Chapter 4

Cardiovascular Monitoring and Therapy

4.1 Immediate Postoperative Care and Monitoring

4.1.1 Care in the Operating Suite

Optimal postoperative care of patient mandates adequate correction of cardiac lesion both by physiological and anatomical standpoint. At the end of operative procedure while the child is still in the operating room one should ensure:

Rewarming to 36.5 ℃. Control of bleeding.

Adequate ventilation. Correction of acid-base and electrolyte balance.

Stabilized cardiac function by ensuring:

Maintenance of correct intravascular volume.

Adequate heart rate.

Adequate myocardial contractility and hemodynamics.

4.1.2 Transfer to Intensive Care

After transfer of the patient to ICU inform the multi-professional team:

Anesthetic type, method of perfusion, surgical technique, and duration of aortic clamping (time in minutes),

Fluids: water and colloid balance, diuresis and urine output,

Invasive lines: venous and arterial catheter placement,

Pacemaker wire and mediastinal or thoracic drainage tube positioning,

Ventilator settings and acid-base balance,

Heart rate, arrhythmias, and use of vasoactive medications,

Any abnormal bleeding or status of coagulation.

4.1.3 Standard Monitoring in Intensive Care Unit

EKG, direct arterial pressure (through invasive line in a peripheral artery), central venous pressure, temperature probes (nasopharyngeal, rectal, toe, and skin.), pulse oximeters, and sometimes capnography.

Direct arterial pressure:

Radial artery catheters are generally preferable.

Catheters in foot vessels may not precisely reflect aortic pressure.

Arterial pressure recorded in infants' feet is less accurate in the presence of hypothermia.

The central venous pressure line:

Catheter is directly placed in the right atrium at the end of an operative procedure / or Seldinger technique is used to position the catheter into the internal jugular vein of choice by a surgeon.

Temperature monitoring:

Assesses the body metabolism.

1) Rectal probe or probe near the esophagus:

Provides estimate of body and core temperature.

2) Nasopharyngeal probe:

Estimates brain temperature.

3) Skin temperature probe:

Ideally connected to a toe or finger.

Increased gradient between the core temperature and skin temperature suggests ↓ perfusion of the tissues. The central-to-toe temperature difference increases in the global blood flow abnormalities.

4.1.4 Supplementary Monitoring

A. Pulmonary artery (PA) and left atrial (LA) pressure lines:

These lines are placed through transthoracic (in operating suite) or transvenous approach.

The severity and type of cardiac disease determines their need.

In small children, the traditional Swan-Ganz 7F or 5F catheter can not be used.

Cardiac output is measured by means of a thermo dilution 2F probe inserted into the pulmonary artery.

B. On-line monitoring of arterial (aO_2) and mixed venous oxygen saturation (MVO_2):

These are usually done by fiberoptic catheters placed in a peripheral artery (aO_2), the pulmonary artery, and / or the mid right atrial location (MVO_2).

C. Serial echocardiography:

D. Pulse contour analysis:

It is done by PICCO device. It is a suitable method to monitor cardiac index over a wide range of indices. Recalibrates with the integrated transpulmonary thermo dilution within a short time frame.

Indications for supplementary monitoring:

i) Significant myocardial dysfunction:

May be encountered in the left coronary anomaly, transposition of the great arteries, and hypoplastic left heart syndrome.

ii) Pulmonary arterial hypertension (PAH):

PAH is common during or prior to the operative period, i.e., total anomalous drainage of the pulmonary veins, aortic arch interruption, and truncus arteriosus communis.

iii) Valvular insufficiency:

Valvuloplasty or correction of the atrioventricular canal defect with a suspected residual defect.

4.2 Postoperative Cardiac Output and Cardiovascular Function

Cardiac output (CO) is considered adequate if it is sufficient to meet demands for cellular oxygen. Adequate CO is an index of adequate organ and skin perfusion.

4.2.1 Determinants of Cardiac Output

A. Preload, afterload, contractility (see also Chapter 1), heart rate, and diastolic function of ventricle.

B. Indirect factors:

These alter the myocardial oxygen supply-demand relationship and cardiac output.

1) Anxiety, pain, temperature, and hemoglobin level.

2) Endogenous and exogenous catecholamines and biochemical composition of blood.

C. Intraoperative factors:

1) Anesthetic type, intraoperative complications, myocardial protection, and degree of hypothermia during CPB.

2) Duration of CPB, aortic clamping time, and deep hypothermic circulatory arrest (DHCA) use.

3) Morphology and structure of the heart (neonatal vs. older child).

4) Surgical procedure, degree of ventricle muscle incision, and placement of corrective patches.

Neonatal heart:

i) Contractile force: It is less than that of the adults and children.

Myofibers are fewer (50%) with chaotic pattern, which limits the systolic capacity. Sarcomeres and mitochondria are small in number.

Calcium storage capacity of the sarcoplasmatic reticulum is immature.

Myocardial contractility (during 1st week) occurs due to circulating catecholamines (epinephrine), because sympathetic nervous system is immature in myocardium.

Cardiac output in a neonate is maintained primarily by ↑ in heart rate and is dependent on circulating epinephrine. Neonate is susceptible to parasympathetic stimuli similar to an adult as enervation is complete.

Myocardial fiber length at baseline level is optimal, so a neonate has reduced diastolic reserve for volume overload.

Myocardial contractility and ventricular compliance are reduced despite increases in volume load.

ii) Ventricular interdependency: Volume or pressure load imposed on one ventricle influences filling characteristics of the other ventricle. The dilated right ventricle increases the filling pressure of the left ventricle. This increased pressure on the left generates elevated pressure on the right sided ventricle in order to maintain transatrial flow.

iii) Substrate use: Carbohydrates, amino acids, glycogen, glutamate, pyruvate, and lactate are used for myocardial contraction.

iv) Adaptation to anaerobic metabolism:

Reduced numbers of mitochondria and elevated stores of glycogen reflect this adaptation. It has greater recovery capacity and tolerance for hypoxic and ischemic insults. But it very vulnerable to hypoglycemia, alterations in pH, lactic acid, glycemia, and temperature.

Complete maturity of the myocardium occurs only after 2 years of age.

4.2.2 Effects of Cardiopulmonary Bypass (CPB)

Exposure of blood to the prosthetic surface interface damages blood elements and proteins and triggers a systemic inflammatory response (SIR).

A. Systemic inflammatory response (SIR):

The important components of SIR are as listed below:

Interstitial edema, activation of coagulation cascade, generation of kallikrein, bradykinin, and consumption of platelets.

Activation of neutrophils and monocytes with release of vasoactive and cytotoxic substances.

Activation of complement proteins and anaphylatoxins (i.e., C3a and C5a).

Activation of tumor necrosis factor (TNF) and generation of free radicals derived from oxygen.

B. The CPB associated edema:

It occurs due to hemodilution and inflammatory process unleashed by CPB.

Permeability of the capillary vessels is greater in immature individuals (infants) than in adults. Therefore, prolonged CPB in infants results in ↑ deleterious effects. Overloads the systemic circulation that is already compromised by the previous heart failure.

C. Deleterious effects of excessive hemodilution in CPB:

i) Greater requirement for blood transfusion and inotropic drugs.

ii) Depressed myocardial contractility with undesirable hemodynamic performance.

D. Treatment of CPB associated edema:

1) Conventional ultrafiltration has limited effectiveness in treatment of CPB associated edema.

2) Modified ultrafiltration (MUF) system and CPB associated edema:

Hemoconcentrator is placed between the aorta and the right atrium following CPB. It reduces total body water content, improves systolic function, and increases diastolic ventricular compliance.

Subsequent to modified ultrafiltration, increases in hematocrit, fibrinogen, and total plasma proteins are noted with an elevated systemic arterial pressure.

MUF also decreases serum cytokines and C3a and C5a associated with CPB.

E. Hypothermia:

Profound hypothermia permits reduction of flow through the CPB.

It reduces trauma to blood elements, and it allows total circulatory arrest.

Hypothermia preserves high energy phosphate stores, and it reduces liberation of toxic cerebral neurotransmitters.

F. PH management:

pH-stat method: Acidification of blood provides vasodilatation and homogeneous hypothermic cerebral protection.

Alpha-stat method:

Cognitive development is poor in the late postoperative follow-up in children compared to those undergoing the pH-stat method.

Alpha-stat has no significant effect on postoperative cardiac function as compared to pH stat method.

4.2.3 Postoperative Cardiovascular Evaluation

The cardiovascular function is evaluated postperatively, by either any one or all of the following methods when indicated.

Clinical examination, hemodynamic parameters, tissue oxygen indexes, echocardiography, radioisotope evaluation, and cardiac catheterization and angiogrphy.

1) Clinical evaluation of cardiac output:

It is performed by examination for the following signs and symptoms.

i) Adequacy of level of consciousness, capillary filling, perspiration, and coloring and temperature of extremities.

ii) Thermal gradient between knees and feet, thermal gradient between core (rectum) and peripheral (toe) temperature.

iii) Amplitude of peripheral pulse, arterial pressure, and urinary output.

Signs of adequate cardiac output:

i) No cold perspiration or psychomotor agitation, colored (pink) and warm extremities, and feet are hotter than the knees.

ii) Central to peripheral thermal gradient is < 4 degree centigrade, easily palpable peripheral pulse, good capillary filling.

iii) Arterial pressure is within the normal limits for the age group* and urinary output is greater than l mL/kg/hour.

Peripheral vasodilatation usually occurs after 4th postoperative hour. Establishment of adequate tissue perfusion occurs around 6th postoperative hour.

2) *Non-invasive blood pressure (BP) measurement in infants and children:

a) Pulse oximeter method correlates better with intra-arterial measurements than those recorded from the oscillometric device.

b) Differences between the pulse oximeter and intra-arterial measurements were significantly smaller than, between oscillometric and intra-arterial measurements in children < 15.0 kg.

c) Pulse oximeter waveform change is an accurate and reliable method to measure BP in infants.

3) Tissue oxygen indices and Biochemical markers:

a) Serum Lactate:

i) If serum lactate is > 2.0-3.0 mmol/L, it is evident of tissue hypoxia and a marker of anaerobic metabolism. It reflects ↓ perfusion, hypoxia, or tissue toxic agent.

ii) After DHCA (< 20 ℃), or a low cardiac output, the levels often exceed 6-10 mmol/L.

iii) Always correlate with (A-V) O_2 differences, O_2 transport and O_2 consumption for evaluation (see Chapter 17).

It is ideal to maintain above indexes to normal levels.

b) Troponin, creatinine kinase (MB):

i) Troponin is a specific marker for a myocardial injury.

In infants with cardiac surgery on CPB, ↑ levels of troponin are observed and remain elevated for 72 hours postoperatively.

In infants with cardiac surgery without CPB, troponin levels are not elevated.

ii) Infants from both groups (on CPB and non-CPB) have ↑ creatine kinase MB isoenzyme levels that progressively decrease over 72 hours.

4) Two-dimensional and doppler echocardiography:

Doppler ECHO is performed postoperatively if needed to evaluate the following:

Analysis of cardiac chambers, operative results, and detection of residual septal defects.

Estimation of pressures inside cardiac chambers.

Calculation of myocardial fiber shortening and ventricular ejection fraction.

Segmental and global myocardial analysis.

Evaluation of position and function of valvular prosthesis.

Evaluation of pericardial space (for a clot / fluid).

5) Cardiac catheterization (angiography / manometry of cardiac chambers):

Evaluation of systemic-pulmonary artery shunt permeability (Blalock-Taussig shunt).

Evaluation of pulmonary vasculature, subsequent to uni-focalization of pulmonary arteries.

Measurement of residual pressure gradients across cardiac cavities.

Evaluation of cavopulmonary anastomosis, e.g., Fontan procedure or modified Glenn (bidirectional cavopulmonary ansatomosis).

6) Direct measurement of cardiac output:

a) Fick method:

It is performed by estimation of oxygen consumption (VO_2) and the arterial-venous (A-V) O_2 difference (in vol %) (see Chapter 17).

Oxygen consumption that is calculated by nomograms is dependent on:

1. a) Heart rate, b) Age, c) Sex, and d) Body temperature.

2. Some of the above factors are variable in the first hours following CPB.

b) Thermo dilution method:

It is most frequently used method in the postoperative care. In small children, it is measured by a thermo -dilution 2F probe inserted in the pulmonary artery.

Infants and children: Normal is: > 3 L/min/m^2.

Moderately reduced: 2.0 to 3.0 L/min/m^2,

Severely reduced < 2.0 L/min/m^2.

Cardiac index (CI) tends to be lower in the 4^{th} hour and increases beyond 9-12 hours postoperatively.

(CI) < 2.0 L/min/m^2 in the postoperative period is associated with a greater number of deaths.

4.3 Low Cardiac Output, Determinants, and Treatment

4.3.1 Myocardial Contractility and Myocardial Edema

Reduced myocardial contractility is the primary cause of low CO, and is often noted in complex congenital heart disease such as in left anomalous coronary artery, hypoplastic left heart syndrome, transposition of the great arteries, severe tetralogy of Fallot, and severe pulmonary artery

hypertension (PAH). Myocardial edema causes ventricular diastolic restriction with ↓ diastolic filling with decrease in stroke volume.

4.3.2 Decreased Preload (Intravascular Volume)

1) Fluids shift to the interstitial space during first 24 hours as result of CPB induced endothelial inflammatory process.

2) Vasodilatation during the rewarming period results in active blood loss and ↑ urinary output, thus decreasing preload.

Right and left ventricular diastolic pressures should equal to respective mean atrial pressures.

Diastolic ventricular volume can be controlled by gauzing both the right and left atrial pressures. During the postoperative period, the right atrial pressure should remain around 15 mm Hg.

The optimal or maximal atrial pressures (right 18 mm Hg, Left 20 mm Hg) may be maintained in following specific conditions:

Ventricular hypertrophy or hypocontractility.

Partial obstruction to ventricular outflow.

Pulmonary artery hypertension.

4.3.3 Increased Afterload

It may be associated with ↑ mean arterial pressure for age * (see below).

It occurs primarily by vasoconstriction subsequent to CPB, hypothermia, excessive endogenous catecholamines, or administration of vasoactive amines.

Increased afterload results in reduction of myocardial fiber length, and consequently leads to decrease in systolic volume.

* Normal arterial pressure limits by age:

1 to 12 months: Mean arterial pressure does not exceed 100 mm Hg.

Doppler systolic pressure does not exceed 113 mm Hg.

1 year to < 10 years: Systolic arterial pressure does not exceed 130 mm Hg.

Diastolic arterial pressure does not exceed 85 mm Hg.

> 10 years: Systolic arterial pressure does not exceed 140 mm Hg.

Diastolic arterial pressure does not exceed 90 mm Hg.

4.3.4 Heart Rate

Optimal heart rate varies with age to maintain adequate cardiac output (see Chapter 1).

The heart rate is influenced by intravascular volume, temperature, pain, anxiety, type of surgery, perioperative rhythm disturbances, anemia, metabolic disturbances, and perioperative use of chronotropic dugs.

4.3.5 Treatment of Low Cardiac Output

1. Reduction of metabolic demand:

Maintain body temperature around 36.5 ℃ to reduce the general body metabolism.

2. Reduction of respiratory workload:

It is accomplished by institution of mechanical ventilatory support.

i) Respiratory mechanics and settings: These are manipulated by use of PEEP, adjustment of $PaCO_2$, and pH to improve homodynamic state.

ii) End-points of mechanical ventilation: The following objectives influence decision to wean from mechanical ventilation.

Control of associated bleeding, maintenance of stable hemodynamics, attaining adequate body temperature (perfusion), correction of metabolic derangement, and acid-base imbalance.

3. Measures to improve tissue perfusion and oxygen transport:

Address the determinants of cardiac output with a concomitant use of inotropic and vasodilator agents. For a choice of an inotrope, see the discussion below.

4. Improvement of right ventricular inotropism and control of pulmonary vascular resistance (PVR):

The pharmacological agents such as sodium nitroprusside, nitroglycerin, prostaglandin [PGE_1], prostacyclin (PGI_2), ultra-short acting vasodilators like adenosine, and ATP may be used. The inhaled nitric oxide, phosphodiesterase inhibitors, and inotropes may also be used to achieve this goal.

In pediatric cardiac surgery, currently, phosphodiesterase inhibitors replaced nitroprusside to decrease PVR. Phosphodiesterase inhibitors have inotropic, arteriolar, and venous vasodilator effects.

4.3.6 Inotropes and Vasodilators

A. Dopamine: The physio-pharmacologic effects of this drug are discussed below.

1. Increased myocardial contractility:

By stimulation of postsynaptic beta-1adrenergic receptors and release of norepinephrine from myocardial presynaptic sympathetic storage sites.

By decreasing enzymatic degradation and reabsorption of norepinephrine.

2. Pulmonary and systemic arterial vasoconstrictor:

Through stimulation of postsynaptic alpha-1 and alpha-2 adrenergic receptors.

3. Vasodilator of renal, spleen, coronary, and cerebral vessels:

By exerting action on dopaminic adrenergic receptors.

4. Dose dependent clinical (pharmacological) effects:

i) Dose of 1 to 2 μg/kg/min:

Vasodilator on the mesenteric and renal circulation.

ii) Doses of 2 and 10 μg/kg/min:

Increased myocardial contractility and coronary flow with ↑ in mean arterial pressure (MAP) and cardiac index (CI).

In neonates, ↑ cardiac output, ↑ heart rate, and ↑ systemic arterial pressure.

Dopamine is used commonly in low cardiac output states associated with water retention (as in post CPB).

In neonates, more elevated doses are necessary to obtain the same effects as neonatal myocardium is less sensitive to the action of dopamine than that of older children,

iii) Doses > 15 μg/kg/min:

At this dosage in postoperative pediatric cardiac surgical patients, it ↑ heart rate, ↑ CI, and ↑ systemic arterial pressure without significant action on systemic vascular resistance.

Since rise in pulmonary vascular resistance occurs, this dosage is not used in patients with pulmonary artery hypertension.

B. Dobutamine: The physio-pharmacologic effects of the drug are as discussed below.

It ↑ cardiac output, ↓ systemic vascular resistance, and ↓ ventricular filling pressure.

it is a beta-1 adrenergic receptor stimulant (agonist).

It does not liberate endogenous norepinephrine from receptors.

It has no effect on alpha-adrenergic receptors. Due to positive chronotropic effect, use is limited in cardiac surgery of children and neonates.

The ideal indication for this drug is in a low cardiac output state not associated with severe hypotension.

It is a drug of choice with norepinephrine (a vasoconstrictor) in sepsis if cardiac compromise exists.

Start at low doses such as 2.5 ug/kg/min.

Range: of 2 to 20 ug/kg/min.

C. Isoproterenol:

It is a synthetic analogue of norepinephrine and ↑ cardiac output and ↑ systolic arterial pressure. It is a myocardial beta-1 adrenergic receptor stimulant with inotropic and chronotropic action. Beta-2 adrenergic receptor stimulation promotes peripheral vasodilatation. It is a vasodilator of renal, spleen, and skeletal muscle vessels, and it decreases systemic vascular resistance (SVR) and ↓ diastolic arterial pressure.

It is ndicated mainly in the presence of sinus bradycardia or transitory atrioventricular block. In neonates, isoproterenol improves low cardiac output secondary to persistent arterial hypertension.

Initial dose is 0.01 to 0.05 μg/kg/min for symptomatic bradycardia.

Initial dose is 0.05 to 0.1 μg/kg/min as a positive inotropic agent.

Untoward effects: ↑ In heart rate and ↑ in myocardial oxygen consumption, which leads to transitory myocardial ischemia.

Elevated doses → sinus tachycardia and ventricular arrhythmias (due to ↓ coronary flow).

D. Epinephrine:

It is an endogenous catecholamine liberated from adrenal medulla, and is derived from norepinephrine. It stimulates alpha, beta-1, and beta-2 adrenergic receptors.

Depending on the dose, it ↑ heart rate, ↑ systolic arterial pressure, ↓ diastolic arterial pressure, and relaxation of peripheral vascular bed.

At high doses alpha-adrenergic effect predominates and ↓ skin, gastrointestinal, and renal perfusion.

i) At doses of 0.03 to 0.1 μg/kg/min, beta-1 and beta-2 effects are predominant.

ii) At doses of 0.1 to 0.2 μg/kg/min, there is a mixed alpha and beta effect.

iii) At doses above 0.2 up to 1 μg/kg/min, the alpha response is predominant.

Epinephrine is indicated in low cardiac output states associated with the following:

Systemic arterial hypotension and compromised coronary perfusion as in post-cardiac surgery and cardiogenic or septic shock unresponsive to dopamine and dobutamine. It is also effective inotrope in cardiac arrest.

Adverse effects:

Ventricular arrhythmia (due to subendocardial ischemia) and peripheral vasoconstriction.

E. Norepinephrine:

It is a local adrenergic neurotransmitter with beta-1 and alpha receptor stimulation.

It increases systolic arterial pressure as much as the diastolic arterial pressure.

Norepinephrine increases or decreases the cardiac output depending on the myocardial reserve.

Starting dose: 0.05 to 0.1 μg/kg/min.

F. Phospho-diesterase inhibitors:

These are non-sympathomimetic agents that inhibit the cyclic phosphodiesterase nucleotide, and increase myocardial and vascular cyclic adenosine monophosphate (cAMP). Elevation of cAMP increases myocardial contraction by activation of the kinase protein.

Kinase protein facilitates rapid entry of calcium through calcium channels and activates calcium stores of the sarcoplasmic reticulum.

i) Physiological actions:

It increases myocardial inotropism and contractility, increases arteriolar and venous vasodilatation, and increases ventricular relaxation during diastole.

ii) Clinical effects (e.g., amirinone):

Increases cardiac index and decreases left ventricular end diastolic pressure,

Reduces ventricular wall stress and impedance to ejection.

Use in congestive heart failure and in postoperative low cardiac output leads to ↓ pulmonary capillary pressure, and ↓ right atrial pressure.

In combination with dobutamine administration, causes greater elevation of the cardiac index and greater reduction in systemic vascular resistance, but does not significantly cause increase in heart rate.

iii) Clinical indications:

Low cardiac output with myocardial dysfunction and elevated systemic vascular resistance (without severe arterial hypotension).

iv) Side effects:

Hypotension, thrombocytopenia (incidence of occurrence with amrinone is 2.6% vs. 0.4% with milrinone), and elevation of hepatic enzymes.

v) Drug potency and metabolism:

Milrinone is 10 to 30 times more potent than amrinone. Milrinone does not elevate myocardial consumption, and is a coronary vasodilator. Milrinone is metabolized primarily in the kidneys (need dose correction in renal failure).

Amrinone is metabolized by the liver.

a) Amrinone:

Dose: 0.75 ug/kg bolus dose (over time for 2-3 doses) is followed by, a maintenance infusion of 5-10 ug/kg/min.

Neonates:

Bolus doses of 3 to 4.5 ug/kg is followed by

Maintenance infusion 5 to 15 ug/kg/min.

The half-lifeof amrinone is 3 to 15 hours, so use caution in systemic arterial hypotension.

b) Milrinone: Dosing in normal renal function:

50 ug/kg bolus dose over 10 minutes is followed by, a maintenance infusion of 0.375 to 0.75 ug/kg/min.

The half-life of milrinone in CHF is 2-3 hours.

G. Vasodilators:

These agents are used as an adjuvant to inotropic therapy to reduce systemic arterial resistance and enhance ventricular ejection.

a) Sodium nitroprusside:

Dose: 0.5 to 8 ug/kg/min. It causes arteriolar and venous vasodilatation, controls systemic and pulmonary hypertensive states, and reduces after load in low cardiac output states post-cardiac surgery. Phosphodiesterase inhibitors replaced sodium nitroprusside in pediatric cardiac surgery due to inotropic, arteriolar, and venous vasodilating properties.

b) Nitroglycerine:

Dose: 0.5 to 20 ug/kg/min. It acts as a vasodilator by releasing nitric oxide. Causes vasodilatation and ↓ ventricular filling pressures.

It is indicated if preload↑ with signs of pulmonary and systemic venous congestion.

4.4 Right Ventricular (RV) Dysfunction and Inhaled Nitric Oxide (NO)

RV dysfunction is often observed in infants and children in the postoperative period. RV output is very sensitive to the variations in afterload, so control of pulmonary vascular resistance (PVR) is extremely important.

Ideal strategies to improve RV function in postoperative period are as follows:

1) Increase RV Inotropism.

2) Optimize RV preload.

3) Control or reduction of pulmonary vascular resistance (PVR).

Pharmacological and ventilatory manipulations are some of the measures commonly used to achieve reduction of PVR.

Common agents used for controlling PVR:

Sodium nitroprusside, nitroglycerin, and phosphodiesterase inhibitors.

Prostaglandin E_1 (PGE_1) and prostacyclin (PGI_2) are pulmonary vasodilators, but these drugs are not specific to the pulmonary circulation.

Newer drugs (i.e., Inhaled nitric oxide and ultra-short-acting intravenous vasodilators such as adenosine and adenosine triphosphate) are used for treating acute pulmonary artery hypertension (PAH) in pediatric cardiac patients.

Inhaled nitric oxide (NO):

Produced by the endothelial cells, and maintains blood vessels of lung relaxed (dilated). It is useful in presence of previous pulmonary hypertension and/or (RV) dysfunction. Rapidly, it is inactivated by hemoglobin, and no vasodilatation of systemic circulation occurs.

Clinical usage:

Commonly used in severe RV dysfunction with PAH.

Improves pulmonary gas exchange by recruiting more number of pulmonary capillary units.

Start with small doses i.e., 3 to 5 parts per million and may ↑ to 20-40 parts per million.

Prior to NO use, do the following:

1) Adequately sedate the patient.

2) Provide adequate ventilation.

3) Correct acid base abnormalities.

4) Use phosphodiesterase inhibitors to decrease pulmonary artery pressures and improve RV function, and to improve the efficacy of inhaled NO, see figure 9 and the discussion below.

5) Monitor ventricular function with echocardiography.

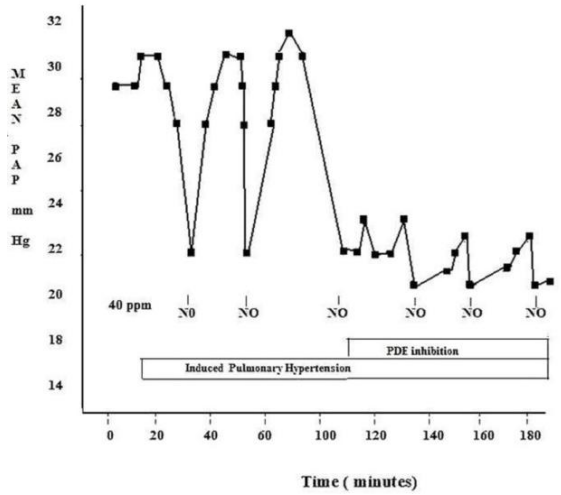

Figure 9 **Prolongation of pulmonary vasodilator effects of inhaled NO by phosphodiesterase inhibitors in pulmonary hypertension. Pulmonary hypertension was induced with U 44619 in lambs. Inhaled NO (40 ppm) was administered intermittently before and after intravenous infusion of PDE inhibitor, zaprinast (C-GMP specific) and mean PAP was recorded. Without PDE inhibition, the reduction of mean PAP produced by inhaled NO persisted only 2-3 minutes. With zaprinast infusion the reduction in PAP was maintained for nearly 90 minutes with only four 4-minute periods of NO inhalation. NO = nitric oxide, PAP= pulmonary artery pressure, PAH= pulmonary artery hypertension PDE = phosphodiesterase.*Adapted from the journal of applied physiology, 1995, Vol 78. The American physiological society.*

Dipyridamole (another PDE inhibitor) has also been used to augment the efficacy of inhaled NO in human trials. Dipyridamole in high doses may worsen regional myocardial perfusion (especially in coronary artery disease). Sildenafil may also be used to prolong and augment the effects of inhaled NO for treatment of PAH.

4.5 Heart Rate, Rhythm, and Conduction Disturbances

1) Normal heart rate:

The normal heart rates for different age groups of children is shown in a table 4.1

Table 4.1 **Age specific normal heart rates (beats/minute).*

Age	2%	Mean	98%	Age	2%	Mean	98%
1st week	91	123-130	155-165	1-2 mon	121	149	179
1-3 weeks	107	148	182	3-5 mon	106	141	186
6-11 mon	109	134	169	1-2 yrs	89	119	151
3-4 yrs	73	108	137	5-7 yrs	65	100	133
8-11 yrs	62	90	130	12-15 yrs	60	85	119
% percentiles. mon= months							

*Modified from: The Harriet Lane Handbook. A Manual for Pediatric House Officers, 1996, 14th edition. Mosby Year Book Inc.

2) Sinus tachycardia:

Heart rate greater than 95 percentile for age with a normal sinus rhythm.

Normal R-R and P-P relationship.

Rate may vary with respiration, normal QRS morphology with sinus P waves.

Usual rate < 230/minute. Causes: fever, hypovolemia, sepsis, pain, etc.

Treat underlying cause.

3) Sinus arrhythmia:

Respiratory variation of R-R intervals, with no change in P-R intervals.

Normal sinus P wave with a normal QRS morphology (see Figure 10).

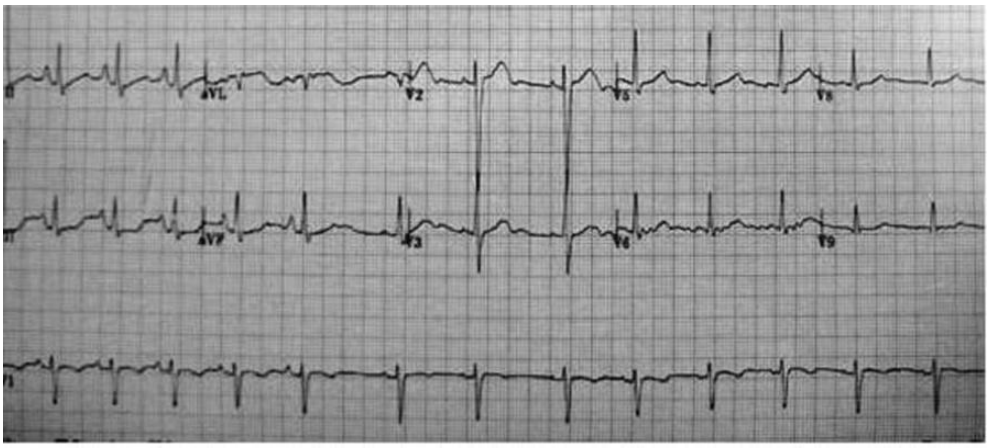

Figure 10 *EKG strip showing sinus arrhythmia. Note the respiratory variation of sinus beats with constant PR interval, but with changing RR intervals.*

4) Sinus bradycardia:

Heart rate less than 5th percentile for age with a normal sinus rhythm.

Causes: Hypoxemia, acidosis, hypercapnia, and increased intracranial pressure.

Treat only in symptomatic patients and correct underlying cause.

Epinephrine: 0.01 mg/kg bolus IV / IO. Repeat every 3-5 minutes prn.

(ET dose 0.1 mg/kg)

Atropine: 0.02 mg/Kg bolus IV, minimum bolus dose 0.1 mg.

Maximum bolus dose: 0.5 mg (child), 1 mg (adolescent).

Isoproterenol drip: 0.1 mcg/kg/minute and increase by 0.1 mcg/kg/minute

every 5-10 minutes, maximum dose 2 mcg/kg/minute.

5) Supraventricular tachycardia:

Normal QRS complexes with or without discernible P waves.

Rate of > 230 beats /minte (bpm); (260-300 bpm in infants).

P waves do not have sinus morphology.

After first 20 beats in the rhythm strip, the QRS morphology is same as sinus rhythm. If QRS is like in bundle branch block VT is considered (see Figure 11).

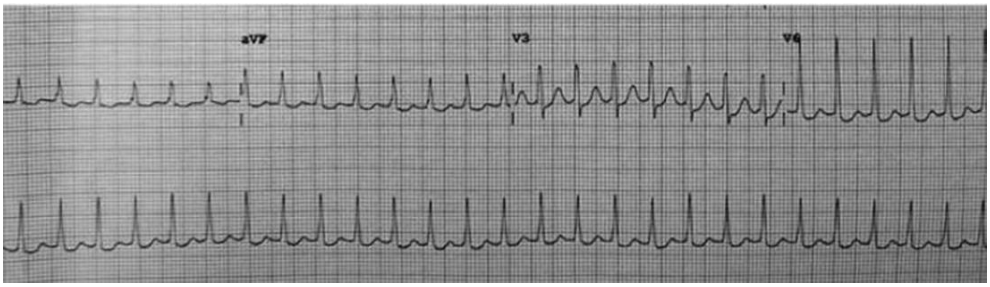

Figure 11 *EKG strip of supra ventricular tachycardia. (rate 188 beats per minute). Note the normal QRS morphology without discernible P waves.*

Treatment:

i) Vagal maneuvers: (ice to face and head, carotid massage, valsalva, knee-chest position).

ii) Adenosine: 0.1 mg/kg rapid IV bolus, if there is no effect, repeat a double dose (0.2 mg/kg): maximum single dose: 12 mg IV.

iii) Other drugs: Digoxin, beta blockers, procainamide (see Chapter 16).

iv) Other measures: Atrial overdrive pacing or synchronous cardio version:

Dose for cardioversion: 0.5-1.0 joules/kg.

In unstable patient, cardioversion should be initially tried.

6) Wolf-Parkinson-White (WPW) tachycardia:

Shortened PR interval and initial slurring of the upstroke of QRS, producing delta wave and prolonged QRS duration (see Figure 12).

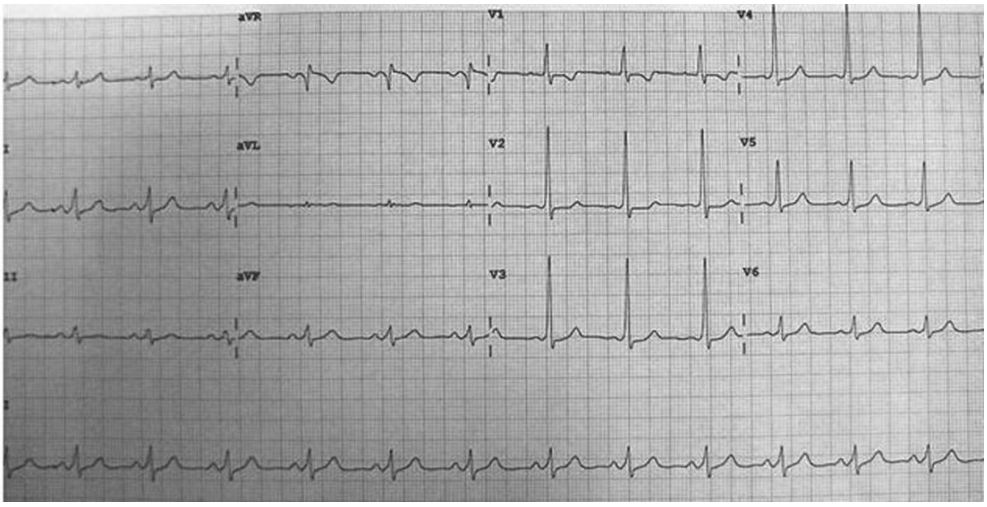

Figure 12 *12 lead EKG of WPW syndrome. Note the delta wave (initial slurring of QRS complex) prominent in lead I. Note also shortened PR interval (100 msec) and prolonged QRS (180 msec).*

Treatment and prevention of SVT:

If delta wave is present: Digoxin or verapamil should not be used.

May use beta- blockers.

Recurrent SVT may require ablation of accessory conduction tract.

7) Atrial flutter:

Normal QRS complexes with flutter (p) waves between QRS.

Atrial rate 250-500 bpm, ventricular rate depends on a variable block, i.e., 1:1 to 4:1 conduction. 92% patients have abnormal heart rate (see Figure 13).

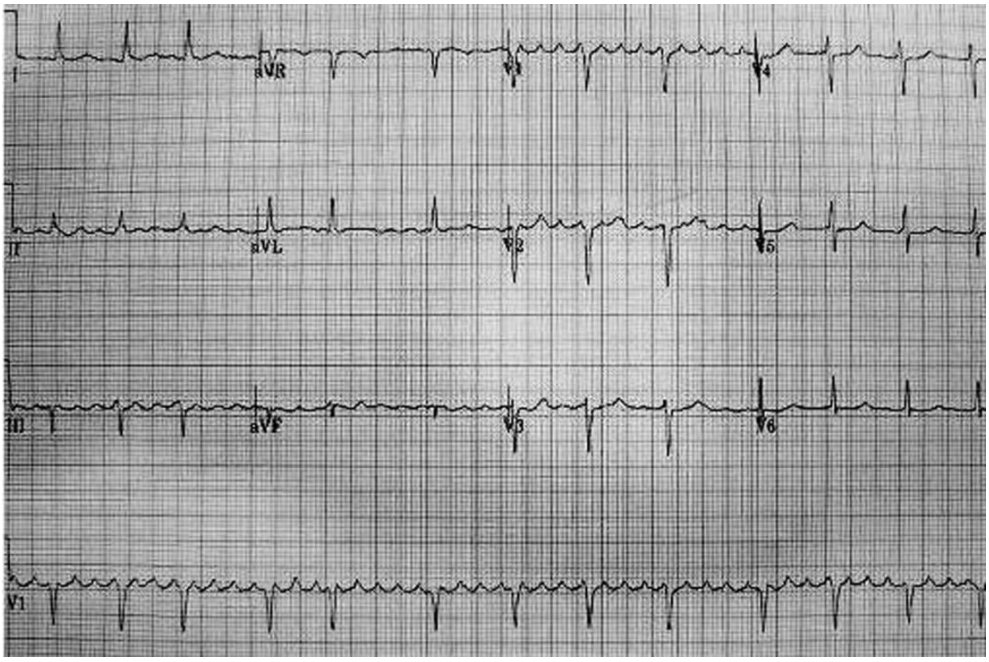

Figure 13 *12 lead EKG of atrial flutter with 3 to 1 atrioventricular block (atrial flutter rate of 300 to 375, ventricular rate 100 to 125 beats per minute).*

Treatment:

Drugs: Digoxin, qunidine (oral dose), and procainamide (see Chapter 16-D).

Other measures: Atrial overdrive pacing or synchronous cardioversion with 0.5-1.0 joules/kg (in unstable patient it is initial Rx)

8) Atrial fibrillation:

Normal QRS complexes with absent P waves and irregularly irregular R-R intervals. The heart rate varies (see Figure 14).

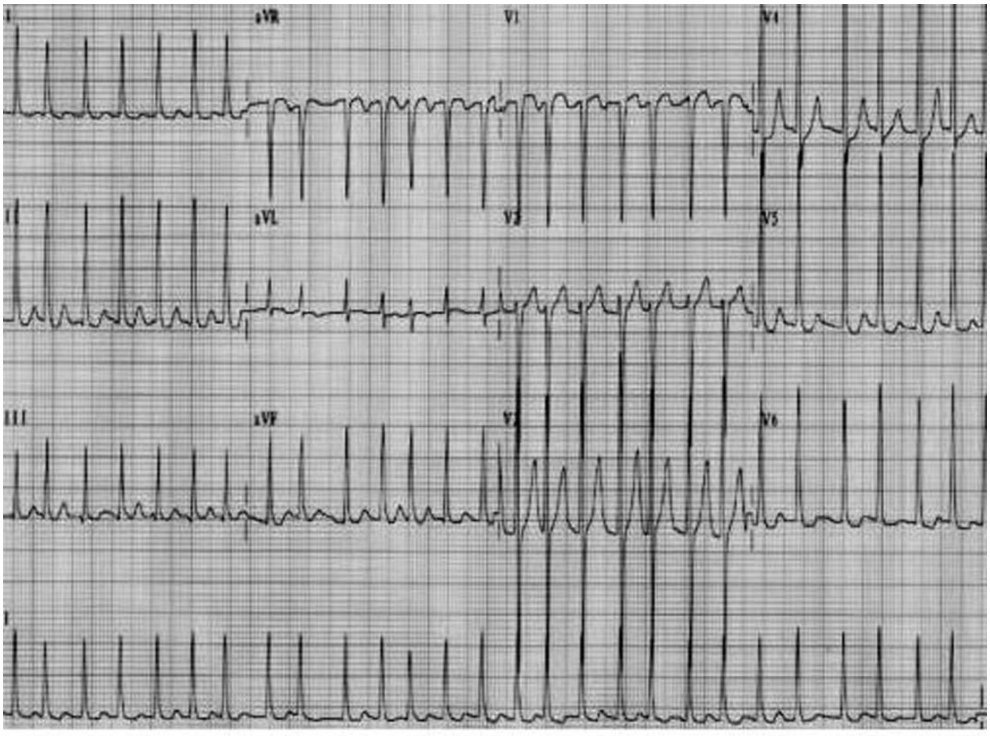

Figure 14 *12 lead EKG of atrial fibrillation with a controlled ventricular rate. Ventricular rate varies from 130-168 beats per minute. Rhythm is irregularly irregular. Note that there are no discernable P waves.*

Treatment:

For control of rapid ventricular rate digoxin / beta blockers are often used.

Quinidine is used to prevent recurrence.

Synchronous cardioversion: 0.5-1.0 joules/kg (in unstable patient it is initial Rx).

The patient is anticoagulated before cardioversion.

9) Premature ventricular contraction:

Wide or prolonged QRS complexes that differ morphologically from a normal (sinus) QRS. ST segment slopes away from QRS, and T wave is often inverted. Impulse is usually followed by a compensatory pause (see Figure 15, top strip).

Bigeminy (alternate normal and abnormal ventricular beat), couplets (two consecutive abnormal beats) or triplets may occur.

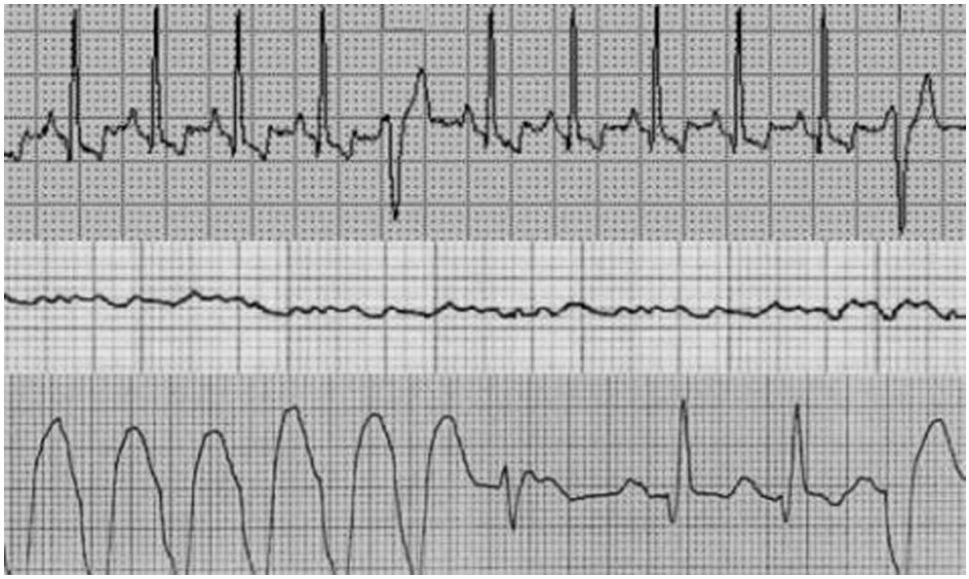

Figure 15 *Top strip: Sinus rhythm with two premature ventricular contractions that are followed by a compensatory pause. Middle strip: Scribble on a rhythm strip representing ventricular fibrillation. Bottom strip: Fusion beats due to ventricular tachycardia (VT): The first of the narrower complexes is a fusion beat; the next two are capture beats. In some cases of VT with AV dissociation the SA node may transiently "capture" the ventricles producing a capture beat, which has a normal QRS duration, or a fusion beat, in which the sinus and ventricular beats coincide to produce a hybrid complex. It indicates that there are two foci of pacemaker cells firing simultaneously: the sinus node and a ventricular ectopic focus.*

Treatment:

Indicated only in symptomatic patients and / or > 6 PVC's per minute.

Address the underlying abnormality: Metabolic disorder, drugs, myocarditis hypoxia, etc.

Lidocaine: 1 mg/kg IV bolus, followed by an infusion of 20-50 mg/kg/minute.

Other drugs: Procainamide and quinidine (see Chapter 16-D).

10) Ventricular tachycardia (VT):

Greater than three serial PVC's occurring in a rhythm strip, or abnormal QRS's occurring at rapid rates.

QRS is prolonged and wider than normal upper limit for age.

P waves, if occur, are dissociated and Q waves are absent in V5 and V6.

Fusion beat may occur before and after termination of VT.

After 10-20 beats, the morphology of QRS differs from a normal sinus beat.

70% have abnormal rate (< 250, in infants 200-500 bpm) (see Figure 16).

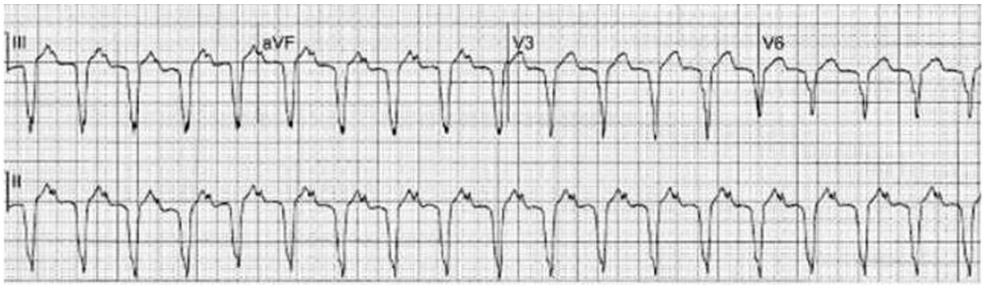

Figure 16 *EKG strip showing ventricular tachycardia with a controlled ventricular rate of 120 beats per minute. Note P waves are seen in some leads but are dissociated from QRS complexes.*

Treatment:

Treat the underlying abnormality, and if hemodynamically stable give lidocaine, bretylium (see Chapter 16).

Hemodynamically unstable patients: Cardioversion is used.

11) Ventricular fibrillation:

Rapid irregular ventricular depolarization of low voltage (without definite QRS complexes).

Mimics asystole that is more common in children (see Figure 15. middle strip).

Treatment:

See cardiac arrest algorithms (see Chapter 13).

12) Fusion beat:

It has characteristics of both a sinus beat and a PVC.

QRS resembles an early activation of a sinus and a late activation of PVC (see figure 15, bottom strip).

13) First degree AV block:

PR interval is prolonged than normal for age and heart rate (see text).

PR interval varies between 0.09 to 0.2 seconds,

PR is short, (i.e., 0.11-0.15) seconds: Occurs with rapid heart rates (140-180 bpm) and in infants and children < 3 years.

PR is longer, (i.e., 0.15-0.2) seconds: Occurs with slow heart rates (60-100 bpm) and older children and adults (see Figure 17)

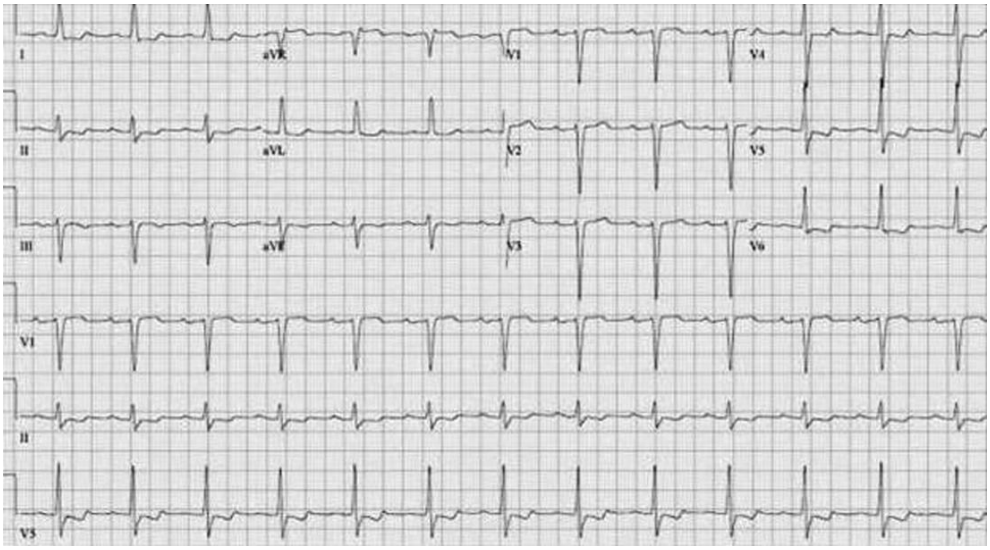

Figure 17 *12 lead EKG of a first degree atrioventricular block. PR interval is prolonged (240 msec).*

14) Second degree AV block: Mobitz (type II) block.

Atrial rate > ventricular rate with regular conduction of atrial impulse to ventricle at varying rate. i.e., 2:1, 3:1, 4:1(skipped ventricular beats).

PR interval does not change (see Figure 18).

Progresses to complete AV block.

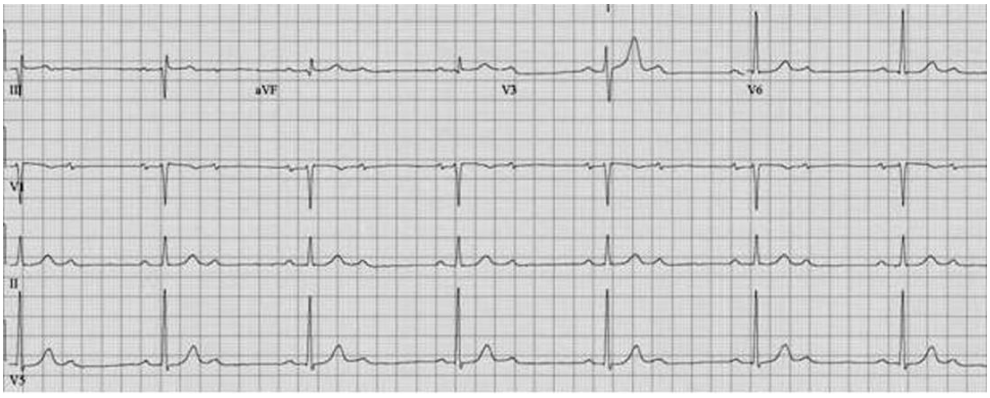

Figure 18 *EKG strip showing second degree atrioventricular block of Mobitz type II.*

15) Second degree Wenckebach block: Mobitz (type I) block.

Progressive prolongation of PR interval until atrial impulse is not conducted to the ventricle (dropped ventricular beat occurs).

PP interval is regular and RR interval shortens (see Figure 19).

Does not progress to complete AV block.

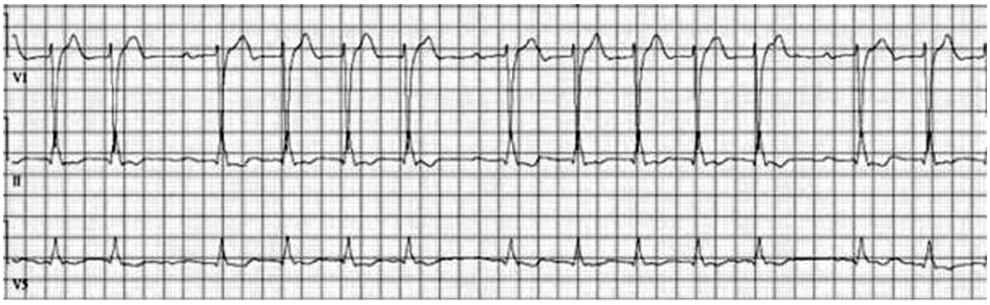

Figure 19 *EKG strip showing second degree atrioventricular block of Wenckebach (Mobitz type I). Patient has sinus rhythm with apical non specific ST segment and T wave changes.*

16) Complete AV block:

Atrial beats are not conducted to the ventricles.

Slow rate ventricular beats (junctional or ventricular morphology) are not related to atrial beats.

Complete AV dissociation and changing PR intervals (see Figure 20).

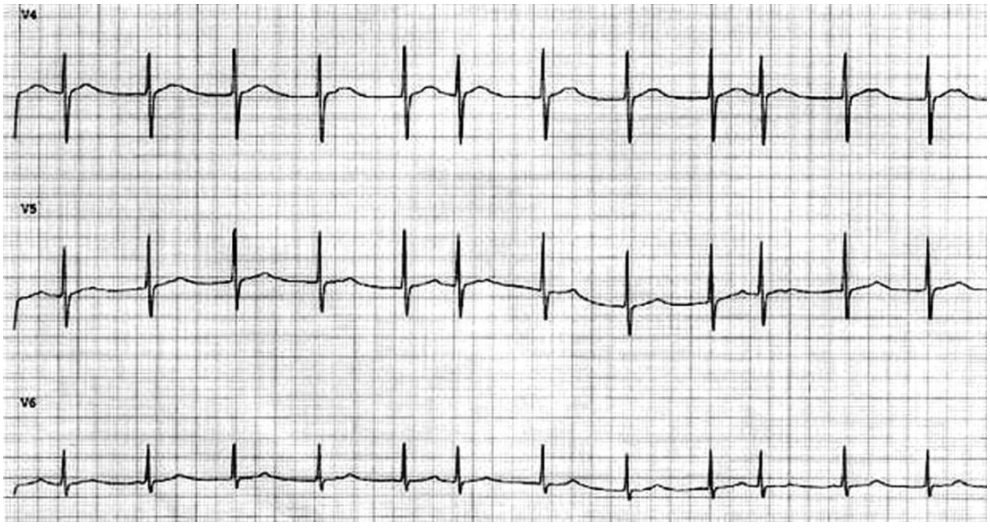

Figure 20 *Lateral precordial leads V4, V5, V 6 showing complete AV block. Note P waves have no relationship with QRS complexes and also note changing PR intervals.*

Treatment:

Epinephrine: 0.01 mg/kg bolus IV/I0 or 0.1 mg/kg ET. Repeat every 3-5 minutes.

Atropine: 0.02 mg/kg bolus IV (minimum 0.1 mg, Maximum 0.5 mg in a child Maximum 1 mg in an adolescent).

Isoproterenol drip: 0.1 mcg/kg/minute, increase every 5-10 minutes by 0.1 mcg/kg/minute, Maximum dose 2 mcg/kg/minute.

If above drugs fail to improve heart rate and/or reverse AV block pacemaker therapy is indicated.

Pacemaker therapy in emergency:

Connect epicardial temporary wires to a pulse generator.

Modes of pacing:

Atrial pacing, ventricular pacing, and AV sequential pacing.

Sinus rhythm improves cardiac output (CO) by 20% as compared to V-pacing alone.

Sinus rhythm improves CO by 50% in a non-compliant heart.

Pacing rates:

Ideal rates vary with age.

Neonates and young infants: 140-160/minute.

Older infants and young children: 120-140/minute.

Older children and adolescents: 100-120/minute.

Modes of pacing and terminology for modes of pacing:

AAT, VVI, VVT, DDD, DVI

Chamber paced (A, V, or D), A= Atria, V= Ventricle, D= Atria and ventricle

Chamber sensed (A, V or D)

Pacemaker inhibits its own output (I) if senses a native impulse, but fire an impulse if no native impulse is sensed (D), D= demand pacing.

Pacemaker triggeres (T) impulse, despite presence of a native impulse.

Temporary pacemaker settings:

Rate: as desired.

Atrial output: 5-10 ma, (milli amperes). Ventricular output: 5-10 ma.

AV interval: 150 to 200 msec.

Ventricular sensing 5-10 mv (milli volts) for demand pacing or VVI.

Use asynchronous mode (> 20 mv) for VVT.