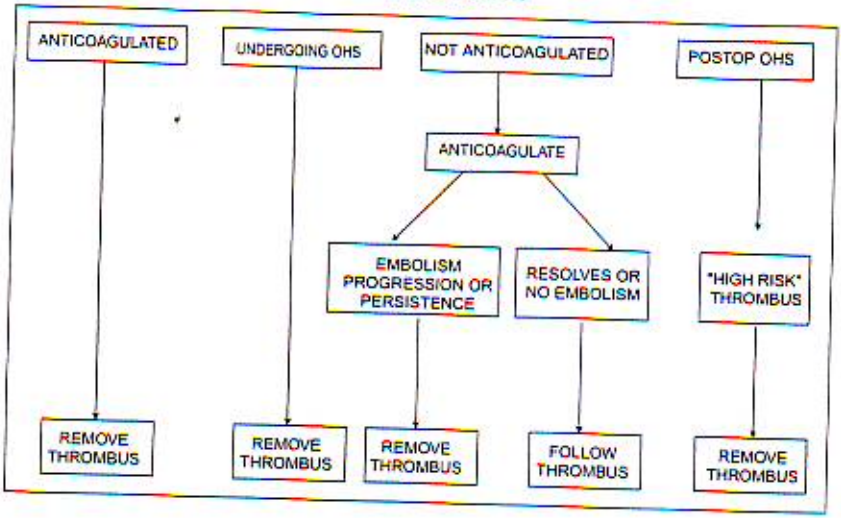
- Flat /Mural thrombus- Adherent to the wall & less embolization potential
- Protruding/Pedunculated thrombi are potentially exposed to high velocity & heigher rate of imbolixation
- Mobile protruding thrombi- have heighest patentiality

FATE

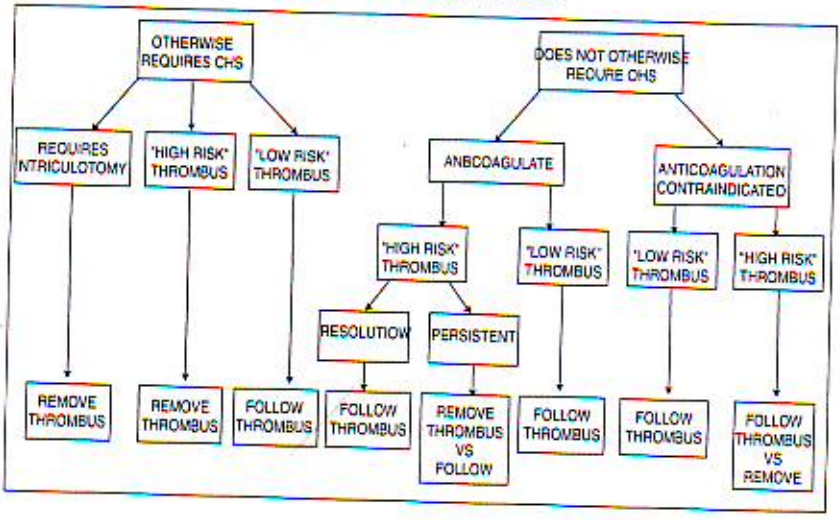
- Majority specially mural resolves with outout embolization
- Embolization happens (irrespective anticoagulation) in mobile & protruding thrombi
- LV aneurysm & ventricular thrombi rarely emblolize.May be the shear forces in the cavity is not sufficient to dislodge the clot in aneurysm
- Indication Surgery indicated in reurrent embolization despite anti coagulation
- Embolization in presence of anticoagulation is medical failure & surgery is advised with resonable low morbidity /mortality

- If managed medically serial followup is necessary & when shows pedunculation surgery is advised

Flow Chart
LVT & Embolism



Flow Chart
LVT without Embolism



MANAGEMENT OF COMPLEX CONGENITAL HEART DISEASES: SINGLE VENTRICULAR HEART

SINGLE VENTRICULAR PHYSIOLOGY
FORMS OF UNIVENTRICULAR AV CONNECTION :

- Tricuspid atresia
- Double inlet Ventricle
- Atretic AV
- Aortic Atresia& Hypoplastic Lt.Heart
- Pul.Atresia with Intact IVS
- Atrial Isomerism(2 circulation difficult)

Most Patients with SV need Staged Palliation

- 1st stage: Neonatal Period
- 2nd Stage: 3m ~ 1y (SCP Shunt) for deloading
- 3rd Stage:1y~5y (Fontan)

INDICATION

- One Ventricle with suficientnAV connection& power to supply 2 circits-Flow needed
- Discordant/Concordant AV connection with small one ventricle to support
- Adequet size 2 ventricles But with complex Great artery relationship & VSD –Difficult to separate circulations
- Hypoplastiv RV(<30% normal size) does not support pulmonary circulation
- 1 & $\frac{1}{2}$ Ventricle Repair – RV >30% but needs unloading for failure (to avoid Fontan procedure)

Other Situations

- Unbalanced AVSD
- Moderate Rt / Lt heart hypoplasia
- DORV with uncommited VSD

- TA ē VSD ē Underdeveloped RV
- PA ē intact septum ē RV hypoplasia/dysfunction
- Ebstain ē moderate RV hypoplasia/dysfunction
- Straddling Av ē discordant heart (inlet/outlet) ē VSD,PA/PS
- TOF ē Rv dysfunction – 1 &1/2 possible
- No definite criteria yet available in these complex morphologies for intracardiac repair
- No data of long term superiority exsist of BiVentricular repair, Fontan, 1&1/2 repair or transplant

Neonatal Palliation

1st stage
Aim:

- To balance Qp/Qs
- To ensure unobstracted shunt in atrial level
- To ensure unobstracted Cardiac Output

CONCORDENT V-A CONNECTION

Presentation : Pathophysiology

- Low pulmonary Flow
  - ❖ Reduced Qp
  - ❖ Duct dependant Qp
  - ❖ Obstracted at/below PV
  - ❖ Palliation (Shunt) indicated
- Excessive Flow
  - ❖ No obstaraction
  - ❖ Increased Qp
  - ❖ Palliation (Banding) Required
- If Qp/Qs well balanced –avoid Palliation

  - follow up necessary to identify resistance accross VSD resulting decrese flow to lung-(Shunt indicated)

Timming of BANDING

- With Normaly decresed Post natal Rp – If presented with elevated Qp Banding indicated

DISCORDANT A-V CONNECTION

MORPHOLOGY:

- PV in fibrous continution Of MV
- PV from LV
- PV obstraction unusual
- Qp increase is rule
- Aorta from hypoplastic RV
- LV output through VSD>RV>Aorta

Management

- If no obstraction of LV output-Management similar to concordant subset with excess (Banding)
- If obstraction is present either:

  *Subaortic-Due to restricted VSD/Hypoplastic RV

  *Arch – Coarctation,Intrrupted arch

Problem of Banding > Increases the obstraction later in life(as reduces volume load)

VSD equal to Aortic orifice is important to evaluate before - Banding/BT Shunt/Fontan

Operations

- Banding & Coarctation Repair
- PA to Aortic Connection with Arch repair

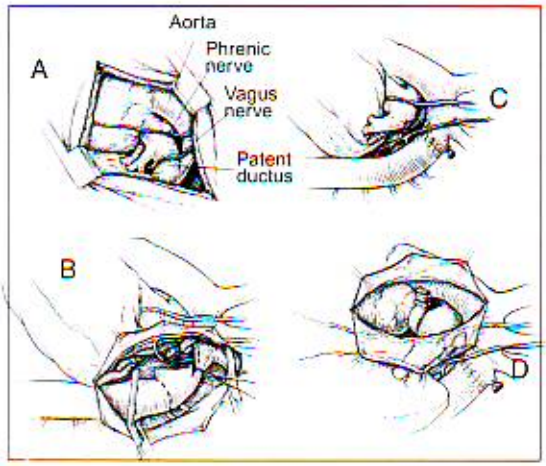


Fig 1. Banding & Coarctation Repair

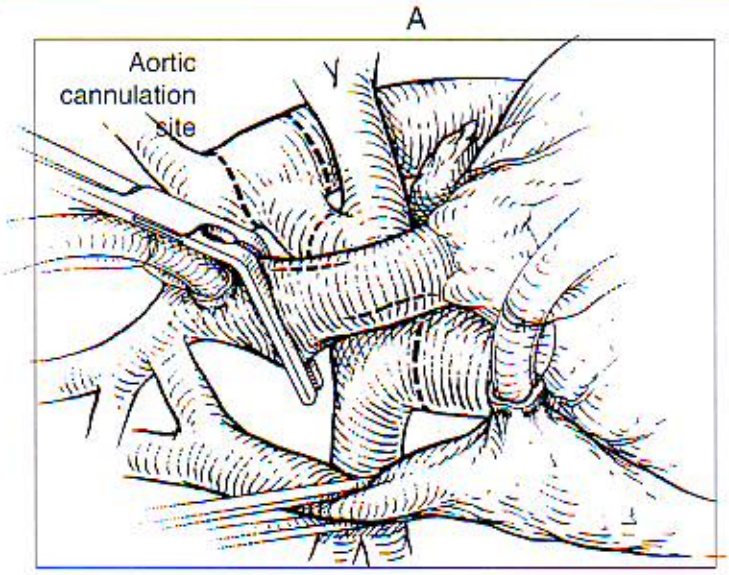
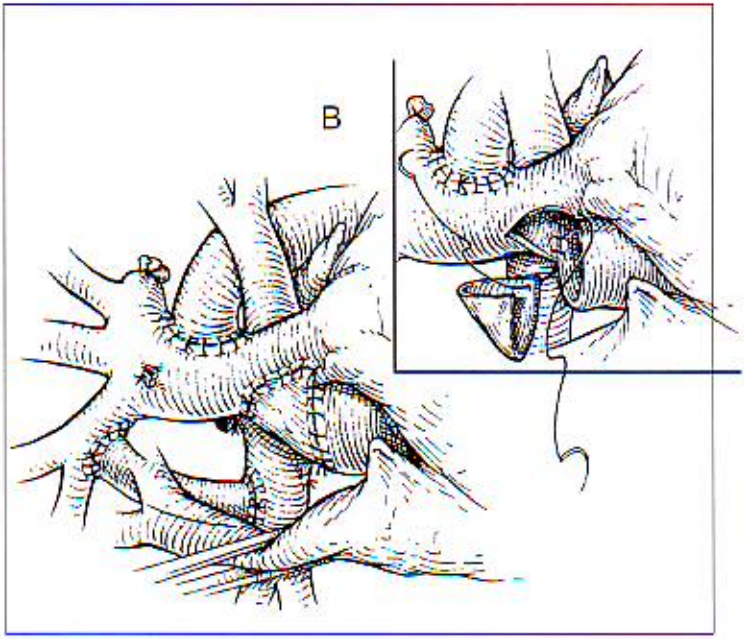


Fig 2



2nd Stage Palliation

Aim

- (1) To eleminate in efficiency of mixed circulation as early possible
- (2) To correct morphologycal abnormalities before Fontan

(1) Elemination of mixed circulation

- Partial seperation by Shunts:
  - » Cavopulmonary (CP shunt)
  - » HemiFontan

Advantage:

- Improves saturation
- Reduces work load of single ventricle
- Preserves myocardium (Important prediction of Fontan)
- Increases Diastolic pressure & Coronary circulation (For removal Of B T Shunt)

CONTRAINDICATION IN NEONATAS

- Elevated Rp
- Possible after 1-2 months when Rp falls to normal
- Indicated at 2-2.5 months (Mortality less than after 6m.)
- Morbidity Related to ;
- Cyanosis than elevated Rp
- Poor ventilation-Perfussion match
- Response to CPB

(2) Correction of Morphology

- Early removal of BT Shunt
- PA hypoplasia ,stenosis
- AV regurgitation
- Sub aortic obstraction, Arch obstraction
- Restrictive ASD,PAPVD

Cardiac Cath

- Priror to 2nd Stage to-

  Evaluate pulmonary circulation

  Rp (Critically important to advice CP shunt)

  2nd Stage Palliation

Characteristics:

Pt have CP shunt /HemiFontan

IVC blood directed to PA

Only CS & Pul Vn blood to RV

Complete seperation of Pulmo/Syst circulation

Some surgeons do Fanestrated Fontan > ASD to fascilitate mixing

Pre Op condition

- Any Syst-Pulm shunt to be taken down
- Other morphologycal anomally to correct at the time of Fontan(PA stenosis,TV regurge,MR,Obstraction of outlet,Fistulae)

v If 1st ,2nd palliation or both not done than CP shunt to be done at FONTAN

Pre Op Preperation in Neonats:

- Should be cardiopulmonary Stable
- Breathing sponteneously with little medical support(PE1) .
- If uncompenseted pulmonary plethora / Cyanosis present > Aggressive management for stabilization (PE1, Inotropes, Diuretics, Ventilation, Nutritional support,Others)

FOLLOW UP NECESSARY TO PREVENT ORGAN DAMAGE BEFORE OPERATION

OPERATIONS

B T SHUNT

- Monitoring : Internal Jugular, Femoral Vn to be avoided
- Incission : Preferded MidSternotomy

Advantages: Both lung ventilated

- Shunt can be placed more proximal PA (to avoid upper lobar Artery stenosis)
- Maximum Flexibility
- Duct ligation/PA plasty possible
- CPB conversion easy

- Disadvantages: Re–do is difficult (can be minimized by opening upper part of pericardial cavity.

- Heparinization 3mg/Kg
- Attention of site (Angle of take off of Innominate)
- No bleeding at PA end
- Make 90˚anastomosis at PA end
- Haemodynamic adjustment:

❖ Attention to SaO2 changes

❖ Judge with base line value

❖ SaO_2 75-85% acceptable> If low –Inadequet shunt/Distal artery problem

> If High - Large shunt (Associated with Diastolic pressure <25-30mmHg)

After Stabilization PDA should be ligated/ PE1 Stopped

Pulmonary Trunk Banding

❖ Incissions: Mid sternotomy / Lateral Thoracotomy / Para sternal Incission on left side

Points of Attention:

❖ Pericardium not oppened over ventricular mass

❖ Tissue plan developed in midway between Sino tubular junction of PA & origin of RPA

❖ Aggresive dissection avoided- to avoid migration

❖ Width of band should be broad (>2.5mm)

❖ Ideal band 3mmX0.3 silicon rubber

❖ Attach to adventitia to prevent distal migration

❖ Gradiebt between Distal PA & systolic Pr. to be measured

❖ Gradual tightening of the band & asses Gr.& Sa

❖ Typical Gr. (in neonats) >40-70mmHg, SaO2 >75-85%

❖ Small change in band circumference changes markedly the Qp

❖ Qp depends on systolic pr

❖ Important for Anaesthesiologist to keep BP at awake lebel (Drug,Volume)

❖ Pacing

POST OPERATIVE CONSIDERATION

B T Shunt :

- Not necessary to reverse heparin/Some advocate Heparine for 24 h (3mg/kg)
- Maintain BP heigher 10-20% than normal
- Inotropes may be indicated to raise BP for better flow
- At 1st night Aspirin (1mg/Kg/day)per rectal
- Some degree of haemodynamic instability & Metabolic acidosis for few hrs. Correection important contd..
- Important to support 1st post Op day with ventilation & Observation
- Watch renal failure (cyanosis).Ensure diuresis
- Chest X ray in ICU immidiate & after 4H to see dense opacification due to haemorregic oedema,cyanosis .> Returne to OT to reduce flow
- Auscultate shunt murmur.If doubtfull patency,ECHO/CINE (if cyanosis not imroved).
- If not functioning >Return to OT to correct shunt

Post Op Consideration

PA Banding:

- Rp decreases after band
- Reoperation needed to tighten further

Result:

BT Shunt:

- Early mortality low
- 5 yr survival with out any thing 90% ,10yr 85% (without Fontan)
- Risk of dyiung heigst in 1st few months
- Risk after 5-10 yrs due to cyanosis(for restenosis & LV myopathy for overload)

Result

PA Banding:

- Mortality in current era <5%
- Early mortality 25-35% due to difficulty in Balabce of Qp:Qs

- Outcome influenced by Anatomy
- Unfavourable in Discordant anatomy (develop subaortic stenosis)
- Before or after FONTAN Sub Aortic stenosis is risk of death (Ventricular Hypertropy /Compliance)

INDICATIONS

BT Shunt :

- Severe Cyanosis (< 70~75%) or Duct dependency

PA Banding :

- Qp large to produce to Heart Failure
- If not sufficient to produce important Heart Failure in early life

Timming :

- If early at birth Rp is high – Tightening is limited by cyanosis
- IF Rp gradually decreases later after band the –Retightening needed
- Repeated Re-do can be limited by appropiriate timing
- Best time 2~4 wks of life
- Low Rp High Qp is ideal situatuion for balance circulation

B.T Shunt :

- Anatomical Variation : Individual Anatomy
  - Rt.arch
  - Situs Tnversus
  - Isomerizm
  - Abnormal Arch banding
- Site : Pulmonary Trunk Segment(to avoid distortion of PA)
- Size : Important determinant of Qp
  - Diameter of Graft(3.5mm in 3.5 kg ideal)
  - Site of the artery
  - Length of graft
- Small PA (High resistance), In large Infant >Tube anastomosis to aorta that brances (resistances reduces across connection)
- In low Rp(Small Infant) > Subclavian best

Types of : Systemic- Pulmonary Shunt

- Ballock-Taussig Shunt(Subclavia-RPA)

- ❖ Watersons Shunt (Ascending aorta-RPA)
- ❖ Potts Shunt (Deascending- PA)
- ❖ Central Shunt (Ascend-Pul.trunk)

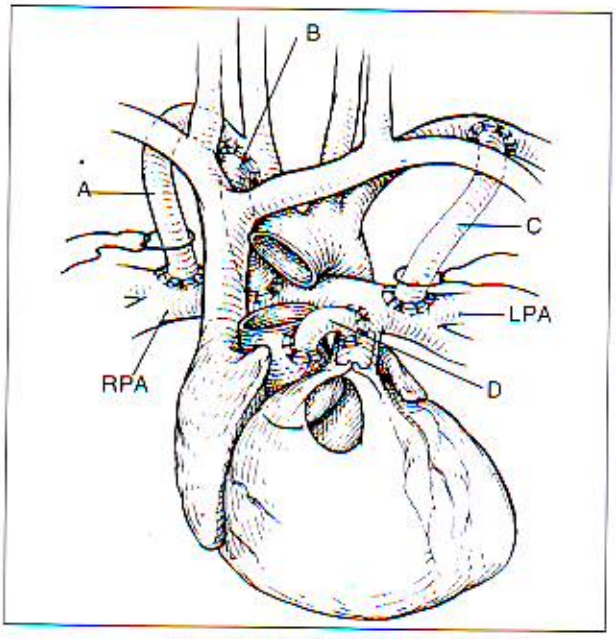


Fig. B T Shunt (A, B, C) Central (D)

Careful evaluation of Pt. size / Pil; .anatomy /Size of tube

Other Palliations:

- Atrial Septectomy (in mitral atresia) with PA Banding / B T Shunt
- Classic Glenn Operation:
  - Low mortality (5%) in > 6m/Children
  - Fascilitate Fontan (1/2 done already)
  - Intrruption RPA disadvantageous
  - Long tern result for pt. not fit fpr Fontan is good (Patency also satisfactory)

Long term result :1) Alteration of Upper/lower lobe flow

2) A-V fistulae lung

2nd Stage Palliation

Aim : *1.Eliminate inadequecy of mixed circulation-*

* Partial Separation of 2circuits

* Early removal BT Shunt

2. Correct/Eleminate existing morphological defect before Fontan

Partial Separation of Two circuits

Methods:

- Superior C-P Shunt
- Hemi Fontan

Benefits:

- Improve Gas exchange
- Eleminate /Reduce flow BT/Banding
- Decrease workload of Single ventricle
- Preserves myocardium(Important for Fontan or not)
- Sao2 remains > 80%
- Diastolic runoff not present improves coronary circulation

Indication: C P Shunt

- 1-2 m when Rp drops to normal
- Limiting factors for mortality
  - Lower age group
  - Mismatched Ventilation Perfusion
  - Exaggerated responnse of CPB

Early Removal of BT Shunt

❖ With Correction of Other Malformations

- PA Hypoplasia
- Stenosis
- Arch anomaly
- Sub aortic obstraction
- TV regurge
- PAPVC

TO BE ADDRESSED CAREFULLY TO PRESERVE CIRCULATION & SIMPLIFY FONTAN

BI DIRECTIONAL CAVOPULMONARY SHUNT

- Echo:To asses morphology
- Car.Cath: PA morphology, Rp (critically important value to advice

CP Shunt)

PROCEDURE :

- Pt usually with BT shunt/Banding
- May be using CPB
- With out CPB
- MPA Pr < 15mmHg & Pulse Pr PA < 5mmHg
- If extra source to be kept-Narrow BTShunt/Readjst Banding

Hemi Fontan

(RA-RPA anastomosis)

- Similar CP Shunt Physiologically
- Requires hypothermic arrest
- If future Fontan if extra Cardiac,no benifit

Post Operative Care & RESULT

- Agrresive pulmonary toilet
- Maintain Hct at 45%

RESULT :

CP shunt :

Hospital mortality 5-10% Survival :

- Long term data not available
- 1yr survival -90%
- 5yr survival -80%

Factors for Mortality (CPS)

- High PA Pr.
- TAPVC
- RV morphology
- Very Young age

❖ Age 0f 2-3 month & appropiate selection not factor for death

❖ Fascilitates more than Classic Glenn for FONTAN

❖ If FONTAN NOT DONE LONG TERM RESULT LIKE CLASSIC GLENN

❖ HemiFontan

- Mortality 5-10%
- Fscilitates lateral tunnel Fontan

INDICATION :

2ND stage

- Not absolute
- Beneficial
- Indicated in most patients with single ventricular physiology (3-6m)
- Both CPS / HemiFontan same physiologic result
- CPS is befitting for extracardiac Fontan
- HemiFontan for lateral tunnel fontan
- CPB absolutely necessary if Sys. Pul shunt needs to remove to connect CPS shunt(Rt side BT shunt when present)

3rd Stage Palliation

- Pt having CP shunt/Hemi Fontan
- Morphology & Haemodynamics should get attention
- Any Syst-Pulmonary connection to be disconnected
- Distortion/Hypoplasia/AV regurge/Obstraction of systemic circulation/ Abnormal A-V connection to be corrected

Pre Op Evaluation

- Card.Cath : 2 reasons
- Diagnostc study for morphlogy that can not be done by ECHO
- Distal Pulmonary Artery
- Haemodynamics: Arterial filling pr.,EDP,PA pr,Qp,Qs.Rs,Rp >> to select appropiate case for Fontan
- Interventional correction of the lessions (Stenting / embolisation etc)

INDICATIONS

- 15 Kg (2mm diameter tube)
- Rarely before 1 yr
- Typically not before 18-24 m (1.5-2 yrs)
- Rp <4 um^2

- Rp 2-4 um^2 – SCP shunt indicated than if pulmonary vascular bed is competant

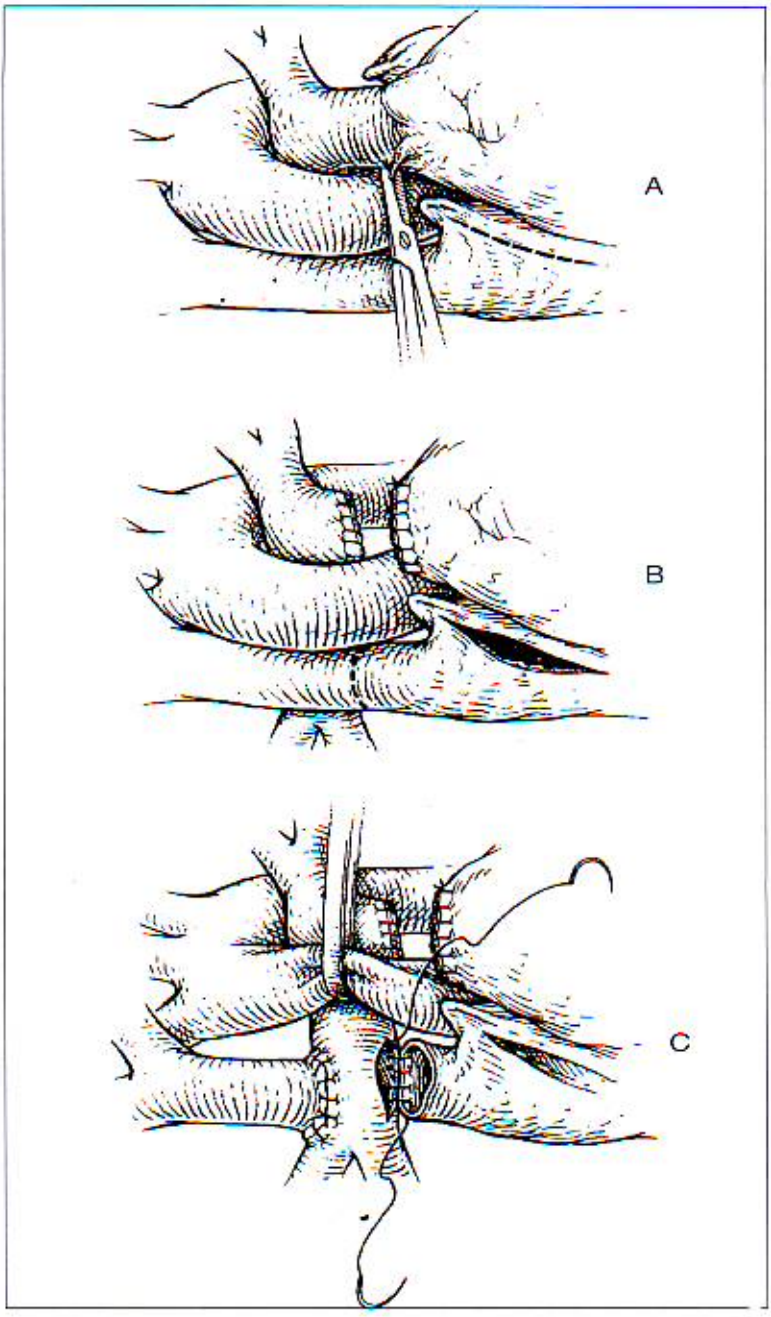
❖ small RPA,LPA contraindicated (High Rp) – Only atrial shunt feasible

- Narrow segment of RPA,MPA not contraindicated (Can be corrected)
- EDF >15 mmHg, EF <45% contraindicates Fontan
- FONTAN TO BE PLANNED FROM Ist palliation, not uncoordinatedly

Fontan Procedure

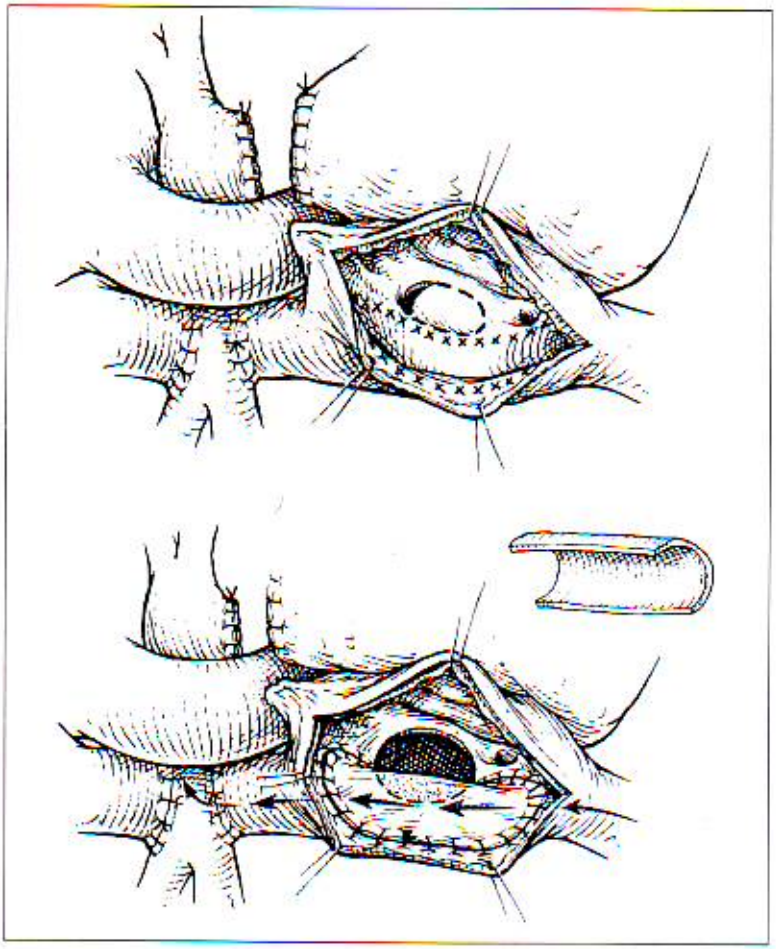
EXTRA CARDIAC :

- Pt uderwent Neonatal & 2nd stage palliation
- Median sternotomy
- Femoral Vn. Cannulation of IVC
- Upper anastomosis can be done without CPB
- L PA is clamped
- Oblique clamp is applied on SCP shunt
- Pulmonary Trunk disconnected,if not done(as in Hemifontan)
- Inferior surface of RPA anastomose to a tube (PTFE), 2cm in > 25Kg / 2.2–2.4cm in >20Kg
- If instability anticipated CPB strarted
- Evacuate graft & restablish CP shunt
- If CPB not used centrifugal pump to decompressed IVC is used
- If CPB used intraposition of IVC necessary for anastomosi
- IVC to RA shunt is alternative


A
B
C

Fontan : Lateral Tunnel

- Mid sternotomy & similar CPB preperation
- Any Vn. cannulae are apt to thrimbosed & should be removed after OT
- Pursting suture on Vn to be elliptical to avoid narrowing
- Hypothermia 25°c
- Pulmonary trunk dissected away
- Atrium opened & tunnel with patch graft biult
- Heart closed deaireated as usual



Peroperative Maneuvers

- Fontan pathway(FP) Pr,LA & PA pr monitoring is important
- If FP Pr high but not the LA/PA pr than Obstraction in FP
- FP Pr should not be > 16 mmHg.If so re operation needed

SURVIVAL

- Early mortality in specialized centre <5%
- Staged operation 1-5 yr survival recently 90%
- Survival decline lately
- These suggest Fontan is excellent Palliation not curative one.
- Multiple morphological/Functional factors for death exists like BDG

MECHANICAL CIRCULATORY SUPPORT

A means of imparting energy for forward flow of blood by artificial devices.

A. Intraaortic ballon pump(IABP) - Percuteneous
Transthoracic

B. Ventricular assist devices(VADs) – Left,Right
Biventricular, Implantable
Paracorporeal
(Partial implantable)
Power Source: Electric
Pneumatic
Type: *Pulsatile
*Non pulsatile

C. Others- (ECMO,CPB portable system)

HISTORY

- 1920-Brynchonenkho first attempted artificial circulation
- 1940-In Russia reported
- 1953- HEART-LUNG MECHINE INTRODUCED
- IABP-Invented in 1962
- 1968-Clinical introduction
- 1982 –Jarvic – 7(VAD) was introduced(Pneumatic,Parmanant)
- 1990 -VAD USED AS TEMPORARY BRIGE TO TRANSPLANT

BI VENTRICULAR ASSIST DEVICEs (VADs) -.TEMPORARY

- Uses:
  1) After Surgery
  2) Bridge to transplant
  3) Bridge to recovery

May be used with IABP

FDA approves 5 devices :

1. ABIO MED BVS5000	Pneumatic	Temporary
2. Thoratec VAD (Rt/Lt)	,,	,,
3. Thoratec Heart Mate (IP1000LVAD)	,,	Parmanant
4. Novacor	Electrical	,,
5. Thoratec Heart Mate (TCI LVAD)	,,	,,

All are pulsatile flo

VADs – Parmanant

VADs - Parmanant

- Paracorporeal : Thoratec VAD (PNEUMATIC)
  LV / RV or both
  Untill recovery LV
  Bridge to transplant
  TO PLACE OVER ABDOMINAL WAL

VADs -Parmanant

- Implantable :
- Pneumatic - Thoratec Heart mate (IP1000 LVAD)
  long pipe line
  out side dreiver console
- Electrical -Thoratec Heart mate (TCI SVE LVAD)
  - Novocor

Use : Normal Life style
Long wait

Total Artificial Heart (TAH)

- Cardiowest C-70 Total artificial heart :
- Ortotropic bi ventricular pneumatic pulsatile blood pump is used to replace Heart. This replaces both the ventricle and use natural atria for inflow.
- New systems of TAH are entering in trials(AbioCor-electrohydrolic, PennState / 3M Electric TAH-electromechanical). Primarily used as a bridge to transplant for patients

- With biventricular failure & high pulmonary resistance or intractable ventricular arrhythmia generally with life expectancy of under 48h.
- Several systems are under development and promising

Counter pulsation : IABP

- Physiology & mechanism :
- IABP only augments cardiac function by reducing afterload & increasing diastolic pressure.This is contrary to VADs that can completely replace pumping function of failing heart
- Basic Strategy to use devices:
- Mostly IABP is firstly used.Can be put in ICU.
- If IABP +Pharmacological support fails adequet tissue perfusion _ LVAD indicated

INDICATIONS:IABP

- Post cardiotomy cardiogenic shock (inability to wean from CPB)
- Cardiogenic shock after AMI,unresponsive to medical therapy

  Primary myocardial dysfunction

  VSD

  MR(papillary rupture)
- Unstable Angina

  Pre MI & Post MI

  Failed angioplasty- travelling to operation
- Ventricular tachyarrhythmias caused by ischemia
- Bridge to transplantation
- High-risk cardiac patients undergoing general surgery
- Adjunct to mechanical ventricular assistance

CONTRAINDICATION

- Aortic Regurgitation
- Dissection
- Thoracic aneurysm

- Peripheral Vascular Disease(severe)
- Blood dyscarasias
- Irreversible Brain Injury
- End stage Ventricular failure

IABP : COMPLICATION

- Limb Ischemia

  Most common (5-19%)

  Related to cardiac output, catheter diameter,intimal injury,thrombosis.

- Perforation –Common in shock,PVD

  Sup.Femoral Artery – thrombosis,leg ischemia

  Abdominal vessel - Retroperitoneal haemorrhage

- Incorrect position

  Viscral ischemia,Aortic insufficiency

- Aortic dissection (<5%)-Usually retrograde ;often seals with own
- Wound complication (1-3%)
- Catheter failure (Gas escape)

VADs / TAH:

- When function of heart remain inefficient inspite of ballon,inotrops use of VAD may be considered
- VADs takes over the function of heart but ballon only optimize cardiac function

Cardiac Transplant & Devices

- Carrel & Guthrie reported heterotropic dog transplant
- World War II revolutionczed the understanding of transplant, developed the concept of immunology in transplant
- Laboratory work started in standford in early 1950 of cardiac surgery
- Late 1966 in Cape town Barnard(a trainee Lillehi,Minnesota) done successful human transplant

- Lack of prevention of rejection technology & poor out come soon leads to disappearance from clinical practice
- With the development of immunosuppresive therapy reappeared again in 1980s as a modality

Histocompitability

- ABO compitability for Donar,recipient are done
- 3 procedures are used:

  HLA typing-Lympho + antisera HLA-A,B,C & some times A2,A3

  WBC Antibody screen-Also lymphocyte toxicity test for antibody

  Lymphocyte cross match-Lympholytic test for hyperacute rejection

Indication

- Cardiogenic shock with Mechanical support & reversible organ damage
- Refractory LOS with support
- NYHA III-IV with progresive functional loss
- Recurrent failure
- HCM / DCMyopathy (NYHA IV)
- Refractory angina,not amenable to CABG / TMLR
- Recurrent threatening arrythmias
- Cardiac`tumors
- Hypoplastic Lt Heart
- Complex CHD not amenable to correction

DONOR

- Brain death patients
- <55 yrs
- CAG should be performed if >45yrs
- Should have no evidence profound hypotension,arrest after injury
- Cardiac contusion of injury or wall motion abnormality are disqualification

- Eveidence of sepsis,malignancy ,hepatitis disqualifies donor
- Cardiac ischemic time <180 min

Ischemic time

- Time required to remove the donor heart after aortic clamping,transport the heart to recepient's OT,suture to recepient,release of recepient cross clamp & start of aortic root perfusion
- Donor Ht.trimmed& carried in ice chest for transport

Surgical Technique

- Classic technique
- Bi Caval anastomosis technique

  Bi-Caval is more anatomic

  Counter probable atrial enlargement

  Better atrial function & less TV regurgitation

  Better haemodynamic performance

Rejection Episodes

- Major part of cardiac transplant – to recognise rejection
- Endocardial biopsy every 7th day for 1-2 months :

  Unexplained fever

  Jt.pain

  Personality changes

  S/S of Failure are to biopsy & to start treatment

Other Complications

- Infection
- Nephrotoxicity(drug)
- Glucose Intolerance
- Hypertension
- Hyperlipidemia
- Osteoporosis
- Malignancy

- Biliary stones
- Impotency

Heterotropic Transplant

- Primarily as assist device to LV
- Method for some Pts. of Pulmonary Hypertension
- Very limited use

Xenotransplant

- Shortage of allograft lead
- Rejection starts within minutes
- Number of scientific & ethical barrier remain
- Remain a dream yet to other sources

Alternatives

- CABG – done in compensated heart,EF >20% with accepatble risk against a good cardiac function–If EF <20% (decompensated ht.) recommendation for CABG rathe than transplant should be careful
- Valve Operation – for Myopathies
- Aneurysmectomy /Ventriculectomy
- Ventricular Devices:

  -Myosplint

  -Prosthetic Jackets > to prevent remodeling

  (Both of Preliminary human use)

- Direct Cardiac - aortic device:(CardioSupport system,Abiomed ,CARDIOVAD) Cuff sorrounds heart & aorta for compression.Principle similar to IABP -simple to use & remove

  -No clinical use yet

- Dynamic Cardiomyoplasty-

  -Latissimus dorsi electrically stimulated to augment function wrapped arround

  -Not yet routine use in HF

  -Under experimentationwith high feasibility

- Bi Ventricular Pacing: Simultineous stimuli of LV, RV

  -Prolong PR activates base eariler to apex,lateral wall resulting paradoxical motion in HF

  -Pacing with AICD appears to be future direction of HF treatment

MECHANICAL VALVES : ANTICOAGULATION

Anticoagulation

Anticoagulation Issues

Why is AC necessary for prosthetic valves?

- All prosthetic valves = foreign body
- Pathophysiological response - encapsulate foreign body with thrombus & exclude it
- Thrombus adherence & possible resulting Thromboembolism (TE) influenced by:
- Patient-related Factors
- Surgical Injury
- Prosthesis-related Factors

Thromboembolism

Patient-related Factors

- Old age
- Poor cardiac output
- Atrial fibrillation with enlarged left atrium
- History of previous embolis
- Prenancy
- Oral contraception
- Cigarette smoking
- Hypertension
- Diabetes
- Coagulation abnormalities

Surgical Injury

TE risk is the highest the first postop month

- Blood contact with injured native tissue & prosthesis
- Blood flow changes (prosthetic turbulence & stagnation)
- Coagulability changes after surgical injury
- Prosthesis-related factors
- Type of prosthesis (increasing risk)

- Homografts, autografts, stentless < Stented biological < mechanical
- Number of implants
- Multiple implants double the risk of TE
- Position
- Mitral > aortic
- Valve thrombosis 5x higher in mitrals
- TE 2x higher in mitrals

Anticoagulation Issues

- Thromboembolism
- Prosthesis-related factors
- Sewing Ring
- Microthrombi > avascular fibrous tissue endothelial coverage > vascular fibrous tissue growth
- The higher the local pressure on the sewing ring the more limited the thickness of avascular tissue.
- aortic valves & ventricular side of mitral valve have less
- than atrial side of mitral valve
- Non-physiologic flow patterns - stagnation, turbulence, high shear stresses
- Highly polished surfaces - insecure attachment of thrombus
- Thrombus location
- Ball & cage - at apex of cage
- Tilting disc & bileaflet - at struts & hinges

Coagulation

Cascade Mechanism:

INJURY

VITAMIN K

(provided by intestinal flora)

Stimulates Prothrombin Manufacture

INTERACTION WITHIN FIBRINOGEN

FIBRIN MANUFACTURE
CLOT FORMATION

Anticoagulant Medications

- Anticoagulants also known as "blood thinners"

  Anticoagulants do not thin the blood but reduce the bloods ability to clot by blockingor reducing protein components required for normal coagulation .

Anticoagulation Medications

Types

- Heparin
- Coumarol derivatives
- Warfarin sodium - Coumadin ®
- Phenprocoumon - Marcumar ®
- Acenocoumarol - Sintrom ®

Antiplatelet drugs

- Aspirin
- Dipyridamole - Persantine ®
- Triclopidine
- Heparin
- Administered intravenously (immediate effect) or subcutaneously (20-60 minutes)
- Dosage dependent on body weight (IU/kg)
- Given during OR to prevent clotting in the extracorporeal circuit (heart-lung machine) and postop to initiate AC therapy
- Antedote = Protamine sulfate
- Coumarol derivatives
- Discovered in 1920's - cattle disease causing bleeding - caused by improperly cured feed = sweet clover
- Dicoumarol identified as hemorrhagic agent
- New coumarins synthesized in laboratory

- Warfarin 5-10x more effective than dicoumarol
- Given orally absorbed by stomach & small intestines
- Antedote - vitamin K or blood transfusion

Initiation of AC Therapy

- Two schools of thought
- Wait until chest tube bleeding has stopped (1-2 days post op) then start oral AC
- Immediate postop with overlap therapy (Baudet)
- Increasing number of centers are using the Overlap Therapy
- Initiation of AC Therapy
- Overlap Therapy - Dr. E. M. Baudet, Bordeaux, France
- Stage 1- Intermittent IV Heparin-start 6 hrs postop then every 4 hrs (WBCT & TE risk)
- Stage 2- Subcutaneous Heparin-start 24 hr postop then every 8 hrs til APPT 1.5-2.0x control
- Stage 3-Oral AC-start 48 hrs postop & maintain s.c. Heparin til PT 1.6-1.9x control,APPT 1.5x control, Fibrinogen level <4g/l

E.M. Baudet, Anticoagulant therapy for aortic prosthetic valves- a French perspective, in Surgery for Acquired Aortic Valves Disease, ed. A. Piwnica, Oxford, ISIS Medical Media, 1997, pp. 304-310

Additional Drug Therapy

Antiplatelet drugs (ASA, Dipyridamole, Triclopidine)

- Action: inhibit the deposition & aggregation of platelets on injured tissue & foreign material
- Why? - greatest platelet activity in first few weeks postop, sewing rings needs to be "covered" (endothelialized)
- When? early postop - first 3 months
- Who?

  pts with stroke risk factors (diabetes, smoking, carotid disease)

  pts with concomitant CAD

  pts with older turbulent prostheses

  pts who have have had TE despite good AC control

Monitoring AC Levels

- Blood tests necessary to control intensity of AC therapy
- Testing done in hospital, dr.'s office, lab or with home monitoring device
- Tests use THROMBOPLASTIN to test how long it takes before the blood clots
- Testing done every 2-4 wks or more often, dosage regulated according to results
- AC levels influenced by:

  diet, drugs, stress, illness, activity

  postop patient education very important

Common AC Test Methods

- Heparin

  Activated Partial Prothrombin Time (APPT or PPT)
- Oral anticoagulants

  Quick value & Thrombotest
  - expressed as a % of prothrombin activity
  - goal - 15-25%

  Prothrombin Ratio (PT Ratio)
  - expressed as a ratio to mean normal prothombin time
  - 1.6-2.0 x control (high), 1.3-1.6 x control (moderate)

  International Normalized Ratio (INR)
  - developed as a way to standardize the results between tests and thromboplastin batches - converts other test results to a standard measure
  - each batch is assigned an International Sensitivitiy Index (ISI) which is used in the INR calculation

ANTICOAGULATION

Prothrombin Time (PT)

PT ratio is determined by dividing the time required for the patient's blood sample to clot by the mean of the normal person's clot time:

$$\text{PT ratio} = \frac{\text{Patient's clotting time}}{\text{Mean of the normal clotting time}}$$

Unfortunatelly, PT times are not comparable form test to test. Labs may use different sources of thromboplastin(from rabbit, cow or human brain tissue) to test theclotting time of blood samples.Therefore, thromboplastin responsiveness can vary frombatch to batch.

ANTICOAGULATION

International Normalized Ratio (INR)

- International Normalized Ratio (INR) was introduced as a standardized way to measure clotting time.
- The INR is calculated by taking the patient's PT ratio and raising oit to the power of the International Sentitivity Index (ISI).
- The ISI is a value assigned to each batch of thromboplastin indicating its sentitivity to the effect of anticoagulant drug.
- INR can be calculated given the patient's PT times and the ISI.
- INR = (patient's PT / mean normal PT) ISI
- The advantage of INR is that it can be compared from lab to lab or from hospital to hospital.

AC Target Recommendations

- Recommended by:

  American College of Chest Physicians (ACCP) 1995 R. C. Becker et al., Antithrombotic Therapy, Achives of Internal Medicine, Jan. 23, 1995,155, pp.149-161.

  European Society of Cardiology - Working Group on Valvular Heart Disease (ESC) 1995 C. Gohlke-Bärwolf et al., Guidelines for prevention of thromboembolic events in valvular heart disease, European Heart Journal, (1995) 16, 1320-1330.

- Edwards employees do not recommend targets

  cite and/or provide references

- Anticoagulants also known as "blood thinners"

  Anticoagulants do not thin the blood but reduce the bloods ability to clot by blocking or reducing protein components required for normal coagulation .

MYOCARDIAL INFRACTION : MECHANICAL COMPLICATION (VSD)

DEFINATION

VSD : Due to ruptured acutely inflammed myocardium

Incidence: 1-2%

Sites:

- Anterior / Apical IVS due to transmural MI
- Posterior IVS due to inferior Infraction
- May be multiple (50%)

Cause

- Mostly compilete acute occlusion Of Coronary artery- LAD
- May be associated with other vessels
- 40% survives early insult, develop aneurysm
- Occurs 2-3 days after infract
- 70% survive 24 hrs,50% 1st wk, Only 10-20% >4 wks
- Mostly are seriosly ill

DIAGNOSIS

- Development of haollow systolic murmur
- BED Side Echo : Can provide diagnosis, site & magnitude of shunt

Cath& LV graphy

- Role is controversial because of sufficient non invasive diagnostic information
- Coronary Angiography is important to identify associated lessions

Management

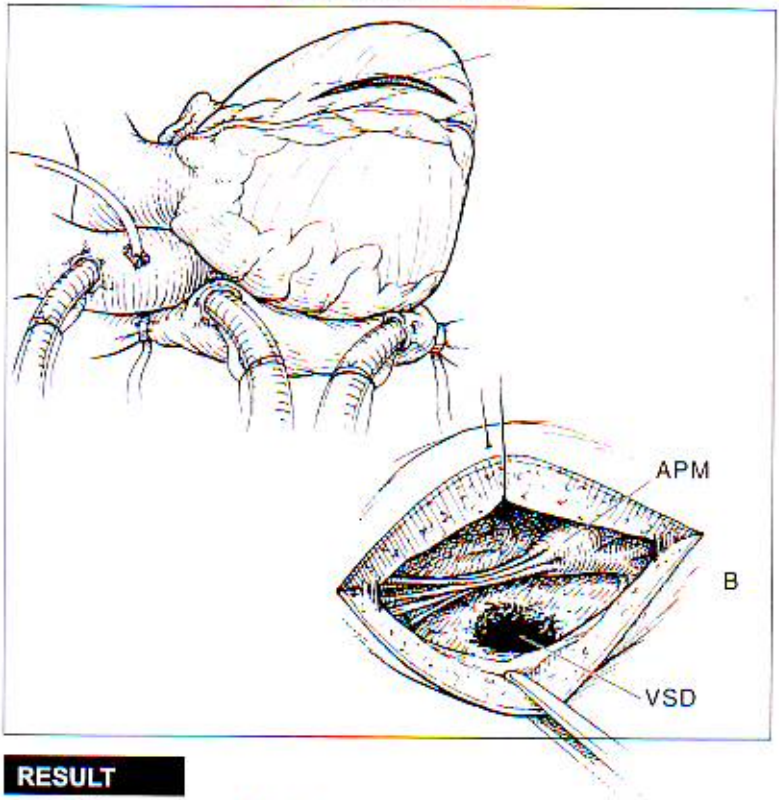
- Management before operation is critical
- Immidiate IABP is set up & transfered to Cath.lab
- When diagnosed Swan-Ganz Catheter is placed. Sample from LA, RV, RA, PA & areterial blood is drawn.Pressures are also measured.
- Shunt usually large.
- If <2:1 elective approach to be considered
- If Pt. is critically ill femoral CPB is started & Pt is broght to OT even if Cath not done
- In OT prompt sternotomy after initial preperation usually done

- CPB started & femoral canulae are clamped & taken out
- Aorta cross clampe, Ante & Retrograde cardioplegia are used

VSD Closure

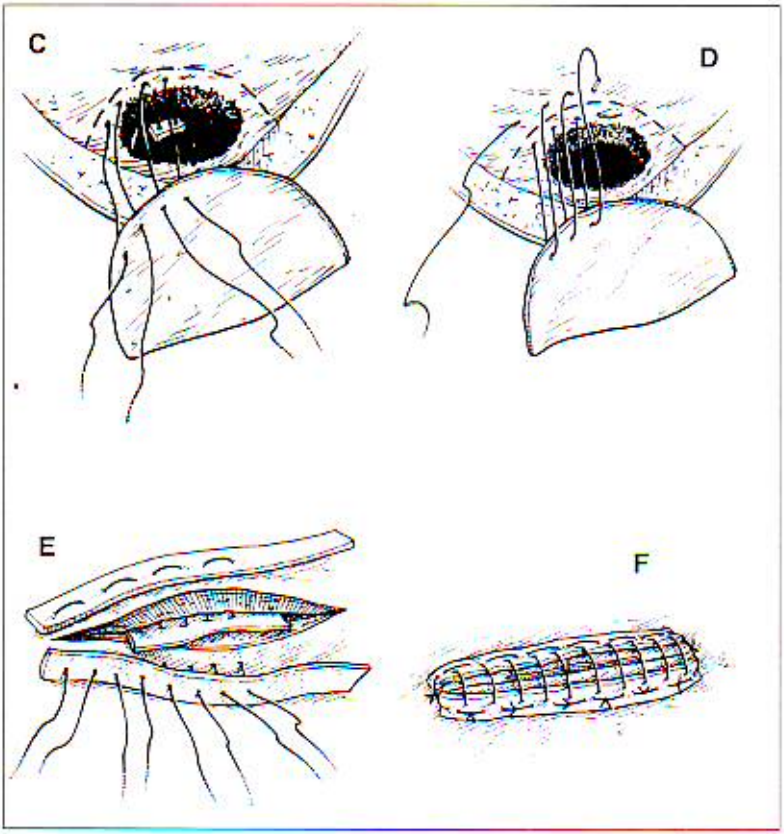
- Anterior Wall /Aneurysm :
- Approach through the area
- No part of IVS resected
- Sufficient patch including infracted area placed
- Controlled aortic root perfusion is essential part
- IABP started during rewarming
- If CPB not possible to stop after 30 mins after normothermic flow then VAD (Temporary) may be required

Operation: VSD closure



RESULT

- Hospital mortality 30-40%
- 5 yrs survival 45-55% (cardiac failure)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OFF-PUMP BYPASS GRAFTING (INCLUDING MAIN CIRCUMPLEX) IN PATIENTS WITH SIGNIFICANT LEFT MAIN CORONARY ARTERY STENOSIS- IS RATIONAL ?

INTRODUCTION

- Since the first clinical description of left main coronary artery disease by Herrick in 1912 studies showed that stenosis of the left main coronary artery is of critical prognostic importance. CABG surgery lessens symptoms and significantly prolongs survival.
- In long term follow-up of patients listed in the Coronary Artery Surgery Study (CASS) registry, the 15-year cumulative survival of those with initial coronary artery bypass grafting (CABG) was 44% v. 31% in medically treated patients; median survival in the surgical group was 13.1 years compared with only 6.2 years in the medical group
- But more than 16 yrs CASS Registry follow up showed that over all Surgery prolonged survival with ≥50% LMCAS,median survival was not prolonged in subgroups : (1) LM <60% (2) Normal LV function,even with RCA stenosis (≥70%) (3) Nonstenotic(<70%) RCA .
- In this registry of >16yrs followupdisproportionate increase in mortality of surgical cases than medical group.may be multifunctional ,mystry in the atherosclerosis(Surgical, noncardiac recordd 20% Vs 6%)
- The presence of critical LMCS has been considered a relative contraindication for the off-pump coronary artery bypass (OPCAB) technique due to concerns over the well demonstrated hemodynamic changes during displacement of the heart
- Recently, there has been renewed interest in the potential benefits of off-pump coronary artery bypass (OPCAB) surgery and now accounts 20% CABG in US, West Europe.
- Development in exposure and stabilization techniques, the introduction of intra-coronary shunts and increasing

understanding of the haemodynamic changes which occur during off-pump surgery encourage the surgeons to do OPCAB surgery in patients with significant LMS disease .

- The idea is that avoiding CPB improves End organ function by eliminating problem of inflammatory cascade & trauma.
- A retrospective review of non-randomized prospectively collected data from July 2004 to Oct 2007 of National Institute of Cardiovascular Diseases registry with Total number of Patient : 207 was analysed
- Patients of the registry with significant left main coronary artery disease who underwent coronary bypass on the beating heart were 121 and patients with the conventional method were 86.

Surgical procedure

- CABG in left main coronary artery disease were performed through median sternotomy

OPCABG:

- We used tape proximally to control flow at the anastomotic site and also used mist air blower (Medtronic)
- Commercial stabilizer and positioner including octopus and starfish/Urchin (Medtronic Inc) were used.
- Exposure of LV surfaces achieved by different combination of elevation, right pleurotomy(later with out pleurotomy), lateral displacement fascilated by starfish/Urchin
- An intracoronary shunt was used (Bio vascular Inc)
- Some proximal anastomoses were made to ascending aorta with side clamp at BP 75-90 mmHg.

CCABG:

Conventional CABG was done with aortic and two stage single venous canultion. Cardioplegic arrest of heart was done.

Results:

PATIENTS PROFILE :

Variables	OPCAB n=121(%)	CCAB n=86(%)	P
• Age	55(45.45%)	48 (55.81%)	0.50 NS
• Female	8 (6.61%)	11 (12.79%)	0.50 NS
• Pre op MI	29 (23.97%)	26 (30.23%)	0.25 NS
• Smoking	67 (55.37%)	49 (56.98%)	0.10 NS
• Diabetes	44 (36.36%)	23 (26.74%)	0.25 Hypertension
35 (28.93 %)	26 (30.23 %)	0.25 NS Dyslipidemia	
54 (44.63)	32 (37.21%)	0.10 NS	
• Renal dysfunction	5 (4.13%)	0.00	
• H/O CVA	7 (5.79%)	6 (6.98%)	0.25 NS
• PVD	13 (10.74%)	8 (9.30%)	0.25 NS
• Chronic Lung D.	21 (17.36%)	6 (6.98%)	0.2 NS
• CCS (I-II)	51 (42.15%)	38 (44.19%)	0.25 S
• CCS (III-IV)	31 (62%)	45 (56%)	0.25 S
• Lt. Main only	20 (16.53%)	16 (18.60%)	0.25 S
• 1-2 VD+ Lt. Main	34 (28.10%)	29 (33.72%)	0.10 NS
• 3 VD+ Lt. Main	67(55.37%)	41 (47.67%)	0.10 NS

OPERATIVE DATA

Variables	OPCAB n=121 (%)	CCAB n=86 (%)	P value
Converted	1 (0.8 %)	--	
Distal anastomosis/ pt	3.3 + 0.7	3.0 + 0.64	0.09
Type of Conduits			
LITA	121 (100%)	81 (94%)	
Long saphenous vein	217	164	
Bypassed arteries :			
LAD	121 (100%)	86 (100%)	
Diagonal	29 (23%)	26 (30%)	
RCA/ PDA	67 (55%)	56 (65%)	
OM	116 (95%)	86 (100%)	
Cx (Main)	05 (4%)	-	
X-CL time (min)	-	45 + 8 min	
ECC time (min)	-	59 + 10 min	
Hemodynamic change	2 (1.65)	9 (10.47%)	0.90 S
Inotropic support	17 (14.05%)	44 (51.16%)	0.50 S
Blood transfusion	1.65 + 1.42 unit	4.2 + 2.2 unit	0.75 S
IABP	0	2	

Post-operative Data

Mortality	4(3.3%)	6 (6.98%)	0.50	S
Perioperative MI	01(0.8%)	6 (6.98%)	0.75	S
Inotrope required	17 (14.05%)	44 (51.16)	0.50	S
Blood loss (ml/24h)	375 (309.92)	485 (563.95)	0.005	S
Reopen for bleeding	3 (2.47%)	7 (8.14%)	0.75	S
Blood transfusion	1.65 + 1.42 unit	4.2 + 2.2 unit	0.001	S
Ventilation time (h)	5.1hours (4.21%)	18.7 h (21.74%)	0.50	S
Chest infection	15(12.40%)	17 (19.77%)	0.25	S
AcuteRenal Failure	0	1 (1.25%)		NS
Neurological Com.	2 (1.65)	9 (10.47%)	0.90	S
ICU stay	48H (39.37%)	72H (83.72%)	0.25	S
Postop Hosp. Stay	7.1+ 2.2	9.5+3.5 days	0.50	NS

- Analysis of variables established that OPCAB patients in comparision to CCAB group had a lower requirements for postoperative inotropes 17(14.05%) vs 44 (51.16%), blood transfusion 1.65 units vs 4.2 units and lower mechanical ventilation time (5.1H vs 18.7H, p =0.50),
- Pulmonary complications, Neuro cognitive dysfunctions were less observed post operatively in OPCAB group and a slightly reduced postoperative hospital stay (7.1vs 9.5 days).
- OPCAB costs was significantly less than CCAB
- The grafts performed was 3.3 + 0.7 vs 3.0 + 0.64 (Table- II).
- In OPCAB the grafted vessels are LAD 100%, RCA/ PDA 67(55%), OM 116(95%), Cx 5(4%),Diag 29(23%)
- In CCABG LAD 100%, RCA/ PDA 56(65%), OM 86(100%), Diag 26(30%).
- There were 1 incidences of intraoperative conversion from off-pump to on-pump surgery due to hypertension.
- The mean intubation period, intensive care unit stay and postoperative hospital stay were 5.1hours vs 18.7hours, 48h vs 72h and 7.1vs 9.5 days respectively.
- Hospital death was 4(3.3%) in OPCAB group & 6(6.98%) in CCAB group.
- Postoperative myocardial infarction was observed in 1(0.8%)& 6(6.98%) patients respectively.

- Neurological complications (1.0 vs 10%) is higher in CCABG group.

Discussion

- Surgical revascularization prolongs life in patients with significant LMCA stenosis compared to medical therapy alone.
- Numerous studies have showed that left main coronary artery disease is an independent predictor of postoperative morbidity and mortality in patients undergoing coronary revascularization.
- The presence of critical LMCS has been considered a relative contraindication for the off-pump coronary artery bypass (OPCAB) technique due to concerns over the well demonstrated hemodynamic changes during displacement of the heart
- Revascularization of the circumflex territory is in most cases difficult because of hemodynamic impairment associated with exposing the vessel.
- Grafting of the main circumflex artery in the posterior atrioventricular groove was of particular interest of this study
- When all obtuse branches are small caliber and/or the lession is only in LMA with /without proximal cirumflex we performed main trunk graft
- By virtue of its large size we found less difficulty in anastomosis using a large size intravascular shunt
- The challenge was in the exposure of Cx not in anastomosis,as AV groove does not contract like ventricle & simple pressure can immobilze the area of anastomosis
- Starfish / Urchin stabilization along with verticalization of the apex provided an excellent haemodynamic toleranance
- Myocardial revascularization without ECC through median sternotomy is a developing surgical strategy today. Recently, however, there have been encouraging reports about the safety and efficacy of OPCAB for patients with LMCA stenosis
- There was a definite trend toward improved in -hospital survival of OPCAB patients with LMCAS
- This may be attribuatble to better myoocardial protection & subsequent cardiac performance as evidenced by lower level of CPK-MB rise (immidiate post operative) in our another study

- On the other hand cardiac complications are the most frequent cause of mortality in on-pump group implies CPB as an indipendent predictor of mortality
- Eliminating CPB in coronary revascularization reduces the incidence of intraoperative blood transfusion requirements. Our study supports obviating the need for CPB and cardioplegic arrest clinical outcomes can be improved particularly in left main artery disease patient
- Technicaly challenging and with a perceived learning curve,reproduceability of the result provides greater significance in demontrating safty & efficacy

CONCLUSION

Despite a significant learning curve, evolution to routine off-pump coronary artery bypass technique good patient outcomes can be achieved with careful patient selection during the "learning curve.

OPCAB in LMCAS is safe and reproducible, and of definite benefit with shorter intensive care unit and hospital stays

OPCAB can be an effective alternative to the conventional method CCABG with same or better early results as well cheap and cost effective. The long-term results are to be evaluated.

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PACE MAKING & PACE MAKERS

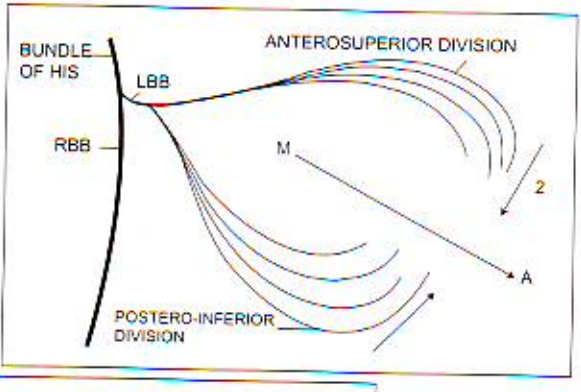
PACING : CONTROL OVER ELECTICAL ACTIVITY OF THE HEART

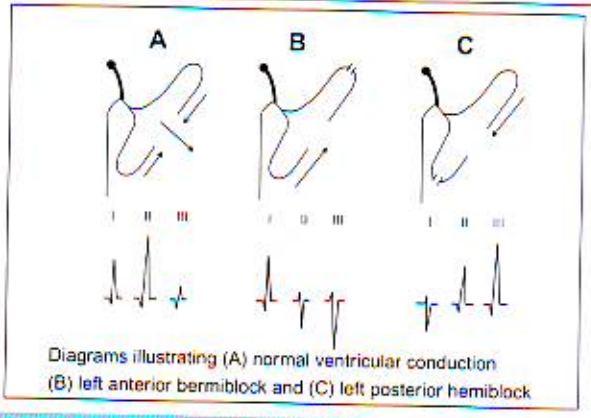
Unit Consists:

- Generator - Batteries with electronic circuit
- One/Two Electrodes

Potentially hazardous if stimualtes at repolarization(T wave)>VF

- External Pacing: Through electrodes over skin
- Temporary Pacing: Short term measure
  Epicardial –In Cardiac Surgery
  Endocardial- Through a veinous access
- Permanant:
  Epicardial
  Endocardial





Diagrams illustrating (A) normal ventricular conduction (B) left anterior bermiblock and (C) left posterior hemiblock

PACING MODES

Alphabetic Codes (3 letters):

- 1st letter refers sites of Pacing- Atrium /Ventricle /Both(Dual)
- 2nd to site of sensing –Atrium /Ventricle / No sensing(O)
- 3rd to response of sensing –Inhibited / Triggered /Dual / No(O)

Additional letter used to inform type of Generator/Lead

Usual Practice: VVI (common), VVIR, AAI, DDD(most sophisticated pacing , sensing atrium with AV counter), VDD(use in AV block with normal sinus)

Dual chamber Pacing (DDD)

- Two intra cardiac(atria,Ventricle) leads
- Maintains norval AV synchrony(Progammable)
- Singel/Both chamber modes
- Usefull in normal atrial rythm
- Prevents 'Pacemaker Syndrom'-retrograde VA conduction

Anti fibrillation(atrial) Pacing- senses ectopic to suppress.

Indications:

Class I :

Conditions with General agreement

Class II :

Commonly used but with difference of openion

Class III :

General agreement of no necessity

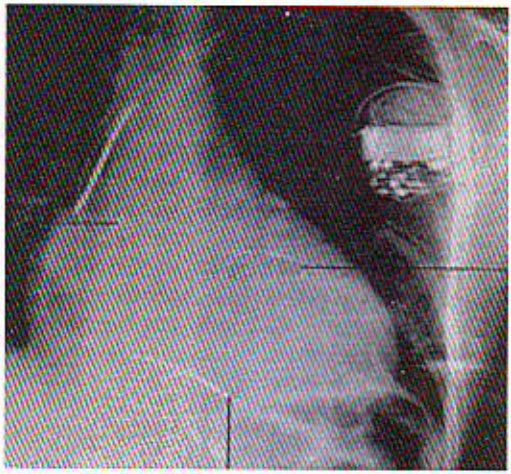
Parmanent Pacemaker(PPG) in Acquired AV block (in Adult)

- **Class I : (Implantaton unequivocal)**
  Complete AV block (Intermitant/Parmanant)- associted with:
  Bradycardia
  HF
  VES- needs medication to suppress foci
  Confusional state
  Second Degree AV block (Inter /Parmt)-
  With symptomatic bradycardia.
  Atrial fibrillation/Fluttar – in association of CHB, bradycardia-(not drug mediated)
- **Class II : L(Implantation questionable)**
  Asymptomatic complete HB
  (Permanant/Intermitant with VR≥40/m)

Asymptomatic 2°AV block Type I
(Intra His /Infra His levels)
Asymptomatic 2° AV block Type II
(Parmanant /Intermitant)

- **Class III : (Not necessary)**
  1° Heart block
  Asymptomatic 2°AV block Type I
  (Av nodal level)

NEW MODE
Resynohronising Page maker/ Implantable Defibrillator (New)



leads - R.A, RV [2 coils, diatal for DC shock], LV [coronary sinus]. Significantly reduces QRS duration

INDICATIONS IN MI

- Class I
  Complete / Persistant 2° AV block
- Class II
  Persistant 1°block with BBB
  Transient AV block with BBB
- Class III
  LAFB With transient /without AV block

Indication in Bi/Tri fascicular block

- Class I :
  Bifascicular + Intermitant CHB with bradycardia
  Bifascicular + Intermitant 2° Type II with symptomII
- Class II :
  Asymptomatic bi/tri fascicular block + 2° Type II

Bi /tri fascicular block with H/o Syncope

- Class III :
  Fascicular block wthout AV block/Symptom
  Fascicular with 1°block but without symptom

Sinus Node dysfunction

- Class I:
  SSS With Symptomatic bradycardia (Sponteneous / If for prolong compulsary drugs)
- Class II:
  SSS sponteneous /due to drug but no definite documented bradycardia
- Class III:
  Asymptomatic SSS with brady <40/m for prolong drug

TACHYARRYTHMIAS

- Class I:
  Symptomatic SVT not controlled or with inability to normal life
- Class III :
  AF with rapid ventricular rate

CAROTID SINUS SYNDROME

- Class I:
  Recurrent Syncope with clear evidence of minimum sinus stimulation
- Epicardial Pacing (Temporary)
  2 RA , 2 RV leads
  Dignostic for exact arrythmias / Theraputic
  Atrial Pacing:
  Bipolar,easier to detect capture function
  Asynchronous mode at expected rate
  Normal AV conduction must present
  Ineffective in AF

Indication:

- Bradycardia / Ventricular or atrial ectopy / SVT suppression
  Atrioventricular Pacing /Sequential:
  Both atrial ,Ventricular leads are connected to 'AV Pacer'
  PR interval can be manipulated for CO
  Demand mode safe
  Asynchronous mode - to avoid 'cross talk'
  Ineffective in AF

Indication: Heart Blocks

- Ventricular Pacing:
  2 ventricular electrodes to Generator -for bipolar or one with skin -for unipolar pacing
  Demand(synchronous) mode - to avoid discharge on T.

Indication: Slow ventricular rate

- Overdrive ventricular arrythmias
  Problems (Epicardial Pacing)
  Faiure to capture
  Faulty cord /Generator
  Changing threshold
  Competition

Management:

- Change output
  Use other cords (if any)
  Use isoproterenol
  Transvenous pacing

BEATING HEART CABG (OPCAB)

INTRODUCTION

Surgical treatment of coronary artery disease is constantly undergoing alteration and refinement to meet the clinical challenges brought on by an aging of population, recurrence of disease and ever increasing competitive age of PTCA and stenting

In addition conventional CABG by midline sternotomy is a gold standard but comparatively a bit extensive surgical procedure

To meet the challenge the fast tract approach to early extubation and less expensive CABG have became popular and there is increased focus on the less invasive surgery. Now OPCAB accounts 20% CABG in US and West Europe.

A preponderent retrospective study have mortality and morbidity benefit in specific subgroup.

Patients and Methods

- Retrospective review of prospectively collected data
- Total patient : 225
- Duration : Jan 2004 to July 2006.
- 10 (4.4%) patients had to convert to on-pump CABG due to arrhythmia and hypotension during grafting
- 25 (11%) patients had undergone MIDCAB and 13 (5.7%) patients had undergone Awake CABG (ACAB)
- 18 (8%) patient came for CABG after the Stent failure.
- Male: Female=6.25 :1.0
- 19 variables were analyzed
- Youngest patient was of 25 years
- Oldest was of 66 years.
- 33 patients were with Ejection Fraction < 40%

Patients Selection

Inclusion criteria of patients for OPCAB

- Patients with suitable anatomy of epicardial non-calcification vessel >1mm
- Patients without history of failure or evidence of moderate to severe cardiomegaly.
- Patient considered high risk for CPB with comorbidity were included.

Excluded patient for OPCAB

- Patients with Intramyocardial vessel / with arrhythmias and/or haemodynamic instability

Ideal anatomical condition for LIMA to LAD In MIDCAB

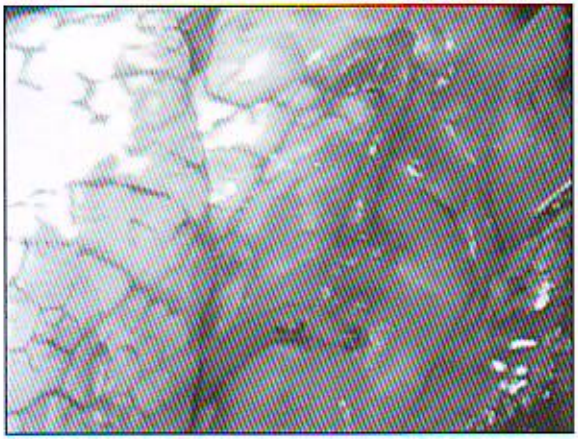
- LAD >1.5 mm in diameter
- Presence of a tubular heart on chest radiography
- Thin chest wall with wide intercostal spaces
- Reoperation coronary artery bypass with
- deterioration saphenous vein graft
- Totally occluded LAD with good clinical collaterals to distal LAD

Contraindication for MIDCAB

- Small sized coronary artery target site (<1.3mm)
- Diffuselly calcified coronary arteries
- Intramyocardial LAD coronary artery
- Stenosis or occlusion of left subclavian artery

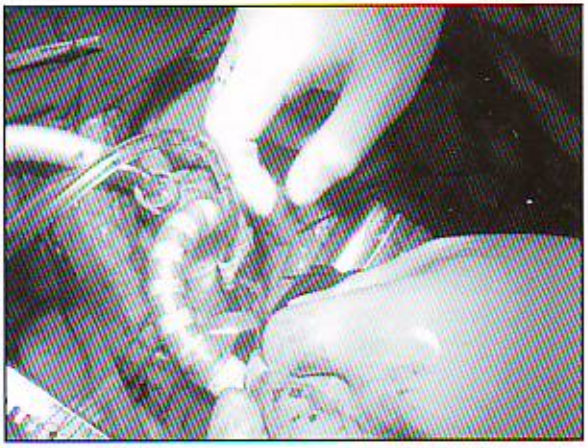
SURGICAL TECHNIQUE

- A median sternotomy in OPCAB and anterior mini thoarcotomy / anterolateral thoracotomy in MIDCAB
- We used tape proximally to control flow at the anastomotic site and also used mist air blower (Medtronic)



Proximal : Aorta to RCA(proximal) / Cx

- Commercial stabilizer and positioner including octopus and starfish (Medtronic) were used.
- Exposure of LV surfaces achieved by different combination of elevation, right pleurotomy, lateral displacement fascilated by starfish.
- An intracoronary shunt was used (Bio vascular Inc)
- Some proximal anastomoses were made to ascending aorta with side clamp at BP 70-80 mmHg.



Distal :Mid RCA

PREOPERATIVE DATA

Variables	n=150	%
Female	31	14
Pre op MI	74	32.67
Re do/stent	18	8
Smoking	158	70
Diabetes	120	53.33
Hypertension	142	63.33
Hypercholestrol	127	56.67
Renal failure	3	1.33
H/o CVA	6	2.67
PVD	7	3.3
Chronic Lung D.	23	10
CCS (I-II)	163	72.67
CCS (III-IV)	62	27.33
1-2 VD	57	25.33
3 or more VD	120	53.33
Lt. Main	48	21.33

OPERATIVE DATA

IMA	224
RSVG	278
Mean graft (Per case)	2.23
Total	**502**

Conduits and Arteries Bypassed

Variables	No	%
LIMA +RIMA	224	44.62
RSVG	278	55.38

Coronary Anastomosis

LAD	225	44.82
DIAG	33	6.62
PDA	87	17.47
RCA	79	15.66
OM	63	12.54
Ramus Intermedius	15	3

POST OPERATIVE COMPLICATION

Variables	No	%
Reexploration for bleeding	5	2.22
Prolong ventilation	9	4
Renal Failure	1	0.44
Neurological Complication	12	5.3
Prolonged Hospital Stay	12	5.3
Sternal Resuture	5	2.22
Mortality	4	1.78

- POST OPERATIVE ICU STAY AVERAGE 48 HOURS
- POST OPERATIVE BLOOD TRANSFUSSION AVERAGE 2.5 UNITS

Discussion

- With perfection of technique and equipments for stabilization benefit of off-pump multi vessel CABG is more apparent
- With availability of stabilization technique our adaptation rate and experience has increased and is becoming integrated
- Until now mortality is 1.78%, it may be due to the election bias in early experience.
- But other larger series had observed mortality of 1.9%.
- The rate is higher in Octagenerian group series (6%,10.3%).
- Our patient were not older.
- Some series addressed influenced of patient selection and surgeons experience on mortality.
- They hold that mortality influenced by adaptation rate also.
- Our study examined morbidity and complication though in small series but similar to other workers.

Conclusion

- Coronary artery bypass grafting on a beating heart is a surgical strategy that has gained popularity.
- Selection of the patients and technical skill are the critical aspect of this procedure. Even though the early result is available by clinical ground the patency rate is comparable with that of graft performing with conventional CPB.

It is cost effective.

- Long term study to reveal patency & quality of life is warrented.
- We advice young surgeon & anaesthetists should be trained to enter the upcoming new era of coronary revascularization

VALVULAR HEART DISEASE (ACQUIRED)

MITRAL STENOSIS : Result of Rh.Ht. Diseases

MSR : Incompetence results from fibrous retraction, rupture

MITRAL REGURGITATION

- Rh. MI – Annular dilatation
  – Chordal clongation
  – Calcification (Uncommon)
- MVP – Myxomatous/Rupture (Barlnw's)-10%
  (resulting reducdancy & non cooptation)
- Idopathic chordal rupture : Localised-Post medial
- Ischemic PM dysfunction/Rupture
- IE-Uncommon for MR
- Sub mitral I. V ancurysm-Distortion post. Cusp

MS:

INDICATION OPR.

- Area <1.5 cm2 with / without symptom unless other disease risks
  Class IV~opertive risk >10%

OPEN / CLOSED BALVOTOMY

Idealy closed should be done : OPENCOMMISSUROTOMY:

- Severe HF with Preg.
- Risk-1% (Embolic risk also less)
- Non availability of H-L mechine

GOAL: of surgery

To open as possible with out insufficiencv

OBJECTIVES

- Not to reduce gradient

- To minimize turbulance

MVO- 2 cm2 good for few yrs

20~25% restenosis in 5yrs

- ***RESTENOSIS AFTER EXTENSIVE VAOVOTOMY THAT ELEMINATES END- DIASTOLIC Gr. IS UNKNOWN***

BALLOON VALVOTOMY:
- All limitation of older closed valvolotomy
- Valuable with associated operative risk

CHOICE OF PROSTHESIS

Complicated factors:

- Rapidly changing models
- Regulatory agencies - prevelance

Selection Policy

- Individual both Surgeon/Pt.
- When anticoagulation safe mechanical recommanded <65y
- Recent Bio- Prosthesis 10-20 yr durable-suitable for older/risk gr.

Factor of consideration

- Durability
- Permanant Anticoagulation
- Current Xenograft degenerates early in youngs(Good candidates for tissue)
- Bio prosthesis degenates rapid in mitral position
- Bioprosthesis in state of evaluation may lead to good durability
- Prosthetics also evolving-but indication that a device will appear that needs no anticoagulation
- Haemodynamic performance influences the choice of .
- Newest Mitral substitute-Cryopreserved Stentless allograft
  - Good haemodynamic performance
  - Adequeat freedom of embolism in abscence of antioagulation
  - Durability ?
- Pulmonary autograft as cylinder ('Top-hat') in mitral position

Bioprosthesis

- A number of series used
  - Stent mounted
  - Leaflet made allograft Av
  - Xenograft AV

- Pericardium
- Fascialata
- Duramater
- AV function improves
- Trans valvar gradients always persists
- Depends upon activity,Pt.size & device
- Mean Diast.Gr - Rest Exer

  Normal o

  Desired <10mmHg

  Orifice >0.9 cm2

ACQ. MR

REPAIR - Method of choice

1) Rupture post chorda < half
2) Combined MSR
3) Non Rheumatic, non cal. , Degenarative (some)
4) MI of BE

Rh. MR (ADULT)

Annuloplasty with Ring

In YOUNG children-measured plasty (Reed`s)

RESULT

OMC:
- Gradual loss of piiability
- Prograssion of subvalvular path.
- Calcification
- *NEED REINTERVENTION*
- Few deaths early without any risk factor, PVR directly affects Survival

- Survivors 86%
- Survivor with out reoperation 70%
- Survivor with out emboli 61%

Recent 20yr survival 70% (CMC)

38%(OMC)-incomplete compare

No difference in multi variable comapre

20% needs reop. in 10yr

50% in 20yr after

- OMC/CMC not risk factor for redo

REPAER in MR

Possible in 30~50% MI

Mortality 0~4%

Better survival (7~94% =5y) repair than replacement

MVR

Mortality 2~7%

Time related survival(68%-10yr) heigher in Isolated case than with associated disease.

RISK FACTOR FOR PREMATURE DEATH

- Old age
- LV enlargement
- NYHA class
- Longer ischemec time
- MV Surgery WITH TVD
  10~20% Tv - plasty
  <2% Replacement

ACQ. AORTIC STENOSIS

- Cal. AS – Dystropic (Unicommisural than Bicommisural)
- Rh. AS – Diffuse thickening & Calcification
- Degenative AS – Commis.free
  – Cusps held closed
  – Calc.deposit

LV Structure & Function:

- LV mass increased
- Mass >300g/m2 - degeneration
- Depressed systolic function
- Loss syst. Function & irreversibily
- Indices of syst. Funct. goes down
- Increase interstitial tissue with pressure overload LV

CHIOCE OF OPERATION

- Allograft AV – Device of choice
  (Primary Contra Indication > 30mm root)
- PV Autograft – Suitable for <15y
  – Needs assesment
- Stent Mounted Xenograft – Appropriate for elderly

- Mechanical – Durable in all circumstances
- AV en Block – Annulo-aortic Ectesia
  – Annurysm
  – Dissection

CHOICE OF DEVICES

- Prosthetic Cylinder (lowest mortality- 50%)
- Autograft PV cylinder-15%
- Allograft valve cylinder –14%
  (Allograft/Autograft prefferred with Prosthetic IE)

Acquired AR

- Rh.AR – Cicatrization of cusp (Slight/no thick)
  – Rolling cusp
  – Annular dilatation
- Ectesia (Cystic degeneration) – Extension progress
  – anneurysmal
- Endocardities – Normal/Rheumatic
  – destruction / prolapse / perforation
- Congenital AVD – Unicusps/Bicusps
- Floppy aortic cusp – Redundant
  – Myxomatous

Aneurysm /Aortitis -Tightening free edge
– (Syphilitic / Atheroma)

- Others – Sponteneous
  – Truma
  – HTN

REPLACEMENT DEVICES

- Allgraft – Cryopreserved banked – not stented
- Allograft PV – Replacement PV to AV – not stented
- Allograft AV cylender – not stented
- Prosthetic

MORTALITY

- AVR with other Procedure – 3-6%
- Isolated AVR – 1-2%
- Overall Survival – 60% at 10yr

Mode of death : Failure / Hge / Infection / Neurological

Risk factor of premature death

- Older age (as other cardiac oper)
- Heigher NYHA class
- Greater LV enlargement
- More AR

❒ 15yrs Survival unrelated to type of prosthesis

HOSPITAL MORBIDITY (AVR)
- CHB
- Embolism

TRICUSPID VALVE DISEASE

- Rh.TVRs – Assoc. MV/AV(not seen isolated)
  – Pure stenosis rare
  – All Comm. Fused / usualy ant. Septal
  – No Calcification
- TV Endocarditis – Staph. Aureas (Drug addict)
  – Candidia
- Trauma – Rupture corda
- Carcinoid Diseases(GIT) – Cicatrical deformity TV/PV
  – Fusion commi/Chorda

TEATMENT

- Ring Valvoplasty
- DeVaga`s Annuloplasty

❒ **TV Replacement:** Early mortality 7%(5~11)
Survival depends upon other lessions.

- **INDICATION:** Isolated TVR- Annuloplasty,
  Replacement-when fails
  Drug addict -Simple Excession

CONGENITAL MALFORMATION OF MV

- Incidence – 0.6%

❒ **Asso. With :**

- Complex congenital – AV canal defect
  – UVH
  – Hypoplastic LV
  – EFE
  – TGA

- Discrete lesion – Coarctation
  - AS
  - PDA
  - VSD
  - TOF
- Valve stenosed/Incompitant /Both

MS : (2gr)

A. With normal papillary muscle
1. Comm.fusion-Valvotomy
2. Excess Valvular Tissue
   - Fenestration
   - Papillary
   - splitting.
3. Annular Hypoplasia
   Extra cardiac conduit
4. Suprvalvar ring
   - Resection

B. Abnormal Papillary Muscle

1. Parachute MV-Splitting – Fenestration
2. Hammock MV – Resection except Marginal chorda
3. Absent – Replacement

MR (3Grs)

A. With normal leaflet motion-
1. Annular dilat.-DeVaga`s/Wooler`s
2. Cleft Leaflet-suture
3. Leflet-defect(holes)-Patch

B. Prolapsed Leaflet motion-
1. Absent chorda –Resection leaflet
2. Elong. chorda-shorteninf/slidding
3. Elong.PM-buried LV wall

C. With restricted motion-

1. Normal PM – Fusion comm.(Commisurotomy)
   - shortchorda(Elongation)
   - Ebstein`s(Replacement)
2. Abnoramal PM – Complex agenesis(Replec.

TREATMENT

- Most require opr. In infancy / early childhood
- Advisable to correct asso. Lesion first

- Even functionally imperfect repair superior to prothesis as long child grow &
- Without refractry failure.

AIM TO RESTORE FUNCTIONAL VALVE THAN NORMAL ANATOMY

INDICATION OF SURGERY

- Refractrory HF
- Recurrent Pul.Infec.
- Pul.Hypertension
- Pul. Oedema

CONGENITAL AS:

- Spectrum of lesions
- Obstracts flow (3-10%)
- Asso. Anomalies (8-30%)
- Coarctation. PDA.
- Endo Fibroclastosis(EFE)
- VSD, PS, MS

Classification (anatomical) :

- Valvular AS
- Sub-valvular AS
- Supra valvular
- Hypertrophic Sub-AS.

Considerations

- Infants with CHF refractory to medical needs surgery
- Older infants & young adult-Early surgery for risk of sudden death
- Systotic gr 50-70 mm Hg & Valve area <0.5 cm2. m2 accepted para meter for surgery
- Gr. 40 mmHg in Subvalv.AS requires early repair to avoid surgically defficult tunnel formation
- Approach & Urgency depends on location. Age & condition of pts.

VALVULAR AS

TRANS VENTRICULAR CLOSED VOTOMY

Usefull in children – lateral thoracotomy
(some may be treated by balloon valvoplasty)

TRANSAORTIC VALVOTOMY – under caval occlusion & various degree of hypothermia (some degree of AR well tolerated than AS)

Goal of Surgery- to reduce gradient (Valves still abnormal)

AVR

- Valvotomy to enable children to grow untill can accommodate Prosthetic valve if obstraction persist (30% children after 10-20yr needs second opr.)
- Smallest available prodthesis 17 mm small for many children.
- Annular Enlarging Procedure available
- Cryopreserevd homograft are now available

SUBVALVULAR STENOSIS

Approach for resection for most forms through –aorta.

A variation (AS Hypert) treated by Myomectomy when medically failed.

- **SUPRAVALVULAR AS** : Localized or Deffuse

Under modorate hypothermia by

- Patch annuloplasty
- E to E anastomosis

Palliative nature of surgery in children tells proper timming, so that current situation ***Not replaced by equal concern***

TRICUSPID ATRESIA

(Includes subarterial & ventriculo-artrerial relation)

I. With normal great arteries – a. Palmonary Atresia

b. Pal.Hypopls & smallVSD

c. Large VSD

II. With d – position – a. Pal.atresia

b. Pal. Stenosis

c. Large Pal. Artery

III. With _ -position – a. Sub pul / aortic stenosis

PALLIATIVE

Infants with – Insufficient pulm. Flow
– Exessive flow
– Inadequet interatrial connection

PROCEDURES :

- Systemic to pulm – Shunts
- Cavopulmonary
- Banding
- Closed / Open Septectomy

CORRECTIVE OPEARION:

Concept : A pump is not necessary for pulmonary circulation

Selection:

- >4yr to <15yr
- Sinus Rhythm
- Normal venous drain
- Normal RA vol.
- PAP not >15 mmHg
- PR index <4 unit/m2
- PA:Aorta=75 (at least)
- EF at least 0.6
- No MR
- No effect of shunt

(Hospital Mortality 10-20%) When selection criteria strict 0-7%

EBSTEINS` ANOMALLY (1%)

SURGICAL CONSIDERATIONS:

Medical management offers little (Arrythmia)

- Prognosis poor in CHF/ cyanosis/ C:T >0.65/ infancy/ Asso. annomally
- Survived infacy does well, needs observation
- NYHA III-IV / Progressive cardiomglaly are definite indication for opr.
- Shunt procedure -Life saving only in Pul. Obstraction.

TOTAL CORRECTION

- ELECTRO-PHYSIOLOGICAL MAPPING
- PATCH CLOSURE ASD
- PLICATION RV
- PLASTIC REPAIR / REPLACEMENT TV
- CORRECTION OTHER ANNOMALLY

Result is favourable when compared with Natural History

- **PS(Congenital):** Lethal Lesion
- Depends on RV size, Age
- Most infant develop symp. Slowly
- Some needs urgent valvotomy (Transventricular valvotomy)
- **Surgery:** Open repair only when balloon valvoplasty fails.

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PCI / CABG

- In 2003 OHS observed 50th aniversary
- Since 1962 (almost 40 yrs)Multivessel &LMD are bypassed–Gives a strong basis of evidence for practical guide line
- PCI appeared in 1977 with a hope to cover 10-15% of bypass pts, suitable
- In late spring of 2003 DES generally available in US with a hypothetical near zero restenosis rate

Trials

- All the trials of PCI with stents (including DES) are underpowered & lack of sufficient follow up
- 1 yr. Follow up:
  Revascularization rare 16.8 Vs 3.5%
  Event free freedom – Equal
  No difference in MI,Stroke or death
  (ARTS, n=1205)
- 2 yrs follow up
  Restenosis- 21 vs 6%
  Freedom Angina-79 vs 66%(p –S)
  Mortality heigher in PCI
  [2 large? trial with n=988, not all multivessels (57%2VD;42% 3VD & stent 71%stent.CABG 81%]
- Largest Series is SoS:
  Vasit Majority SVD, DVD, normal EF
  Mortality -2.5% vs 0.8%
  Restenosis -21% vs 6%
- Over all 6 trial by 2004, 11 trals by 2006 are published–All are under popwered. Even some includes 121 (SIMA) for DES,127 (ERACI)
- These trial shows the gross disparity in need of revascularization favours CABG
- Particularly with Diabetes long term survival significantly better in CABG

Considered cases for PCI-S

- Diffuse diseases
- Restenosis after surgery
- Small narrow vessel
- Even in ACC guide line is modified by 2005:

  CCSI/II with 1,2/3 or even in some multivessel recommandation of PCI-S changed to ClassIIa(not evident) from ClassI(evident) & Level of evidences are B (from single randomized/ non randomizes).

PCI /BMS

- 6m Efficacy is heigher in BMS(58% vs 73%)
- Restenosis rate is not Significant

❖ 3 major RCT onPCI-S give similar report-"At 5 yr(ARTS trial) no difference in mortality between stenting &CABG in MVD" *(Undisclosed fact of ARTS trial is that the Governer of the group is Vice president of Cordis,division of JONSON &JONSON)*

British (NHS) assessment on DES

- Used 2 model(DVD/SVD & MVD)
- Opine "Given CABG is the standard for most with MVD. It is difficult to justify substitution by cheaper PCI-s....This argument remains valid also in case of DES...***Hence we found no grounds for substitution of CABG by DES in MVD"***

Meta analysis DES / BMS

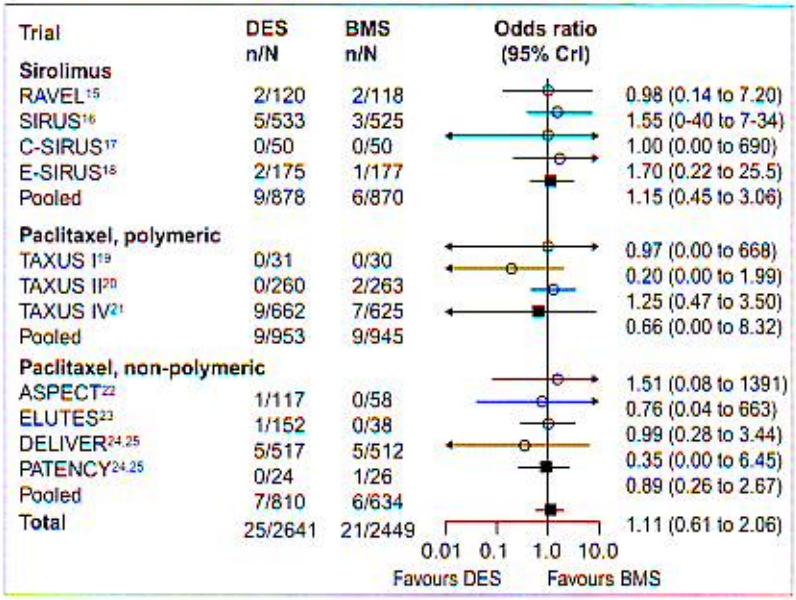
- Restenosis- 10%(simple.le)-30%(complex.less.)
- Uptill now 11 DES trial Metaanalysis > n = 5000, 1yr followup, Excluded High risk lession (Multivessel, Long lession, Restenosis. Small vessel, DM)
- No Mortality benifit
- Restenosis - 9% vs 29%
- Mortality - 0.9% vs .9%
- MI - 2.7% vs 2.9%)

  Risk of Late thrombosis due to sudden absence of Antiplatelate as prevents endothelization

❖ Not a single trial demonstrated mortality benefit for DES over BMS & even myocardial infraction

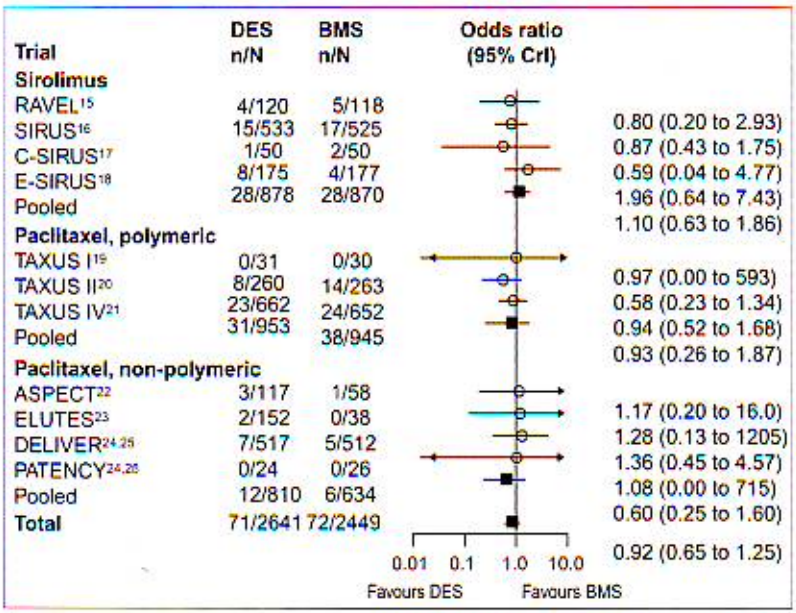
Meta analysis RCT (DES vs BMS) : Mortality (Forest plot)

(Ann Thor Surg 2006;81:1949-57 / Lancet 2004;364:583-91)



Meta analysis RCT (DES vs BMS) : Myocardial Infraction (Forest plot)

(Ann Thor Surg 2006;81:1949-57 / Lancet 2004;364:583-91)



Myths with PCI /DES

- Higher risk of Post PCI MI, evidenced by deteriration LV after repeat PCI
- Restenosis much heigher
- DES does not improve outcome over BMS although a significant restenosis reduction observed

CABG ideal in :

- LMD
- TVD
- TRUE PROXIMAL LAD
- Ch. Total Occlusion
- Impaired LV
- Need of Other Cardiac surgery

Effective in terms of :Survival, Freedom,Reintervention,Durability

Consideration of PCI/BMS/DES Still in :

- Diffuse lession
- Restenosis
- Small coronaries

WHY?

- CABG:

  CABG offers more complete revascularization with durable graft in multivessel /LMD

  Can protect development of new lession

PCI:

- Deals with immidiate culprite lession(assuming suitable)
- No protection of failed procedure
- No protection of development of new lession

No debate in 2 issues:

- Pt. wants less invasion
- Appropriate treatment modality

Keeping this in mind Physician to move ahead for operaton and patents-friendly procedure & devices with combined stategy (Hybdride revascul arization) to achieve better Goal

Stent/MIDCAB/ACAB/ROBOTICS/TMR-ALL included in common pool to avoid invasion & to achieve durable revascularization

Last Remarks

- PCI used appropriately (including subset multivessels/LMD) is worthwhile & effective
- Hybrid approach is reasonable & needs to be addressed with programmed trials
- Grafts are more durable & natural

PERCUTENEOUS VALVE REPLACEMENT PRESENT & FUTURE

- ENDOVASCULAR VALVE REPLACEMENT MAY BE A ALTERNATIVE FOR DISEASED HEART VALVE
- FOLDABLE VALVE MOUNTED ON STENTS DELIVERED CATHER-BASED TECHNIQUE
- IMPERATIVE TO PERFECTION:
  - STANT SHOULD HAVE STABLE INTRALUMINAL POSITION
  - ADEQUET HAEMODYNAMICS
  - NON COMPROMISED CORONARY FLOW

This novel concept evolved extensive animal model trial with success

RESCENT REPORTS ON PERCUTENEOUS PVR IN CHILDRTEN & AORTIC REPLACEMENT IN ADULT INDICATES

- IT MAY BE AN EFFECTIVE & VERSATILE PROCEDURE OF BENIFIT TO A LARGE POPULATION OF VALVE DIESEASES

EVOLUTION

- 1992 Attempted transluminal implant in animal-2 groups
- Anderson first demonstrared aortic in pig
  -resulted coronary flow disturbance
- Pavcnik same yr. implanted caged ball
  -failed due to escape of ball

EVOLUTION

Investigator	Stent	Complication
Anderson et al,1992	Porcine AV/Stent expandable retrograde Des.aorta in Pig	Coronary Obst.
Pavenik 1992	Cage Valve Retro.Carotid in dog	AR
Bonhoeffer 2000	Boviine jugular valve on stent-PV,Antigrade.Jugul in lamb	Pannus leaflet
Lutter 2001-2002	Porc.AV/Stent in Pig	Coronary obst.
Boudjemline 2001	Bov.Jugl.Vn/Stent to AV Retro.Carotid in Lambs	Coronary Obst.

Bonheffer	2002	Commercial Biolog.valve,Stent Antigrd.femoral PV in 7 child,1Adult	PR/RVOT obst.
Cribier	2002	Biolog./Stent expandable in Old Man,AV Antigrd.transseptal	Para valvular leak
Crebier	2004	Bov.Piericd./stent Antigrade Transseptal AV in 6 pts.1-Migration,death	Para valvular leak

PRESENT SITUATION

- CURRENT SURGICAL PROCEDURE INVOLVES CPB
- NEW INNVATION FOSTERED:MICS
  - * Roboticalley assisted Video- enhanced valve repair
  - * Sutureless technique/Annuloplasty band

BASIS OF PRESENT SEARCH

- MINIMUM INTERVENTION
- WITHOUT G.A
- COSMETIC SCARRING
- LOWER OVER ALL COST

VALVE REPLACEMENT PRESENT REALITY

- Until 1982 Surgical valvotomy was choice
- After Ist report of Percuteneous Pul.valvotony by Kan in 1982-largely replaced for better success
- Percuteneous becomes the choice of Cong. stenotic PV
- Percut.transseptal MV-tomy supplemented for most pts,who had previo -usly none.
- Works going on for 'Percuteneous edge to edge' operation for prolapse MV
- All experiment failed for obstraction/MV dysfunction
- In 2002 Crebier performed Ist AVR in Man percuteneously avoiding problems
- Successful Mid-term result achieved
- These preliminary studies suggest-Percut.valve stent in AV can be achieved with End-stage AV calcific Pts.
- Can be option in non surgical cases inspite of defects in model

LIMITATIONS : Number of difficulties

- Collapsible valve-stened can be crippled in cath.tip
- But needs large vascular access & delivery system
- Potentially limits to apply in [illegible]tric population

Vascular access:

- Impotant to select site
- Carotid artery used with high risk plaque mobilization /embolism

SIZE OF STENT

* DIAMETER MUST BE LARGER THAN VESSEL WALL-to prevent displacement
* Berbs/Sutureless tech. gaining popularity

HUMAN EXPERIENCE IN AVR

Sample size(n=11)		Detail
Clinical status	11/11 3/11 8/11	NYHA IV SHOCK CO-MORBODITIES
Approach	8/11 3/11	Ante/Fem.Vn. Retro/ Fem.Art
Tech.Failure	2/11	Migration/To cross AV
Mortality	5/11	1-Migration/4-co-morbidity
Morbidity	9/11	Para valv.leak
Follow up	9/11	Average increase Area(0.6-1.7) Decrease Gradient(50-8 mmHg) Increase LV funct. (2 fold)

Limitation....

- MAJOR OBSTACLE of stent TO FIT IN ANNULUS IN ORTHO-TROPIC POSITION (previously was heterotropic)
- ONLY HUMAN SERIES OF Cribier SHOWED THAT IT IS NO LONGER DIFFICULT WITH GOOD MID TERM SESULT
- Nobel approach to annulus - cather based ablation tech/leser (with Abla-tion chamber) FUTURE DEIRECTION-

CLINICAL APPLICATION

- EXTRAORDINARY ADVANCE GIVE CHANCE TO PERSUE INTERVE NTION-WHERE SURGERY WAS ONLY OPTION
- YET PERCUT.AV-PLASTY NOT SUCCESSFUL IN AQUTE.AS (CAL)
- CURRENT PROSTHESIS BURDENED WITH
  - * ANTICOAGULATION
  - * IE
  - * GROWTH FILURE
  - * DYSFUNCTION (AUTO REPAIR)

PERSUED : IDEAL TISSUE-ENGINEERED VALVE

- USE OF BIODEGRADABLE SCAFFOLDS SEEDED WITH STEM CELLS
- S-CELL PROLIFERATE,ORGANISE SHAPE OF VALVE, SCAFFOLD DISAPEARES
- CLINICAL EVALUATION IN PROGRESS & OUTCOME PROMI-SING FOR INTERVENTION

BENEFICIARY OF NEW TECHNOLOGY

- PAEDIATRIC Pt. WITH Pul.Regr /RVOT
- Pts. WITH AR & agressively in AS
- BESIDES CHILDREN Cal.AVD
- Pts. WITH CO-MORBID RISK SUITABLE FOR NON SURGICAL REPLACEMENT BETTER THAN PALLIATIVE BALLOB AV-TOMY

CONCLUSSIONS

- TO DATE ENCOURAGING IN ANIMAL STUDIES
- PERCUTENEOUS VALVE REPLACEMENT BECOMMING A REALITY

  PROVEN FEASIBLE & PROMISING. ALTHOUGH MANY OBSTACLES STILL EXIST.
- AVAILABILITY TO COVENTIONAL THARAPY DEPENDS ON ABILITY TO DELIVER IN HUMAN IN COMPARE TO GOLD STANDARED OF SURG.VALVE REPLACEMENT

PULMONARY ATRESIA (PA) WITHOUT VSD

Introduction

- Congenital anomally with atretic pulmonary valve along with RV & TV hypoplasia.Constitutes a spectrum of abnormality
- PS without VSD is in some part similar.
- Probably the stenosis after normal development of RV in fetal life.
- On the other hand atresia starts before RV develop normaly

Pulmonary Arteries

- P.Trunk is ususlly normal but rarely may be hypoplastic
- RPA,LPA normal/mild hypoplastic(in severe RV hypoplasia)

RV :

- Variable in size
- 5% enlarged
- Majority(60%) severely reduced in size due to wall hypertropy
- Infundibular obstraction is visible
- Hypertropied muscles are fibrotic. RV shows fibroelastosis

Right Atrium & Tricuspid Valve

- RA Enlarged particularly when TR present
- Tricuspid Valve is smaller in size.Agenesis & incomplete separation of cusps occure

PRESENTATION

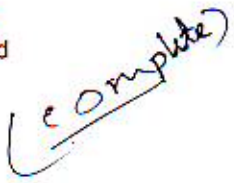
- Cyanosis in the 1st day of life
- Abscence of RV impulse at birth should raise suspicion of the spectrum
- No typical murmur
- Clear chest film with concave 'pulmonary bay' & diminished / normal vascular marking
- ECHO: USE FULL FOR DIAGNOSIS & CONFIDANT IN EXPERIENCED HAND
- Fetal echo is also diagnostic

Cardiac Cath

- Indicated for Diagnosis
- Determines Coronary Anomalies/fistulae/sinusoid
- Establishment of LV dependence is necessary
- RV pressure may be heigher/ equal to Systemic Pr.
- No procedure is standard
- Procedure is entity specific
- Transanular Patch with BT shunt – Can be done under CPB / Beating Heart
- Open Valvotomy /Transanular Patch

RESULT

- Early mortality 20%
- Result of Shunt good



PULMONARY DISEASE : CORONARY ARTERY SURGERY

Introduction

- Association of pulmonary Diseases with patients for surgery of ishemic heart diseases are not infrequent
- Smoking a important risk factor for coronary diseases is also primary cauśe for pulmonary diseases
- Management is complex due to interrelationship of cario-pulmonary physiology
- Patient selection, preperation for surgery& respiratory-anaes-thetic management is essential for good outcome

Respiratory Function:Evaluation

- Special attention to be paid for tobacco use,chronic cough, productive sputum & intolerance of physical activities
- Special exercise test(6 min walk or climbing flights with assesments of SpO2) can assists the degree of impairment
- Pulmonary function tests to identify restrictive/obstractive patern are not routine & not substitute of clinical evaluation
- FVC,FEV1, FEF 25-75% are predictors but frequently difficult to interpretate
- Preop blood gas may be necessary in increased risk of pulmonary disease
- CO_2 retention is indicator of high risk than hypoxemia

Selection of Cases

- Pulmonary disease is not a direct cause of operative mortality
- But an important cause of morbidity
- Mortality is secondary to complication of ventilation
- Timing if surgery is important
- Early surgery is indicated with sever unstable angina who does not response to medical therapy & not candidate of PCI (with respiratory insufficieny)

- These pts. have no absolute respiratory contraindication to surgery
- But Terminal respiratory disease despite critical ischemia rules out surgery
- Patients with severe repiratory disease & stable angina with non critical coronary anatomy should be medically treated with an attempt to optimize respiratory function for a later surgery
- Less sever respiratory disease need CABG as best option may have electively after optimizing lung function

Measures

- Smoking to be stopped at least 8 wks before surgery(minmum time require to improve ciliary & secretory function)
- A controlled respiratory exercise along wih control of infection is essential

Factors

- Cardiopulmonary bypass
- Lung deflation during arrest
- Blood products
- Hypothermia
- Sternotomy
- IMA hervesting – all produce pain,stiffness and increase lung water interfer with respiratory function

Caution: Maneuvers

- Carefull dissection,gentle handling of tissue, carefull chest tube placement to prevent pleural collection
- Risk of surgical procedure should always be weighed against benifit & long term survival
- In emphysema damage to lung must avoided
- Bullae may necessitate ligation /resection to prevent air leak

Goal of Management : Intra operative

- Adequet Oxygenation
- CO_2removal – to prevent MI,CCF,Pul oedema
- Restricted fluid management with diuretics (Minimum of Haemo-dynamics)

POST OPERATIVE

- IMV +PEEP can improve FRC
- PEEP >5 cm H_2O affects CO
- FiO_2 & PEEP routinely modified for SpO_2
- RR &TV are routinely modified for CO_2 wash out

(PEEP improves O_2 transfer & hypoxemia but not underlying pathology)

Physiotherapy

- No chest physiotherapy- Causes Bronchospasm (Except in Lobar atelectasis ,High Sputum)
- Recommanded nebulizer/I.V dilators

Ventilator Dependency

- A small group develops after Coronary suregry
- May be for single factor only(Heart failure)
- May multifactor be responsible
- Needs co-ordinated rehalilitation avoiding infections & optimizing nutritional status

PULMONARY STENOSIS(PS)

INTRODUCTION

- Incidence: 10% of Congenital Heart Diseases with female dominance
- Spectrum consists pinhole stenosis with severe PS with normal /dialated RV to moderate /mild PS

Morphology : Pulmonary Valve

- May be myxomatous,deformed & thickened
- In adults may be calcified
- Infundibuluar hypertropy or stenosis(secondary) may develop Pulmonary Atereries(PAs)
- Post stenotic dilatation of PT is characteristic(70% in children)
- LPA may also be dialated

Right Ventricle(RV)

- Important hypoplasia of RV is uncommon
- Hypertropy is found
- Results subvalvular stenosis (so called suicidal tendency of heart)
- Low lying stenosis is also seen.
- Valves may be normal (10-20%)

Right Atrium(RA)

- IAS intact in the patients
- PFO with R>L shunt results cyanosis
- L>R shunt results when large ASD with mild / moderate PS is present

Left Ventricle(LV)

- Mascular sub aortic /Sub pulmonic obstraction may be associated (Noonan Syndrome- when with typical facies)
- Altered LV function due to buldging of IVS

Features

- 30-40% are a symptomatic
- Cyanosis when IAS defect is present
- RV pr. is high (supra systemic)-RV hypertropy
- In older children RV hypoplasia is present when presented with cyanosis and heart failure (without evidence of HF in ECG)

ECHO: Is definative & diagnostic

Natural History

- In infant with RVOT(severe) Heart Failure (HF) is common & prognosis is poor. RV hypoplasia is predominant factor
- Mild RVOT(isolated PS)- When Pr.Gr <25 mmHg or RV Pr. 50 mmHg, normal life expectancy is seen
- Moderate RVOT (Gr. >25mmHg/ RV pr. 25-80 mmHg)-25yrs survival is expected
- Severe PS prone to RF & premature death

OPERATIONS

- Ballon Valvotomy is treatment of choice in all patients
- Open valvotomy under CPB

Result

- Early mortality is nill after Ballon
- RV hypoplasia increases risk of death particularly in adults _ HF
- Restenosis is 10% like regurgitation
- Reoperation is uncommon if properly done
- Cyanosis may persists when ASD is not closed,may be due to fibrosis following hypertropy or hypoplasia RV (non compliant RV)

INDICATIONS

- Surgical intervention is special circumstance
- In infancy PCI indicated in critical stenosis/asymptomatics(Not in mild /moderate stenosis
- Older pts. with moderate PS may not need intervention

- In hypoplastic RV in older children/adult intervention indicated unless severe HF refractory to medical treatment (These are high risk group)

RV hypoplasia with PS (in Children /Adult)

- Ballon is not effective
- Organic infundibular obstraction present
- ASD closure necessary
- Enalargement of RV cavity by surgery is beneficial
- HF carries poor result in these cases
- BDG Shunt is usefull to unload RV

PULMONARY STENOSIS (PS) WITHOUT VSD

Morphology

Pulmonary Valve

- Fibrous & stenotic
- Due to increased myxomatous tissue

P. Arteries (PAs)

- Moderate /Severe Hypoplasia - due to reduced flow

Right Ventricle

- Concentric hypertropy
- Increase number of myocytes
- Diffuse fibrosis is prominant
- Raerly enlarged.When enlarged associated with myopathy & critical
- Incontrast to PA,hypertropy is secondary to stenosis (after development of RV)
- PFO with L>R shunt

Associated lession: UNKNOWN

ECHO:

- Near Definite dignosis can not be done

CINE ANGIOGRAPHY:

- Provide orecise details

NATURAL HISTORY:

- Die without treatment within few days to months

OPERATION

- Ballon valvotomy
- Closed Valvotomy / Open Valvotomy

  (with/ without CPB & Cardioplegia)

  A B.T shunt may be an adjunct if PaO2 <30 mmHg after CPB & PGE1 infusion

TRANSANULAR PATCH

- May be needed when RV cavity is small
- At primary operation when Z= <-4, it is better option

Post Operative Care : VALVOTOMY

- PGE1 stopped after hours on ventilation
- If after 24 h PaO2 >30 mmHg, stable haemodynamics found ,Pt. is extubated
- If PaO2 <30 mmHg & if residual PS mild/absent - a BT Shunt to be done
- If important stenosis is present Transanular Patch with BT Shunt is choice
- It is followed for peak Pr.gradient (RV-PA)
- BT shunt may need to be closed at followup to prevent Heart Failure

RESULTS

- Early Mortality 6%,comparatively favourable with Ballon Valvoplasty (8%)
- RV enlargement with Myopathy(Genetic) is lethal risk factor for death
- Surgery without BT shunt is a risk factor for death when anular hypoplasia is present
- Without shunt Pt becomes hypoxic & prone to marked R>L shunt through PFO due to acute RV failure
- Rerely 2 ventricular circulation is attainable.Fontan ultimately is required besides repeated Ballon & all surgical options

SURGRY OF: ABNORMAL ORIGIN OF CORONARY ARTERIES

CA :Connectiong to Pulmonary Arteries

- LM or any branch may arise from proximal pulmonary trunk
- RCA communicate LCA & drain to PA
- Rarely both CAs connect to PA by single trunk

Embryogenesis

- Proximal CA grow from the peritruncal area into aorta & maat at a single orifice for both CAs
- Abnormal LCA ariese above the left or Post cusps
- Cx may arises fr9m Lpa
- LCA,RCA WITH SINGLE STEM CONNECT PULMONARY TRUNK/Rt. PA
- LCX/LCA may arise from Rt. PA

Indication : Operation

- Infants are seriously ill & urgently required
- Diagnosis is indication of Surgery in adults

Operations

Under CPB

Cardioplegia to aorta & PT

- 1. Tunnel operation
- 2. Left Coronary Transfer
- 3. Subclavia to LCA anastomosis
- 4. CABG

Result : Prediction valueless because of small reported series

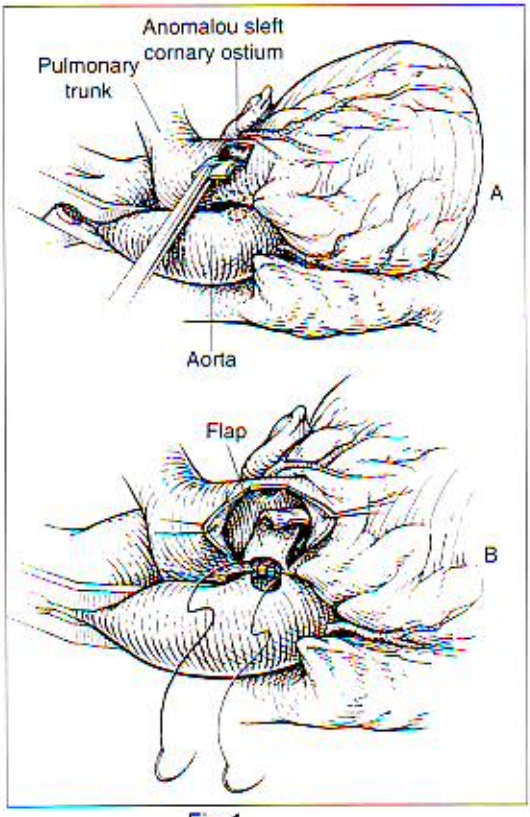

Anomalou sleft
cornary ostium
Pulmonary
trunk
A
Aorta
Flap
B

Fig-1

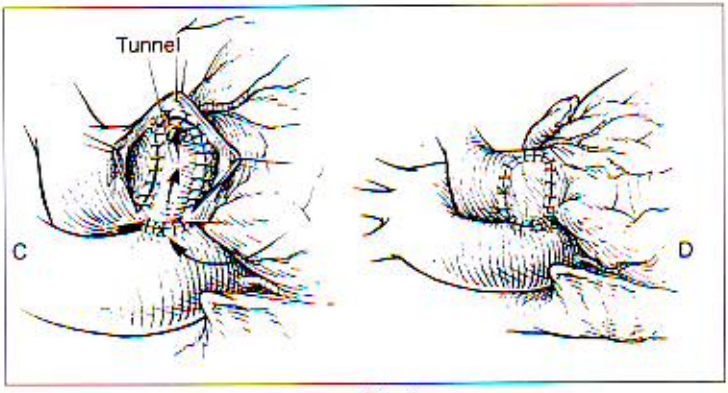

Tunnel
C
D

Fig-2

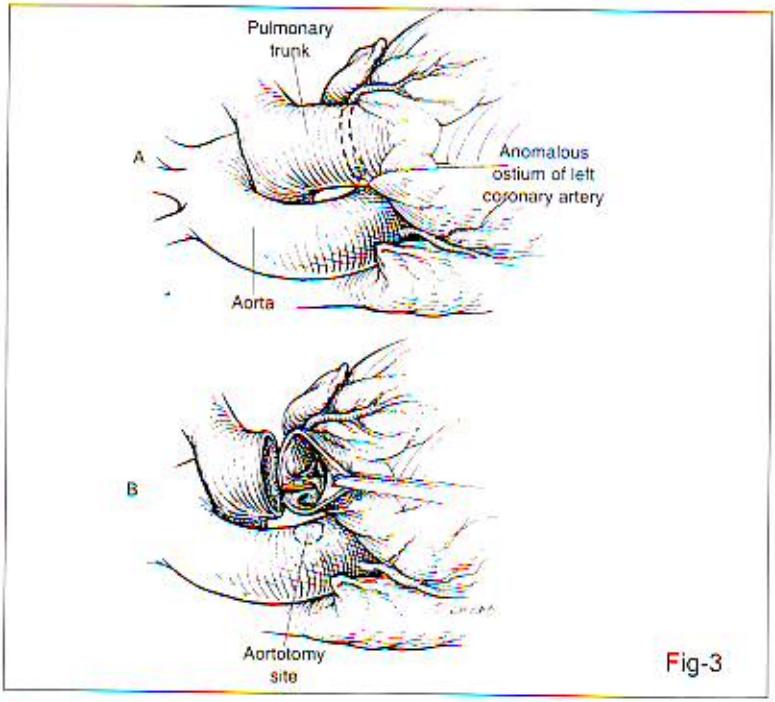

Pulmonary trunk
A
Anomalous ostium of left coronary artery
Aorta
B
Aortotomy site

Fig-3

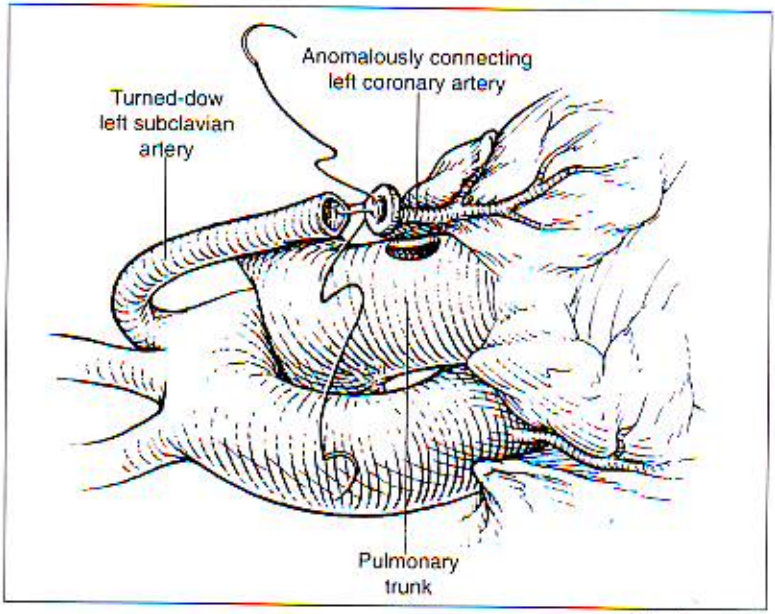

Anomalously connecting left coronary artery
Turned-dow left subclavian artery
Pulmonary trunk

Fig-4

Abnormal LMA to Aorta

- LCA/RCA to a area other than Lt /Rt coronary sinus
- Results compression between Aorta & PT

Operations

1. CABG
2. From with in the aorta re-position
3. Reconstructive surgery(Intramural cases.RCA in LCA sinus)- by unroofing the tunnel & placing the osteam at rt. sinus

Coronary Arteriovenous Fistula

- Direct communication between a coronary artery & chambers (any) of the Heart or any veins arround the heart
- Probably represents persistant embryonic trabecular space

Site : Morphological

- RCA 50-55%
- LCA55%
- Both 5%
- Usually CA & branches are normal
- May beend to side or e to E anastomosis
- Dilatation/Aneurysmal appearenceof the involved CA found
- Rupture is rare

Opening

- 90% Rt sided chamber (40%RV,25%RA,20%PA,1% SVC,7%CS)
- Results L >R shunt. Qp: Qs >/=1.8
- Lt sided fistulae have no L>R shunt. Only Closes on systol giving overload to LV (Arterio arteri al fistulae)
- May be multiple in rt. Side
- single in lt.side

Associated lessions

- May be associated with other lessions
- Mostly isolated

Diagnosis:

- Mostlt accedental/ may present symptomat late in life.
- MR is probably associated with Ischemia

ECHO:

- Dilated vessels are present
- Large fistulae can be demonstrated

Angiography

- Selective procedure is necessary for definite diagnosis

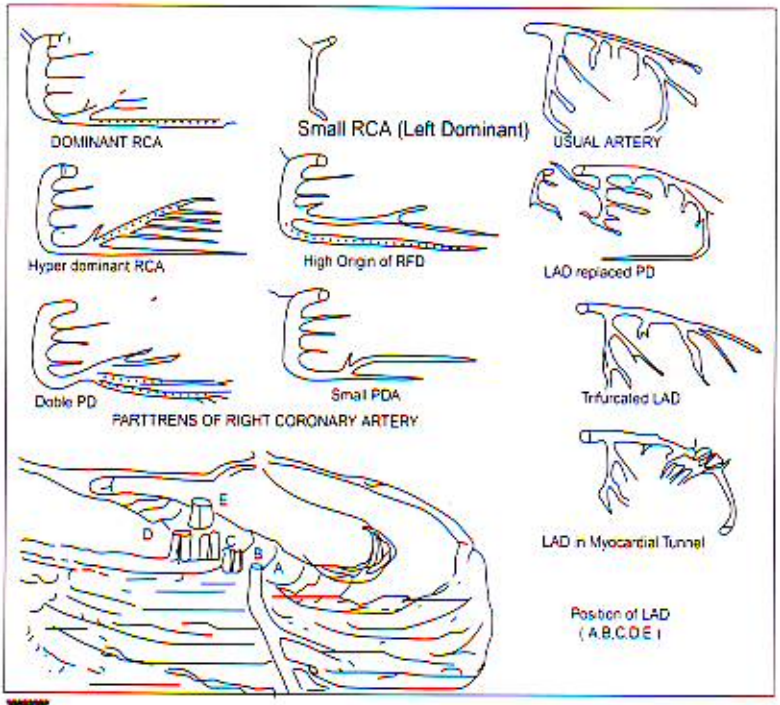

DOMINANT RCA
Small RCA (Left Dominant)
USUAL ARTERY
Hyper dominant RCA
High Origin of RFD
LAD replaced PD
Doble PD
Smal PDA
Trifurcated LAD
PARTTRENS OF RIGHT CORONARY ARTERY
LAD in Myocardial Tunnel
Position of LAD
(A,B,C,D,E)

Fig-5

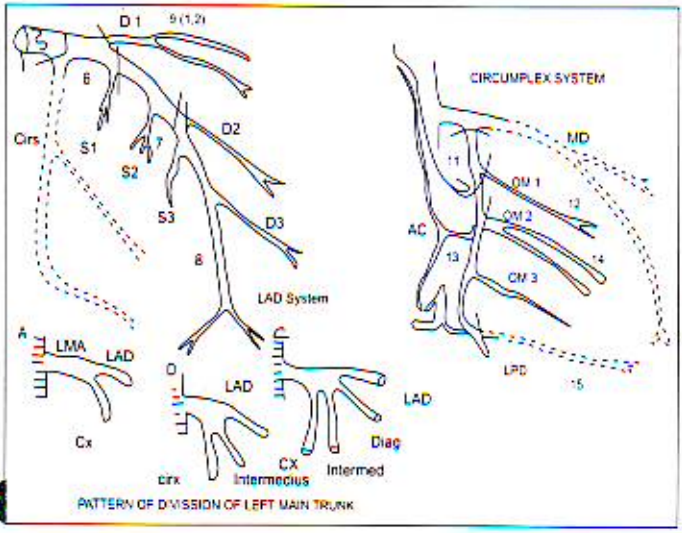

D 1
S(1,2)
6
Ors
S1
S2
7
D2
S3
D3
8
LAD System
A
LMA
LAD
Cx
B
LAD
crx
C
CX
Intermedius
LAD
Diag
Intermed
CIRCUMPLEX SYSTEM
MD
11
OM 1
12
OM 2
AC
13
14
OM 3
LPD
15
PATTERN OF DIVISSION OF LEFT MAIN TRUNK

Fig-6

Operation

- Both with /without CPB
- Closure/repair may bedone
- Giant aneurysm - Excission & reconstraction of the artery
- If distal is good - CABG

TECHNIQUES IN SURGICAL CORONARY REVASCULARIZATION

- Surgical management of atherosclerotic CAD evolved for stable coronary syndromes, primarily
- At present mid sternotomy with total CPB mostly used technical procedure
- For its more than half century extensive experiences all other technics to be compared with it.
- Extensive invasineness, Post op complications, Complications of CPB, Ventilation draw attention for others.

Other Techniques

1) Endaterectomy

- Some use frequently in distal RCA
- Most prefer this artery to use distal graft
- Some continue to use in long segment even in LAD & report good
- Lower patency reported (64% Vs 92%)
- Some reported favourable patency result
- Frequent periop MI & complete revascularization is controversial

2) Angioplasty

- Abandoned years ago
- Some used for LMA,RCA ostia

  -Under CPB &anterior approach the ostia incised with aorta & LM.Enarterectomised & patch repair done(pericardiun/ sephanous Vn)

Contraindication: Calcification,Old age

3) Transmyocardial Leser (TMR)

- CO_2 or Holmium laser used

- Mini thoracotomy
- 10-15 channels in myocardum created
- Without CPB
- Done in primary/Re-do CABG with CPB or with OPCAB
- Areas not correctable by bypass graft
- Mechanism remain unclear
- Improves symptoms but does show improvement in perfusion/LV function
- Mortality 5-10% in selected pts.

4) Gene therapy

- Genetic manipulation to enhance microcirculation of ischemic muscle/ Proliferation of endutheliun
- Vascular Endothelial Growth Factor(VEGF)-Direct injection through mini Thoracotomy/ during CABG(Hybrid)
- Human fibroblast growth factor(FGF)- used similarly
- DecoyOligonucleotide-block gene transactivation to prevent hyperplasia and atherosclerosis,used transfection of vein graft to prevent primary graft failure

Currently used technics :

- OPCAB - Sternotomy without CPB
- MIDCAB-Thoracotomy/Sternal/Parasternal with/without use CPB
- ACAB – Thoracic epidural without Mechanical ventilation & CPB
  - Epidural with normothermic CPB & without mechanical
- Heart Port – CPBwith Endoscopic (ports) for LIMA/Vn conduit & direct vision Anastomosis through mini incision
- Robotic CABG-Robotic consle with chest ports(3)to harvest LIMAs & direct thoracotomy approach with Stabilizers for anastomos is.

Thoracic Incissions:

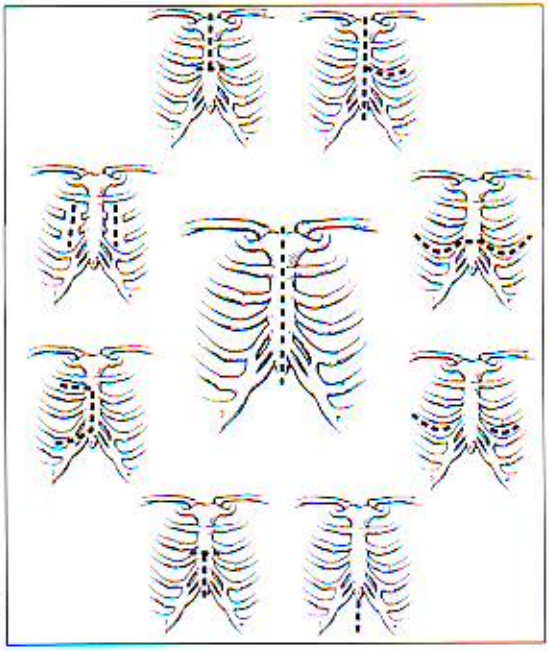


Fig-1

OPCAB

Inclusion criteria of patients for OPCAB

1) Patients with suitable anatomy of epicardial non-calcification vessel >1mm
2) Patients without history of failure or evidence of moderate to severe cardiomegaly.
3) Patient considered high risk for CPB with comorbidity were included.

Excluded patient for OPCAB

- Patients with Intramyocardial vessel / with arrhythmias and/or haemod-ynamic instability
- Surgical treatment of coronary artery disease is constantly undergoing alteration and refinement to meet the clinical challenges brought on by an aging of population, recurrence of disease and ever increasing competitive age of PTCA and stenting

- In addition conventional CABG by midline sternotomy is a gold standard but comparatively a bit extensive surgical procedure
- To meet the challenge the fast tract approach to early extubation and less expensive CABG have became popular and there is increased focus on the less invasive surgery. Now OPCAB accounts 20% CABG in US and West Europe
- LAD >1.5 mm in diameter
- Presence of a tubular heart on chest radiography
- Thin chest wall with wide intercostal spaces
- Reoperation coronary artery bypass with deterioration saphenous vein graft
- Totally occluded LAD with good clinical collaterals to distal LAD

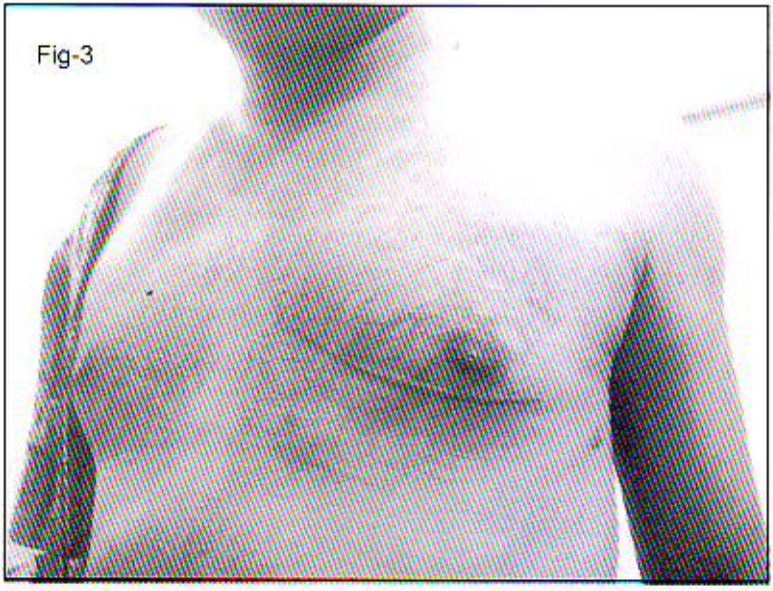
MIDCAB / OPCAB

Contraindication for MIDCAB

- Small sized coronary artery target site (<1.3mm)
- Diffuselly calcified coronary arteries
- Intramyocardial LAD coronary artery
- Stenosis or occlusion of left subclavian artery

Awake CABG:

Fig-2



ACAB

- Off-pump CABG (OPCAB) was introduced to reduce surgical trauma by avoiding extracorporeal circulation(ECC). High thoracic epidural anaesthesia(TEA) reduces intraoperative stress and postoperative pain .
- This allows Awake coronary artery bypass graft surgery(ACAB) avoiding the drawbacks of mechanical ventilation and general anesthesia particularly in high-risk patients.
- High thoracic epidural anesthesia (TEA) leads to stress-response atten-ution, intense perioperative analgesia, cardiac sympatholysis and thus improved vascular graft bloodflow, and improved postoperative pulmonary and gastrointestinal function.
- Patients with certain risk profiles, including chronic obstructive pulmonary disease, coagulation disorders, and aberrant neurological conditions, get benefit from operations without cardiopulmonary bypass

- TEA reduces hemodynamic compromise as a result of narcotic med-cation before intubation in some patients.

- Complete median sternotomy :

  The chest opened with an standard pneumatic saw and particularly careful LIMA dissection necessary to avoid pneumo-thorax in the spontaneously breathing patient.

  After dissection of the LIMA in conventional technique without opening the pleural cavity, the pericardium opened.

- Left anterior mini-thoracotomy/ antro-lateral thoracotomy: Left thoracotomy made through a incision in left 5th intercostal space. LIMA harvested. Pericardiotomy done vertically and parallel to the phrenic nerve.This procedure used when only the left sided grafts implanted.

TOF WITH PA

Defination

- Tetralogy of Fallot with no luminal continuity (Extream form of displacement of Conal septum).
- May be acqiured also

MORPHOLOGY

(TOF with PA - congenital)

- Differs from TOF with PS
- No flow crosses from RV to lung
- Lung flow through PDA,Collaterals or fistulae
- Pulmonary artery(PA) anonalies are common
- Aortopulmonary collaterals are common

RVOT

- Atresia may be at anulus or infundibulum
- Infundibular lession is common(70%). May be totally absent
- VSD is large
- Massive RV hypertropy is present
- In anular atresia infundibulum is present but hypertropied

Pulmonary Trunk

- It is hypoplastic,may be a cord or absent (5%)

Right /Left PA

- 20-30% patirnts have discontinuity(non confluent)
- Usually absent central part of one / two arteries
- May be distal MPA or origin of LPA,RPAs are atretic
- RPA origin involved in (10%),LPA origin (20%)
- TF with PS rerely have these anomalies
- Confluent RPA, LPAs may have patent / atretic trunk

Arborization of PA

- Failure (frequent) of PA to distribute 20 pulmonary segments
- Patients with PT,normal cofluent RPA,LPA and valve atresia get flow from PDA. Rarely distribution abnormality found.Also rarely have Large collaterals
- Complete distribution found only in 53% with confluent RPA,LPA (with normal calibre)
- 80% nonconfluent/confluent but hypoplastic PAs have incom-plete distribution of one or both branches (1/3rd have <10 pulmonary segments supplied by PAs)
- Disconnected segments are supplied by AP collaterals (large)
- Most patients with TOF with PS have 20 segments central or proximal PA connection.

Stenosis of PAs

- Stenosis after origin of PAs are found in some percentage of cases
- Flow increases if pressure is increased by palliation

Size of PAs

Prebranching size varies widely

- TOF with PA have samaller size than TOF with PS (also varies in normal individual)
- Hilar branching is abnormal with large AP collaterals

Alternative sources of Pulmonary Flow:

- 2/3rd TOF with PA have large Collaterals (embryological)
- AQUIRED COLLATERALS ARE SMALL & NUMEROUS
- AP collaterals are large & 1-6 in numbers from upper/mid desc-ending thoracic aorta
- Course is curve & terminate at interlobar/Intralobar PAs that dis-tribute normally
- These are prone to stenosis (60%)
- When join end to end these become histologically PAs

- 50% join end to side at hilar complex junction
- In some cases does not communicate wth central PAs
- May terminate end to side at central pulmonary arteries
- Rerely single collateral at each sideconnect end to side with hilar arteries
- AP collaterals are present when there is abnormal distribution & assoc-iated with stenosis /dysplasia of PAs

Para mediastinal collaterals

- Mimics BT shunt
- Arrises from Rt/Lt subclavia opposite the aortic arch
- Connects end to side to central/hilar arteries
- Found in TOF with PAs

Bronchial Collaterals

- Both in TOF with PA / PS are present
- These are diffuse network
- Intercostal collateral / Coronary collaterals
- Are also fond in association in PAs

Ductus Arteriosus (PDA)

- Size & position differs
- May arries from subclavia /other aberrant origin
- When PA confluent with large collaterals usually no PDA found
- When non-confluent PDA may/ may not be present
- When PDA is present supplies the whole single lung & gets no collaterals
- Opposite lung has collaterals

ACQUIRED PULMONARY ATRESIA

- Sponteneous after birth in TOF with PS
- Usually valvular or infundibular
- May be stimulated by palliative operations

PRESENTATIONS

- When present TOF with PA cyanosis is evident during 1st days of life & progressive
- If large AP collaterals /PDA present cyanosis is mild
- Some presents with Heart Failure

ECHO

- Diagnostic but definative only if PDA & confluent normal pulmonary arteries are present

Angiography

- Should be performed at birth in all patients of TOF with PA & no duct dependant flow
- Origin ,flow & distribution are assesed along with extra/intra paranchymal (lung) stenosis
- Identification of true central arteris by Pulmonary Wedge Injection of dye is necessary
- Only after these assesment true surgical plan is possible
- Management may need repeated angiography even in months to identify development & further action
- Clinical stability (thriving infant, SaO2 80±5%) is not a criteria of non invasive investigation
- Stable TOF with PAs may be at risk of overcirculation of some lung segment & collapse of others
- Repeted Cath not necessary in Duct dependent Confluent PAs
- Absolute necessity of Angio in TOF with PAs in pre-op work-up.
- New additions are 3D-CTscan /MRI may in future be an alternative in dianosing Complex HD with Pulmonary anomalies
- Ecdo has limited subordinate role in assesing arterial details

Natural Course of Disease

- Varing morphology changes the courses
- Has short lasting early death phase

- May survive 50yr (constant hazzard to late phase) is disputed
- No morphology yet identified tha corresponds to early death.

❖ Confluent normally distributrd to pul.segments +PDA

- Belongs 50% patients.PDA closes slowly
- Half die by 6 month with hypoxia
- 90% die by 1 yr

❖ PDA + confluent LPA,RPA with normal distribution :

- INCLUDE 25%
- With out intrvention 10% die in few years, 90% by 10yrs

❖ Confluent/nonconfluent RPA,LPA supprting minority segments:

- Belongs 25% with AP collaterals to majority/all segments
- Large Qp at birth presents with HF
- Good health upto 15 y than DEVELOP CYANOSIS
- Die by 30 yrs (Eisenmenger like)

PULMONARY ARTERY DISEASES

- Differs than TOF with PS in this subject
- Due tro high registancer for hyhypoplasia/stenosis of the distal arteries/ collaterals (Not like hypertensive vascular diseases)
- Limited to central segments
- Also in Ap collateral segments in fetal life
- Develop haemoptysis in 2nd /3rd deacde with large APC if not intrvein

OPERATION

- TOF with confluent normal RPA,LPA distribution +PDA:
  - Neonats / Young Infant / Older infant / Children (if shunted)
  - Anular atresia, MPA, confluent RPA, LPA good

  SURGRY SIMILAR TO TOF WITH TRANSANULAR PATCH
- TOF with PA + large APC

– No single operation is applicable (although claims available)
– One stage (Unifocalization + ICR)
– Infants (3-4m)
– Neonates (unstable)

PROCEDURE

- STERNOTOMY
- DISSECTION MOF THE PA TOM THE PARANCHYMA
- DISSECTION OF THE APCs
- CONTROL OF ALL SOURCES OFPULMONARY flow before CPB
- CPB at 34°c with strong LV vent
- Unifocalization

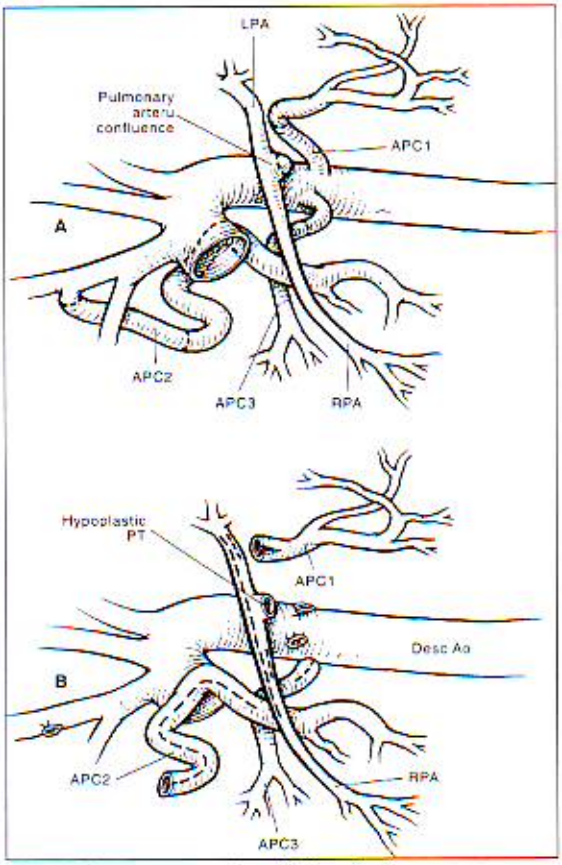


Fig.1: One stage complete Unifocalization

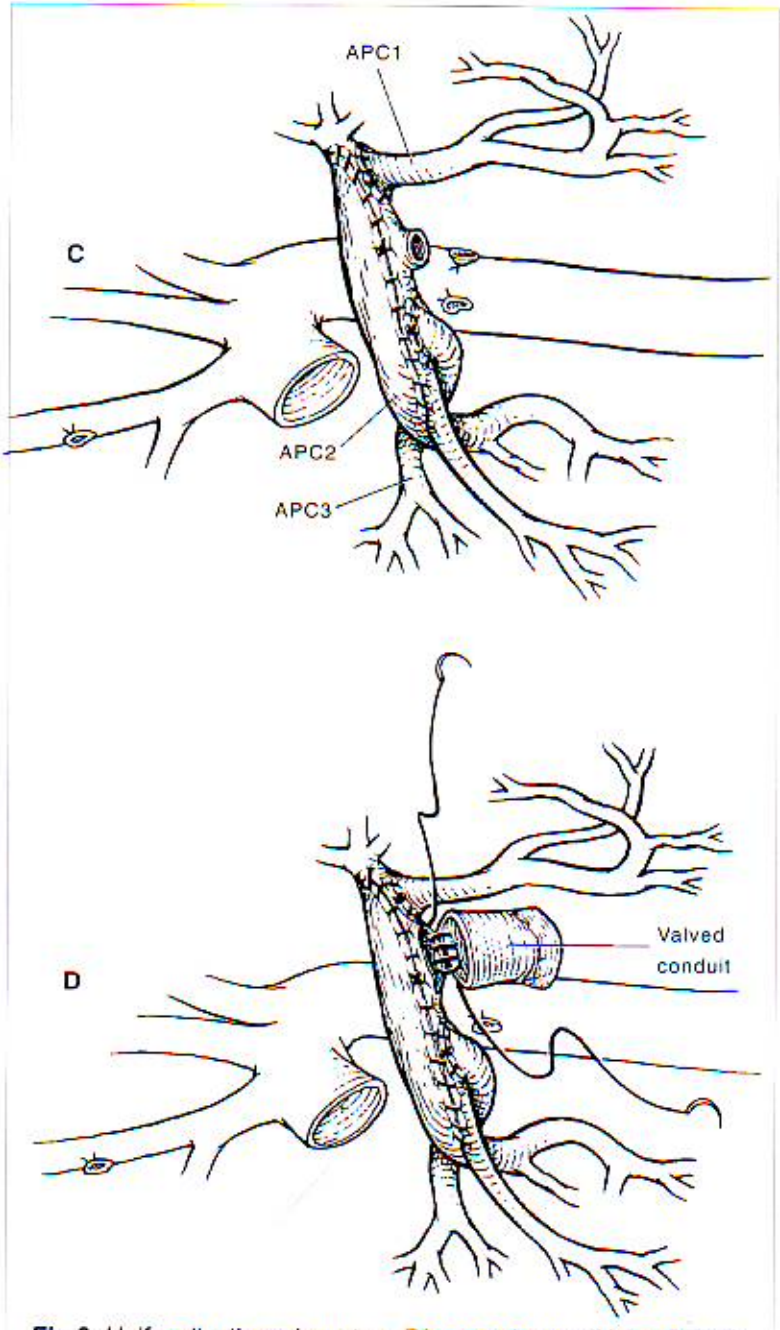


Fig.2: *Unifocalization where true PAs are abscent(Using PTFE)*

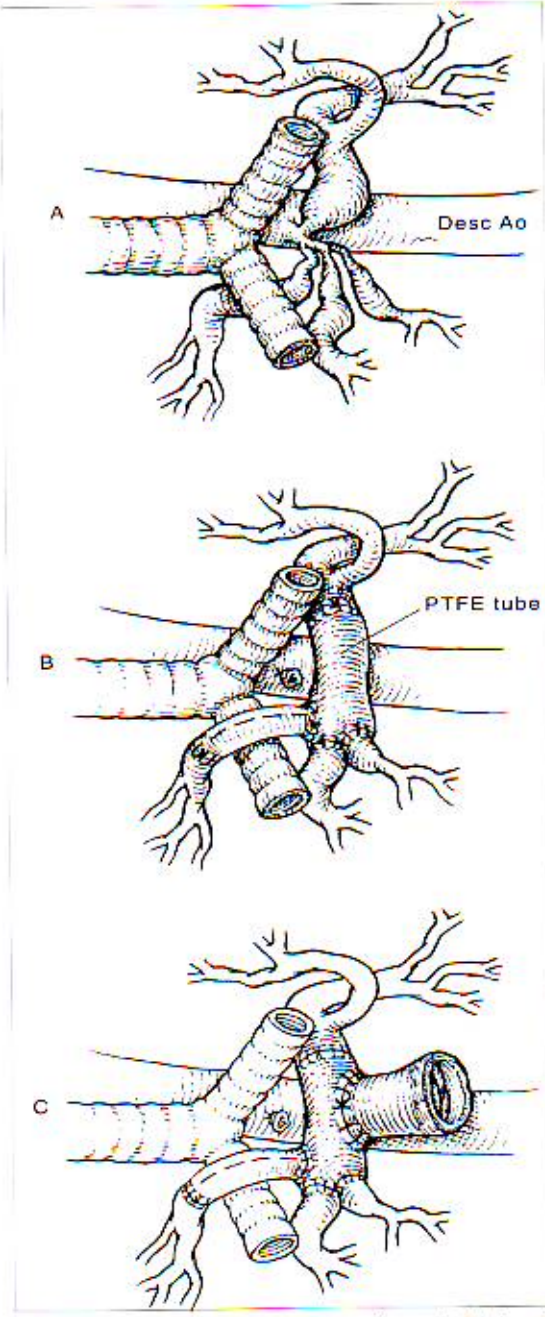


Fig.2: Unifocalization where true PAs are abscent (Using PTFE)

Assesments

- RV Pr <60% LV Pr
- Ifnot unifocalization (Intracardiac) sufficient to low RPA (based on expe-rience) >>Decide to move wheather ahead for ICR or not

Preoperative Assesment

Intraoperative

- Perfuse seperate cannula (vn) TO newly UNNIFOCALIZED SYSTEM
- Controlled perfusion 20-100% CO (CI) done
- APC Pr measured
- If PPA <215-30 mmHg, (PRV/LV <0.5)full CO than ICR possible
- If PPA >30 abandand the ICR
- AO to APC shunt created

Intracardiac Repair (ICR)

- If RPA is accepetable>> with valve conduit(allograft preferable)

Assesment

- Measure PRV?LV (accepted <0.9)
- If >0.9 than VSD patch perforated (Prbability of death conside-rable)
- Close as usuaL

Indications

- Not certain in all grouph for pt.specific risk factors
- Normal RPA, LPA +PDA
- One stage repair indicated
- 75% are alivewith good health for10 yr
- Promary shunte Pt. are also in this catagory
- Neonates aftre iy with APCs is also indicated

Arborization defect

- Rerely achieve adult age with out intrvention
- 1stage / 2stage operation not yet evaluated

2 stage Unifocalization

- Indicated in 10-20%with APCs
- No pulmonary 'Raw Materials' found (underdevelop PAs/ collaterlas)
- Lateral thoracotomy with single lung unifocalization to Ap shunts are choice
- Similarly at the contra lateral lung at next time
- 3rd stage mid sternotomy & recontraction of PA

Note

- Wide different of attemt are found for these Pt. among experience Surgeons
- COMMON APPROACH
- Provide flow & pr in cental artery system when confluent pulmonary arteries are hypoplastic
- Syt-Pul Shunt
- RV-PAconduitleaving VSD open
- Modified BT Shunt

CONCERN

- WHEN ARBORIZATION IS LIMITED & COLLATERALS UN ATTENDED
- DAMAGE TO LUNG FROM OVER CIRCULATION IS INEVITABLE

ALTERNATIVE APPROACH

- Confluent + normal Rt&Lt PAs + PDA
- 80% survive complete repair at early life
- ICR as early as possible & indicated
- If with palliation also possible with transanular patch
- But if conduit appears to be needed with good palliation than deferraluntil 3-5 m is advisable
- Confluent Rt & Lt PAs to majority but not all segments:
- When > 15 segments are connected to central artery unifocalisation is not indicated
- These pt needs valved condit at 3-5y
- A primary Sys-Pul Shunt at 3-4y (when with reasonable PAs size but with cyanosis& s/s and no more that 2 AP collaterals are present)

- When Transanular patch is feasible theas early indicated
- When found APCs can be closed without jeoperdising lung segment than PCI closure 2-3 days before can be done
- If PCI not possible than to be done at the time of operation
- When 11-15 segments connected to cental arteries there is risk of post of PAH & increased surgical mortality
- These have > 2-3 APCs
- Than 3 Optións available to surgeons:

❖ 1. Treat as having > 15 segments connected centrally

❖ 2. Treat as like 10segments connected centrally (vide infra)

❖ 3. Treat palliatively to accept natural history(detemined by flow of collaterals OR aorotopulmonay shunt may be made but not desirable

- CONFLUENT / NONCONFLUENT DISTRIBUTING TO MINORITY SEGMENTS (<10-12) :

❖ These pts can be consider primary ICR at any age dur incompitable high arterial/vascular resistance incompitable to survival

❖ Multi stage procedure with placement of valved conduit with PCI closure/surgical closure of APCs

❖ As musch possible collteral are to be centralized

❖ Than after 6-18m or at 3 yrs Repair is done if with reasonable chance of success

❖ These procedure to be undertaken only in centres properly prepare for this work

- Insfficient data available of all these procedures to say one is optimal & acceptable

Percuteneous closure of

Large APCs

- With precaution effectiveness & safty demonstrated
- Wire coil with uniformly coated thrombogenic decron strands occlusion by thrombosis usually appears with in 10 min

- Complete occlution 70%
- Other devices alkso applicable.

CAUTION

- A LARGE APC SHOULD NOT BE CLOSED SURGICALLY OR PERCUTENEOUSLY WHEN IT CONNECTS END TO END TO DISTAL PULMANARY ARTERIES MAY BE THE SOLE SOURCE OF FLOW TO ONE /MORE SEGMANTS
- IF NOT SO, YET IT IS TO BE PROVED THAT ITS PRESENCE IS NOT IMPORTANT TO MAINTAIN REASONABLE PaO_2

TOF with PS

Defination

- Malformation due to underdevelopment of RV infundibulaum for anterior & leftward displacement of conal septum & its extension. With concordant Atrioventricular connection& bi-ventricular origin of aorta (Overriding).

Morphology : RVOT

- Stenosis is hallmark of TOF
- Conal septum displaced leftward &anterior to the septal band(ant.division),not in between two division
- Conal septum merges with the free wall in sagital rather coronal plan
- When narrowing localized (Os infundibulum)–72% in transverse line,may present infundibular chamber (3rd ventricle)
- When absent(in infants) a tubular narrowing through out OT

Pulmonary Valve

- Stenosis found in 75% TOF
- 3 cusps are fused in non-stenotic
- But in both area is smaller than aorta
- Valves may be vestigial & ring stenotic or non stenotic
- If no valve with regurgitation & annulus not narrowed Known as TOF with absent Pul. valve
- Narrow valves are tethered/no fusion
- Pulmonary valves can not open & pull down the walls giving localized narrowing

RV –PT Junction

- Almost always smaller than than aortic annulus may or may not be obstructive
- If small obstructive ,associated with diffuse outflow hypoplasia
- Low lying infundibular stenosis does not show junction. May not be obstructive.

Pulmonary trunk

- Always smaller than Aorta
- Less than half and short
- Usually hidden by the Aorta
- When Valves are tethered ,the Trunk too (corserted)

Bifurcation:

- LPA direction continuation with trunk
- RP at right angle
- It may be Y shaped and narrowed

RPA / LPA:

- Abnormality common with TF with PA
- Uncommon ToF with PS
- Any anomalies Of PA may be in PS

Distal arteries and Veins:

- Pulmonary artery /vein beyond hilum normal in most cases
- Parenchymal arteries are smaller and thinner
- Lung volume and alveolar size and total number tends to be less

Pulmonary Pathway:

- Hypoplasia marked in infundibular and PT
- On an average RPA LPA are not abnormal. Though origin of stenosis may be narrow
- Great variability in dimention is present & should be carefully studies pre operatively.

VSD

- Large VSD is juxtaaortic (Membranous/conoventricular)
- Malalingment of infundibular septum
- Due to anterior displacement of conal septum at the base of IVS
- Parietal band is displaced toanterior& leftward.(Makes part of VSD heigher to get before deviding the band)
- Posterio-superiorly NCC bounds the defect

- Posterior margin is variable
- Inferior formed by septal band
- When conal septum abscent VSD is large & Juxta arterial
- Aortic & Pulmonary valves are separated by a thin fibrous ridge
- This is morphologically DORV with doubly commited VSD (juxtra arterial)
- Variation : Multipe VSD,Inlet septum VSDs are also available

Conducting System:

- Position is normal
- Course similar to perimembranous VSD
- Follows inferior margin of VSD to left side
- When aorti root rotates more clock-wise the His bundle comes on margin of VSD
- When muscle ridge is present on margn,safely suture can be placed

Right Ventricle

- Sinus of RV is large dye to hypertrophy resulting clockwise rotation of LV
- RV trabeculae are propinant resulting reduced EF & EDV of RV

Left Ventricle

- Usually normal
- Uncommonly LV & MV is hypoplastic , and contradicts repair

Coronary Arteries

- Conal branch may cross RV wall
- LCA may arise from RCA (5%) & cancross RV wall

AORTA

- Origin is bi-ventricular & anteriorly placed due to rotation & overridding
- Overridding may be 30–90% (Generally 40%)
- Clockwise rotation carries NCC right way & superior at the posterior superior margin of VSD, may be away from the base of the AMI, (aortic curtain) & continuity is lost & lies below the conal septum

- LCC moves resulting continuity with the AML
- Simulteneous movement of RCC (posterior superior) to the left brings it at the anterosuperior margin of VSD
- Degree of rotation relates to the degree of RVOT development & malalignment of infundibular septum
- If rotation is minimal (Low lying stenosis)
- If extrem becomes dextro position,called DORV with PS
- Overriding is obvious in Cine angio (80° LAO, 40° RAO)
- If AR present aortic root may be enlarged/ dialated

ASSOCIATED ANOMALIES

- PDA
- Multiple VSD
- CAVSD

Presentation:

- Infants with defuse hyoplasia does nat develop failure but breathlesness on feeding.No hypoxic spell
- Infants with dominant infundibular stenosis cuanosis delayed & spell may occur on spasm
- Infants (10%) with purely infundibular stenosis needs surgical care with large VSD & pulmonary plethora with heart failure at 2-3 month with increasing cyanosis & spell
- A minor group with minimal R > L shunt, mild stenosis or predominant L > R shunt is acyanotic at rest & without spell presents at 1st /2nd decade with increasing symptoms
- Severe cyanosis leading to crerebral thrombosis with ploycythemia can get hemiplegia at any stage
- Failure to thrieve & respiratory infection is less marked than isolated VSD

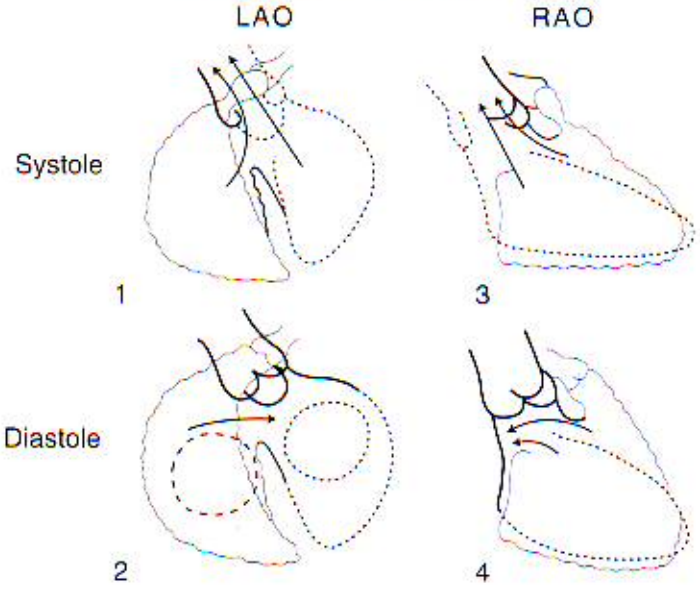
ECHO:

- VSD, overriding, RV infundibulum are well deliniated in echocardiography with experienced hand
- Morphologic details of pulmonary trunk,LPA are not reliable in these study
- For risk if invasivenee in sicks Echo can be a definite diagnostic tool

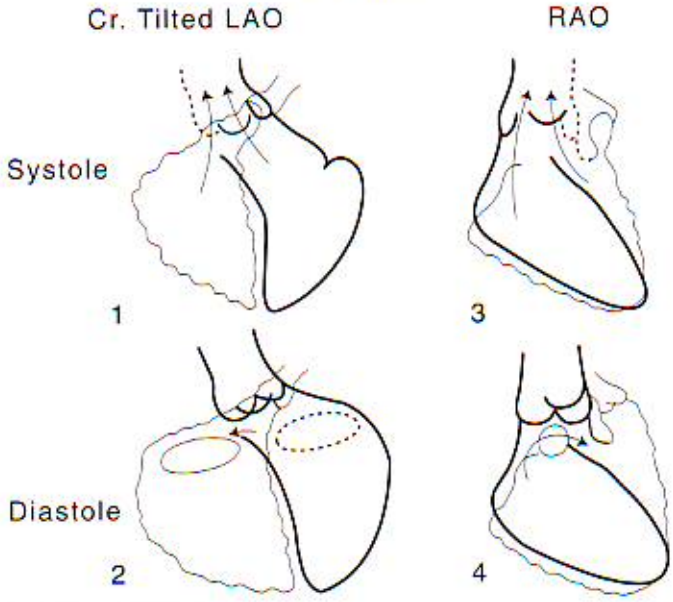
Card.Cath & Angio:

- Expertly demonstrated echo can precisely describe morphology & haemodynamics
- Biplan cine can give morphologic details.Oblique &angle views are used(70-80° LAO,30-40°RAO in RV-graphy,50-60° LAO with 40°Cr.tilte in LV-graphy)
- Sitting up (Cranianlly tilted frontal view) gives origin of PA branches view well
- Presence, size and different portion of RPA/LPA are viewed critically
- AP collaterals are selectively studied if present
- If LPA,RPA not seen then PV wedge injection for retrograde filling of pulmonary tree is to be done to mark absent central pulmonary vessels

Cine profile (RV Injection)



Cine (LV Injection)



COURSE OF DISEASE:

- 25% die in 1st year/month with severe pulmonary obstraction
- 40%,70%,90% die by 3,10 40 yrs respectively
- High risk at 1st yr & after 25yrs again risk rises
- Few survive 4th & 5th decade & die to Myopathy

OPERATION:

- Ensure Excellent Exposure(EEE)
- Avoid damage to CA
- Avoid damaging RV band
- Must relieve RV hypertension cutting parietal,septal &crista
- VSD must be closed completely avoiding damage to conductive system

Transannular Patch

- Not necessary when z value >(-3), preoperatively
- If VSD subarterial chance of Patch is high
- Reasses with Hagar after VSD closure,passes through easily

- During patching distal level of incision is important to reduce gradient
- If distal PT is narrower than annulus than patch should be upto LPA & may need to be extended to mid LPA
- Important consideration is the proper sizing of the patch with a 'square cut' distal end
- Too large patch increases regurgitation
- Roof of the neo-PA/Annulus & its sizing carefully judged,not to make a larger than the diameter of the ascending aorta(3/4th).
- Aleternately placing the measured Hagar on the open annulus width of patch require to make roof over it is measured(Usually 50X15mm) with both end transversely cut.
- A monocusp may be attached while roofing

Measuring PRV/LV

- After repair ,with the canulae in position PRV/LV is measured
- If transannular patch is not used & ratio >0.7,CPB restarted & patch is used
- If patch placed & ratio>0.8,localization of area of gradient in RV to be done
- If gradient identified between sinus RV& distal end of the patch, must be corrected
- If repair properly performed than gradient should be at the distal end of the patch(if it at the widest part of the trunk), nothing more can be done
- If no correctable cause of high ratio is found,if not extreem & patients condition is good, should be taken into ICU with an expectation that after a few hours ratio will fall down
- If pt is not good in OT and CVP is much elevated than the situation is very bad & requires correction
- CPB to be restarted & a hole is made in VSD patch to save life and brought to ICU
- PRV/LV related to RP , Size of LPA , RPA, Stenosis & Incomplete distribution of Pulmonary arteries

*(Some times PFO is kept open to decompress in neotats/Infants)

RESULT:

- Mortality 2-5% (IN HOSPITAL)
- Mortality reported 12% in all patient (Paed.consortium)
- Death occurs largely in sever/complex morphology

Long term Survival

- One stage & staged procedures show equal survival at any age.
  Reports vary.

1 month survival	94%
1y "	92%
5y "	91%
10y "	90%
20y "	87%

- To be a 'curative'operation hazzard for death must be not greater than match population
- In essence long term survival in most TOF with PS is excellent but risk of death is slightly heiger tha general popullation

Risks of Death after operation:

- Young age at repair:
  Risk may be related to inability of very young (<3m) RV to adjust overload after trans anular patch
  Risk was heigher earlier but with expertise in myocardial management now a days is reduced in very young
- PRV/LV & Z value (anular):
  This has clear association for death after repair
  Inversly corelated with size of anulus & trunk

INDICATION:

- When diagnosed operation is indicated
- Whe presented at 3rd monthh & symptomatic prompt ome stage indicated to aoid unfavourable ist year of life
- When sevrely symptomatic in 2m,initial shunt than in 1yr Total correction is reasonable
- Asymptomatic/ mild with simple morphology deferred until 3m-24m
- 2 stage repair is prudent for centres not well prepared for infants & post- op care required be Neonates & Small children

TOTALLY ANOMALOUS PULMONARY VENOUS CONNECTION (TAPVC)

Defination

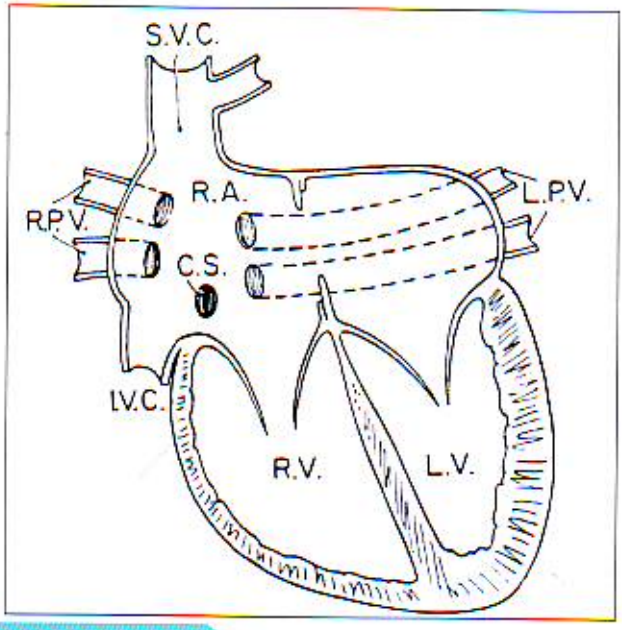
- Defines anomaly where pulmonary veins have no connection with left atrium.Either they connect to RA or to any other systemic veins

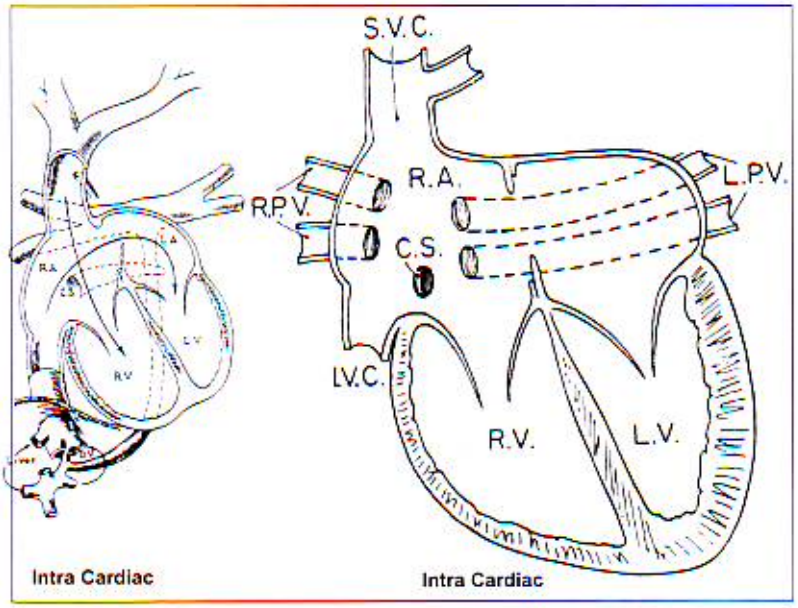
Types:

- Supra Cardiac ~ CPV connecting to Left Innominate Vein by Vertical Vein (lies anterior to LPA)
- Cardiac ~ 2 (Two) Sub types:

  CPV draining to Coronary Sinus (CS)

  PVs collectively / individually open to RA
- Infra Cardiac~ Connecting vein passing through the diaphragm infront of oesophgus(or acessory hiatus) to join inferior Vena Cava or its branches
- Mixed ~ One lung connecting to systemic vein & another to cardiac chamber (Coronary Sinus)





Natural History

- Presence of pulmonary obstraction influences the course of the disease
- If present hardly reaches lyr of life
- Contra indication of Operation
- Only irreversible pulmonary venous obstractive changes (PVR > 75% SVR) is contrindication ~ a rare condition

Diagnosis:

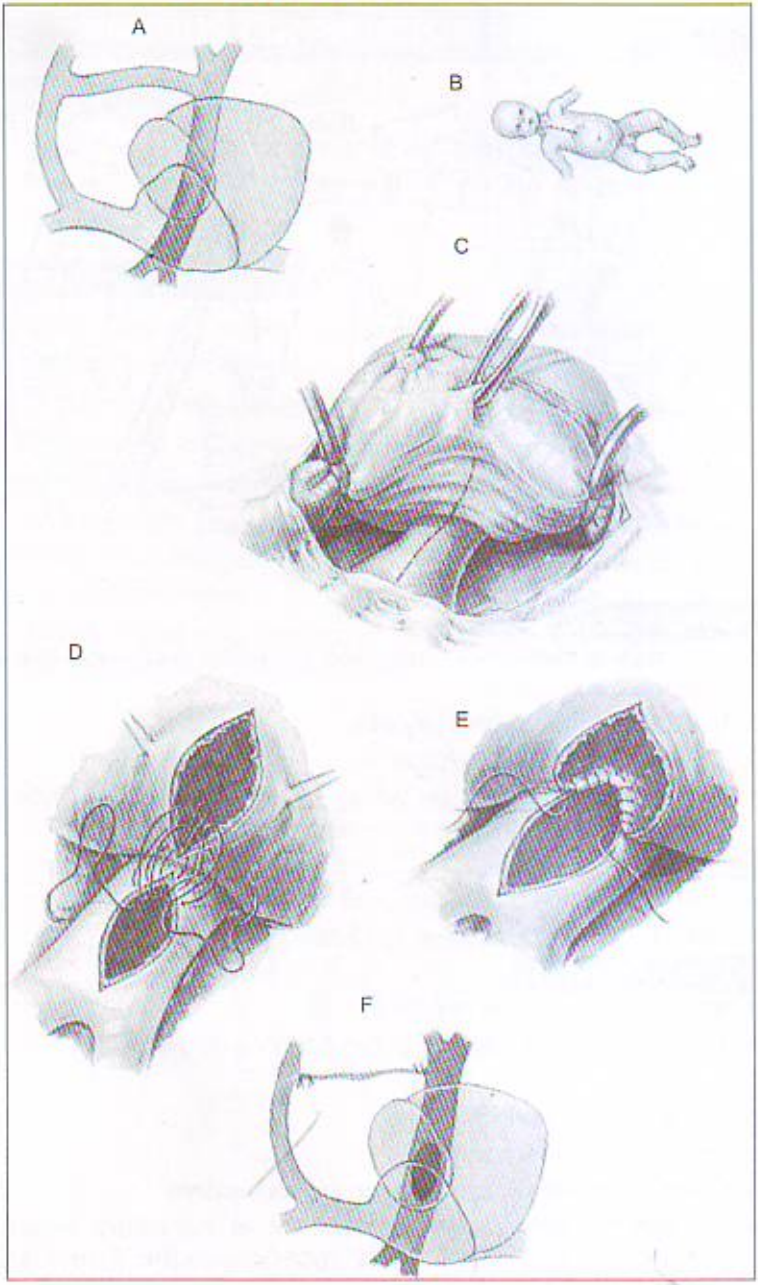
- Severity depends on obstraction of connections
- Patients with out obstraction can attain adulthood

Echocardiography:

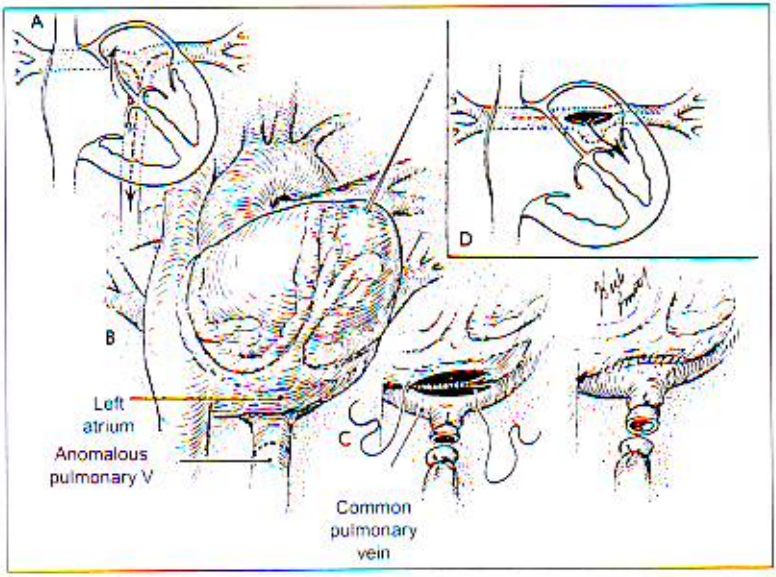
- Right Ventricle volume overload
- A echo-free space posterior to Left Atrium is diagnostic

- Similar to ASD if with out PH
- Only PaO2 is lower(cyanosis)
- Dye at Pulmonary artery vidualize the connections
- In patients with obstraction~ (similar to pulmonary venous obstraction- Shows pulmonary hypertension.But Pulmonary wedge pressure &PVR is low

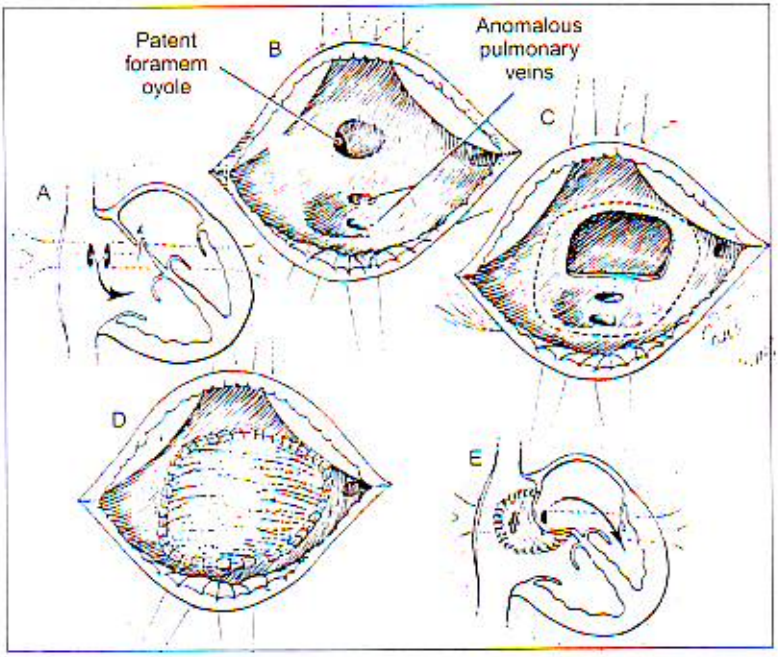
Surgical Procedure : Supra Cardiac



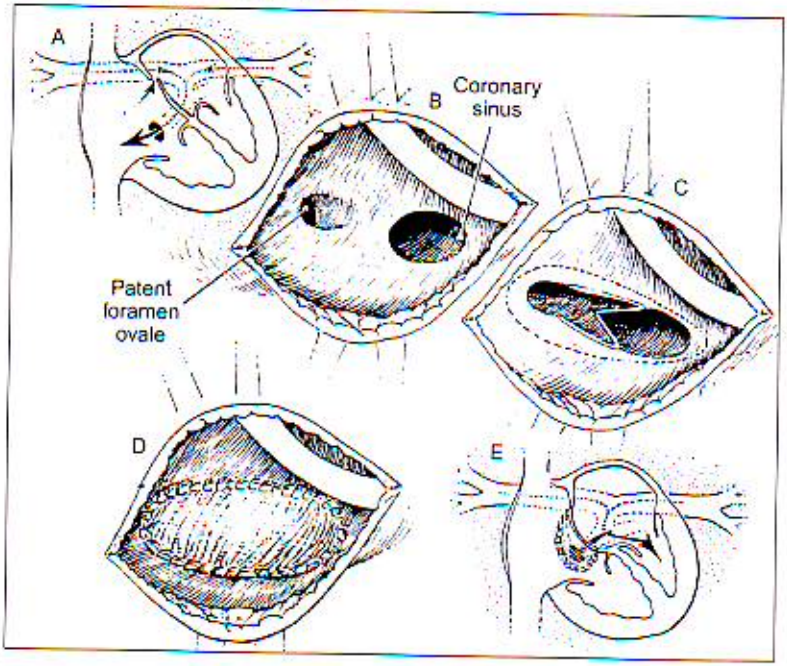
Surgery : Infra Cardiac



Surgery : Cardiac type



Surgery : Cardiac type (CS)



TRADITIONAL PROTECTION : MYOCARDIUM

- Traditionally,hypothermia has ben employed to arrest the heart in order to reduce metabolic requirements and permit operative procedures.
- Intermittant cold blood crdioplegia & solutions containing potassium have been administered to arrest and cool the heart and to rid the tissue of anaerobic metabolism.But with these the heart continues to metabolize aerobically & high energy phosphate continues to decrease in cool diatolic arrested heart.Therefore
- Hypothermia & K+induced Cardiac arrest in Traditional gold standard of "Intermittant cold blood/crystalloid cardioplegia does not maintain or improve myocardial integrity rather minimizes injury during cross clamp
- Quite adequet for low risk/short clamping time,may not be sufficient for high risk patients
- Hypothermia itself is deleterious,when rapid can cause Ca + sequestration leading to "stone heart"
- Although metabolic rate reduced but not always effective
- Areas distal to stenotic vessels not cooled uniformly
- Heart gets warmer with time for noncoronary flow
- Surgeon face warming heart with increasing metaolism getting intermittant cold cardioplegia whose O2 delivery curve shifted to left
- Furthermore the heart faces the negative effect of hypothermia

Disadvantages of Hypothermia

Shitf of oxygen-hemoglobin dissociation curve	Intracellular Ca^{2} sequestration (esp. with rapid cooling)
Myocardial enzyme activities altered: ATP-ase, G6PD, and oxidative phosphorylation	Intracellular edema
Glucose utilization reduced	Reduced platelet function
ATP generation reduced	Increased blood viscosity, rouleaux formation, sludging, vessel occlusion
Intracellular H^{+}regulation altered	Activation of platelets and ieukocytes, thromboxane release, complement activation, myocardial damage
Temperature-dependent phase changes in myocardial membranes	
Reduced membrane stability and repair	

Antegrade : Limitation

- If aortic insufficency- little /no flow
- If Lt.main stenosis/ TVD/ Diffusediseaseor when IMA is used the whole territory is underperfused
- O2 demand of arrested heart at 37°c is 1.9ml/100gm/min; at 20 °c lowered to 0.9ml/100gm/min

Retrograde Cardioplegia

- Retrograde infusion is superior to antegrade in maintaining nutrient floe,subendocardial flow& septal cooling distal to stenoses
- Technique:
- CPB started
- Systemic .Temp 34 °c -37 °c
- Arrested with high K+(100mEq/L)in 4:1
- Low K+(30mEq/L)in 4:1 retrogradeat not >250ml/min at mean pr.40 mmHg

Conceptual changes : New technique Protection to Resuscitation

- Developed Improvement in myocardial protection from cold intermittent crystalloid cardioplegia to intermittent cold blood cardioplegia folloed by additives stillleaves out the harmfull effect of hypothermia

- Worker in the field no longer consider "cardioplegic infusion" but "resuscitating the heart" during cross clamp. Ideas shifted from ischemic time to "resuscitating time (perfusion of the arrested heart with normothermic blood

Technique of retrograde cardiac resuscitation (Normothermic)

1. Rule out contraindications for retrograde cardioplegia
2. Standard cannulation:
   - Ascending aorta perfusion cannula
   - Double-stage venous connula
   - Ascending aorta cardioplegia cannula
3. Six-foot saline-filled line attached to pressure gauge and zeroed
4. Pursestring suture in right atrium, immediately next to venous pursestring suture
5. Insertion of cannula prior to cardiopulmonary bypass
6. Position of catheter confirmed by palpation and blood flow
7. Distal catheter placement
8. Cardiopulmonary bypass at 34-37^{0}C

Cardiac Arrest

1. Antegrade arrest with 1 liter high K^+ blood cardiopelgia:

   50 mEq K^+/500 cc Fremes solution

2. Switch to retrograde with pressure monitor:

   90-250 cc/min

   30 mEq K^+/liter Fremes solution

Advantages of Retrograde Cardioplegia (normothermic)

Delivery of usable O_2 and nutrients to myocardium

No detrimental effects of hypotherima

Better delivery to myocardium distal to coronary artery lesions:

- severe triple-vessel disease
- multiple coronary lesions in one vessel
- left main stenosis

Immediate and simultaneous delivery to entire myocardium

Quicker reperfusion of ischemic regions

- LIMA, RIMA
- acute evolving MI
- coronary dissection in cath lab
- angioplasty failure
- unstable angina

Repaor pf cells and repayment of oxygen debt while surgery proceeds

During aortic valve surgery:

- short left main coronary artery
- anomalous origins of coronaries
- coronary ostial lesions
- "jet lesions"
- type A dissection

Better delivery of cardioplegia in patients with mild to moderate AI

Less interruption of operation (to give additional doses of cold cardioplegia)

Reoperative surgery. Early ligation of diseased conduits. No embolization distally.

Single cross-clamp technique prevents injury to calcified or heavily diseased aorta

Adequate visualization of arteriotomies for CABG

Longer cross-clamp times not detrimental to heart

Allows for safety margin especially when complex procedures performed

Cold agglutinis

Potential Disadvantages of Retrograde Resuscitation

(Continuous Retrograde Normothermic Blood Cardioplegia)

Systemic normothermia
- oxygenator failure
- ?storke; cerebral hypoperfusion during hypotension

Catheter displacement

Inability to induce cardiac arrest

Coronary sinus injury

Intramyocardial hemormage

Venous hypertension, superficial hematomas

Right ventricular perfusion

Perfusion pressure iess than 40 mm Hg

Perfusion rates 50-90 cc/min

Shunting; subendocardial blood flow

Renal fialure; high K+load

Large positive fluid balance

Congenital cardiac lesions
- left SVC
- unroofed coronary sinus

Many lines obscuring/complicating surgery

Obscuration of field with blood

Regional venous pressure disparties within heart

Myocardial edema

New Technique & Interventions

Clinically Utilized	*Clinically Evaluated*	*Experimentally Effective/ Not Yet Used Clinically*	*Speculative*
I. Retrograde Cardiopiegia • Right atrial • Coronary sinus • Combined antegrade/ retrograde	I. Retrograde Cardiopiegia • Pressure-controlled • Intermittent coronary sinus occlusion	I. Prevention of Free Radical Injury • Alpha-tocopherol • Ascorbate • Catalase • Dismutase • DMSO • glutathione perioxidase	I. Modification of Calcium Metabolim • Trifluoperazine

II. Continuous Blood Cardioplegia • Cold antegrade • Cold retrograde	II. Continuous Blood Cardioplegia • Warm antegrade • Warm retrograde	II. Substrate Enhancement • Branched-chain amino acids (leucine, isoleucine, valine)
III. Substrate Enhancement • Glutamate and asparate warm induction and terminal "hot shot" cardioplegia	III Prevention of Free Radical Injury • Deferoxamine	III. Nucleotide Precursors • Orotic acid

Applicability of Resuscitative Modalities

Clinical Situation	Currently Useful	Potentially Useful in Future
1. Previous coronary artery bypass	Retrograde cardioplegia +/- Combined antegrade/retrograde	Continuous blood cardioplegia
2. Associated valvular disease	Retrograde cardioplegia +/- Combined antegrade/retrograde	Continuous blood cardioplegia
3. Failed angioplasty and/or ischemically depressed ventricle	Retrograde cardioplegia +/- Combined antegrade/retrograde Glutamate/aspartate warm induction, terminal "hot shot"	Continuous blood cardioplegia Prevention free radical injury Modification calcium metabolism (e.g., trifluoperazine)
4. Proximate transmural infarction	All of above	All of above Preoperative administration of nucleotide precursors

TRANSPOSITION OF GREAT ARTERIES (TGA)

- Congenital anomaly with aorta arises largely from the right ventricle & pulmonary trunk largely or entirely from the left ventricle (Ventriculoarterial discordant connection)

Morphology

Right Ventricle:

- Hypertropied, large & in normal position in TGA. Inflow & sinus portion are essentially normal.The infundibulum not deviated to the left like normal. It is directed posteriorly from the sinus part
- Less wedging of PT between mitral and TV than normal heart

L.Ventricle:

- Pulmonary-mitral continuity is present.Aorta anterior & at right mostly.May be left & posteriorly
- In embryo LV wall thicker.At birth LV wall thickness increases progressively& RV decreases relatively
- In TGA RV increases with age. Septum & LV becomes static.With age LV becomes thinner relatively
- When VSD present LV thickness but less than normal heart.Yet within normal thickness in 1st year
- If LVOT obstraction (PS) without VSD – LV thickness more than RV
- As a whole LV thickness indicates LV functional capacity
- LV becomes Banana shaped (Ellipsoid at birth)

RV function:

- RV EDV increases & RV function(EF) decreases, may be due to hypoxia

LV function :

- LV EDV increases & RV function(EF) decreases but EF normal
- RV,LV EDV ratio is normal 1.0- 1.46
- Inlet VSD are more common

LVOT:

- May be dynamic / anatomic
- Dynamism is simialr to obstractive cardiomyopathy without septal myopathy
- Sub valvular stenosis/valvular anomalies may be present
- Valvular stenosis may also be present

PDA:

- Common in TGA than others

MV /TV:

- Anomalies are present among which are dialatation, Hypoplasia. Cleft, Stradling & overriding

Arch anomaly:

- Coarctation,hypoplasia,Right arch(5%)are present

RV Hypoplasia:

Found 17% in heart with TGA

Clinical Physiology & Diagnosis:

- TGA with concordant AV 2 circulation are parallel & life is not possible without any shunt
- Shunt must be present that detemine the degree of mixing and symptom
- When high mixing Qp large.symptoms are less(unless PV hypoplastic)
- Anatomic factors that reduces mixing also produces cyanosis

Clinical Features

3gr of TGA –depending on presentation:

a. Some presents with "Poor Mixing" :

- No ASD (PFO)
- Cyanosisainfantsppears shortly& progressive
- Baby critically ill.cyanotic,acidotic with frank failure
- Atrial Septectomy does not help increasing cyanosis in surviving infants

- Normal weight with good circulation despite cyanosis .Clubbing appaers at 6 months survives
- Oval/egg shaped cardiac shadow with moderate plethora(Less marked when with LVOT)
- Narrow mediastinum

b. With good mixing:

- Large VSD with PDA
- Present late first month with cyanosis
- Signs of Heart Failure
- Plethora & widen mediastinum
- Early development of PVD(Pulmonary Vascular Diseases)
- May be associated with coarctation

c. With poor mixing with out high pulmonary flow: (Large VSD + LVOT)

- Less common
- Poor mixing for LVOTO
- Features similar to TOF
- Cyanosis sever after birth
- Chest X-ray may be normal

Diagnosis:

Echo:

- 2D sufficient for morphology

Card.Cath/Cine:

- Not done routinely
- Can provide important information of Parallel Circulation

Natural course:

- Incidence 7-8% of CHD
- Survival:

  55% alive 1 month

  15% ,, 6m

  10% ,, 1y

- TGA with out VSD : survival 1 wk - 80% (Cause –anoxia)

- TGA with VSD : survival 1m – 91%,1 yr- 32% 1 wk (cause-recurrent pulmonary infection)
- TGA with VSD with LVOT -Better survival ,70% - 1 y,29% 5 y (Cause : Hypoxia)

Effect of PDA closure :

- Closure lessens flow of pulmonary bed
- Result flow through ASD
- Ptatients deteriorates sharply

Dynamic LVOTO:

- Appears later
- Cause cyanosis & short life
- Usually not present when impotant VSD is there

Pulmonary Disease:

- Rare in TGA with out VSD and PDA
- Die in 12 m (50%)

Operation:

- Arterial switch is choice (if no important LVOTO)
- Atrial switch rarely in highly selected cases
- PE1 priror to OT resluts adequet mixing & stability

PROCEDURE

- Exposure, Sternotomy are usual
- Support to be used are

(1) Continuous CPB at 18-25°c, bicaval canulation,low flow after temp achieved

(2) Near continuous CPB 18-25°c with low flow & single arterial cannula

- Total circulatory arrest for ASD closure
- Then annula reintroduced & full flow warming done

(3) Circulatory arrest after 18°c. Operation primarily performed during cardiac arrest & then rewarming with CPB

Myocardial protection: Varries, Preferable with cold, hyperkalaemic blood

Repair of LVOT :

- Arterial switch is used with intact IVS
- In LVOTO,LV Pr ≤ RV (systemic) nothing done directly on LVOT
- LV Pr > RV Pr surgical relief is needed
- Local excission/Valved conduitnecessary + Arterial switch
- Intraventricular repair (like Taussig Bing)
- Rastelli operation : (VSD to aorta,PA closed inside&RVOT attached with valved conduit to MPA)

Post Op care

- Arterial switch PA Pr should be <12mmHg
- Patient to be in ventilator 24-48 H
- LV function to be monitor & if lrss filling ,caution to be taken
- Arterial switch – PEEP not needed (obstraction SVC). CVP should be low,support is needed for early days

RESULT

- Institution properly prepared- 2-7% early (low risk institute)
- MultiInstitutional 30 day mortality -16%
- Survival falls after 1 y
- 5 y survival – 82%(including hospital mortality)
- Long term(20y) survival - not available
- Mode of death :Acute/Subaute cardiac failure

RVOTO (of Arterial Switch):

- Complication 10%- most after 6m
- Usually at Pul.trunk /Infundibulum
- Freedom of intervention 94%, 1yr

Indication of Operations & Procedure

- Neonates (Simple TGA)
- Itself an indication
- Severe cyanosis BAS(Ballon Arterial Septostomy) to be done
- Arterial Switch i in 1st wk – 30 days
- LVOTO is not contraindication like Rp in earlies
- Risk lowest in 1st wk

- Arterial swith is better than Atrial switch

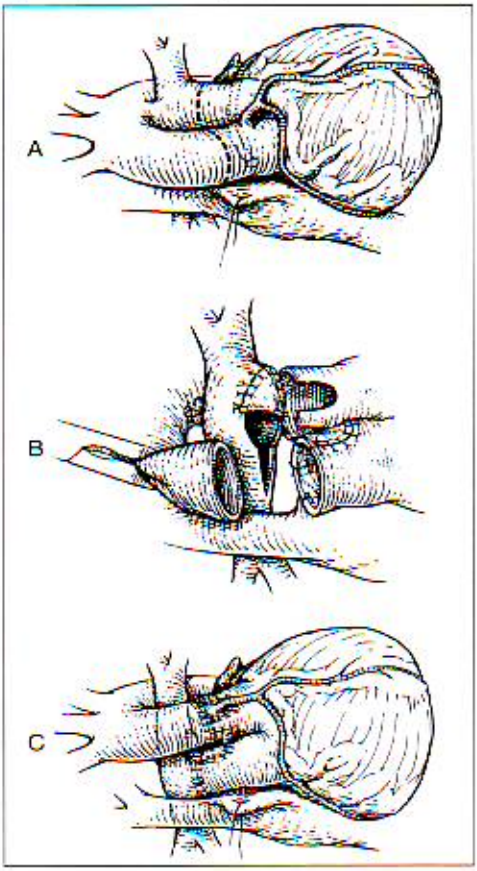
After 30 days:

- LV adapted to low pr.- Arterial Switch heigher risk
- May safely be done (Arterial switch) in <8wks if VAD is available)

Alternatives:

- PA banding +BT shunt followed by arterial switch in 1-2 wks
- Atrial Switch
- PA banding approach has low risk
- If delayed more than months after banding mortality heigher
- Less Experience hospitals- Atrial switch is appropiate
- LV funtion after Arterial (98%) than Atrial (79%)

Arterial Switch Procedure

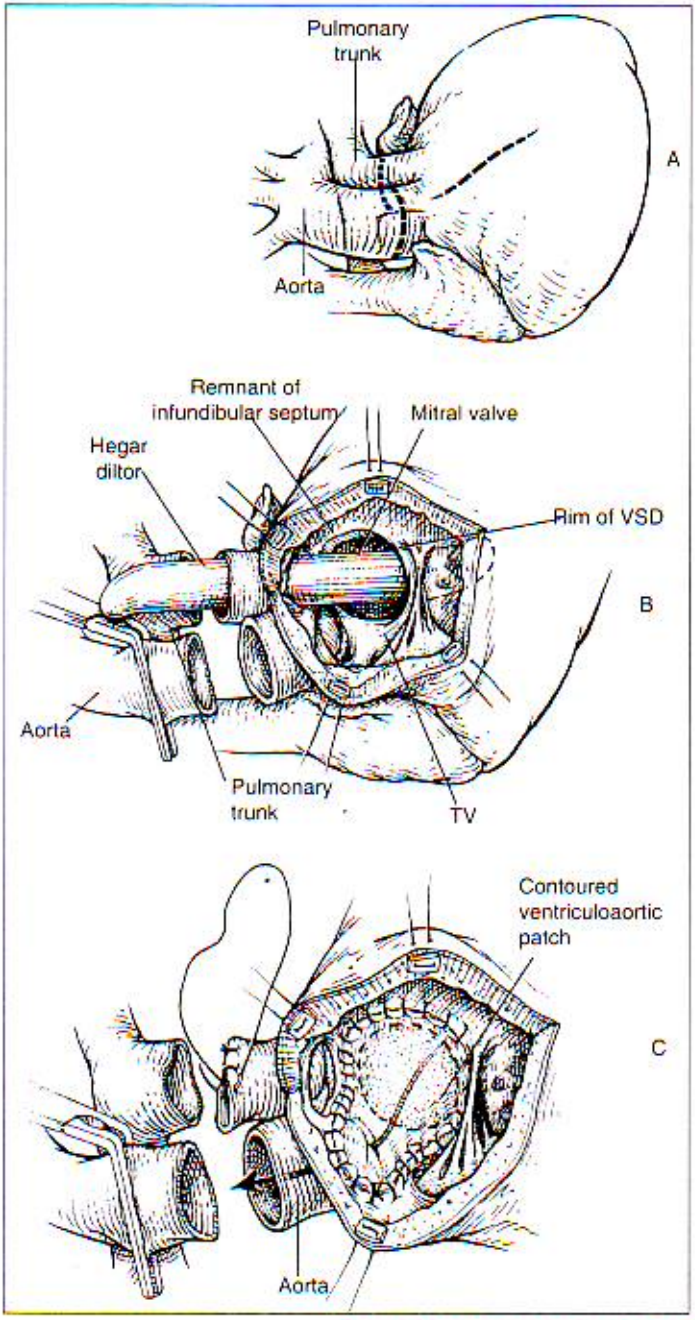


Indication:

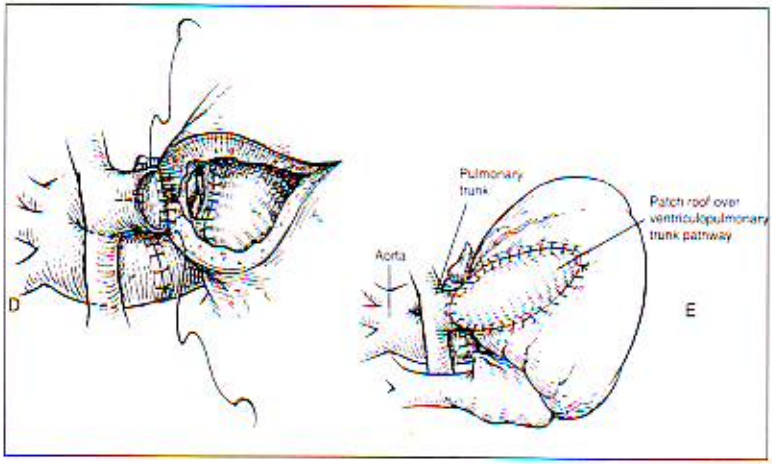
- TGA with VSD
  - Arterial Switch + VSD repair
  - When Patient is first seen
- TGA +VSD +LVOTO
  - Timing & procedure are controvertial
  - If not cyanotic , early intervention
  - LeCómpte indicated operation between 6m-5y
  - If important cyanosis , <6m then
    - BT Shunt followed by Leompte in 6-18 m
    - Primary LeCompte

Choice is according to Capability

Lecompte Procedure



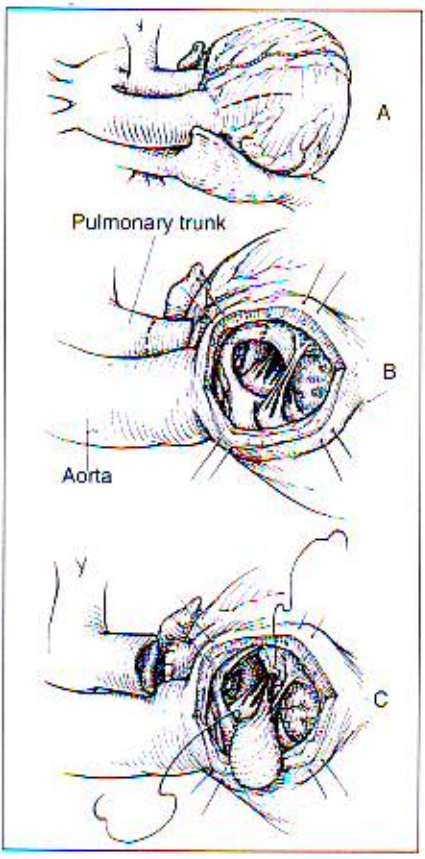
Lecompte



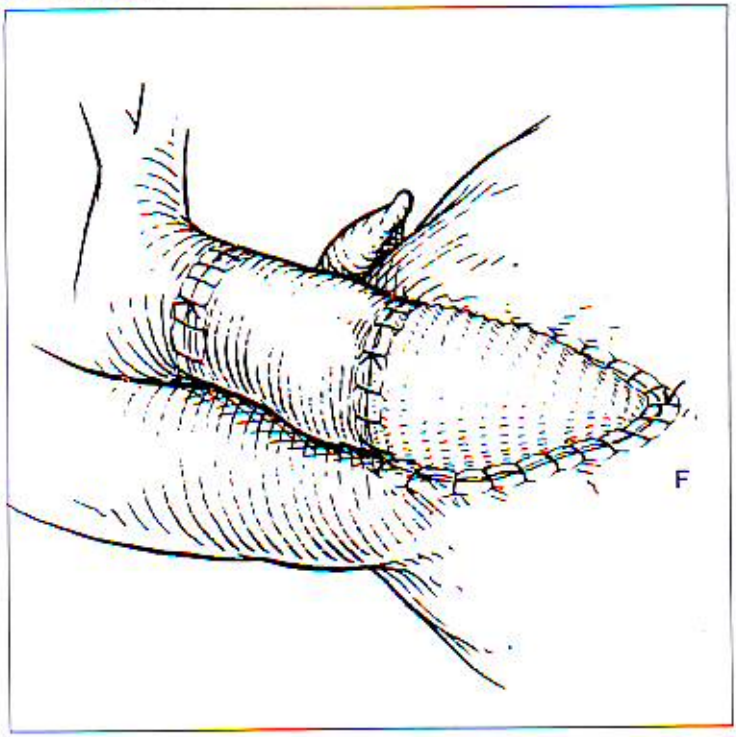
Children (3-5y)

- LeCompte &
- Rastelli–good result

Rastelli Procedure



Rastelli Procedure



- Mortality: Now a days 5%(earlier 20-30%)
- Survival:
  - 20y – 52%
  - 15y - 68%
  - 10y - 95%

VALVE REPLACEMENT

Factor of consideration

- Durability
- Permanant Anticoagulation
- Current Xenograft degenerates early in youngs (Good candidates for tissue)
- Bio prosthesis degenates rapid in mitral position
- Bioprosthesis in state of evaluation may lead to good durability
- Prosthetics also evolving- indication that a device will appear that needs no anticoagulation
- Haemodynamic performance influences the choice of .
- **Newest Mitral substitute-Cryopreserved Stentless allograft**
  - Good haemodynamic performance
  - Adequeat freedom of embolism in abscence of anticoagulation
  - Durability?
- Pul. autograft as cylinder ('Top-hat') in mitral position

Bioprosthesis

A number of series used

- √ Stent mounted
- √ Leaflet made allograft Av
- √ Xenograft AV
- √ Pericardium
- √ Fascialata
- √ Duramater

- AV function improves
- Trans valvar gradients always persists
- Depends upon activity,Pt.size & device
- Mean Diast.Gr - Rest Exer

Normal	o
Desired	<10mmHg
Orifice	>0.9cm2

Considerations

- Infants with CHF refractory to medical needs surgery
- Older infants & young adult-Early surgery for risk of sudden death
- Systotlic gr 50-70 mm Hg & Valve area <0.5 cm2.m2 accepted para meter for surgery
- Gr. 40 mmHg in Subvalv. AS requires early repair to avoid surgically defficult tunnel formation
- Approach & Urgency depends on location. Age & condition of pts.

VENOUS DRAINAGE ANOMALIES: PULMONARY & SYSTEMIC

Introduction

- Represents the forms of congenital heart diseases with normal valves & ventricles

Embryogenesis

- Splanchnic plexus drains lung & connect with cardinal,Umbilico Vitellin vein
- Common pulmonary vein (CPV) invaginates from LA & connects splanchnic vein
- As splanchnic connects the LA the primitive connection disappears
- With the development of individual pulmonary vein that drain to LA ,CPV disapperas
- If atresia of CPV develops than systemic connection is maintained & Totally
- Anomalous Pulmonary Venous Connection (TAPVC) to systemic venous system develops (fig-1)
- When right /Left portion of CVP is involved PAPVD(Partially Anomalous Venous Drainage) occurs
- Persistant cardinal vein develops – SVC,Innominate,Coronary Sinus, Azygos vein
- Umbilicovitelline vein forms IVC, Portal vein, Ductus venosus

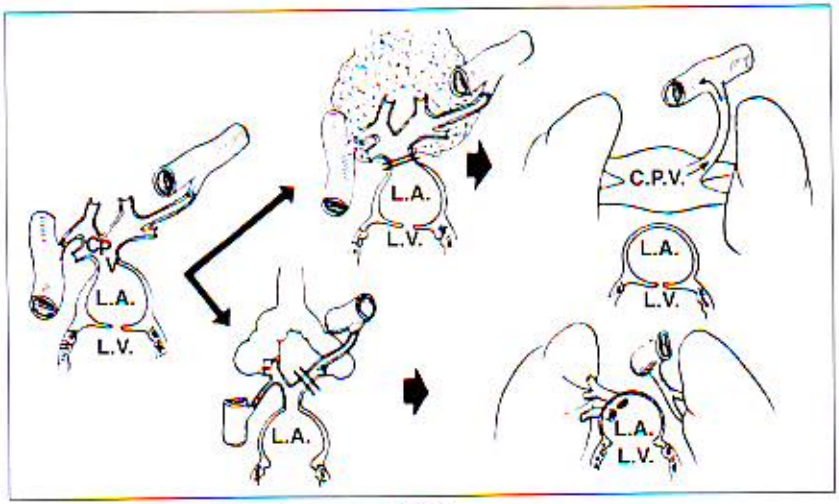


Fig-1 : Abnormal development of CPV

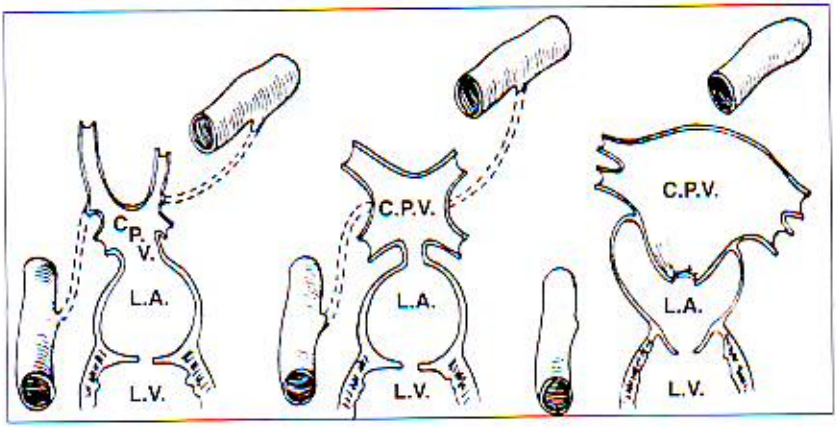


Fig-2 : Cor-triatriatum

- Direct RA connection of pulmonary veins are best attributed to abnormal septation of two atria
- Stenosis of CPV results cor-triatiatum
- Occasionally Cor-triatiatum is associated with APVC because of the presence of stenosis when systemic primitive veins are still present
- Similarly failure of right subcardinal vein to connect hepatic vein develops intrrupted IVC (fig-3)

Cor-Triatriatum:Embryogenesis

- Stenosis of Common Pulmonary Vein (CPV) that fails to be incorporated in LA results The anomally

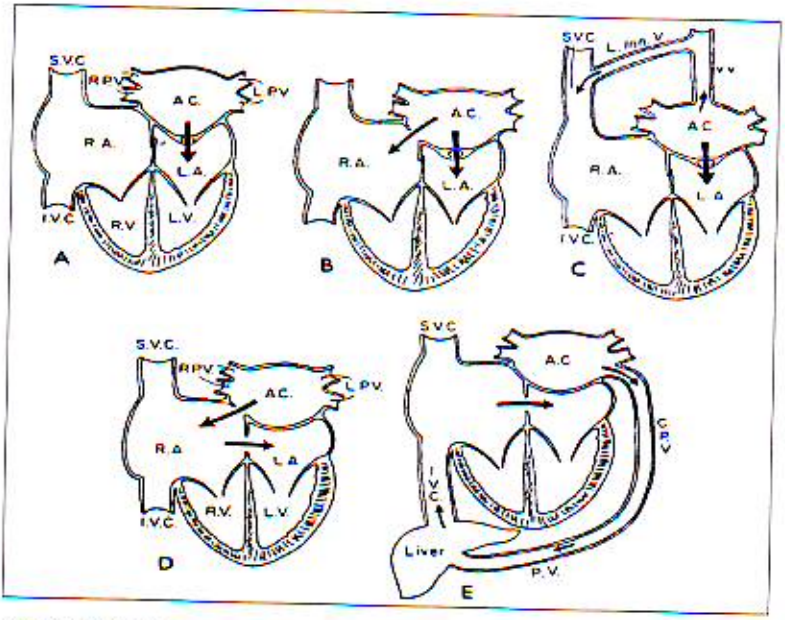


Fig-3 : Types

Morphology

- Classically an acessory chamber receives all pulmonary veins & drain to Left Atrium (LA)

Diagnosis

- Majority present sign of pulmonary obstraction in early life
- X-ray shows pulmonary venous engorgement
- Echo: In most cases it is difficult to identify the accessory chamber to CS or PLSVC to CS

Cath:

- Pressure gradient between pulmonary (wedge)& LA is the hallmark
- L~R shunt & Pulmonary hypertension are rule
- Slelective Pulmonry arteriography in venous phase(prolonged transit time)opacifies the chamber & LA

Surgery

- Successful therapy for the anomally
- Open correction under CPB & resection.
- Post operative is good

Systemic Venous System:

- Cardinal Venous Anomally

Persistant Left Superior Vena Cava:

- Common in association with other lessions
- Part of superior caval system
- Passing infront of the aortic arch crossing left pulmonary veins receives hemiazygos vein & superior intercostal vein and than penetrates the pericardium to reach atrial groove

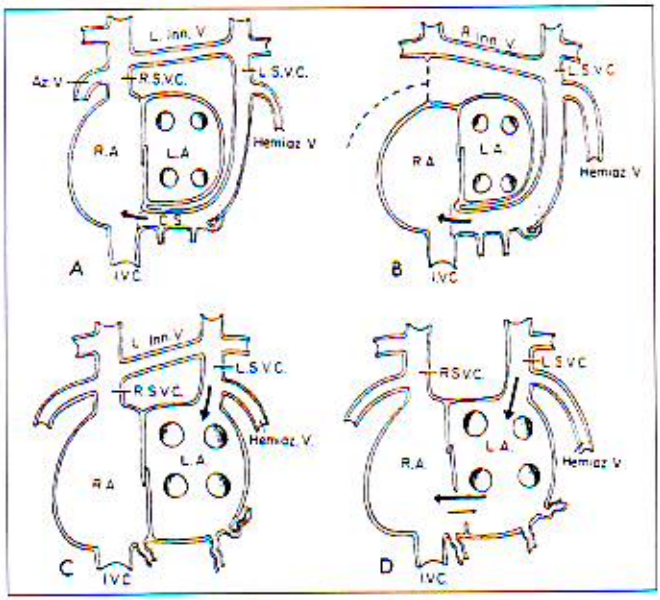


Fig-4 : Anomalous Left Superior Vena Cava (PLSVC)

PLSVC With Unroof Coronary Sinus (CS)

- LSVC does not descend posterior to LA to reach the atrial groove
- Instead enter into LA in between LA appendage & LSPVs
- Coronary Sinus fails to develop

- Almost invariably associated with other anomalies
- Small R ~L shunt is the only finding

Cine Angio

- Finding of the route of contrast media in the face of peripheral cyanosis gives suggestion

Surgery

- If bridging vein exists then ligation
- If no bridging vein exists then roofing of Coronary Sinus

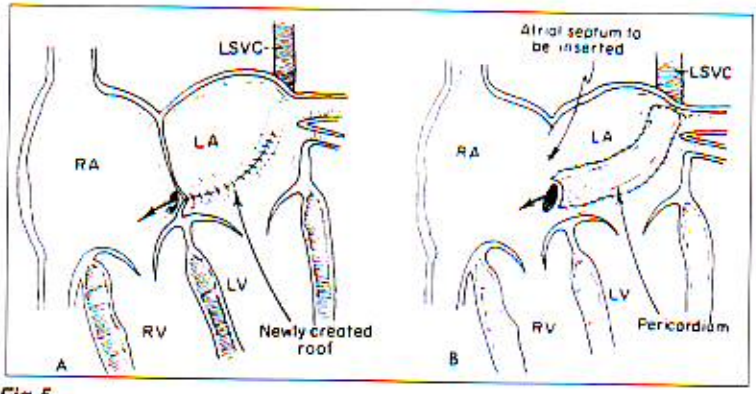


Fig-5

Failureof right subcardinal vein to connect hepatic vein develops intrrupted

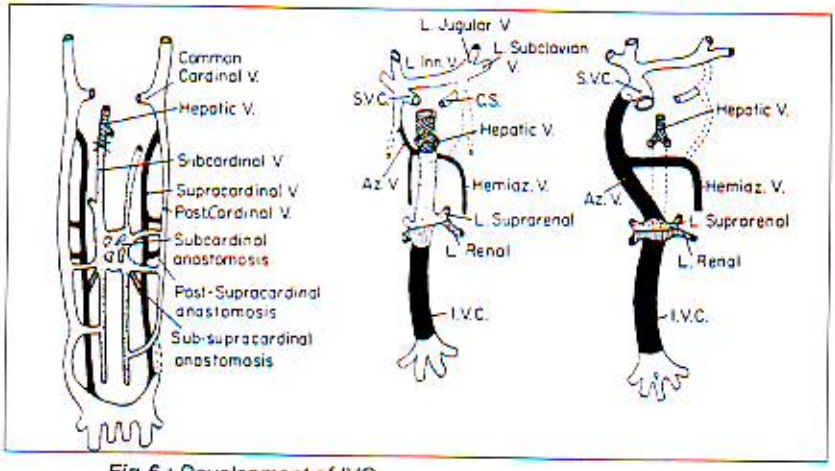


Fig-6 : Development of IVC

VENTRICULAR SEPTALDEFECT (VSD)

Definition

- A hole or multiple holes in the interventricular septum
  May be primary
  Associated with other major defects

Morphology

(Morphologic Classification based on CHS nomenclature)

- Perimembranous
- Mascular
- Inlet Septal
- Subarterial Doubly commited

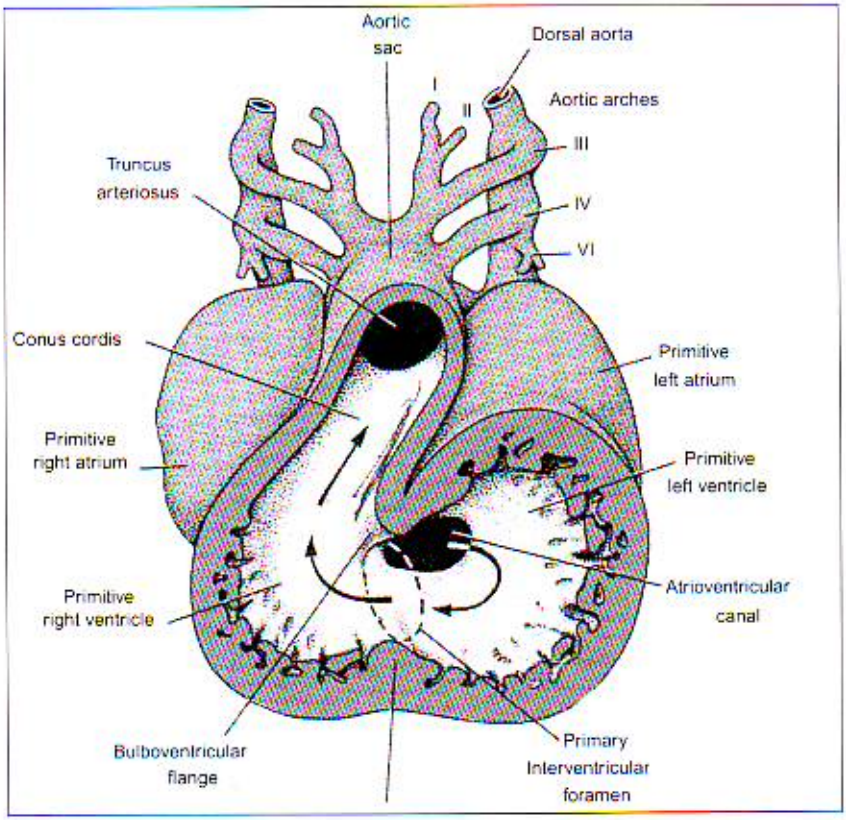


Fig.1 Development

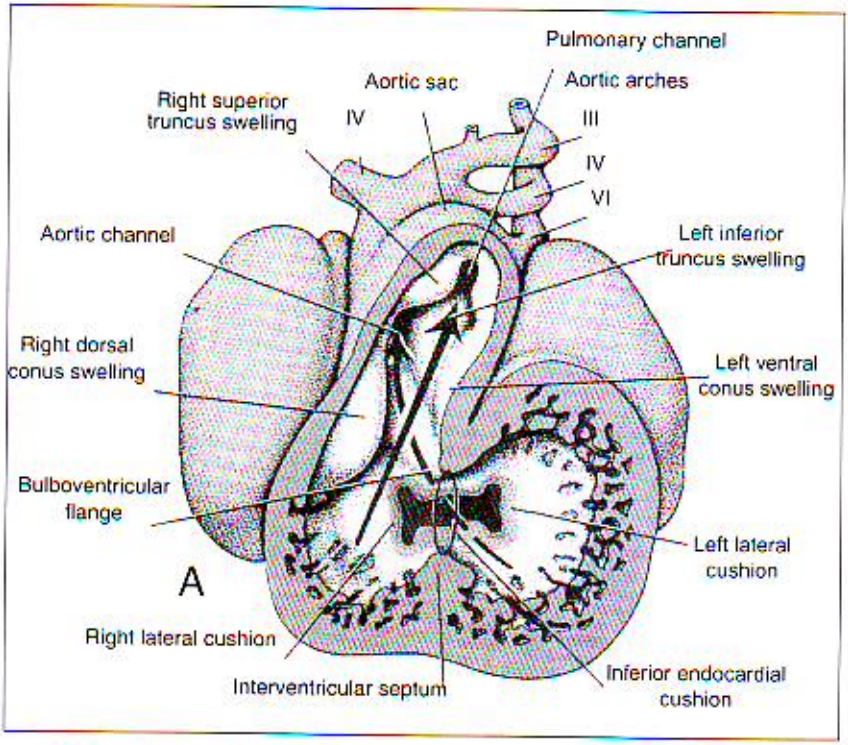


Fig.2 Development Tranco conal septum

- 1 Doublly commited.juxtra arterial VSD.
- 2 Cono Ventricular (membranous) or juxtra arterial / juxtra tricuspid.
- 3 Inlet septal (juxtra tricuspid/mitral), associated with atrial/ventri septal malalignment&Overridding TV.
- 4 Mascular / Trabicular VSD

Arbitrary Divisions

SIZE:

- LargeVSD–Aortic orific/Larger.RV/LV pr. approximates.Qp: Qs depends on PVR
- Moderate –Retritive.High RV pr.Qp:Qs >2 or greater
- Small VSD- RVp not raised.Qp:Qs <1.75

Subarterial Catagory: (juxtaposed to)

- Juxta-aortic
- Juxta-Pulmonary
- Juxta-arterial
- Juxta-truncal

Catagory as to commitment

- Subaortic
- Subpulmonary
- Doubly commited
- Non commited

These are not morphologic & creates confusion

Malalignment VSD

- VSD associated with Malalignment of parts of septum or atrial septum
- Echo terminology- regarding ailgnment of trabecular & conal (outlet) part of septum
- Anterior- Outlet septum anterior to trabecular septum,interposed VSD (TOF)
- Posterior-Intrpt.arch
- Rotational-Taussig-Bing

Perimembranous VSD

- Also called Junctional VSD
- BETWEEN INLET & OUTLET SEPTUM
- Juxtratricuspid/mitral/aortic when abutting valves
- When abuts RCC, NCC-Conoventricular VSD
- Some separated from TV by TSM-Inlet septum VSD

Conductive tissue

- AV node , His normal location
- Penetrates trigon at base of NCC & lies post inferior boarder

- LBB leaves as croses inferior margin & only RBB gets the muscle of Lancisi

Doubly Commited Subarterial VSD

- More beneath the RCC
- Also known Conal/ supra crystal
- Boardered space overlied by AV, PV-subarterial
- RCC/NCC may prolapse with / wothout AR
- Well separated from TV, His bundle DORV with doubly commited
- When associated with severe over riding aorta
- DOLV when PA overrides contd..
- VSD in RVOT when abut RCC, frequently with AR
- VSD Rarely Juxtapulmonary to left

Post Malalign VSD

- RVOutlet- VSD with muscular boarder lies in muscular septum, superior the defect
- When malaligned & displaced left causing subaortic stenosis (found in interrupted aortic arch)

INLET VSD

- (AV canal type VSD)
- Involves RV inlet & LV outlet
- AV septum intact whereas in AVSD involved
- Abuts TV
- Superioly extends to membranous septum
- May have Mitral cleft
- His bundle lie in postinferior margin & to the left
- A muscular VSD at inlet separated by band where lies His bundle Muscular VSD
- May be multiple
- Often associated with others
- Mostly in infants
- Common in middle part overlying TSM

- In anterior part multiple, may whole length with more RV opening
- Both involes- Swiss cheese defects
- His bundle at apart

Associated Lesions

- PDA
- Coarctation
- AS
- MVD
- ASD when VSD large in infants

Features:

- Large VSD
- Heart failure
- Growth failure
- Pansystolic murmur (3/4 ics, 2/3rd in subsrterial) with apical diastolic murmur
- Cardiomegaly with sign of huge Qp

Eisennenger Complex

- High Rr >changed haemodynamics
- No large shunt
- Bidirectional of equal magnitude
- Heart not enlarged, not hypertropied /hyperactive
- Systolic murmur soft/ absent
- No apical diastolic murmur
- P2 loud/thrill

ECG RVH than BVH

- Small VSD
- Small shunt
- Only murmur often
- Other normal
- Moderate VSD

- LV enlarged
- RV over load

Echo sufficies:

- If neonates/infants with large Qp/Qs, echo defines morphology with arch / PDA
- When surgeon experienced in surgical identification
- Cath not necessary
- Muscular VSD escapes refined Eho, PM VSD may acompany > Does not necessitates Cath
- Malalign VSD can be diagnosed Other tools
- MRI – at present

Card.Cath:Angio

Assesment Rp indicated

Sometimes in assocoated lesion

PVR:

<4 u	Normal
4-5u	Mildly elevated
5-8u	Moderate.
8u	Severe

Indication

- Infant,large VSD with failure in 3months prompt repair
- Excepton is rare swiss cheese defect-for high risk- Banding indicated until compor 3-5 yrs
- This plan indicated when coarctation,straddling associated VSD
- Not indicated electively in 3m (may close)
- At 6 m & thriving closure unlikely.If Rp >8 repair advisable.If <4 with good clinical condition delay untill 12 m. Risk simila
- Infants considered primary for PVR.
- If Rp <8 opr indicated, if Rp>8,isoprin inj.given to see response.If fall > opr In older Pt
- Qp/Qs 1.5-1.8 at rest-probably inoperable

- Simple finding Fall in SaO2 at exercise –suggest inoperable,more investigation
- O2 inahalation is not useful to determine

Moderate VSD:Children

- Does not raise PPA >40–50–will not develop Rp. Qp/Qs–3, cardiomegaly, plethora
- Can wait upto 5 yr

Small VSD : Children

- Not indicated
- Controversy at 10y
- Surgery for :

Endocarditis

Empact at youn/adult age

Vent.dysfunction

possible complete cure

Subarterial

- Prompt opr if diastolic murmur develop

Compliations:

- Early death-Aute Cardiac failure for inadequet myocardial protection in sicks

Young age partcularly when associated with other lesion

Multiple VSDs

- RBBB
- RBBB+LAHB
- CHB
- Ventricula arrythmias
- Residual shunt

RESULT

- *REPAIR CURATIVE WITH FULL FUNCTIONAL CAPACITY WITH NORMAL LIFE EXPETANCY, IF AT 1-2Y*

(WHEN Rp < 5units)— ' Surgical cure'

CRADIAC REHABILITATION

Defination

RECOVERY FROM PHYSICAL & EMOTIONAL DISABILITY TO A NORMAL OR NEAR NORMAL LIFE AS SOON AS POSSIBLE IN A SCALED MANNER

INDICATIONS

- Following Heart Attack
- After Bypass Surgery / PCI
- After Heart Failure & other Cardiac ailment

CONTRAINDICATION

- POOR EXERCISE CAPACITY
- ANGINA ON MINIMUM EXERTION
- UNSTABLE BP
- FRESH ECG ABNORMALITY
- SEVERE HEART ENLARGEMENT

Factors underlying Disability

- Heart Diseases per se
- Physical deconditioning due to bed rest
- Emotional stress
- Nutritional stress
- Side effect of drugs
- Medical disorder per se(HTN,DM,Musculoskeletal disorder etc)

GOALS

IMMIDIATE GOAL

- Restoration & enhancement of exercise capacity after operation /attack
- Control OF Adverse emotional consequence of active illness
- To Identify Pt. at high risk of recurrence
- Early return to gainfull employmment

Late Goal :

Control of underlying

- Risk factors
- Prolong gainfull life

Components of Rehabilatation

- Psycological counselling
- Stress control
- Vocational counselling
- Pt & Family Education
- Dietary Counselling
- Assesment of future risk
- Control of risk factor
- Exercise training

contd..

- Key point of rehabilatation is Supervised exercise training to enable quickly to resume normal activities without symptom
- EARLY MOBILIZATION AFTER SUREGRY/ATTACK/PCI:to prevent secondary complications :–

  Physical Deconditioning

  Pneumoniaq / Lung complication

  Thromboembolic disorder

  Musculo skeletal disorder

  Psycological setback

Rehabilitation

Immidiate Post operative

1st day after Surgery

- Goal for today:
  - Activity :
- Get pt. out of bet for each meal
- Walking in the room
- Weight measurement
- Transfer to step down unit
- Make sure to maintain privacy,respect[sex,personality]

- Diet :
- Clear liquid progressing to low salt, low cholestero & animal fat
- Medication :
- Take care of Pain & ensure painless breathing
- Encourage coughing from the 1st day& strat breathing exerc - ise quitly
- Ensure Vist by Physiotherpisi, Diatician, Social workers

2nd day after surgery

- Acivity :
- Allow walking in the hall with nurse/therapist/family member
- Weight measurement
- Insist meal in the chair today
- Encourage to do as much self - eating bathing,refreshing
- Diet ;
- Progress to low salt,low animal fat
- Medicine :
- Allow pt to ask for pain medicine
- Encourage to cough & breath deep[ly
- Some previous medicine be restarted today- Review & ensure
- Teaching:
- Try breathing exercise/spirometry
- Write down question to ask - to visitng Thrapist,Dietacian
- Tiring easily.Allow frequent rest
- Most Uncomportable day today

3rd day after surgery

- Activity:
- Allopw Walking at least 3 times a day,with assistance if necessary
- To Do as much self as possible
- Diet:
- Diatery visit toninform diet at home
- Medicine:
- If no bowel movement – Laxative
- If Valve replacement/Af then –Blood thinner to add
- May need diuretics still today
- Teaching:

- Now Pt. & Family have important role
- To continue coughing /Exercise
- Allow paperwork
- Instruct to feel secure about going home

4th Day after surgery

- Activity:
- Allow self- walking & daily activities(bathing,eating,etc)
- 2 new GOAL today
  - Stair climbing
  - Taking Shower –after nurse takes care of wounds[Nurse to let know concern & care of wound today]
- Diet : Any question to dietician
- Medicine : Again To Evaluate TODAY
- Teaching:
- Provide discharge instruction & prescription
- To write down question about doctor visit,drug & diet again
- Tomorrow Pt may be going home

5th Day

- Activity:
- Encourge to self walk & climbing
- To do everything self as possible
- If no shower yet,Get it today
- Diet : Get answers of your all questions
- Medicine: To be reviewed again
- Teaching:
- Review questions about discharge to day
- Assure phone call/contact when pt at home

[It is not routine prctice of tredmill before discharge after operation/Ht.attack]

After discharge : Schedule of WALKING

- When to walk:
  - Any time of the day but space session evenly throughout the day.DO NOT PERFORM ONLY MORNING/EVENING

- Not after a meal
- Warm up:
- Perform sitting/Stending exercise before walking
- Pace:
- Medium pace that does not make breathless
- Be able to talk with fellows while walking
- Increase pace with time
- Try walking for a perticular period on ground level with pause / rest if so.
- Increase period weekly [exm:1st wk-10minX3 times/2nd wk-15min X 3 times & so on]
- Make 1 hour once a day after 6 weeks

Supervised Rehabilitation : OPD

- 6-8 WEEK AFTER OPERATION
- Individual to patients fitness,decided by the surgeon
- Should be 30 min daily/1 hour thrice weekly
- Exercise is Dynamic- involves whole body
- Includes:
- Warmup exercise
- Endurance - [BiCycle,Treadmill]
- Cooldown
- Relaxation

Should be under medical monitoring & supervision

Rest & Sleep

- During initial period personal activities may be tirying.Needs more rest necessary
- Recovery in some cases may be slow paced
- Activities to be well paced to avoid fatigue
- Sleep at lest 8 hrsd in 24 H.Avoide sleeping in the day.Avoid awaking at night
- When feeling stronger start out side shopping/social visit

Sex & Rehabilitation

- Sex after operation/Ht.attack needs to be address
- No reluctancy expected in the subject
- Spouse mostly nervous about Pts. health

PROBLEM

- Exhaustion
- Decreased Libido
- Avoidance
- Impotence
- Ejaculation problem

Causes

- Fear of Angina
- Mental depression
- Drug side effect
- Fear of injury of BREAST BONE

Teaching

- Every cost of orgasm is equal to climbing 1~2flight of stair(Heart rate at peak increases 30~35 beat/m
- If recovery normal and feeling normal & Incissions heal normaly & adequetly _ Sex can beperformed with in 8~12 wkafter surgery/Attack
- Sex can be more safe by change of position comfortable
- Avoid sex when tense.after meal or when unsocial

Stress

- Nonspecofic natural arousal response of individual
- Also associated with heart disease, operation,ect
- Recognition & Management necessary
- Expressed variedly including physical /psycological manifestations
- Avoidance of undue stress by changing personal life style ,work Togather with
- Relaxation exercise can metigate stress respone duringrehabilitation–Family & *social worker must paly important role*

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To encourage understanding
Medical Science