Intensive Care UNIT ICU

manual

Cardiac Surgical Care

N.A. KAMRUL AHSAN

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ICU Manual : *Problems, diagnosis, management, indications & practical guides in post operative cardiac care.*

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Edition : March, 2003 (1st) : April, 2007 (2nd)

Publishers : Ahsan Sabik & Ahmed Saifee
HD-18, Suhrawardy Hospital Qr.
Shere Bangla Nagar, Dhaka.

Printed by : Helpline Resources
2nd level, Sobhan Mansion
46/1, Purana Paltan, Dhaka-1000
Phone : 9571027, 7170132

Concept & Cover Design :
Insight
research, development & media services

Price : Tk. 200.00

Preface

Care in ICU is complex with postop cardiac patients. Purpose of this text is to presents principles and practical fashion activities in algorithm for ICU personnel with an emphasis on the development of sound judgement based on thorough evaluation of clinical picture than the derived numbers and calculations.

This manual is based on and brings together the experience of Japan NCVC, The Johns Hopkins, Stanford UMC, New England Medical Centers Faculty of Cardiac Surgical patients care and NICVD, Dhaka.

We hope this should help to develop a basis of an effective team approach of paramount importance for safe convalescence of Surgical patients.

We tried to be more concised to provide all practical things together for easy accessability to guide.

This mannual was first published to meet the need of bedside clinical care of our own patients on March 2003

With the growing need for surgical ICU care throughout the country,we hope this manual will help to build an uniform responsible approach to surgical patients of our country.

We wish to acknowledge all of the physicians, anaesthetists, nurses,students and other personnel involved in the care of our surgical patients

Prof. N.A. Kamrul Ahsan

Contents

Appendix

Cardiogenic Shock 1

Systolic failure

LOS *(Low outputSyndrome)* **:**

1. Myocardial Infraction

2. Severe arrhythmia

3. After cardiac surgery.

Diastolic failure :

Cardiac tamponade

Pneumothorax (Tension)

Shock : Defined as a state caused by Cardiac dysfunction signaled by

CI < 2.0 L/min/m^2 & elevated PCWP.

Diagnosis :

1. **BP < 90mm Hg** (if previous pressure unknown)
   or **25 to 30mm Hg** BP lower than before (measured).

2. **Decrease in Blood circulation**
   (a) Urine **20 ml/h**
   (b) Consciousness Disturbance
   (c) Constriction of peripheral Vessels (cold pale extremities) < 3°C
   (d) Cardiac Index **< 2.0 1/min/m^2**
   (e) CO **< 25 ml/beat/m^2**

ETIOLOGY :

TYPES OF SHOCK

CARDIOGENIC :

(INADEQUET OUT PUT)

- Large MI (> 30 to 40%LV), Acute MR, Acute VSD, ARVF, Acute Cardiac Tamponade
- Arrrythmias
- Congestive Heart Failure

HYPOVOLEMIC :

- Hemorrhage
- Trauma
- Burn
- Fluid loss

LOW RESISTANCE (SVR)SHOCK :

- Sepsis
- Anaphylaxis
- Neurogenic

OBSTRTACTIVE SHOCK :

- Tension Pneumothorax
- Pulmonary Embolism
- Cardiac Temponade
- Cardiac tumor

Pathophysiology of Cardiogenic Shock :

1. **LV functional failure (a pump failure)**

→ LOS →→ Cardiac output ↓

PAP ↑

PAWP ↑

(By Swan gauz Catheter)

2. **Proxymal temp. (Rect. /Oesph.)** ↑

Peripheral vessels constriction →→ no superfical radiation of profund heat

3. **LV failure** → **Diastolic Volume** ↑

→ **LVEDP** ↑ → **LAP** ↑ → **PAWP** ↑

(> 15 mmHg)

4. **C V P : > 15 cm H_2O**

5. LOS → Anoxia in Brain → Consciousness → Spzasm ↓ Cerebral edema.

Myocardial Contraction ↓

↓

Cardiac output

↓

B P

↓

Peripheral Circulatory failure

↓

Hypoxia in the tissue

↓

Metabolic acidosis

Monitoring and exam :

1. **Physical findings :**

   (a) Pulmonary congestion ⟶ moist rales

   (b) Arrhythmia
   Shallow and irregular breathing

   (c) B P : Monitoring (continuously).

   (d) Consciousness disturbance
   Peripheral Cyanosis (Lips and Extremities)
   Peripheral coldness - due to peripheral vascular Sweat

Urine :

Baloon Catheter : **Vol/h**
(1 ml/Kg/h)

Specific gravity
Osmotic pressure
Electrolytes
} Check

E C G monitoring

C V P (Central Venous Pressure)

Body temp.

Proxymal temp. > Peripheral temp. **difference :**

↓ **Better condition** ↑ **Worse condition.**

Chest film :

CT Ratio

Heart shape and size

P A, Atrium

Lung congestion, pulmonary edema

Atelectasis

Biological exam :

Serum electrolytic (Na, K, Cl, Ca)

BUN, Creatinine

Blood exam :

R B C, W B C, Hct, Hgb.

Fluid Balance :

Blood gas :

AO_2 -

Base excess.

Intracardiac Pressures :

Swan gauz Catheter

- R A
- R V
- P A W (after inflating balloon) ⟶ LA

Thermodilution ⟶ **Cardiac output (If available)**

Indication of Ventilation :

Breath ing	:	**> 55**	**0r < 5 times / min**
Tidal Volume	:	**< 150 ml**	
Vital capacity	:	**< 500 ml**	
Pa O_2 (mmHg)	:	**< 70**	**(under FiO_2 1.0)**
Pa CO_2 (mmHg)	:	**> 80**	**(except chronic respiratory failure)**
A-a DO_2 (mmHg)	:	**> .450**	**(FiO_2 1.0)**

TREATMENT OF THE CARDIOGENIC SHOCK :

Principle & measures

Clinical Symptom (Myocardium contraction)

Shock → Congestive heart failure

Pump action of the heart ↓ → Coronary Blood flow ↓

ADDRESS : ABC's of resuscitation

Restoration of these failure (main problem) :

- Measures for anoxia
- Correction of acidosis
- Blood transfusion
- Fluid with electrolyte Balance

I. Place in Trendelenberg Position

II. O_2 Inhalation - 100% O_2 via face mask or intubation of indicated

(FiO_2 depends on PO_2 & PCO_2)

III. Correct maintenance of the air way :

Prevention :

From 1) Drop of the tongue root.

2) Pneumonia due to aspiration.

⟶ **Endotracheal intubation**

⟶ **Tracheostomy.**

IV. I.V. fluid

V. CaCl one ample IV bolus (repeat if needed)

VI. If in early Post Op. with dopamine **renal dose** (0.3 mg/ Kg/min), increase for **pressor effect** (15 - 20mg/ ug/min)

VII. Notify fellow / surgeon

SPECIFIC ETIOLOGY :

I.	Hypovolemia	:	Add Vol.
II.	MI / Ischemia	:	See appendix, P 80
III.	Temponade/Acutely Vol. loading	:	See appendix, P 91
IV.	Arrythmia	:	Vide infra P 10
V.	Pneumothorax	:	Evacuation

VIII. Mechanical Ventilation : Indicated

Tidal Volume (< 150 ml)

Frequency of respiration < 6 times/min.

> 55 times/min.

$PaCo_2$: > 60 mmHg

PO_2 : no or small change after FiO_2 ↑

IX. Decreased SVR :

a. Volume expansion

b. Vasopressor therapy

1. Phenylephrine (Neo-Synephrine 10 - 100 mg/min (10mg/250 ml mixture)

2. Norephrephrine 1 - 10 mg/in (4mg/250 mix)

or lower than 90 mmHg (if the previous pressure unknown.)

X. Treatment of Cardiac Failure.

1. Blood Transfusion → **CVP** ↑

Fluid Infusion ↗

no change in S/S → Cardiogenic& Vasogenic Drugs.

Assesment :

BP 20 - 30 mmHg lower than previous pressor

Mixes and Dosage Ranges of Vasoactive Medication :

Medication	Class (Stimulation)	Mix.	Dosage
Dopamin	α_1/β_1/DA	**200 mg/250 ml**	**2 - 20 mic/kg/min.**
Dobutamine	α_1/β_2	**200 mg/250 ml**	**2 - 20 mic/kg/min.**
Epirepsrine	$\alpha_1/\beta_1/\beta_2$	**1 mg/250 ml**	**1 - 4 mic/min.**
Isoproterenol	$\beta_1/\beta_2(\beta_1>\beta_2)$	**1 mg/250 ml**	**0.5 - 5 mic/min.**
Amrirone-P	α_1/β_1 Esterase	**200 mg/200 ml**	**.75 mic/kg Bolus than 15mic/kg/min**
Novepirephromic(Lovophed)	α_1/β_1	**4 mg/250 ml**	**1 - 10 mic/min.**
Phenyl ephrine (Neo-Syn ephrine)	α_1	**10 mg/250 ml**	**10 - 100 mic/min.**

Note : X - mg in 250 ml gives an infusion rate X micrograms in 15 drop e.g. 200 mg/250ml mix gives

200 mg in 15 drops (60 drops = 1 ml. 15 drop/min = 15 ml/hr)

	Initial Dose	Myocardial Contraction	Heart beats	Arrhythmia	Peripheral Resist	Renal Flow	Coronary Flow	Cardiac output	Other Ref.
Isoprotarenal	005	↑↑	↑↑	→	↓	→	↑	↑↑	good for *
Dopamin	3	↑↑	↑	↑	↓	↑↑	↑	↑	**
Dobutamin	3	↑↑	↑	→	↓	→	↑	↑	
Adrenalin	0.1	↑↑	↑↑	↑	↑	↓	↑	↑↑	
N or Adrenalin	0.1	↑	↑→	→	↑	↓	→	↑	

* Cardiac resuscitation,
Heart block, bradycardia,
Bronchial → dilatation
Pulmonary Vessels

** Dripping speed
↑ → ↑↑↑
Renal artery
spasm → Urine ↓

2. **Digitalis** good for congestive heart failure with tachycardia

Heart contraction ↑ Cardiac output ↑↑

↓

Peripheral Vessels spasm ↓

↓

↑ Venous return ⟶ Cardiac output ↑↑

Dose : 0.25 mg I.V.q_4 – 6 hr. for 4 dosage than 0.25 mg. I.V/P.O. qd.

3. **Frusemide**

20 - 40 mg i v every 6 - 12 hrs

4. **Vasodilator :** good for the heart by taking off the after load.

Nitroprusside : 0.1 - 10 mg/Kg/min (50mg/250ml mix)

Cyanide poisoning Treatment :

(care for BP ↓↓) _ Hydroxyecobalami + infusion + Na thiosulphate

Nitroglycerine 0.1 - 10mg/Kg/min (50mg/250ml mix)

6. **Adequate Fluid transfusion** (kind of Fluid under the careful attention to CVP, Vital signs,electrolytes and acid-base balance).

7. **Ant arrhythmic treatment :**

i) PVC (Premature Ventricular Contraction)

Lidocaine 50 - 100 mg IV

if not effective 50 mg 3 - 5 min later

if effective 1 - 4 mg/min observe 1 - 2 days under lidocaine dripping

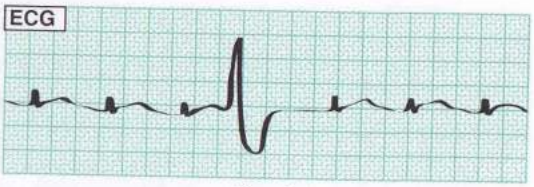


Fig. 1

If lidocaine is not effective

a) **Procainamide** 200 mg IV (Over period of two hours)
100 mg/H dripping

b) **Pay attention to** ⟶ R on T
Frequent PVC
Multi form of PVC

ii **PAC (Prematured atrial contruction :**

a) Atrial over drive pacing

b) Digoxin : 0.5 mg IV stat then 25 mg I.V. q 4 hr 2 doses (Total 1 mg loading over 8 hrs.) then .25 mg PO maintain (adult).

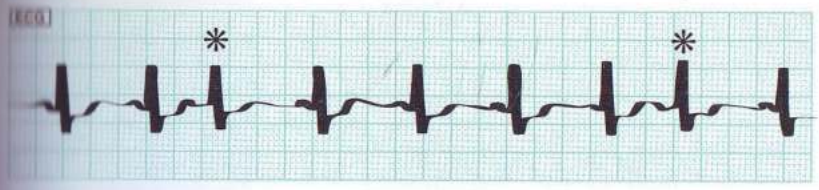


Fig. 2

c) Quinidine

d) Procaniamide

e) Verapamil

b. Tachyarrhythmias

b-1 Supra ventricular Tachycardia (Uncommon after Cardiac Surgery, Needs D/D Atrial Flutter with 2 : 1 block)

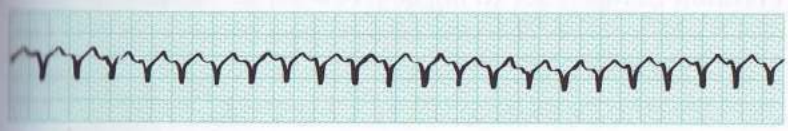


Fig. 3

1. Rapidatrial pacing overdrive
2. Veraparnil 5 - 10 mg IV slowly
3. Propanolol /Esmolol

b-2 Atrial flutter (250 - 350/min) :

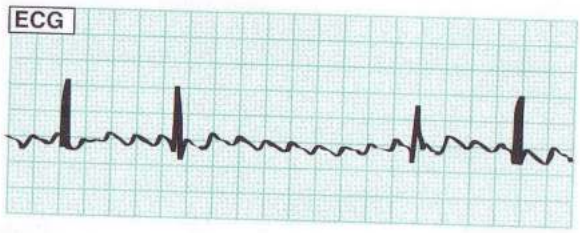


Fig. 4

Fibrillation (> 350/min) :

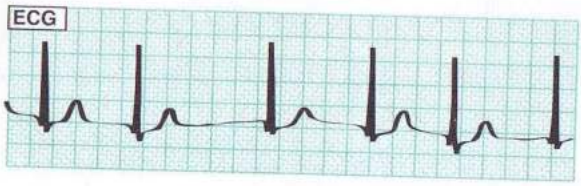


Fig. 5

Etilogy :

1. Poor preservation in cross clamp
2. Rebound with b blocker
3. Metabolic (Hypoxia, K +↓)
4. Atrial distention
5. Surgical Trauma

Prevention : Propanolol 10 mg qd /Atenolol 25 mg qd

a) Digoxin .25 - .5 mg IV
⟶ .5 mg I.V. q 4 - 6h
for 3 doses then .25 mg PO qd.

DC defibrillation : (50 - 200 Jules) Dangerous if digoxin level is acutely raised

b) **Verapamil** 2.5 - 5 mg IV q 1 - 2h prn
⟶ May cause hypotenuse if bolus of > 2.5 mg (without test dose)

c) **Esmolol** – 500 mg/Kg IV over 1 minute then infusion 50 - 200 mg/kg/mi

d) **Proponolol** 0.5 mg/I.V. to .1 mg/Kg increasing every 30 min.

e) Once ventricular response has been controlled Type 1A Anti arrhythmic drugs (quinidine or procainamide) ⟶ useful to sinus rhythm.

Dose : **Quinidnie** 200 mg q 2h × 4
Procanimide I.V. 500 - 1000 mg load (100 mg every 5 min.) then (2 - 4 mg/min infusion)
(P.O) P. Hcl 100 mg, then 500 - 1000 mg q 6h procanimide SR

if converting I.V. to P.O. stop infusion after P.O.

b-3 Premature Ventricular beats (PVC) :

Fig. 6

Evaluate

KCL @ 20 mEq/h (Each 2 MEq increases K + by 0.1 mEq/L
Lidocain/Procainamide

Ventricular tachycardia :

Fig. 7

Over drive pacing

Lidocain 1 mg/Kg IV repeat 1/2 times after 10 minutes to start infusion

(2 mg/min of mi x 1 gm/250 ml).

Procainamid 100 mg I.V. every 5 minute upto 1000 mg ⟶ Infusion (2mg/min of 1gm/250 ml mix) use if ectopy persists or despite Lidocain

Amiodarone (Cordarone) : Indication - Recurrent VT/VF, AF

Dose : 600 - 1200 mg PO for 2 Wk then 200 - 400 mg qd.

IV : 150 mg iv bolus for 10 mins then 900 mg in 500 ml DNS @ 30 ml/H for 24 H

DC shock in Persistement VT :

b-4 V.F : DC shock **(Protocol of Cardiac arrest to be started in Pulseless VT/VF)**

c. Bradycardiac arrhythmia :

c-1 Sinua bradycardia

First line of therapy ⟶ RA pacing.

RV pacing next if without atrial wires

If pacing unavailable or fail :

Atropin IV 0.25 - 0.5 mg IV/Isoproterenol 0.3 - 4 mg/min **(Start at 5 micro drops/min of 1mg/250ml mix)**

If HR < 60 beats/min ⟶ **RA Pacing**

c-2 **S - A Block** → R A Pacing
Atropin 0.25 - 0.5 mg IV

c-3 **A - V Block**
RV Pacing

c-4 **Bundle Branch Block**
Bifascicular Block → RV Pacing

8. Corticosteroid :

Use this in the early stage of Cardiogenic shock (end stage no effect)

Prevention of myocardial damage

- cardiac contraction ↑
- constricted peripheral Vessel → dilatation
- stabilization of the cell membrane

RBC
Endothelium
Blood Platelet agglutination
→ Cell membrane. ↑
↓

9. Correction of metabolic acidosis :

BE > 6 then NH CO3 = BW (Kg) × .3 × less deficit

(usual Preperation) 1 ml - 0.85 mEq

7% " 20 ml - 17 mEq.)

1) initial ½ dose
Check BE again

2) Emergency 1 mEq/Kg.

10. Fluid infusion :

CVP and PAWP (For assessment)

Correction of Electrolytes

11. ME HANICAL CIRCULATORY SUPPORT

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 - Bryncho nenkho 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

B. 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 flow		

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) : Cardio West 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/3MElectric 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.

A. Counter pulsation : IABP

Physiology & 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 then 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 O_2 consumption. 3 parameters can be adjusted accordibg to changing requirement of Pt.

Ballon size : 4.5 - 12.0F (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)

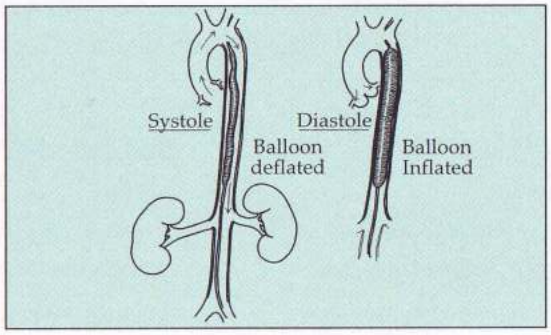


Fig : Correct positioning IABP

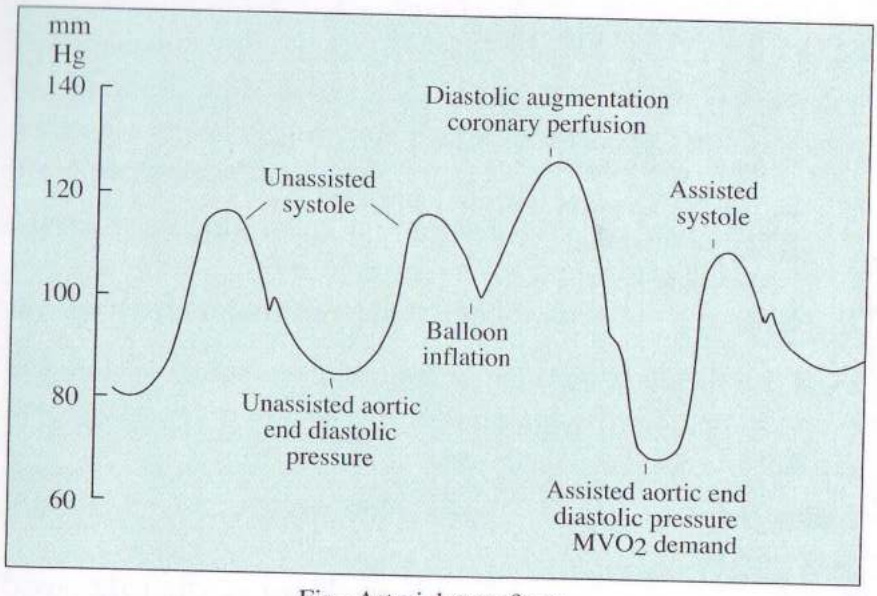


Fig : Arterial waveform

Inflation must occur just after aortic valve (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.

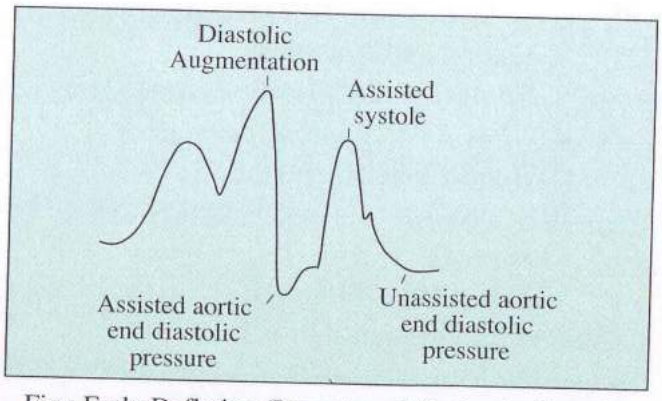


Fig : Early Deflation (Premature deflation in diastole)

Characters :

1. Sharp drop after diasl. augmentation
2. Suboptimal augmentation
3. Assisted EDP equal/greater
4. Assisted systolic pr. may rise

Effects :

1. Suboptimal CA perfus.
2. Retrograde CA perfus.
3. Angina
4. Suboptimal afterload
5. Increased O_2 demand $\downarrow$

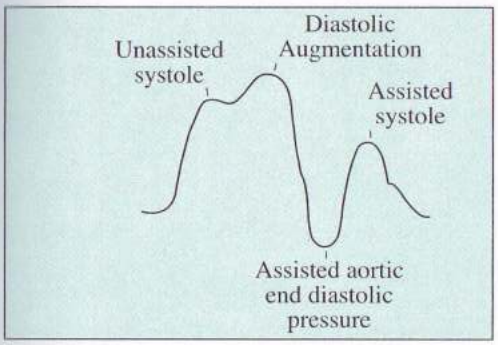


Fig : Early Inflation

Character :

1. Inflation before notch
2. May encroach systole

Effects :

1. Premature closure AV
2. LVEDP/PCWP ↑
3. Increased MVO_2 demand
4. Increased LV wall stress/ after load
5. Aortic regurgitation

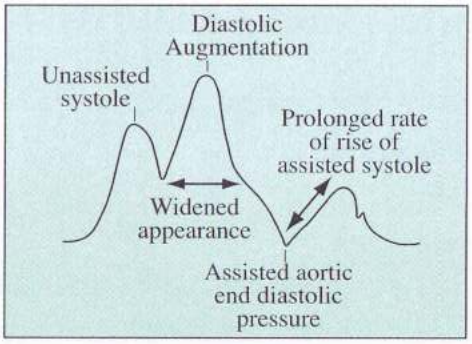


Fig : Late Deflation

Characters :

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

Effects :

1. Afterload ↓ essentially absent
2. Impade LV ejection
3. MVO_2 ↑ prolonged

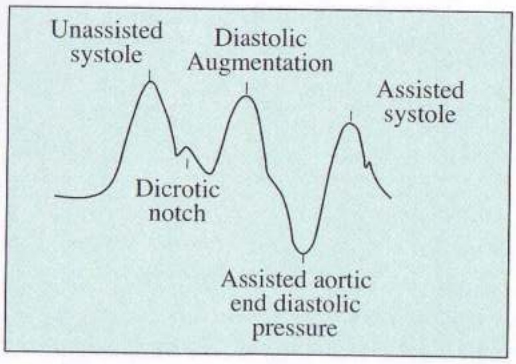


Fig : Late Inflation

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 FREVEALS CORRECT TIMING

WEANING FROM IABP :

PRINCIPLE :

To withdraw support incrementally & to asses haemodynamics at each step

- Pt must be stable with minimum inotrops
- CI > 2L/min/m_2
- Systolic Pressure > 90 mmHg
- LA RA pressure < 20 mmHg
- HR < 100/min
- Urine > 0.5 m_l/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 6 hrs, 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)

C. 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

Fluid Transfusion 2

MOST Pts. GAINS Wt. IMMIDIATE POST OPERATIVELY DUE TO CAPILLARY LEAKAGE WITH VASODILATATION THAT OCCURS WITH WARMING (5 - 10Kg more than preop weight). Concept of fluid admin is to maintain intravascular vol. without contributing interstitial oedema. With stable core temp. this phenomenon ceases.

Diuresis should be initiated if after stabilization, appears overloaded during early post operative period.

With out any temptation to expand volume preload should be increased only to maintain satisfactory CO & tissue perfusion.

Standard Volume and contents of transfusion fluid

Blood & colloid are preferable to hypotonic or even isotonic crystalloid sol. to expand vol.

After open Heart : 5 - 10% Dextrose

Wt.	Standard	Maximum
< 10 Kg.	50 ml.Kg/day	60 ml/Kg/day
10 - 20 Kg	20	40
> 20 Kg	10	30

Remember transfused fluid from injection line

After closed heart operation (Electrolyte fluid)

Wt.	Standard	Maximum
< 10 Kg.	50 ml.Kg/day	100 ml/Kg/day
10 - 20 Kg	30	60
> 20 Kg	20	40

Dose :

KCI :

Open heart 2 mEq / Kg/day

non-open heart 1.5 mEq / Kg/day

If Serm potassium < 3.5 mEq / 1KCl start 0.2 mEq / Kg/h for 3 hrs. into CVP then check serum potassium

GIK Treatment (Glucose, Insulin, Karium)

Indication :

1) Within 24 hrs. after the open heart
2) If Frequent P V C is observed
3) Sepsis

Contents and doses :

50% Glucose	200 ml.	
Aqua	250 ml	20 ml / h
KCI	50 mEq.	adult
Regular insulin	20 units	

Within 24 hrs. after the open heart

50% Glucose	400 ml.	
KCI	100 mEq.	20 ml / h
Regular insulin	40 units	adult

In children, KCl of G I K is controlled at 2 mEq / Kg / day

Serum Potassium

Hyperpotasemia

1. **5mEq / 1 > Serum K + < 6 mEq / 1**
   i) Discontinue K + administration
   ii) Diurectics is to be given (Lasix)

2. **Serum Potassium > 6 mEq / 1.**

   i) G I solution Therapy

50% glucose	400 ml	40 ml/h.
Agua	100 ml	
Insulin		40 units

   ii) $NaHCO_3$- to corret acidosis at pH 7.40 - 7.5

[7% $NaHCO_3$ (Maylon) ⟶ 1ml contains > 0.85mEq/ml (1mEq = 80mg)]

Dose : 1 ml/Kg IV (1ampl = 50 mEq)

iii) CaCl 0.1 ml/Kg (IV)

iv) Regular Insulin 10 U/25 gm 50% Dextose IV

iv) Digitalis

v) Peritoneal Dialysis

Hypopotasemia

Serum K < 3.5 mEq/1

K loading

KCl > 0.2 mEq/Kg/h for 3 hrs. C I V

check serum K after loading

Serum Na.

1. **Hypernatremia** (Wt_1 - Wt_2 = Present Wt.)

   Serum Na > 145 mEq/1

   Free water (5%) (1)

   Deficit Wt = Wt x 0.6 x $\left(1 - \frac{145}{\text{Serum Na}}\right)$

   ..... C I V / 2 day.

2. **Hyponatremia** ($W_2 - W_1$ = Present Wt)

   Serum Na - 135 mEq/1

   NaCl (mEq) = Wt (Kg) x 0.6 x (135 - Serum Na)

   ..... C I V / 1 day

   [10% NaCl 10 ml = Na 17 mEq.]

   [3% NaCl 500ml

[Hypernatremia only water has been lost

Normal Wt $\times$ 0.6 $\times$ 140 = Pt Wt $\times$ 0.6 $\times$ (Na+)

(Normal Wt = W_1) (Pt Wt = W_2)

$Wt_1 \times 0.6 \times 140 = W_2 \times 0.6 \times (Na+)$

Body fluid $Wt_1 \times 0.6 = \dfrac{Wt_2 \times 0.6 \times (Na+)}{140}$

Deficit Vol $= \dfrac{Wt_2 \times 0.6 \times (Na+)}{140} - Wt_2 \times 0.6$

$= Wt_2 \times 0.6 \left(\dfrac{(Na+)}{140} - 1\right)$]

Serum Cl

1. **Hyperchloremia**

   Serum Cl > 110 mEq / 1

   non-electrolyte Fluid (1)

   $= \text{Body weight} \times 0.6 \times \left(1 - \dfrac{100}{\text{Serum Cl}}\right)$

   ..... C I V/2 day

2. **Hypochloremia**

   Serum Cl < 90 mEq/1

   Cl (mEq) : Wt $\times$ 0.6 $\times$ (90 - Serum Cl)

   : Wt $\times$ 0.2 $\times$ (100 - Serum Cl) ... C I V / 6 hrs.

Serum Ca

1. **Hypercalcemia**

   Serum Ca > 12 mg / dl

   1) Na loading 10 mEq / Kg / day C I V
   2) Diurectics

      Lasix 0.2 mg / Kg IV

2. **Hypocalcemia**

   Serum Ca < 8 mg/dl

   1) Ca loading 40 mg / Kg / day C I V
   2) 8.5 % gluconic Ca 0.5 ml / Kg / day
   3) 2% $CaCl_2$ 2ml / Kg / day

Acid-Base Balance 3

Diagnosis of Acid Base Balance :

Blood gas

	PH	HCO_3	HCO_2	BE
Metabolic Acidosis	↓	↓↓	↓	↓↓
Metabolic Alkalosis	↑	↑↑	↑	↑↑
Respiratory Acidosis	↓	↑	↑↑	↑
Respiratory Alkalosis	↑	↓	↓↓	↓

Serum

CI	K
↑	↑→
↓	↓
↓	↑
↑	↓
↓↓	↑

Primary change

Secondary change

Normal Value of Fluid Electrolytes

Na+ (serum)	3.5 - 5.0 mEq / 1
Na+ (serum)	155 - 145 mEq / 1
Cl	95 - 10 mEq / 1
Ca	8.5 - 10.5 mg / a1
Hg	1.5 - 2.5 mg / a1
PH (artery)	7.0 - 7.45
PaO_2	80 - 100 mmHg
$PaCO_2$	35 - 45 torr
HCO_3	25 - 50 mEq / 1
BE	± 3
(Serum osmotic Pressure)	275 - 295 m Om / 1
(Collagen osmotic Pressure)	25.0 - 30.0 mmHg

Treatment of Acid-Base imbalance

A. Metabolic Acidosis

1) Exclusion of the Cause
   Hypoxia, shock, Diabetes mallitus
   Renal Failure.

Treatment : Should be started when $HCO_3 < 20mEq/l$
$NaHCO_3$ (mEq) = Base Deficit (mEq/1) x Wt (Kg) x 0.3
It should be given, divided into 2 dose

Effect : Myoardial contractility reduced, attenuates inotropic action of catacholamines

Sudden change to Alkalosis, may cause
Arrhythmia and cardiac ardest due to serum
Ca++ decreasing
$CaCl_2$ ready
Gluconic Ca

B. Metabolic Alkalosis

Excessive dose of $NaHCO_3$
Excessive Loss of K + $\longrightarrow$ due to diuretics
Loss of gastric Juice
Cl Loading
Chloride deficit (mEq) = (100 mEq / l - Cl mEq/l at present) × Body Wt (Kg) × 0.3)

[**Base-excess method :** mE HCl = Base excess × Body weight (Kg) × 0.3]

Treatment : Usually KCl at rate not exceeding 20 mEq / hr

If not effective - [5% G1 lit + 2 N. HC 60 - 120 ml]
0.12 -0.24 N HCl (120 – 240 mE/l) into CVP line for more than 12 - 24 hrs

{Normal Saline (Cl : 154 mE / l), at hypopotasemia (NS + KCl) soln not more than 40 mEq / H }

C. Respiratory Acidosis

Cause Ventilatory Volume ↓

Alveolar diffusion ↓

During mechanical ventilation TV, RR alteration to be done

($PaCO_2$: 40 Torr 60 Torr

Cerebral Flow ↑ → ↑ Cerebral Pressure → Cerebral Ocdena

Intubation → Ventilation (Tidal Vol. = 10 - 19 ml/mg).

D. Respiratory Alkalosis

Hyper Ventilation due to shock condition

If on ventilation RR/TV to be adjusted

A low PCO_2 should be evaluated

$PaCO_2$: 40 Torr → 20 Torr

Cerebral blood flow ↓ → Unconsciousness
irreversible damage in the brain.

Intubation
Muscle relaxant
Diazepam to
stop hyperventilation
} Control the respiration

IV. Blood Gas and O_2 treatment

Hypoxemia : O_2 in the circulating Blood ↓

Hypoxia : O_2 in the tissue ↓

Asphexia : O_2 Supply ↓

CO_2 discharge ↓

anoxia : Hypoxia

Hypoxia 4

Hypoxia (classification by Saklad)

1. Hypoxic Hypoxia

a) Atmospheric Hypoxia

O_2 in Inhalation

air O_2 21%

N_2 79%

760 mmHg

(normal Atmosphere)

PO_2 in the air : 760 mmHg x 0.21 : 159 mmHg

PO_2 at an altitude of 6000 m 70 mmHg

Atmosphere at an altitude of 15000 m 87 mmHg

(Vapour alveolar : 47 mmHg

CO_2 40 mmHg

> 87 mmHg

No space to O_2

b) Tidal Hypoxia - (O_2 tidal Volume) ↓

Ventilation in the lung ↓

Emphysema, obstruction in the airway,

Hypofunction in respiratory center,

Paralysis of respiratory muscle due to anesthesia and relaxant inj.

c) Alveolar Hypoxia

O2 distribution ↓ through alvaolar to the blood

Pulmonary edema

Pneumonia

A - V shunt in > atelectasis./ Emphysema ↓ PO_2

2. Anaemic Hypoxia

Hge ↓ O_2 in Blood ↓ e.g. Anemia

O_2 combinning drafunction ⟶ CO intoxication of R B C methamoglobinemin

3. Stagnant Hypoxia

R.g Cardiac failure

Blood circulation ↓ ⟶ O_2 Supply to the tissue ↓

Emboli, thrombus

4. Demand Hypoxia

O_2 demand in the tissue even in the normal ventilation

e.g. High fever,

Hyperthy roidism

5. Histotoxic Hypoxia

Tissue intoxication causes disturbance in O_2 intake and consumption

e.g. Cyanide intoxication

TABLE (Hypoxia)

PO_2 O_2 Vol.. % and Saturation in each hypoxia

	Arterial Blood			Venous Blood			O_2 (Vol.) % gradient between A and V
	PO_2 mmHg	O_2 (Vol. %)	SO_2 sat (%)	PO_2 mmHg	O_2 (Vol. %)	SO_2 sat (%)	
Normal	90 - 95	19 - 20	95 - 97	40	14 - 15	70 - 75	5
Hyoxic	↓	↓	↓	↓	↓	↓	↓
Anemic	→	↓	→	↓	↓	↓	↓
Stagnant	→	→	→	↓	↓	↓	↑
Demand	→	→	→	↓	↓	↓	↑
Hestotoxic	→	→	→	↑	↑	↑	↓

Increasing Factors of Hypoxia

Patient	Age, Obesity, Cardiopulmonary Diseases, Anaemia, Decreasing of Blood circulation, Acidosis, Injury.
Mechanical Factors	Pain Abnormal position of the Body, Sprint Dressing Bandage, Abdominal distension
Medical Factors	**Narcotics, Sedatives, Anesthesia drugs, Muscle relaxant**
Others	Vasoconstrictor, inappropriate positive Pressure Ventilation.

Symptom of hypoxia

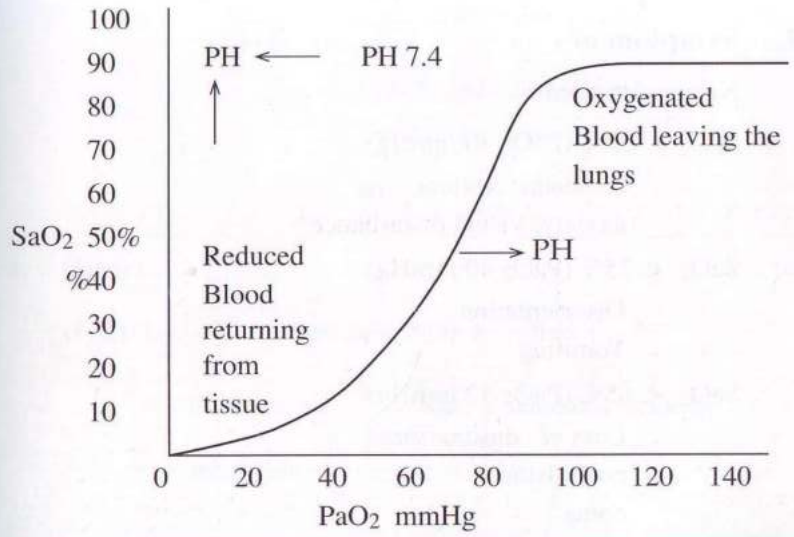
PaO_2 > 50 mmHg — Symptom (+)

(-O_2 Sat < 85%)

Cyanosis (+) — Hgb without O_2 > 5 gm/dl

= O_2 sat < 67%

Disociation curve of Hgb - O_2



Normal person's breathing in the Room air

PaO_2	100 mmHg	PaO_2	50 mmHg
SaO_2	95	SaO_2	85%
$< PO_2$	50 mmHg ⟶	Hgb O_2 ⟶	Hgb + O_2
	unloading point		

Factors shifting to the right

PCO ↑
PH ↓ (acidosis)
Body Temp. ↑
PH ↓ even in the same PO2
↓
SO2 (%) lower
PH ↑ even in the same PO2

SO2 (%)higher (Blood oxygenation easily go-up) ↑

HYPOXIA

1. Symptom of -

Nervous system

SaO_2 < 85% (PaO_2 50 mmHg)
Headache restless
anxiety, Visual disturbance

SaO_2 < 75% (PaO_2 40 mmHg)
Disorientation
Vomiting

SaO_2 < 65% (PaO_2 32 mmHg)
Loss of consciousness
convulsion
coma

Hypoxia ⟶ Cerebral Vessels dilate

Cerebral Blood ↑ + Permeability of cerebral capillary ↑

circulation ⟶ cerebral edema

Brain damage : irreversible

2. **Respiratory symptom** 3 minutes anoxia (PaO_2 60 mmKg)

Carotid body stimulate ⟶ Resp. Rate ↑

Aortic body stimulate Ventilation Volume ↑

Hypoxia ↑↑ ⟶ Ventilation Volume

irregular Resp. > apnea.

3. **Circulatory** : Pulse rate ↑ is related to SaO_2

SaO_2 70% / 30% ↑ in PR ⟶

several minutes O_2 treatment PR 10% ↓

= hypoxia was existed before

BP temporarily ↑ ⟶ BP ↓

at the late stage

Brady cardia arrhythmia

Conduction disturbance

BP ↓

4. **Others**

Hypoxia ⟶ Catecholamine secretion ↑

Blood sugar ↑ ⟶ metabolic acidosis

renal flow ↓ ⟶ Urine ↓

Treatment with O_2

Best range in PaO_2 : 100 - 150 mmHg

Cause of Hypoxia	Treatment
1. Inadequate oxygenation in the normal lung	
a) shortage of O_2 in the air	a) O_2 Inhalion -
b) obstruction of the air way	b) Removal of cause
c) neuro-mascular disturbance	c) adequate ventilation with air
2. Inadequate oxygenation in the abnormal lung	
a) Ventilation decreases	a) adequate ventilation with air
b) Imbalance between alveolar O_2 and pulmonary blood flow	b) O_2 inhalation Hyperventilation with air
c) Diffusion disturbance	c) Same as the above
	3) Closing of the shunt
3. A - V shunt	
4. Inadequate distribution of O_2 by Circulation	
a) Anemia and abnormal Hgb	a) increasing of acting Hgb Hyperbaric O_2
b) Shortage of circulating Blood	b) Blood transfusion
5. Inadequate oxygenation in the tissue	
a) Abnormal O_2 demand in the tissue	a) O_2 inhalation
b) Intoxication of tissue enzyme	b) O_2 inhalation is not effective

Inhalation of the air by normal condition :

PaO_2 = 100mmHg (room air)

SaO_2 = 95%

Hgb = 15 g/dl

1 gm of Hgb combines 1.34 ml O_2

1.34 ml × 15g × 0.95 = 19.1 ml/dl

O_2 Volume desolved in the plasma

0.3 ml/dl/100 mmHg

O_2 total in Blood = 19.1 ml + 0.3 ml = 19.4 ml

Inhalation of 100% O_2

SaO_2 = 100%

Hgb : 1.34 × 15 × 100 = 20.1 ml/dl

Plasma :

A-V shunt in the lung minimal

PAO_2 in alveoli at inhalation of 100% O_2

PAO_2 = 760 - (40 + 47) = 673 mmHg

PAO_2 = 40 mmHg

PAH_2O = 47 mmHg

0.3 × 673/100 mmHg = 2.02 ml/dl (100 mmHg O_2 0.3 ml/dl)

Total in O_2 blood (dl)

20.1 + 2.02 = 22.12 ml/dl

O_2 in Hgb 20.1 - 19.1 = 1.0 ml/dl

O_2 in plasma 2.02

(100% O_2 760 mmHg O_2) 0.3 (95 /O_2 100 mmHg O_2) = 7

2.02 - 0.3 = 1.72 ml/dl

1.72 ml

+ 1.0 ml = (20.1 ml - 19.1 ml)

= 2.72 ml

O_2 Vol.% in Venous Blood = 15 ml/dl

A - V gradient of O_2 Vol. % = 4.4 ml/dl (19.4 - 15 ml/dl)

4.4 ml/dl + 2.72 ml = 7.12 ml/dl

7.12 ÷ 4.4 = 1.6

Hgb ↓ 10 g/dl + 100% O_2 = 118 % (18%) ↑

7 g/dl + 100% O_2 = 124 % (24%) ↑

Effect of 100% O_2 in Hgb

	Room air		O_2 100%	
Hgb g/dl	PaO_2 =	100 mmdlg	PaO_2 =	673 mmHg
	SaO_2 =	95%	SaO_2 =	100%
15	H B O_2 =	19.1	H B O_2 =	20.1
	dissolved O_2 =	19.1	diss O_2 =	2.02 14% ↑
		19.4		22.12
10	H B O_2 =	12.7	H B O_2 =	13.4
	dissolved O_2 =	0.3	diss O_2 =	2.02 18% ↑
		13.0		
7	H B O_2 =	8.91	H B O_2 =	9.38
	dissolved O_2 =	0.3	diss O_2 =	2.02 24% ↑
		9.21		11.40

[O_2 to the tissue in 1 minute
cardiac output × O_2 Vol % in the arterial Blood
Room 5000ml × 19.4/100 ml (ml O_2 / ml Blood)
Air = 970 ml
100% O_2 5000ml × 22.12 /100 ml (ml O_2 / ml Blood)
Usual O_2 consumption at rest 250 ml / min
In Room air, at rest only 1/4 consumed]

Effect of O_2 Inhalation

Effect on Blood gas

a) Oxygen

O_2 ⟶ Peripheral tissue (Usually 5 ml.dl O_2 needed)
(Hgb dissolved O_2 in serum)
100% O_2 under one atmosphere

$PAO_2 = 760 - (40 + 47) = 673$ mmHg

3 Atmosphere O_2

$PAO_2 = 673 \times 3 = 2000$ mmHg

Room air (O_2 : 21%)
Dissolved O_2 in Blood serum
0.3 ml/dl/100 mmHg usual condition

Hyperbaric (under 3 atmosphere)

0.3 ml/dl × 2000/100 mmHg ÷ 6.0 ml/dl

usually O_2 Vol % on A - V gradient 5 ml/dl

Only O_2 dissolved in blood serum under 3 atm. is enough to O_2 consumption in the tissue

(CO_2 carrying disturbed)

CO_2

Metabolism in tissue ⟶ CO_2 ↓ Produced

85% of CO_2 : HCO_3 in the Lung ⟶ out

Blood

$Hgb - O_2 \longrightarrow Hb + O_2 \longrightarrow$ tissue

$CO_2 + H_2O = H_2CO_3 = HCO_3 + H+$

In high PO_2 (lung) ⟶ $Hgb_3 - O_2$

Low PO_2 (tissue) ⟶ Hgb

In high acidity or High PCO_2 (tissue)

$Hgb\ O_2 = Hgb + O_2$

In low PCO_2 or low acidity

$Hgb \longrightarrow Hgb - O_2$

Measures of O_2 administration

1. Mask and. endotracheal tube

2. Nasal Catheter

3 l/min 30% 30% O_2

5 l/min 35%

7 l/min 40% (75 1/min discomfortable)

3. Vinyl mask

$O_2 < 60\%\ O_2$

Venyl Bag > 60% O_2

4. O_2 Tent O_2

Temperature, Humidity > are controlled in comfortable condition.

Difficult to keep O_2 above 50%

O_2 = 8 - 15 l/min 25 - 50%

5. Hyperberic therapy - CO intoxication . Cason disease etc.

Complication :

1. Dryness of the airway

Nasal cavity

Muous mem brane

Function of mucociliary blanket in the trachea, and bronchus is disturbed in the exposure of humidity under 70%.

Dry O_2 1) disturbs the function of mucociliary blanket

2) dries the secretion of the airway

3) Infection easy

a) Humidifier

(only) O_2 ⟶ not effective

H_2O ⟶ not enough

Cascade Humidifier

O_2 ⟶ good ↑

H_2O

+

Heater

b) Nebulizer

Production of aerosol

(Add Bronchus dilater, Sputum dissolver, cortison etc.)

Aerosol size

100 μ < Nasal cavity ↓ good for

Phrynx ENT therapy

3 - 100 μ Trachea

Big Bronchus ↓

1 - 3 μ Bronchus ↓ good for Bronchitis therapy

1 μ > To alveolus

In O_2 therapy, 3 μ > Aerosol required

a) **Jet type**
O_2 Venturi effect, H_2O size 1 - 4 μ
70%. 4 μ

b) Ultrasonic Nebulizer
0.8 - 1 μ

2. Lung Collapse

Sputum, Bronchus → Secretion → obstucts tbe Bronchus → peripheral

alveolar O_2 completely absorbed → lung collapse.

3. Oxygen apnoea

In severe disturbance of Ventilation
Much accumulation of CO_2
Produces disturbance of respiratory center ↓

CO_2 ↑↓ stimulates Respiratory Center

only Carotid and Aortic body control the maintenance of respiration by O_2 ↓

Injury of respiratory center has Hypoxic condition of long period and CO_2

Accumulation ↓
O_2 → PaO_2 ↓↑

Carotid body, Aortic body → function ↓

↓
Apnea
↓
CO_2 Accumulation
↓
Hypoxia

PaO_2 not 100 mmHg, adjust 80 mmHg around
By checking PaO_2 FIO_2 gradually should be elevated.

4. O_2 Intoxication

High FiO_2 and period of O_2 therapy causes the O_2 intoxication (usually more than FiO_2 O.8 and more than 12 hrs. of O_2 therapy causes it).

Nasal stopping cough, substernal pain, sore throat, conjunctivities ear pain, muscle pain of the legs vertigo, Headache.

Lung : mild and moderate degree of pulmonary congestion, effusion excretion in the lung.

Atelectasis.

Vital capacity is good to check it.

Prevention :

By FiO_2 0.4 - 0.5, maintain the PaO_2 100 - 150 mmHg.

When PaO_2 does not increase°» not only FiO_2 increasing, but also think of to prevent O_2 intoxication and lung collapse.

Why PaO_2 does not go up. PEEP is also good to prevent A - V shunt in the lung.

Management of Poor Oxygenation

1. Examine pt, ventilator setting & function arterial gases, chest x-ray.
2. Hand ventilate with 100% oxygen and increase FiO_2 on ventilator until problem is sorted out.
3. Ensure alveolar ventilation by correcting mechanical problem

(adjust ventilator, reposition endotracheal tube, insert chest tube)

4. Assess and optimize hemodynamics.
5. Add PEEP in 2.5 - 5 an H_2O in creament while decreasing FiO_2 to less than 0.5 ; continually assess cardiac output at higher levels of peep to adequate tissue oxygenation.
6. Treat bronchospasm / infectious pulmonary disease
7. Use diuretics if interstitial edema present.

Artificial Ventilation 5

Indication of Artificial Ventilation

Respiration rate	>	*35 /min*	*5 /min*
Tidal Volume	<	*150 ml*	
Vital capacity	<	*500 ml* (<	*10 - 15 ml /Kg)*
Pa O_2	<	*70 mmHg*	*(100% O_2)*
P CO_2	>	*60 mmHg*	*(except chronic respiratory Failure)*
A - a DO_2	>	*450 mmHg*	*(100% O_2)*

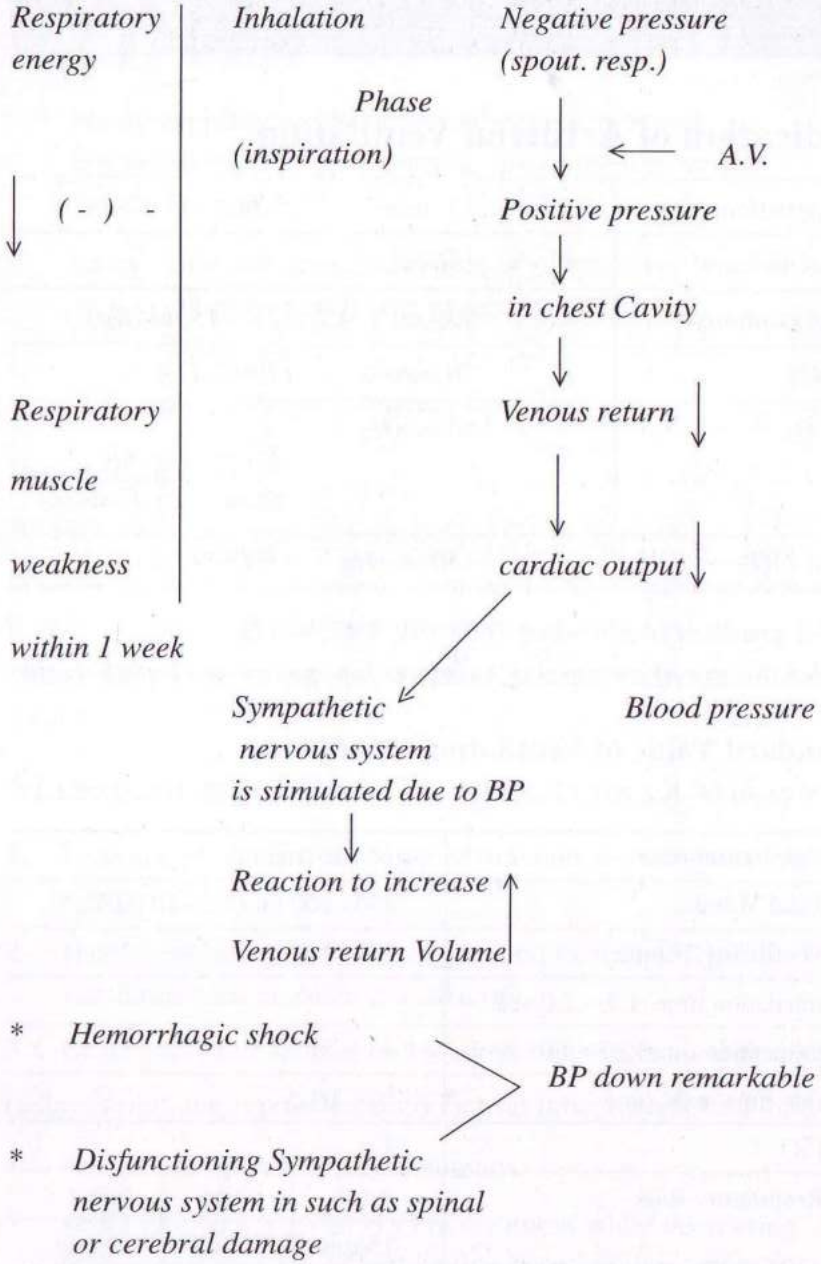
(Al-A gradient in alveolar) 760 - (40 + 47) = 673
under the use of analgesics, relaxant for spasms or Hypothermia.

Standard Value of Ventilation Condition

(Pt Weight 60 Kg.)

Respiration Rate	12 - 15 /min		
Tidal Volume	450 - 600 ml (7.5 - 10 ml/Kg)		
Ventilating Volume	5.4 - 9.0 l/min		
Inhalation time 1.3 - 2.0/sec.			
Exhalation time 2.7 - 3.0			
Inh. time /exh. time	$^1/_2$ - 1/1.5		
FiO_2	1.0		
Respiratory Rate	Adult	Child	Infant
	15/min	20/min.	30/min.
PEEP (Positive End Expiratory Pressure)	0 mm H_2O		
Maximal Inspiratory Pressure	25 mm H_2O		

Circulatory change right after artificial ventilation



Caution !

Usage of morphine or tranquilizer to control spontaneous breathing (lowers BP suddenly ↓↓)

Laboratory Exam. during A - Ventilation

Hemodynamical — Check Vital sign
Temperature
C V P

Hematology

Chest film

Urine Volume Urinalysis

Blood gas (PH, PaO_2 $PaCO_2$ HCO_2 HCO_3, Base Excess)

Electrolytes

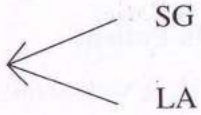
EKG monitoring

* **Chest Film :** Respiratory infection often easy to go to sepsis.

A. **Mediastinum :** Size of the heart and mediastinum
(+ pericardium)
Shape of the heart and mediastinum

B. **Lung :** Congestion of the lungs
Pulmonary edema
Pneumonia
Atelectasis
Pleural effusion
Pneumothorax
other abnormal shadow.

C. Confirm the position of a endotracheal tube
Confirm the cuff of a endotracheal tube

D. Confirm the position of each Catheters 

E. Electrolode Catheter of the pace maker

F. Confirm the position and no-flexion of the chest tube.

G. No abnormal shadow (Foreign body)

H. Conform the position of gastric tube and gas.

Management during Artificial Ventilation

1. Regular check of respiratory sound of the chest and chest wall movement.
2. Postural exchange and tapping or vibrating the chest wall to prevent the stagnant lungs. (Postural drainage) (Make the patient cough and deep breath with route-off if possible)

Watch the endotracheal tube and the route

3. Tracheo-broncheal suction with a sterile tube and hand Inflate the lungs after it with a bag endotracheal saline irrigation if thick discharge exists.
4. Pressure of the cuff of endotracheal tube is about same as the inspiratory pressure of A - Ventilation.
5. Prevention from infection
   oral irrigation with Anti- Biotic
   Ventilatory route : 1/week
   Sputum culture.
6. Regular check of ventilator :
   a) leakage b) route c) fighting d) confirm the respiratory rate e) FiO_2 f) endotracheal pressure g) tidal volume
7. Removal of the excessive water in the Ventilatory route
8. Chest film
9. Blood gas

The Cause of Fighting

1. In Patient

a) Spontaneous respiration
cough, (due to the tip of the tube deep)

b) Ventilation stimulating Factor
Stagnant secretion in the airway, Fever, Oxygen needed

(FiO_2 changed)

2. **In the airway**
   a) Endotracheal tube: obstructed/stenotic
   b) Tube-cuff : flat
3. **In Ventilator**
   a) In the route
   b) in the valve
   c) in humidifier and Nabulizer
   d) in trigger
   e) O2 source, driving system.

Procedure at fighting :

1) FiO_2 is up to 1.0
2) Suck cleanly the airway quickly.
3) Give sigh 1 - 2 time with manual way.
4) Repeat (2) again
5) Confirm and check the position of the tube, and the cuff.
6) * Return back FiO_2 to the previous setting.
7) Relaxant or tranquilizer or morphine given, prn.

* Never forget (6)

When weaning from the artificial ventilation starts.

1) **Blood Gas** *(FiO_2 1.0 PEEP = O cm H_2O*
   Tidal volume 5 ml/kg - 18ml/Kg)
   PaO_2 > 300 torr
   A a DO_2 < 350 torr (300 torr)
   QS/QT (intrapulmonary shunt) < 25%
   Pa CO_2 < 40 torr

2) **Vital sign**
   Hemodinamics > **Stable**
3) **Clear consciousness**
4) **No post operative bleeding**
5) **No abnormal findings in Chest X-ray.**
6) **No inhabitant factor for spontaneous respiration such as pain, muscle weakness or diseases.**

Steps to wean the Ventilation

Complete Ventilation
PEEP
↓
Assistant Ventilation
(IMV with PEEP)
Intermittent Mandatory Ventilation)
↓
Spontaneous respiration
(Voluntary) with CPAP (Continuous positive airway pressure)
↓
T Piece
↓ *Pa O2 > 80 torr*
Pa C O2 < 45 torr
Extubation

Cares in the Artificial Ventilation for long period

Watch the type of respiration chest movement

1. **Special types of respiration**
   Chene stokes ect.
2. **Shape of the thorax**
   Beer barrel like shape in
   Emphysema, Bronchiolitis, Asthma.
3. **Movement of the thorax**
   Asymmetric :- Pneumothorax
   Emphysema.
4. **Seasaw movement of the thorax and abdomen**
   At inspiration, abdomen is raised.
   chest wall down
   At expiration, vise visa
   Bronchial spasms.
5. **Convexity of the laynx or supraclavicular region at inhalation**
   Obstruction or stenosis in the upper or peripheral airway.

6. Auscultation of the breathing sound

Repeat with careful observation

a) Decreasing of respiratory sound
b) Prolongation of expiration and inspiration
c) Bronchial sound audible except at the physiological area
d) Abnormal sound or dry rale (+) e.g bronchial asthma
e) Moist rules bronchial secretion, sticky pus, blood, or pulmonary edema and pneumonia.

Not only check the rate of respiration but also watch adequate ventilation being performed :

- Thorax movement smooth and symmetric
- Respiratory muscle diaphragm and intercostal muscle move stabilly and naturally
- Depth, types of respiration.

Observation points on Artificially Ventilation patient.

1. *At postral exchange*

Care to – Kinking, excessive tension
Regurgitation of the airway (endotracheal tube, circuit etc)
Urinary catheter, chest tube, EKG wire, PM wire, material line, gastric tube, electrode of rectal temperature etc.
Devices : 1) Enough length of the route
2) Adjustment of the level of equipments
3) Loss or non-loading to the airway
4) Marking on the tubes
5) More adequate support
More adequate holding

2. Ventilation

Check the tidal volume
Ventilation volume of minute
Vital capacity, Rate of respiration, Pressure gauze, FiO_2, controlled or assisted
Confirm the present condition of Ventilation upon the machine.
Do not always rely upon mechine
Regularly calibrate equipment.

3. Heating and Humidifing:

* Be careful at and after changing a cascade or parts check quantity, and quality of secretion of the airway dry or excessive moisture of the airway
* Change the ventilator - and nebulizer circuit, and humidifier circuit every day.
* Use sterilized water for Humidifier to prevent the respiratory infection.

4. Vital sign

a) Body temperature

electric electronic / temperature
(check regularly itself)

b) E K G Monitor

Record the time and frequency of arrhythmia

c) Good accommodation of the Ventilator to the patient
no abnormal sound.

d) Blood pressure

in a direct method

Keep aseptic

Keep open

Keep in good position without dislocation, or Kinking.

e) C V P

No removal without special reason

(No use of IV injection through the tip of Swan Gauze Catheter).

f) General observation

Pt is in the abnormal and enforced position, without moving the hand, foot and speaking

Try to decrease mental anxiety pain, and understand what the Pt. wants.

With hearty and warm patients.

Tight fixation of the patient is dangerous and is to be avoided.

5. Emergent measures at disordered ventilator

Beside the Ventilator, ambu bag or an anesthesia machine is ready.

Practical procedures to the patient under Controlled Ventilation

I. Endotracheal suction (watch EKG always during procedures)

1) Wash the hands with 0.1% Hibitan solution
2) Put a disposable glove on the hand to holds the suction tube.
3) Taka a suction tube out of hibitan solution in a sterilized glass.
4) After suction, wipe the tube with alcohol sponge and clean inside with sterilized water by sucting,, and soak it in hibition solution.
5) Put the cap on the sterilized glass with sterilized water.
6) Use another suction tube for oral suction.
   i) Inform a conscious patient prior to suction.
   ii) Be careful of damaging mucous membrane of the airway, such tenderly and gently, time of each suction is limited less than 10 - 1 5 sec.
   iii) Sucking pressure is above 200 torr.
   iv) Do not use a suction tube, of which diameter is larger than a half of diameter of the endotracheal tube to prevent stelectasis of the lung.
   v) Such with rotating the tube to prevent the wall of the airway.

Before and af ter suction, FiO_2 is to be 1.0, to prevent hypoxia of the airway (suction for 30 sec decrease 25 torr of PAO_2, (50 mmHg $\longrightarrow$ 25 mmHg).

Tube : Less than $^1/_2$ diameter of the Endotracheal tube

Time : Less than 15 sec.

Pt. also holds breath during suction, which produce hypoxemia additionally.

II. Endotracheal Irrigation.

Where secretion sticky to such out. use 10 - 20 ml of warm N. Nacl of about 30°C, after enough administration of O_2 under FiO_2 1.0 with enforcing ventilation with a bag. Information and explanation are necessary to the conscious Pt. to get understanding and cooperation prior to this procedure.

After injecting the normal saline, ventilate with a bag, and tap the chest wall or back and suck finally. Usually 3 - 4 times for 24 hrs. It should be done under an well schedule.

III. Postural drainage.

Changing the position of the pt. to move secretion in the peripheral air way with a help of gravity to cough out the secretion more easily.

* Prevent the secretion in the healthy side.
* Tap the chest wall of the affected lung.
* Vibrator or percusser use if available.
* Inform the Pt. about the purpose and procedures.
* Finish the procedure adequately early.

IV. Suction under the direct vision

Using Broncheal Fiberroscope
Adequate O_2 before
adequate explanation to the pt.

Infection

Site of infection:
Endotrachea
Lung
Urinary Bladder
Inserted site of IV
Wounds of the operation
Inserted site of drainage wounds
Decubitus etc.

Bacterium of infection

gram (+)	*Staphylococcus*
	Strept coccus (Resistance (-)
	Pheumococcus
gram (–)	*Pneudomonus*
	E. Coli
	Kleshealla
Fungus	*Candida.*

Endotracheal infection

1) Keep clean around the site of Tracheostomy
2) Dressing on the operation wounds should be changed, if not clean.
3) Use a disposable globe for the endotracheal procedures.
4) At exchange the tube of tracheostomy enough suction, such around the tube by gradual deflation of the tube to prevent the secretion from following into the peripheral airway.
5) Exchange the tube of tracheostomy 48 hrs. after the tracheostomy, and then every 24 hrs.
6) Never forget to clean the hand prior to management.

2. Prevention of infection on the on the Urinary tract
   Change the balloon Catheter - every 5 days at least.
   Special care around the mentus of the urethra.
3. Prevention of infection on the various tubes connected to the blood vessels.
   * Sterilize the connection sites every day and cover them with a septic gauze.
   * Exchange the tube or 3 way cocks if possible.
4. Others.
   Request the X-ray technician, other paramedical staffs, and the family of the patient to wash the hand and put on the gown and cap and mask if necessary.

Nutrition :

Inform the patient the PO nutrition is more effective and necessary (1500 cal/day).

In 5% glucose solution

5% 4500 ml 25g glucose : 100 cal.

7500 ml is needed ⟵ 1500 cal.

1) Before PO nutrition is started check the peristalsis of the gastro intestine, and no obstruction.
2) Tell the patient not to pull out the gastric tube.
3) Require the family to encourage the patient.

At tubing nutrition

1) Semifowler position on the right side.
2) Give the nutrition similar to the time of the usual meal.
3) 3 hrs later after infusion, such the gastric contents and check how well it is digested.
4) During infusion, regurgitation into the oral cavity and the air way due to abdominal pressure is observed. Watch the infusion speed and the general condition.

Oral route :

1. Before it is started, give a swallow of blue or violet colored water to rule out aspiration.
2. After adequate explanation and communication with the patient, give the patient strong confidence of taking oral nutrition even under controlled ventilation.
3. Let the patient swallow slowly little by little, by putting the meals in small amount.
4. May give favorite meals, dilute it, if anxiety of regurgitation exists.
5. A nursing bottle is avoidable because the patient control the speed and the amount by himself.

Criteria for Weaning

1) Tidal Volume is more than 150 ml in Adult
2) Vital capacity is more than twice of the tidal volume.
3) General condition is stable hemodynamically, mentally & spiritually.

* Repeat to give the patient confidence of breathing by himself. (The patient is apt to rely upon the ventilator excessively).
* Instruct the patient to move the hands and legs, and do himself in minor work (to wipe the mouth, and hand with a towel).
* Spend adequate time in mutual communication with writing by slow speaking

  Radio, record tape of family voice give hopes of living.
* Enforcing deep breath, cough and postural exchange should be performed every 2 hrs if possible.

Standard Value of Blood gas during artificial ventilation

PaO_2	80 - 150 mmHg
$PaCO_2$	35 - 40 torr
PH	7.35 - 7.45

- GIVE THE PATIENT CONFIDENCE OF LIVING AND SURVIVING

Tracheostomy

Indication of Tracheostomy :

I. Difficulty in installation into the trachea, and keeping the trachea open

   a) Prolonged artificial ventilation necessary :
   Chronic Paralysis of respiratory muscle
   unconsciousness due to cerebral accident or injury.

b) Difficulty in incubation into trachea :
Foreign body in the oral cavity
Severe facial damage

c) Excessive secretion in the airway

d) Aspiration pneumonia.

II. Stenosis or obstruction of the upper airway

III. Laxynx paralysis

* Dfficulty of coughing out the secretion in the airway
* Apiration into the airway due to paralysis.

Tracheostomy can not be done too early

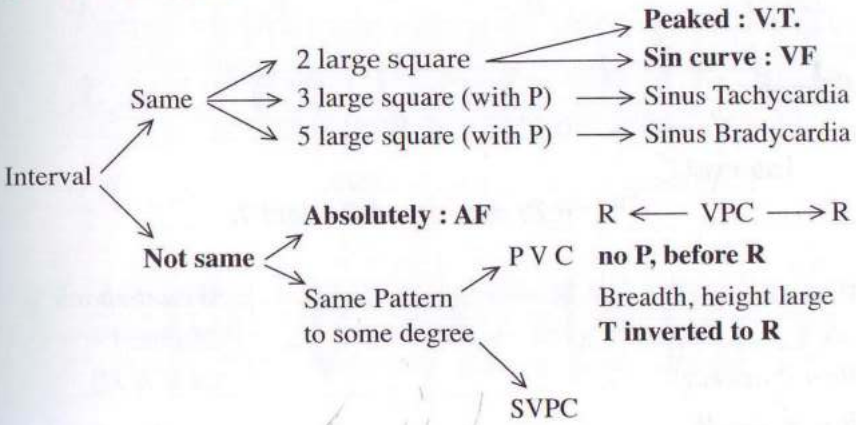
Standard Value of Blood gas during artificial ventilation :

PaO_2	80 - 150 mmHg
$PaCO_2$	35 - 40 torr
PH	7.35 - 7.45

EKG 6

Point to be checked

A. R – R Wave



VT VF PVC AF

Low : < 0.5 mV in I II III **: Low voltage**

Height

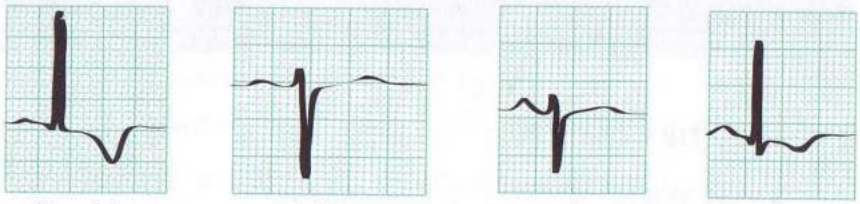
High : in what lead

R VH : (R > II III aVF aVR Large
$RV_1 \geq 7.0$ mm $SV_1 < 20$ mm)

L V H :

$RV_5 + SV_1$	> 35 mm
RV_5 or RV_6	> 26 mm
RaVL	> 11 mm
RaVF	> 20 mm
$R_I + S_{III}$	> 25 mm
R_I	> 16 mm

LVH : RVH :



Lead I *II* *I* *AVF*

P — R :

Interval → 0.12 sec. > ***WPW***

0.20 sec < AVB Block I.

P → R → R ↔ R → No R after P | ***Wenchebach Mobitz 1 (A V B II)***

then P appear

R → ← R

Suddenly No R after P *Mobiz II* *A V B II*

No R after P

P - P - R 2 : 1

P - P - P R 3 : 1 → A V B II

P - P : Same

R - R : Same → but no relation between P and R A V B III

P moves from R : A - V dissociation

No P : nodal Rhythm

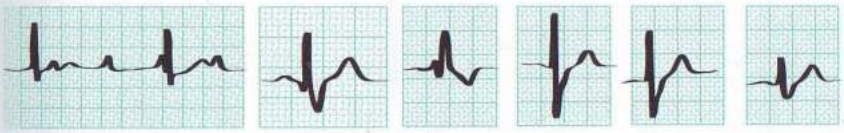
QRS

Breadth → R in V1 and I has two summits → 0.12 ≤ CRB BB, 0.10 < ICR BBB, 0.12 >

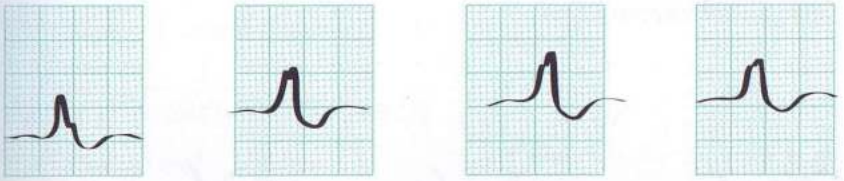
Breadth → ST in I

QRS in V_5 V_6
inverted T
0.12 sec. <
QRS in trapezoid shape
ST in V_1 slurred
→ L B B B

WPW *AVB I* *AVB II*

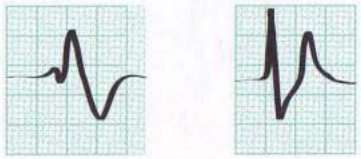


AVB III *I* *V_1* *V_4* *V_5* *V_6*
[RBBB]



I *V_4* *V_5* *V_6*
[LBBB]

Hyperkalaemia >



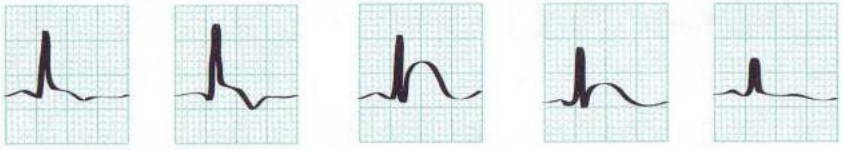
ST - T *V_1* *V_6*

ST segment 0.1 mv < myocardial disturbance

Anterior infarct in V_1 - V_6 < ST ↑ V_1 - V_3 / Q V_1 - V_3

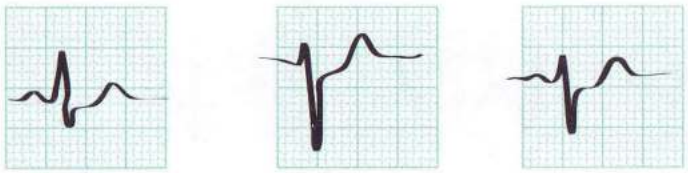
Inferior infarct change in II III aV_F
Upper infarct a V L

Ant. Infraction :



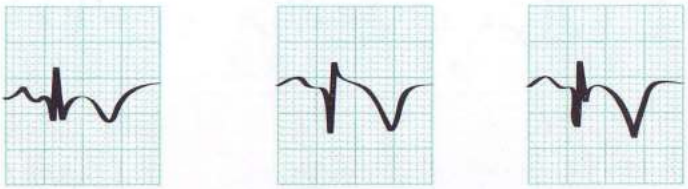
I *aVL* *V_4* *V_5* *V_6*

Receprocal ST changes :



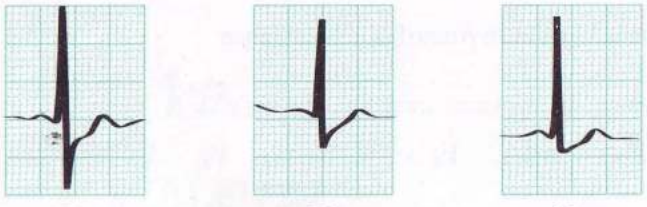
II *III* *aVF*

Inferior Infraction :



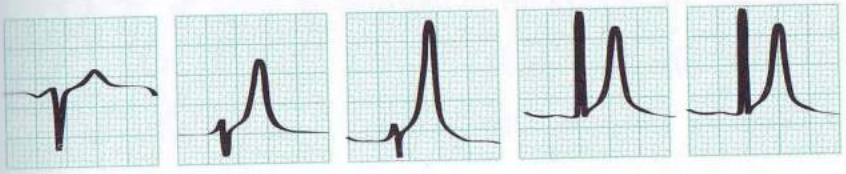
II *III* *aVF*

Receprocal ST changes :



V_4 *V_5* *V_6*

Posterior Infraction :

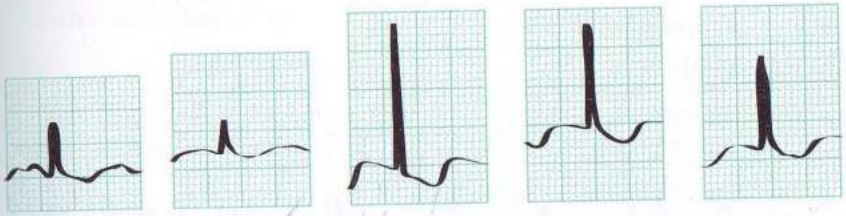


V1 *V2* *V3* *V4* *V5*

Lateral Infarct I AVL

* ***Subendocardial infarct*** ST↓ in V_3 - V_6

Cardiac ischemia :

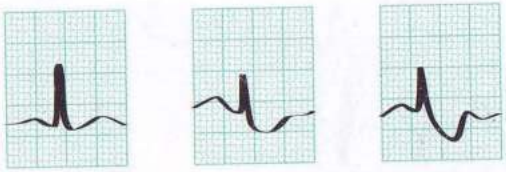


II *I* *V4* *V5* *V6*

Oblique / horizental depression ST - T

Acute coronary insufficiency

PR elongation

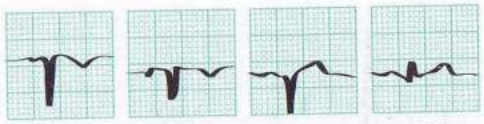


aVL *V4* *V5*

ST ↓

T ↑

Q Wave



Antiseptal : V_1 - V_3 *V1* *V2* *V3* *V4*

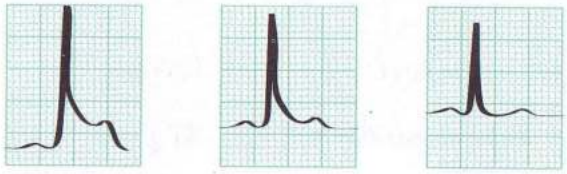
lateral Infarct : I, aVL Q (+) ST

Q in II III
ST in II III → *Posterior (inferior)*

II *aVF* *III*

Q in II III a VF V5 V6
T inverted in V4 - V6 → *acute Post MI (wide)*

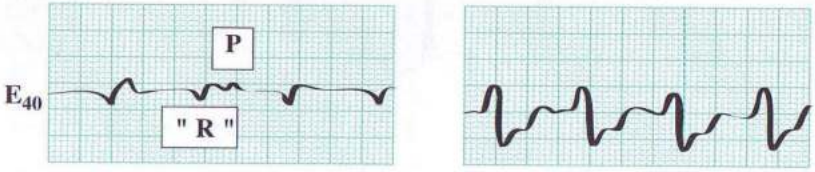
Angina :



V4 *V5* *V6*

A - V dissociation

Nodal rhythm



V1

Supra Ventricular

Stimulus occurs above the His Bangle which divide into two at the Tawara's node

Sinus Arrythmias :

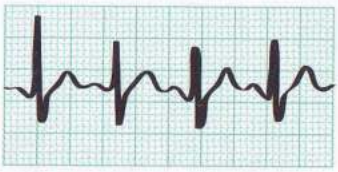


Figure 1 : Sinus Tachycardia

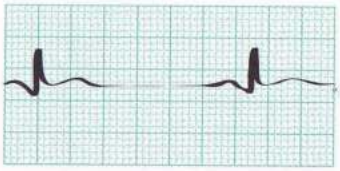


Figure 2 : Sinus arrythmia

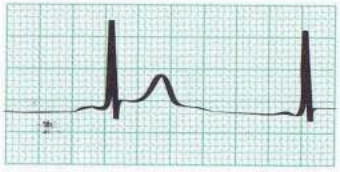


Figure 3 : Sinus bradycardia

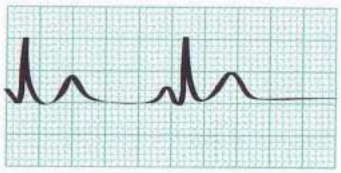


Figure 4 : Sinus arrest

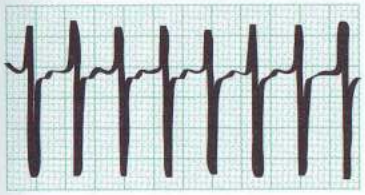


Figure 5 : Sinus premature beat

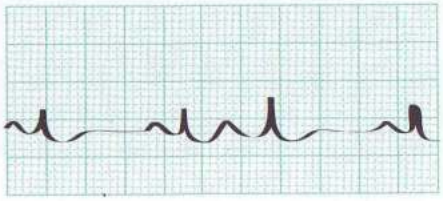


Figure 6 : Tachuycardia (1 : 1 conduction)

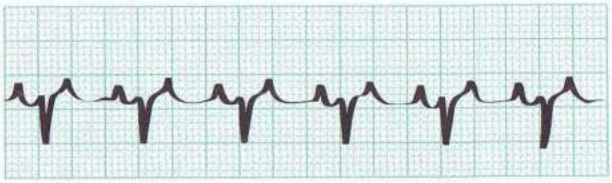
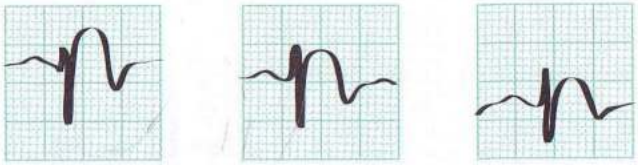


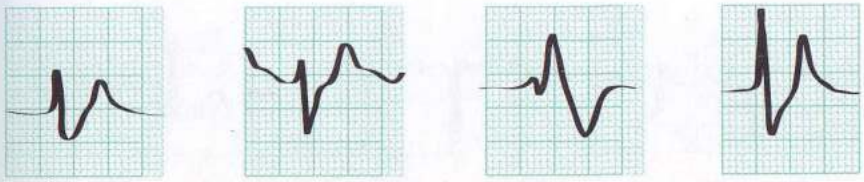
Figure 7 : Tacycardia (AV block)

Ventricular Aneurysm :



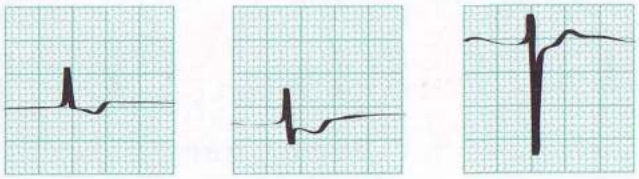
V4 *V5* *V6*

Hyperkalemia :

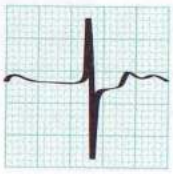


I *aVF* *V1* *V6*

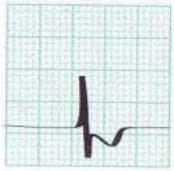
Digitalis Effect :



I *II* *V3*

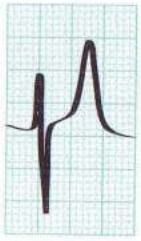


V_4

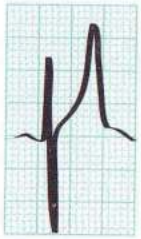


V_5

Hyperkalemia

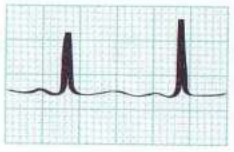


V_2

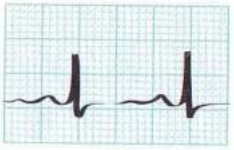


V_3

Hypocalcemia :

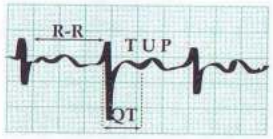


I



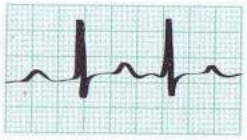
aVF

Hypokalemia :



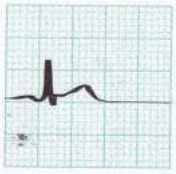
V_2

V_3



V_4

Pericarditis : ST changes in all leads except V1 and aVL



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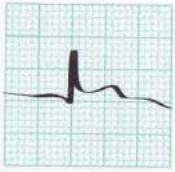
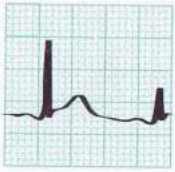


II



V5

Cardiac / Angio Catheters 7

Coronary Catheters

A. Diagnostic :

a. Judkins - Right & left

Judkins	Size in French (Fr)	Curvature
Right Left	5, 6, 7	3 3.5 4

b. Amplatz

c. Multipurpose

B. Interventional – Guide Catheter

Pigtail Catheter :

LV Graphy

Aortography

C. Right heart Catheters:

a. Cournand end-hole presant

b. NIH side hole only

c. Swan Ganz

D. Catheter for electrophysiological study

E. Miscellaneous.

a. Renal Catheters

b. Internal mammary Catheters.

c. Sephenous Venous Catheter

Counter Shock 8

"D C Defibrillator" - delivers large flow of electrons (current in amperer) through heart. The pressure pushing electron is potential

1. Electric Power (volt). The resistance (thoracic) PO the flow is measured in

1) For internal Use Ohms. (70 - 80 Ohms) One watt is the power produced 0.2 - 0.6 W sec/Kg.by 1 ampere (of current).

2) For external Use following pressure of 1 volt (Potential). This power 2 - 6 W sec/Kg. over a duration is total Energy (Joules) Internal use x 10 = external use

2. Indication

1) Atrial Fibrillation **
2) Atrial Fasciculation **] 100 Joules ⟶ Stepwise
3) Supraventricular Tachycardia
4) Atrial flutter – 50J

** For these use, the dial in to discharge synchronous to R (to avoid VF in refractory period)

4) Ventricular Tachycardia (Mono 100J, Poly 200 or more with stepwise increase)

5) Ventricular Fibrillation 200 J Stepwise ↑

At use, a cord always must be connected to the ground.

Pace Making 9

A. ***Pace Maker*** 1) Internal PM
2) External PM

B. ***Site*** 1) Epicardium
2) Endocardium

Pace making Indication

A. Atrial pacing

1) Sinus Brady cardia
2) Extrasystole (over drive)
3) Sick sinus syndrome

B. Ventricular Pacing

1) A - V Block
2) AF (Bradycardiac)
3) Extrasystole (over drive)
4) Sick sinus syndrome.

C. Cardiac Synchronization therapy

D. Prevention of atrial fibrillation

Low Cardiac Output Syndrome (LOS) 10

Low Cardiac Output Syndrome (LOS) :

The achievement of a satisfactory cardiac output without hypertension or hypotension is the objective of hemodynamic management. Hemodynamic norms for patients with uneventful recovery after cardiac surgery are a cardiac index (CI) above 2.5 L/min/m^2 with LA or PCW pressures below 15 mm Hg and a heart rate below 100/min. When hemodynamics indicate marginal myocardial function, such as a cardiac index below 2.0 L/min/m^2, LA or PCW pressure above 200 mm Hg or SVR > 1500 dyne 0 sec cm intervention is usually indicated.

It is worth remembering that myocardial function declines for about 6 - 8 hours following surgery and recovers to baseline by 24 hours.

It is during this period that temporary inotropic support is often considered to optimize hemodynamic periormance.

Low Cardiac Output Syndrome

Management of Hemodynamic Problems

BP	PCWP	CO	SVR	Plan
↓	↓	↓	↕	Volume
N	↑	N	↕	Diuretic
↓	↑	↓	↕	Inotrope
↑	↑	↓	↑	Vasodilator
↓	↑	↓	↑	Inotrope/vasodilator/IABP
↓	N	N	↓	Alpha-agent

↑ = increased

↓ = decreased

N = normal

↕ = variable

CAUSES : (LOS)

1. Decreased left ventricular preload

a. *Hypovolemia (bleeding, vasodilatation from warming, fentanyl, calcium-channel blockers)*
b. *Positive pressure ventilation and PEEP*
c. *Cardiac tamponade*
d. *Severe right ventricular failure*
e. *Tension pneumothorax*

2. Increased afterload

a. *Vasoconstriction*
b. *Fluid overload*

3. Decreased contractility

a. *Poor ejection fraction (EF)*
b. *Myocardial "stunning" ischemia or infarction:*
   - *Poor intraoperative myocardial protection*
   - *Incomplete myocardial revascularization*
   - *Coronary artery spasm*
c. *Hypoxia, hypercarbia, acidosis*

4. Tachy- and bradyarrhythmias

a. *Atrial arrhythmias with loss of atrial contraction*
b. *Tachycardias with reduced cardiac filling time*
c. *Bradycardia with diminished cardiac output.*

5. Other (often hypotension with decreased SVR)

a. *Sepsis (hyperdynamic early and hypodynamic later)*
b. *Anaphylactic reactions (blood products, drugs)*
c. *Adrenal insufficiency (on preoperative steriods)*
d. *Protamine reaction.*

Table : Management of Low Cardiac Output Syndrome

1. ***Look for noncardiac correctable causes (respiratory, acid-base, electrolyte).***
2. ***Optimize preload (PCWP or LA pressure of 15 - 18 mm Hg)***
3. ***Optimize heart rate at 90 - 100 beats/min with pacing***
4. ***Control arrhythmias***
5. ***Assess cardiac output and start inotrope if cardiac index is less than 2.0 L/min/m^2***
6. ***Calculate SVR and start vasodilator if SVR over 1500 dyne-sec cm^{-5}***
7. ***Inotrope selection***
   a. *Dopamine (if low SVR) or dobutamine (if high SVR)*
   b. *Epinephrine unless arrhythmias or tachycvardia*
   c. *Amrinone*
   d. *Insert IABP*
8. ***Vasodilator selection***
   a. *Nitroprusside if high filling pressures and high SVR and BP*
   b. *Nitroglycerin if high filling pressures or evidence of coronary ischemia or spasm.*
9. ***Blood transfusion if hematocrit less than 30%***

ASSESSMENT (LOS) :

Assessment requires a detailed evaluation of the cardiac and respiratory systems to allow for the rational selection of a course of treatment.

1. Bedside physical examination
2. Evaluation of cardiac hemodynamics by Swan-Ganz measurements of filling pressures, SvO_2, and cardiac output calculation of SVR.
3. Analysis of ABGs, hematocrit, potassium
4. Electrocardiogram
5. Chest x-ray
6. Urinary output
7. Chest tube drainage

Table : Hemodynamic Effects of Vasoactive Medications

Medication	SVR	HR	PCWP	C1	MAP	MVO_2
Dopamine	↓↑	↑	↑	↑	↑	↑
Dobutarmine	↓	↑	↓	↑	↓↔↑	↑
Epinephrine	↓↑	↑↑	↑	↑	↑	↑
Isoproterenol	↓	↑↑↑	↓	↑	↔↓	↑
Amrinone	↓	↔	↓	↑	↔↓	↓
Calcium Chloride	↑	↔	↑	↑	↑	↑
Norepinephrine	↑↑	↑	↑	↑	↑↑	↑
Phenylephrine	↑↑	↔	↑	↔	↑↑	↔

MAP = mean arterial pressure, MVO_2 = myocardial oxygen consumption, CI = Cardiac index, ↑ = increased, ↓ = decreas ↔ = no change.

Effect varies with dosage level; relative effect is indicated by the number of arrows.

The effects of amrinone and calcium are not mediated by a and b receptors.

Table : Mixes and Dosage Ranges for Vasoactive Medications:

Medication	Receptor	Mix	Dosage range
Dopamine	($\beta_1\beta_2$ α)	200 mg/250 ml	2-20 µg/kg/min
Dobutamine	($\beta_1\beta_2$)	200 mg/250 ml	2-20 µg/kg/min
Epinephrine	($\beta_1\beta_2$ α)	1 mg/250 ml	1-4 µg/min
Isoproterenol	($\beta_1\beta_2$)	1 mg/250 ml	0.5-5 µg/min
Amrinone	(no $\alpha\beta$)	200mg/200 ml	0.75 µg/kg bolus, then 10-15 µg/kg/min
Norepinephrine	($\alpha\beta$)	4 mg/250 ml	1-10 µg/min
Phenyelephrine	(α)	10 mg/250 ml	10-100 µg/min

X milligrams placed in 250 ml gives an infusion rate of X micrograms (mg divided by 1000) in 15 drops of solution. For example, a 200 mg/250 ml mix gives a drip of 200 mg in 15 drops, 60 drops = 1 ml 15 drops/min = 15 ml/hr.

Table : Management of Low Urine Output

1. Ensure that Foley catheter is in the bladder and is functioning
2. Optimize cardiac function:
   Treat hypovolemia
   Improve contractility
   Lower elevated afterload
   Control arrhythmias
3. Low-dose dopamine 2 - 3 mg/kg/min
4. Diuretics: increasing doses of furosemide (up to 500 mg IV) or ethacrynic acid 50 mg IV.
5. If above fail :
   Limit fluid to insensible losses
   Readjust drug doses
   Avoid potassium supplements
   Essential amino acid diet (enteral or TPN
6. Consider dialysis

(Vide-Infra)

IX. Vasodilators and Antihypertensive Medications

Table : Mixes and Dosage Ranges for Antihypertensive Medications

Medication	Mix	Dosage range
Nitroprusside	50 mg/250 ml	0.1 - 10 µg/kg/min
Nitroglycerin	50 mg/250 ml	0.1 - 10 µg/kg/min
Trimethaphan	500 mg/500 ml	1 - 4 µg/min
Prostaglandin E_1	5 mg/250 ml	30 - 150 µg/min
Esmolol	2.5 mg/250 ml	500 µg/kg/min bolus, then 50 - 200 µg/kg/min
Labetalol	200 mg/200 ml	1 - 4 µg/min
Verapamil	120 mg/250 ml	0.1 µg/kg bolus over 2

Special Care of TC for TOF/Cyanotic Pt.

1. Care for haenoconcentration & consequent pleural & peritoneal collection if develop ⟶ evacuate.
2. Early period shows tendency ⟶ hypotention (10% lower than normal pt) watch warm feet & good pedal pulses (CO) : If good other & signs are normal, observe. Treatment of BP not indicated.
3. Watch Desaturation
   Causes • R ⟶ L Shunt (in PFO)
   • Pulmonary dysfunction

   R - L : Usually improves after 48H as RV improves & excludes pul. dysfunction.
4. a) Measure LAP/RAP ⟶ usually equal or 2 - 4 mmHg higher if LAP > 5 - 10 mmHg ⟶ indicator L-R shunt should be reopened.
   b) if no shunt ⟶ LV dysfunction ⟶ inotropes & vaso dialators indicated.
5. If RAP (5 - 10 mmHg) higher than LAP ⟶ overload or RV dysfunction
   - If transanular patch not given ⟶ reopened and put patch.
   - If patch placed & good correction done. Pts condition is modest ⟶ catacholemine support reasonable for some hours.
   - If no improvement death is 50% ⟶ intervention indicated ⟶ Angio (RV graphy) ⟶ reopen to correct gradient.
   - If correct patching done then gradient usually at distal sature line (1 - 2% Pt.) with hypplasia. Large perforation in VSD patch is done ⟶ out look back to ICU survival good.
6. After leaving ICU ⟶ measure leody wt to avoid retention ⟶ Digoxin & diuretics to start early.

Special Care of CABG

- May be extubated after a few hours or in OR (operating room) and discharged from ICU at the following morning.

Post operative care is simple in most patients.

1. BP may ↓ (10 - 20% below) after 6 - 12 hrs of operation.

⟶ Watch Pedal pulse ⟶ if good & CI > 2.0 then no treatment or low dose (2.5 mg.Kg -1, min -1) Dopamine.

⟶ Oral b blocker (Propanonol) after 6 hrs for SVT Prophylaxis

⟶ Digitalis should not be routinely used ⟶ may invite atrial/ventricular arrhythmia .

⟶ On the day aspirin (325 mg) 1 H after operation ⟶ then after 7 H Continue once daily thereafter for at least 1 years.

⟶ Alternatively Inj. Claxane s/c prophylactically for 7 days.

2. Pt. with excellent LV function may need vasodiator (GTN/Nitropruside)
3. Tachycardia may be present. Use sedation, if fails, b blocker when CO is OK.
4. Look for Tachycardia in hyperdynamic LV Syndrome due to vasodialator ⟶ then allow BP to 140 mmHg (Mean 90 - 100) & use b blocker.
5. I.V. Lignocain prophylactically may be used from OT after CPB for ventricular ecotopy. Antiarrhythmic treatment to be continued in ICU with Pt. of chronic ectopy.
6. AF ⟶ may be due to poor preservation & for withdrawal of b blockers ⟶ use propanol 10 mg qid on the first morning (Post operation) in pt. with b blockers (See dose table -).
   - Digoxin lowers incidence further.

7. Close eye to ECG for Ischemia due to : (Spasm, incomplete revascularization, poor protection, acute graft occlusion).
   - Regardless of causes IV GTN is indicated
     ⟶ Resolves the changes or minimise infract size if is there.
   - IABP sometimes indicated
   - If severe ischemia Angiography or exploration indicated.
8. - Dopamine/Doluctamine are first urine of inotrope.
   - CI, if marginal ⟶ Low dose epinephrime (1 - 2 mg/min) is effective.
   - If little response to catacholamine ⟶ Amrinone (Inocor) is very useful.
   - Some persists "stunned" with poor CO but no infarction ⟶ recovers after several days with mechanical or drug support.
9. If MI occurs Produces ⟶ Haemodynamic problem increases mortality & long term survival. Otherwise no influence of peri Op. MI Treatment is routine care. The only haedynamic problem is low SVR that persist for several days ⟶ Vasopressor (a) to support needed.

Rarely (1%) Pt with CABG, IABP is required for few days

INDICATIONS : IABP

1. LOS with ↑ LAP
2. Malegnant ventricular Arrhythmia intraoperative or post operatively.

Additional

CARE FOR OPCAB / ACAB Pts.

- Optimize Pulmonary function, ensure expantion of both lung.
- Watch serial blood gas through out procedure. Ensure early notice of hypoxaemia & its consequence. Correct acidosis

- Monitor Electrolytes both intra & post operative period. Attempt correction without delay
- Recognise haemorrhage and drainage specially collected in chest cavity and complete evacuation before the chest drain is out.

Late Care/Facilitation of CABG Pt.

During Convalescence

Pt. suffers
- Poor appatite
- Insomnia
- Depression
- Visual, memory, intellectual deficit
- Loss of sexual ability.
- Lack of desire to work.

These are transient in nature

→ Reassurance

→ Excessive medication predispose these → so minimize

→ Encourage rehabilitation.

Post-Op Renal impairment after CABG

Nonoliguric Renal Dysfunction

Causes :

a) Renal hypoperfusion for low CO
b) Nephrotoxic drugs
c) Haemolysis
d) Emboli
e) Sepsis
f) Excess bleeding
g) Masive transfusion
h) Urinary obstraction

Investigations : To dignose tubular dysfunction/ hypovolemia

SerumCreatinine
BUN
Serum Osmolarity
Urine Osmolarity
Urinary Na

MEASURES :

1. Optimize Cardiac output &
2. BP with judicial use of drugs& IABP
3. Maintain pre-load
4. Use loop diuretics
5. Continue intraopertive infusion of low dopamine (2.5 - 5mg/Kg/min)
6. Continue use of frusemide/Mannitol (0.5gm/kg) in the form of **"renal cocktail"**

   Frusemide 400mg

   \+

   20% Mannitol 100 ml (at 1mg/kg/hr)

If serum osmolarity > 320m Osm/kg water then add Frusemide 80 - 200mg i.v anr afeter 30 mins i.v Chlorothiazide 250 - 500mg tostimulate

OLIGURIC RENAL FAILURE

Some patient can not maitain renal function & progress from non-oliguric to oliguric failure

Pathology :

Reduce plasmaflow over time

Decreased GFR

Tubular Necrosis >> evidenced by isothenuria

> leads to tubular obstraction & renal faiure

Note : **BUN & reatenin starts rising by 3rd day & cintinue until dialysis**

Indications for Dialysis :

1. Volume overload
2. Hyperkalemia (> 6 mEq/dl)
3. Increase createnine (> 8 - 10 mEq/dl)

Types & frequency :

Depends upon catabolic & haemodynamic stae of patient

Post op haemodialysis is usually done without heparine / minimum heparine to avoid risk of bleeding in CABG **3 times weekly**

In our practice, **peritoneal** dialysis is used.

Disadvantage :

1. *Iatrogenic defect in diaphragm allows fluid in thorax*
2. ***Reduced splancnic blood flow after surgery decreases efficacy of dialysis***

Protection of Renal Dysfunction

Creatinin > 1.5mg/dl

1. *Pre of More Indration*
2. *Avoid ACE inhibator K sparing diareties*
3. *Intraop Post-Op Low dose dopamine Frusemide*
4. *Oliguria with out elevated cretinin Frusemide 10 - 100mg > i.v*
5. *Oliguria with elevated cretinin – "Renal cocktail" - 2gm Frusemide in 250ml NSa 10ml/H + Mannital 500cc (20% sol a 20ml/H)*
6. *If failed Dialysis*

Post Op Cerebral complications

Features vary. Includes :

1. **Stroke** - Focal neurological deficit last for > 24H. Mostly intraoperative. May occur post operatively
2. **Seizures** - Intraopertive damage manifests as isolated seizures post operatively
3. **Imparied conciousness** - Manifests from mild drowsiness to irreversible coma. Absence of motoractivity, brainstem dysfunction & extensor posturing are grave.
4. **Fatal cerebral injury.**
5. **Spinal cord injury** - rare (IABP insertion, Valve surgery)
6. **Peripheral Nerve injury** - Brachial, Horner's facial, recrrent laryngeal
7. **Neuropsychic - Congnitive changes. Does not include mood or post Op delirium**

Mechanism :

1. Macro / Micro embolisation
2. Inadequet cerebral perfusion pressure – due to reduced flow, low BP, misplaced aortic cannula

Management :

- Neurological consultation is appropriate – to make a base line to follow changes & prognosis.
- Prognosis depends, degree, mechanism, area of injury
- Hyperventilation, Steroids – for brain oedema
- Cognitive dysfunction may persists for months, most resolve completely
- Seizures for structural damage or metabolic encephalopathy – Benzodiazepines, Phenobarbitals

Management Summaries 11

Management of Postoperative Low Cardiac Output Syndrome

I. Key principles

A. Constantly reevaluate hemodynamic status (particularly after intervention).

B. Follow trends in hemodynamics (not absolute numbers).

C. Seek assistance early whenever one is uncertain about management.

D. Use systematic and physiologic approach to optimization of determinants of cardiac output (CO). One should address these hemodynamic parameters in the following sequence: Heart rate/arrhythmia → preload → afterload → contractility.

II. Listed here are possible therapeutic interventions (or goals) for the most common causes of low cardiac output syndrome (L/OS). It is meant only as a brief review for those who already have a thorough understanding of the principles governing CO.

A. Dysrhythmias :

1. Bradycardia : Atropine, isoproterenol, pacer

2. Other arrhythmias : See P

B. Inadequate preload : Administer fluids, control mediastinal bleeding, rule out cardiac tamponade

C. Elevated afterload : Warming light or blanket, fluids, vasodilator therapy

D. Reduced contractility : Rule out myocardial ischemia (vide infra); administer O_2; correct acidosis and optimize ventilation; use warming light or blanket; administer inotropic therapy, mechanical assist device.

Management of Postoperative Myocardial Ischemia

I. ABC's of resuscitation (airway, breathing, circulation)

II. Admit patient to monitored bed

III. Immediate therapy

A. O_2 (100% face mask or intubation)

B. Nitroglycerin sublingually; 0.4 mg STAT and q 5 min and nitropaste 1 - 2 inches)

C. Establish intravenous (IV) access (large-gauge if hypotension present)

D. Morphine, 1 - 2 mg IV STAT

IV. Notify attending surgeon or fellow !

V. Acute therapy

A. Follow-up 12-lead electrocardiogram q $^{5-10}$ min until ischemic changes resolve

B. If blood pressure (BP) adequate, consider nifedipine, 10 mg SL, and/or nitroglycerin infusion, 10 - 100 mg/min.

C. If hemodynamically unstable, initiate invasive monitoring (intraarterial and Swan-Ganz catheters)

D. If hematocrit < 30% and no evidence of congestive heart failure, transfuse packed red blood cells. If pulmonary edema is present, diuretic (furosemide) before transfusions.

VI. Determine cardiac hemodynamics and optimize determinants of CO (see section on L/OS). In brief, usual goals are treatment of tachyarrhythmias and bradyarrhythmias, reduction of preload and afterload, and judicious use of inotropes to maintain adequate contractility (while also minimizing myocardial O_2 consumption).

VII. Consider mechanical assist device (intraaortic balloon pump) early in setting of myocardial ischemial !

VIII. Ventricular arrhythmia prophylaxis

A. Correct K^+ and Mg^{++}

B. Lidocaine, 1 mg/kg IV bolus (procainamide, bretylium, or amiodarone for refractory arrhythmias)

IX. Consider heparinization

X. Emergent coronary catheterization and/or surgical intervention.

Management of Postoperative Hypertension

I. Warming lights or blankets

II. Sedati on and analgesia; verbal reassurance

III. Optimize oxygenation and ventilation

IV. Determine cardiac hemodynamics (cardiac index [CI] and systemic vascular resistance [SVR])

A. If CI < 2.0 L/min/m^2, follow for treatment of L/OS

B. If SVR elevated, initiate vasodilator therapy

C. If CI > 3.0 L/min/m^2, consider b-blocker therapy for hyperdynamic syndrome (consult attending surgeon or fellow).

V. Others

A. Foley catheter or nasogastric tube if visceral distention is suspected

B. Antipyretic agent if patient is febrile

C. Paralytic agent (Pavulon) if intubated patient is shivering

Management of Postoperative Hypotension

I. **Imediate management :** Address ABC's of resuscitation (airway, breathing, circulation)

A. Place patient in Trendelenberg position

B. O_2 (100% face mask or intubation if indicated)

C. IV fluid bolus (through cordis port or large peripheral IV)

D. Calcium chloride 1 amp IV bolus (may repeat)

E. If in early postoperative period, dopamine is often already infusing at renal dose. Increase dopamine rate to achieve pressor effect (15 - 20) mg/kg/min).

F. Notify fellow or attending surgeon.

II. Definitive therapy

A. Assessment (physical examination, 12-lead electrocardiogram and telemetry, arterial blood gases, chest x-ray, Swan-Ganz catheterization)

B. Evaluate and optimize hemodynamic parameters (preload, afterload, and contractility)

C. Etiologies of acute postoperative hypotension

1. Hypovolemia*
2. Decreased contractility*
   a. Myocardial ischemia or infarction

POST OPERATIVE 12
Special Concern :

A. Coronary Vasospasm :

It can affect normal coronary's, laypassed vessels, SVGs or IMAs.

Causes : Speculative : Ca-infusion
Myocardial Ischemia
Rebound (withdrawal Ca + blockers)
platelets $T \times 4_2$

Diagnosis : Difficult, Manifested by

a. ST ↑
b. Haemodynamic Collapse (LOS, BP)
c. Arrythmias
d. Heat Block.

Treatment :

1. Optimize O_2 delivery, acidosis, minimize O_2 demand
2. Optimize determinates of CO.
3. Nifedifine 10ms S/L, then 30ms NG tube 96h.
4. I.V. GTN 0.5mg/kg/min as tolerated.
5. I.V. Verapamil ⟶ 0.1 mg/kg bolus than 15 - A5 drop/ min (2 - 5mg/kg/min) of 120 mg/250 ml mix.
6. When taking PO : GTN 20mg 94h
   Nifidifine 30 mg 96h
   (or) Diltiazim 30 mg 98h.

B. Perioperative MI : (5%)

Prognosis : a) Mimimal/no effect on motility or long term survival
b) Result in hemodynamic compromise

Factors : a) Left main disease

b) UA

c) Poor LV

d) Long Cross Clamp

Mechanism :

1. Undetected Preop MI
2. Ischemia during Induction/before coronary perfusion
3. Inadequate myo-protection
4. Incomplete revascularization
5. Graft thrombosis/emolism
6. Reperfusion injury

Diagnosis : Not clear cut always

a. Appearance of New Q (20% False Positive).New Q or disappearance of precordial R are suggestive(3 - 5%).

b. Enzyme elevation of CK-MB > 40 units or persistant elevation > 8% for more than 24 H have been associated with Peri-MI.

c. Elevated Troponin I

d. ST-T changes, VES, BBB if persists over 24h suggestive

e. Technetium Scan (after 24 H)

Management :

1. **LOS :** Optimise determinants of CO, GTN decreases infract size
2. **SVR Low with good CO :** Lagents may be necessary till SVR normal.
3. **VES :** Standard treatment with Lidocaine and oral antiarrhymics.

Pt. with 'Stunned myocardium' recover after several days of Pharmacological / IABP support.

Indication of Surgery :

I. Indicated in hemodynamically or electrically unstable pts. (Probably due to graft failure or insufficient revascularization).

II. If Pt is stable with drugs/mechanical support & surgeon confident about graft then CAG in suggested option.

III. If Pt is unstable and some suspicion exists about particular graft, emergency surgery is medicated.

C. Management Of Aortic Valve Surgery

Have substancial decreased ventricular compliance due to ventricular hypertrophy for pressure overload

- Filling pressure fluctuates with little change in LV volume
- LV cavity is small& cardiac out put depends upon atri al contribution
- ***So maintaince of filling pressure & sinus rhythm is critical***
  - Hypertophied LV is prone to arrythmias & treatment should be aggressive :
  - IV Lidocain 2mg/min is usually employed
- Critical AS are prone to develop hypotension & arrythmia during intubation with depressent drugs. May suffer hypotensive episode that may lead to post operative faiure.
- Retrieve BP with a agents (Neo-synephrine 500 - 1000mg bolus & repeat) until CPB started
- Only an occational patient will tolerate this series of insults along with faulty presevation in CPB & survive with out neurological deficit. Mortality approaches 100%

'Hyperdynamic LV syndrome' after AVR is characterized by :

- Immediate post operative hypertension unresponsive to nitropruside
- Widening pulse pressure, low diastolic pressure

- Decreased myocardial oxygen supply
- Associated with high systolic pressure & tachycardia

- ***If allowed acute LV failure may result***
- ***Management :***
  - *Reduce rate of nitropruside infusion*
  - *Add trimethaphan for hypertension*
  - *Judicious use of Propanolol / Esmolol*
  - Patient after replacement may remain in peripheral vasodilatation with low mean pressure. Should be treated with a agonist (neo-synephrine)

Loss of sinus rhythm compromises cardiac performances & should be electrically converted

D. Management Of Mitral Valve Replacement

Patients with MS have protected LV (no pressure/volume overload) than **MR and faces less challenges post operatively**

- To be observed for both LV & RV failure.
- RV failure may manifest at discontinuation CPB or at early post operative period
- Surgeon should get alert :
  - Low CO-despite adequet preload
  - Elevatyed CVP
  - Low / normal PA pr.
  - Rt. Ventricular distension/hyperconractility

Causes of RV dysfunction may be secondary to : Coronary Air embolism(improves with additional bypass time), **Pul. vsoconstriction**

- Acidosis/Hypercarbia/Hypoxemia-to be investigated and promptly corrected

If persists pharmacological measures to be taken or PA ballon counterpulsation / Centrifuga pump to bypass Rt. Hreart is indicated

Follow critically both LV & RV filling pressure

Once RV failre is diagnosed-

- IV Nitroglycerine (0.5 - 3.0mg/kg/mim) > decreses preload & Pulmonary afterload
- If inotrop is needed > Isoproterenol (1 - 4 mg/min) Dobutamin may be good alterative if tachycardia is present
- If all measures fail :

  Prostglandin E (10 - 100 ng/kg/min) with/ without Norepinephrine

- Others : Produce mild alkalosis increasins minute Ventilation
- Keep LA pressure below 16mmHg

MITRAL REGURITATION :

Have enlarge LV accustomed to low pressure

Developes post replacement "afterload mismatch"

- Nitropruside IV indicated to counter new load imposed to LV by competent valve (to be replaced orally with ACE inhibitor for 8 - 12 wks or longer till LV is normal)

Sinus rhythm is advantage if Af for 6 month or less and persude electroversion when discharged /after first 2 months.

E. Post operative bleeding

Etiology :

1. **Anticoagulant effect**
   a. Heparin rebound
   b. Platelate dysfunction
   c. Deplation of Coagulation factors
   d. Primary fibrinolysis or DIC
   e. Coagulopathy
2. **Surgical : raw area, suture lines, vessels etc.**

Assessment :

1. Quantitate chest drainage
2. Portable CXR
3. Coagulation study : PT ⟶ for extrinsic cascade
   PTT⟶ for intrinsic cascade
   Platelets - count
4. Suspect other if surgical bleeding is not suspected.
5. Assessment of heparin effect
   ACT ⟶ Qualitative and not always accurate
   TT ⟶ elevated. (D/D Heparin, Fibrinogen low, Fibrin Split Product)
   Specific test - HPT (Heparin Protamin filtration)
6. Assessment of Platelet functions :
   BT - Elevated if count < 100,000 m/L
7. Assessment of Fibrinolysis/DIC.

TABLE : Tests to Assess Fibrinolysis and DIC

Test	DIC	Primary fibrinolysis
PT	Elevated	Normal to elevated
PTT	Elevated	Normal to elevated
Platelets	Low	Normal
Fibrinogen	Low	Low
Factor V	Low	Low
Factor VIII	Low	Low
Clotlysis	Normal to prolonged	P. apid
FSPs	Normal to elevated	Very high
Paracoagulation test	Positive	Negative

TABLE : Management of Postoperative Mediastinal Bleeding

DIC	Primary fibrinolysis
1. Ensure that chest tubes are functioning	
2. Check results of coagulation studies	h PT then FFP h PTT then FFP/ Protamine
3. Control hypertension and shivering	
4. Warm patient to normothermia	
5. PEEP in 2.5 – 5 cm increments	
6. Protamine 25mg IV for 2 doses	
7. Desmopressin (DDAVP) 0.3 mg/kg over 20 minutes	
8. Fresh frozen plasma 2 units	
9. Packed red cells if hematocrit is below 30%	
10. Platelets 8 – 10 units	
11. Cryoprecipitate 8 units	
12. Epsilon-aminocaproic acid if fibrinolysis is confirmed	
13. Avoid and 5% albumin	

Blood Products

A. FFP :

1. Contains all factors and colloid of choice in bleeding if Hct is < 30%. (If PTs are normal, Clotting factors may be boarder line or Low due to ongoing bleeding). So should be used if PT/PTT are normal.
2. Transfuse within 6H of thawing with filter (170 micr)
3. Should be ABO compatible.

B. Blood :

Should be given if Hct < 30% & bleeding persists with filter (170 mier)

1. **PCV** - No factor. Necessitate factors if transfuse > 5 unit. (Each unit hct 70%. Increases Hct of 70 Kg pt. by 3%, 70% cell survive 24H)
2. **Banked whole blood** - deficient in factors. V, VII, XI (50% VIII lost after lunk 80% in 3 wks). Platelets are absent after 24 H. (Rarely used in Open Heart Surgery).
3. **Fresh whole blood** (< 6 Hs old) - contains all and the best product.
4. **Cell saver blood** ⟶ No factors, platelets and with heparin additional protamine, FFP, Platelets also necessary.
5. **Autotransfusion** - Shed pericendial, mediastinal blood collected in bag should not clot.

   Add CPD in bag also (1 ml CPD/7 ml blood)

   Transfuse within 4H of collection

   Contains more factors 4 platelets than blanked blood but no fibrinogen (no harm due to rapid liver generation)

   If 1 > 1 lit then factors, fibrinogen to be added to avoid DIC.

6. **Platelets :** Should be given if < 100000/ml or no hasitation in ongoing bleeding no need for the count.
   - One unit contains - 70% platelets in one fresh blood unit
   - Each unit increases 7 - 10000 m/l
   - Dose : 0.1 unit/kg (iu.e. 7 unit for 70 kg)
   - May be stored in room up to 5 days

     Whereas at 4°C are useful for only 24H (only 50 - 70% activity is present at 6H).

   - ABO compatibility no essential.
7. **Cryoprecipitate :** Rich in I VIII XIII

One unit (10 - 25ml) from one donor contains 30% plasma content

- Should be given rapidly within 6H
- ABO not essential
- Should be given if PTT can not be corrected with FFP & Protamine and bleeding continues.
- Specially useful in Von Willebrand's diseases.

F. AORTIC SURGERY : Post operative Measures

Similar to all major cardiac operations.

- Maximize CO by optimizing filling pr. preferably by colloids/PCV
- Afterload reduction by Nitroprisside/Nitroglycerine
- Use inotropic agents when needed
- Avoid post op hypertension with afterload reduction and fl blockers. Hypertension not only invites bledding from fragile suture sites, also redessection or rupture of false channel
- Start oral fl blocker ob 2nd postop day
- Monitor carefully the neurological function
- Evaluate any sensory deficit & level of conciousness, NOTIFY neurologist.
- Care for acute tubular necrosis (ATN) & Paraplagis :

  Adequet Preload & CO

  Maintain optimal urine output

  Use low dose dopamine

  - ♦ Carefull long term followup with MRI/CT along with control of hypertension anr use of fl blocker is essential. Recurrence is problem but aggressive approach to diagnosis and redo gives low late morality.
  - ♦ These pts. are at special risk to develop post op MI

G. Special Care Issue

SURGICAL WOUNDS :

Most of the aquired wounds of CABG, MVR, other thoracotomies heal similarly, modified by individuals disease status. Wounds have inert tendency to restore un healthy tissue to prewound condition.

Physiology of Wound healing :

- Inflammatory phase > up to 5 days
- Proliferative phase > 5 – 20 days
- Maturation phase > 21 – 2 yrs

MEDIAN STERNOTMY / Thoracic incision:

- Post op dressing should be left 24 hrs & to be reinforced if socked
- Any excessive drainage should be reported
- Dressing should be changed every 24 hours
- Dressing should be protedted from contamination with secretions

LEG INCISIONS :

- Elastic bandage in the theater should be removed in ICU to asses the vascular status and reapplied.
- On 1st Postop day to be removed & if no discharge, reapplied.
- On 2nd day, if dry site may be left to air.
- Inspect wound daily.

CHEST TUBE SITE :

- 1ST post op day change the dressing.
- Atter removal of the tube put an occlusive dressing & should be changed evry 24 hrs until stitches removed.
- Wound then left open to air .
- Inspect any sign of infection daily.

PACING WIRE :

- Keep until priror to discharge.
- During these days take care with dirt dressing every 24 hrs.

IABP SITE :

- Site should be monitored routinely for bleeding & infection when in place or removed.
- After removal for haematoma.
- Dressing should be changed every 24 hrs.

MEDIASTINITIS :

The dark side of cardiac surgery of repotrd varing incidence rate.

Risk factors :

- Patients factor (diabetes, obesity)
- Post operative haemorrhage
- External Cardiac massage
- Prolong CPB and opertive time
- Bilateral mammary use
- Low cardiac output
- Prolong Ventilation/Tracheostomy

General Considerations :

- Majority of cardiac infections originate in the operation theatre
- Early dignosis is significant to less morbidity.
- General nutritional state is important to hea.l
- If happens, emotional support & early consultation to family/Patient is important
- When additional surgery required-Pt should be allowed to ventilate his/her concern & anger about additional surgery.
- Social worker should participate the patients course.

PREVENTION :

"Ounce of prevention is worth a pound of cure". A few cardiac center is able to accumulate the full comlement of preventive measures.

- Incidence is higher in patients given no prophylactic antibioticconerage.
- It is agreed that antibiotics must be providedduring operation
- Controversy exsits over the duration
- Accumulation of blood & air beneath sternum is potential media for microbes & should be darined properly
- Instability predisposes so stable closure must be sought for.

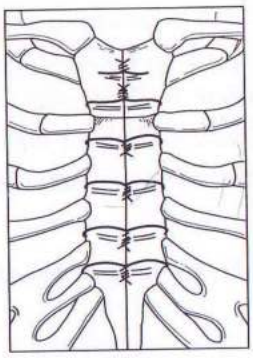


Fig : 1. Single parasternal closure

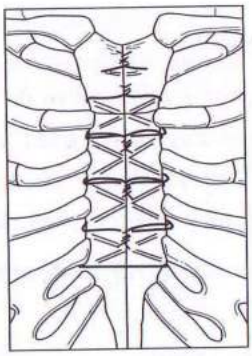


Fig : 2. Figure of eight sternal closure

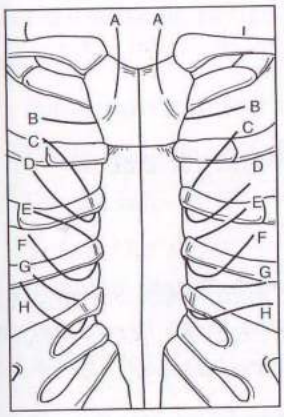


Fig : 3a. Horizental mattress

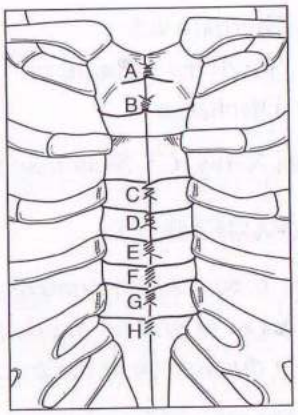


Fig : 3b. Horizental mattress

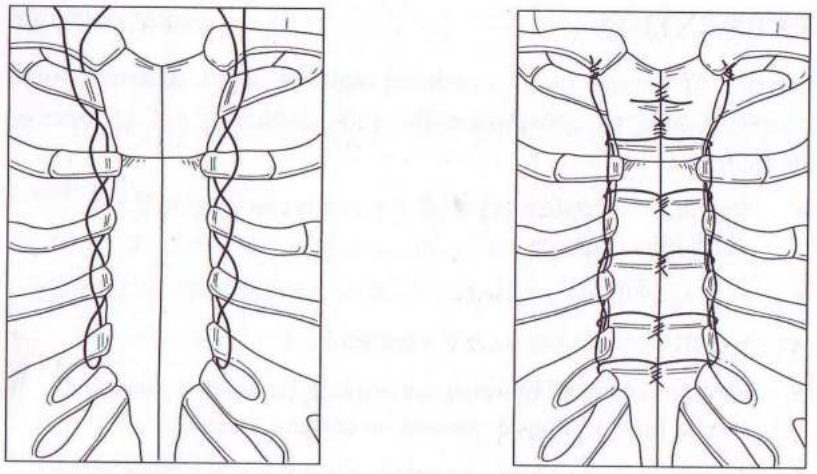


Fig : 4a & b. Robicsek pericostal lattice closure

Diagnosis :

Develop as early at 3 to 60 days after operation. Diagnosis usually needs nothing other than physical examination. Sternal instability & purulent discharge from presternal wound with or without high temperature heralds infection.

Sometimes it is difficult to diagnose. Sign/symptoms associated are :

- Chest pain
- Fever
- Leukocytosis
- Sternal click
- Tenderness/Redness
- Discharge

Chest X-ray, CT Scan may be used to determine the extent.

MANAGEMENT :

Once diagnosed treatment should be prompt. When instability & discharge is present gently probe the draining sinus to see sternum lies in the bottom of the tract or the tract extend deep.

- *In all such situation prompt operation to be undertaken.*
- *If discharge from deep open further to eliminate possibility*

- *When minimal instability with no discharge, observe the patient.*
- *When minimal instability with nonpurulant discharge, appears to be superficial to be probe carefully to confirm the drainage not from beneath the sternum.*
- *When the origin proved deeper, reopen the sternum in theatre to evaluate extent.*

Treatment :

No uniform method available.

Common strategies are

- Open dressing change > control of infection > healing by secondary intention
- Open debribement with closure over irrigation tube
- Debridement (redical) and muscle/omental flap closure

Combination/variation are also possible.

- **There is no acceptable 'best method'**. So it is better to treat all patient with a simpler method (debridement & irrigation) with an intention that some may need extensive method. This principle will lead not to subject all patients to extensive procedures.

CHRONIC MEDIASTINITIS / STERNAL INFECTION :

Easier but difficult to treat.

Causes :

- Inadequet treatment of aute infection.
- Underlying destroyed cartilage.
- Fungal infection
- Tubercular focus.

A chronic draining sinus conferms diagnosis.

TREATMENT :

- Complete excision of all infected tissue, sternum & cartilage. The entire cartilage ach may need to be removed.

- Unsually chronic infection without involvement of cartilage is found.
- After debridement -
  - healing by secondary intention
  - primary overage by skin & soft tissue (muscle flap to fill gap)

H. CARE AT CONVALESCENCE

Goal : Prevention and early detection of post operative complications like Infection, Arrythmias, post pericardiotomy syndrome, Temponade, pleural effusion & other system functions.

Care includes at this phase :

- Physical Rehabilitation
- Nutrition
- Risk modifications
- Anticoagulation
- PHYSICAL REHABILITATION :

Early mobilization is proved safe and beneficial. It decreases the effect of prolonged bed rest, increases self-confidence, shortens the length of hospital stay. Integrated individualized rehabilitation program is ideal.

Benefit :

- Increases strength & endurance
- Decreases lung congestion
- Increases energy, tone & stretching of chest and leg muscles
- Increases the level of tolerance
- Increases confidence & fitness to discharge

General Protocol should includes : 3 parts

- Warm up exercise
- Endurance/Confidence activity
- Cool down excercise

Warm up Exercise :

Sitting	*Standing*
Knee extension	Toe rise
Hip flexion	Shoulder shrug
Shoulder abduction	Side leg lift
Shoulder flexion	Knee touches
	Arm cirles
	Side bends

Endurance :

Purpose is to stimulate oxygen transport system. Walking is common form for inpatient setting. Intesity, duration & frequency is individual within patients limit

Cool-down exercise : Necessary to prevent light headedness, syncope, arrythmias with abrupt cessation of exercise in cardiac surgical patients. To cool down pt should perform endurance acyivity at slower pace for some minute before stop

- **NUTRITION :**

Malnutrition increases morbidity and mortality of surgical patients. Dietitian advice is recommended. Reduction in caloric intake is discouraged than eating habit.

Basic principle of diet :

- Reduction of total fat intake (saturated) - not > 30% total calore
- Replacement of some saturated (10%) fat by poly unsaturated fat
- Reduction of cholesterol intake to < 300mg/day
- Acheivement & Maintenance of body weight
- Increased intake of soluble fiber

- **RISK FACTOR MODIFICATION :**

Factor are identified at admission. Modifiable factors are to be addressed postoperatively. These includes : Diet, Inactivity, Smoking, Hypertension, Stress with emotion& behavioral changes. Sone times need Therapist/Psychiatric consultation

APPENDIX :

Protocols in ICU

Protocol for Optimizing Preload

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

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

a. Judge or measure cardiac output

b. Decide on the most appropriate P_{LA} (or right atrial pressure P_{RA})

c. Set an upper P_{LA} (or P_{RA}) limit

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

e. Limit risk of overinfusion.

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

1. A P_{LA} 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 P_{LA} 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 must have two parts :

1. The P_{LA} limit

2. The parameter to limit infusion isto be clearly mentioned. For example :

   P_{LA} = 12 mmHg. arterial blood pressure (PAO) ≤ 120 systolic

P_{LA} = 8 mmHg. infuse 250 mL m^2

P_{LA} = 10 mmHg. infuse to limit of 2x chest drainage

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

P_{RA} = 16 mmHg. infuse to PAO 100 systolic.

Protocol for Reducing Blood Pressure and Afterload

Sodium nitroprusside 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 10µg. 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 100µg 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-I 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 ($g.kg^{-1}$ of 25% solution of sodium thiosulfate (10 ($g.kg^{-1}$ min^{-1}).

Nitroglycerin : ***Dose : of 0.5 to 3.0μg. kg^{-1} min^{-1} are recommended.***

It decreases venous lone but also decreases coronary resistance and useful when myocardial ischemia is present

(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 α-adrenergic receptor blocking effect, produces direct smooth muscle relaxation, and reduces pulmonary vascular resistance. Its recommended infusion rates are 1.5 to 2$μg.kg^{-1}$ min^{-1}.

Phenoxybenzamine is a noncompetitive blocker of α-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 α-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.)

Milrinone and amrinone (phosphodiestrease inhibitors) also produce important vasodilatory effect.

Protocol for Inotropic Agents

The rate of infusion is calculated and recorded in micrograms per kilogram body weight per minute ($\mu g.kg^{-1}\ min^{-1}$). 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 ($\mu g\ kg^{-1}\ min^{-1} \times$ body weight hour 60 and concentration of calecholamine is that h^{-1} (or drops per minute) =

$$\frac{\text{(Infusion rate } (\mu g\ kg^{-1}\ min^{-1}).\ \text{weight (Kg).}\ 60}{\text{Concentration } (\mu g\ mL^{-1})}$$

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..

Rate of infusion based on past experience is 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 $\mu g.\ kg^{-1}\ min^{-1}$
Debutamine	10.0 $\mu g.\ kg^{-1}\ min^{-1}$
Isoprotoreonol	0.05 $\mu g.\ kg^{-1}\ min^{-1}$
Epinephrine	0.1 $\mu g.\ kg^{-1}\ min^{-1}$
Norephinephrine	0.1 $\mu g.\ kg^{-1}\ min^{-1}$

Protocol for Intravenous Fluids

A. *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.

B. *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.

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 requirement

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

ii. Proce ed 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.]

Other Protocols :

After Open Heart Surgery :

(5 - 10% Dextrose Soln.)

Body Wt.	Standared	Maximum
< 10 Kg	30 ml/Kg/day	60 ml/Kg/day
10 - 20 Kg	20 ml/Kg/day	40 ml/Kg/day
> 20 Kg	10 ml/Kg/day	30 ml/Kg/day

After Closed Heart Surgery :

Body Wt.	Standared	Maximum
< 10 Kg	50 ml/Kg/d	100 ml/Kg/d
10 - 20 Kg	30 ml/Kg/d	60 ml/Kg/d
> 20 Kg	20 ml/Kg/d	40 ml/Kg/d

Contents :

1. **Kcl**

Open Heart Surgery- 2 mEq/kg/day

Closed Heart Surgery- 1.5 mEq/kg/day

2. **Vitamins**

   Vit B_2 20 mg

   Vit C 500 mg

Protocol for Metabolic Acidosis

Indication :

Metabolic acidosis exists if the base deficit is > 2 mFq . 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. Give $NaHCO_3$ 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, much larger doses of $NaHCO_3$ are indicated (44 mEq for adults, 1 mEq . kg^{-1} for infants and children)*

Protocol for Severe Metabolic Alkalosis

Diagnosis :

- 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 × 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_2 retention 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 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. 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 ($mEq \cdot L^{-1}$).

3. 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 HCI contains 150 mEq of H+ and 150 mEq of Cl^-)

4. Administer the chloride deficit as 0.15N HCI over 16 to 24 hours.

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

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

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

(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 $mEq \cdot L^{-1}$.

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 Hyperglycaemia

(IMMIDIATE POST OPERATIVE PERIOD)

DIABETIC CHART

Name age Sex Wt. Date

Date	Time	Urine					Blood sugar	Acetone	Insulin Actrapid/H
		B	G	Y	O	R			
	6AM								
	8AM								
	10AM								
	12PM								
	2 PM								
	4 PM								
	6 PM								
	8 PM								

Protocol of Insulin dosage

Urine Color	Blood Sugar	Dose Actrapid Insulin
Green	100 - 200	1 IU /hr
Yellow	200 - 300	2 IU/hr
Orange	300 - 400	3 IU/hr
Red	> 400	4 IU/hr

1. Insulin to be given intervenously till the patient is on I.V fluid to be stopped soon after oral feed started and other chart to be used
2. Target Blood Sugar 160
3. Every 2 hourly blood sugar monitoring with capillary Blood Glucose method twice in day chacked with laboratory
4. Urine sugar every 4 hourly, 10% Dextrose for maintenance of calories (upto 100ml)
5. K+ will be monitored separately

PROTOCOL FOR DIABETIC MANAGEMENT IN ICU & WARD

Chart

(AFTER ORAL FEEDS STARTED)

Name Age Sex Wt. Date

	BB	AB	BL	BD	AD
Blood Sugar					
Urine sugar acetone					
Insulin Given					
Blood Sugar					
Urine Sugar					
Insulin Given					
Blood Sugar					
Urine Sugar					
Insulin Given					

1. Insulin to be given subcutaneously
2. Blood Sugar :

160 - 200	4 IU
200 - 260	6 IU
260 - 300	8 IU
> 300	10 IU

3. Target level < 160 before each meal
4. Once pt. started` oral feeds, blood sugar level can be monitored before each meal
5. Urine sugar should be chacked 2 hours after each meal
6. Blood sugar to be done 45 min before each meal and insulin to be given 30 min before each meal acordibg to blood sugar
7. If before dinner blood sugar > 200 aqnd previous day fasting >160 then administer only LENTARD at bed time according to blood sugar
8. Oral Hypoglycaemic to be started on 6th day of operation in NIDDM patients

Protocol for Hyperthermia

Indication :

Hyperthermia exists if rectal temperature is ≥ 38°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 / Paracetamol 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^{-1} IV every 6 hours.
4. Give sodium nitroprusside. 1µg · 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 Ventricular Arrythmias

Interventions :

1. If the arrhythmia is premature ventricular contraction (PVC) or ventricular tachycardia (VT) with a good hemodynamic state >>> 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 >> VT and reduction of cardiac output > immediate DC cardioversion (100 and then 200 J).
2. Measure serum K^+ and 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^+ dose

   d. Recheck serum K^+ level. If it is < 4.0 mEq-L^{-1} >>> give oral K^+ supplement as 20% KCI, 10 mL twice a day in orange juice (60 mEq approximate daily dose).
3. **Pacing -** If the ventricular rate is less than 80 to 90 beats/min.
4. Use atrial pacing - If basic rhythm is sinus or atrioventricular (AV) junctional
5. Use ventricular pacing - When cardiac rhythm is other than sinus or AV junctional or atrial pacing fails to result in 1 : 1 AV conduction.

6. AV sequential pacing - In the presence of second or third degree AV block.

Control of the Arrythmic Urgent Situation

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

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

$$\text{Drops per minute} = \frac{\text{Dose - weight (kg)} \times 60}{4}$$

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

2. Other 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 × 24 h^1

Protocol for Acute Digitalization

Digitalizing Dose :

If digitalis has been given in the past 10 days, estimate digitalizing dose of digoxin (provides a convenient guideline for the cardiac surgeon)

The estimated dose of digoxin = **0.9 mg × m^{-2} intravenously and 1.6 mg. m^{-2} orally.**

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

Atrial Fibrillation :

If not controlled *& ventricular rate is at least 110 beats.min-1 and no contraindication to digitalis is present, small doses of digoxin are given by the following schedule unit the ventricular rate is 90 to 110 beats × min^{-1} :*

1. Heart rate (HR) 110 - 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. HR 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. If 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.
5. If 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 :

In case of irregular heart rate, AF, 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 $\mu g \cdot ml^{-1}$.)

[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.]

Others measures :

- ***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 × 1

Adverse effect : Infrequently hypotension

Esmolol : 0.5 mg $\times$ 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

Rapid Atrial Pacing

***Used for 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.*

(Almost 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 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.

***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 started at a rate 10 beats.min^{-1} faster than the atrial flutter rate. Increased gradually. 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

Started 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

In case of recurrence, 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 with 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 :

Paediatrics patients

Excessive capillary leakage

Raised temperature

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. Administer 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), ≥ 16g . 100 mL^{-1}, repeat (steps 1 and 2.)

Protocol for Patients in Ventilator

1. The ventilator should be with heater and humidifier and the valves for intermittent mandatory ventilation with the positive end expiratory pressure (PEEP) functioning.
2. The patient to be well-positioned and well-secured orotracheal tube(in infants and young children) and in 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}

   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. Criteria for extubation :
   Except in case of severe preoperative pulmonary dysfunction

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 $PO_2 \geq 70$ mmHg or $SaO_2 > 90\%$ to 92% (in the absence of intracardiac right-to-left shunting)

On intermittent mandatory ventilation (IMV) of 6 breaths min^{-1} and FiO_2 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 $PaCO_2$ and pH ($PaCO_2$ 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. Insert a urinary catheter if not already done.
2. Exclude low cardiac output as the cause of the oliguria.
3. Give 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 found with higher doses. However, doses of up to 180 to 240 mg may be necessary in patients with chronic heart failure, cirrhosis, and the 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

4. Consult a nephrologists experienced in cardiac surgical cases

Protocol for Acute Renal Failure

(Serum K^+ Level > 5.5 mEq $\cdot L^{-1}$)

1. Give Sodium bicarbonate IV

   For adults, >>> 1 ampule (44 mEq) IV.

   For infants and children >>> 1 mE1q $\cdot kg^{-1}$

2. 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.)

3. If **Potassium levels exceed 6.5 mE · L^{-1}** and the patient is not receiving digoxin :

   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.]

4. ***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 :

Seizures are an infrequent but potentially serious after 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. Seizure wheather generalized or focal is not helpful diagnostically.

2. Respiratory arrest, discoordinate respiratory activity, or sudden inability to adequately mechanically ventilate can be an evidence 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.
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 likely 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.***

Evaluation and Management :

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^{-2}

f. Body temperature > 38.6°C (> 101.5°F).

Initial Anticonvulsant Therapy :

If seizures are first noted, steps are taken to terminate or 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 (if on ventilation) and

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

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

   (The result of **phenobarbital** may not be apparent for several hours, but if problems continue at this stage, consider to give 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 :

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 Infant Feeding

Catabolic state is rapid in infants after major surgery. Caloric intake to be raised to adequate levels as soon as possible.

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

The steps are as follows :

1. Ensure the endotracheal suction catheter at hand
2. Ensure 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 stomach aspirate is not >10 to 15 mL of fluid then initial feedings can be begun.

   Administer feeds sl owly over 2 to 3 minutes and allow to enter by gravity, preferably with the infant sitting upright.

Otherwise, the infant is placed on right side, with the head inclined.

4. The gavage feeding is given every 3 hours on the following schedule :
   a. Give sterile water, 10 to 15 mL
   b. If tolerated, give 10% dextrose in water, 30 mL
   c. If tolerated and if the residual is less than 5 mL, give Baby formula 30 mL. and,if well tolerated, full strength Baby formula or in increasing amounts.
5. Attention :
   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 Autotransfusion

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

   The two general types are

   a) Provides shed blood directly from the drainage system and
   b) Shed blood collected in 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

Protocol of 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 these instances, warfarin is recommended.
- If elective cardioversion is planned, intravenous heparin is recommended and is prescribed in therapeutic doses.

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To encourage understanding
Medical Science