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Postgraduate Topics in ANAESTHESIA

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Preface

A book of short notes! That too when you have umpteen numbers of textbooks and CME notes. The present examination system is entirely different from the past. The long hours of essay writing are replaced by precise short answer questions. You cannot write volumes on a particular topic. The answers have to be precise.

The students of anaesthesia are confronted with many avenues of learning which include textbooks, journals, lectures, refresher courses and their own clinical experience. The book represents a set of short answers from the selected questions asked for postgraduate examinations in standard institutions in the last five years. This is not intended to replace the textbooks, but to augment them. The book is meant to help the postgraduates in preparing for the examination and to give them an insight into the art of short note writing and say serve as a quick reference book before the examination.

V Mahadevan

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I acknowledge with gratitude the supports provided by my teachers, colleagues, postgraduate students.

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Contents

1 Oxygen–Haemoglobin Dissociation Curve

Oxygen–haemoglobin dissociation curve relates the saturation of Hb (Y-axis) to partial pressure of O_2 (axis). Hb is fully saturated (100%) by a PO_2 of about 70 mmHg. The normal arterial saturation of 95-98% with oxygen occurs by a PaO_2 of about 95-100 mmHg (Fig. 1.1).

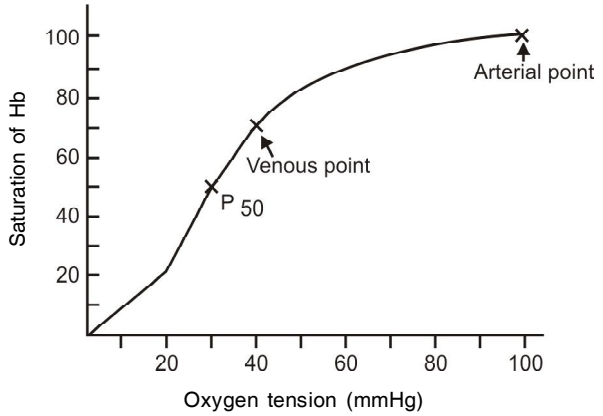

Fig. 1.1: Oxygen–Hb dissociation curve at normal pH

As blood passes by the alveolus, oxygen diffuses into plasma, increasing the partial pressure of O_2 (PaO_2). As PaO_2 in blood increases, O_2 diffuses into RBC combining with the ferrous iron (Fe^{2+}) in the haemoglobin. Each Hb molecule has 4 Fe^{2+} atoms. As each Fe^{2+} combines with O_2, affinity of the Hb to Fe^{2+} increases, till it is completely saturated.

Shape of ODC

The sigmoid shape of the curve reflects the physiological adaptation of Hb to take up oxygen at higher partial pressures (i.e. in alveoli) and release O_2 at lower partial pressure (in tissue). When PO_2 is less than 60 mmHg (90% saturation), the saturation falls steeply, so that for a given decrease in PO_2 amount of Hb uncombined with O_2 increases greatly. Mixed venous blood has a PO_2 of about 40 mmHg (i.e. 75% saturation).

Significance

Oxygen–Hb curve can relate oxygen content (ml O_2 / 0.1 L blood) to PO_2.

- Oxygen is carried in solution in plasma = 0.003 ml O_2 mmHg PO_2 / 0.1 L
- Oxygen is combined with Hb–1.39 ml O_2 /gm of Hb

- Therefore, oxygen content = 1.39 × % saturation + 0.003 × PO_2
- Arterial oxygen content in a patient with Hb 15 ml/0.1 L comes to 21.2 ml of O_2 / 0.1 L
- $C\bar{v}\ O_2$ comes to 15.2 ml of O_2/0.1 L blood.
- Normal arteriovenous oxygen content difference = 5.5 ml / 0.1 L
- Oxygen–Hb curve can also relate the oxygen transport to peripheral tissues to PO_2
- Oxygen transport = O_2 content (Ca O_2) × cardiac output
- Oxygen consumption amounts to 250 ml/min.

Oxygen–Hb curve can relate O_2 available to tissues as a function of PO_2–of the 1000 ml of oxygen going to the tissues, 200 ml cannot be extracted because it will lower PO_2 below the level at which brain can survive. So O_2 available to tissues is about 800 ml/min. With lower arterial oxygen saturation, the important thing to be remembered is that tissue demand of oxygen can be met only by an increase in cardiac out or in long-term by an increase in Hb concentration.

SO_2–consumable oxygen–defined as percentage saturation of Hb when oxygen tension is 20 mmHg–lowest $P\bar{v}\ O_2$ at which tissue oxygen tension is thought to be possible. Normally SO_2 is 33% Hb available.

The position of oxy-Hb curve is best described by P_{50}–partial pressure of oxygen at which haemoglobin is half saturated with oxygen at 37°C at pH 7.4. The normal adult P_{50}–26.7 mmHg.

SHIFTS IN ODC

The effect of a shift in the position of oxy–Hb curve on Hb saturation depends greatly on PO_2. In the region of normal PaO_2 (75-100 mmHg) curve in relatively horizontal, so that shifts of the curve has little effect on saturation. In the region of mixed venous PO_2 (40-50 mmHg) curve is relatively steep.

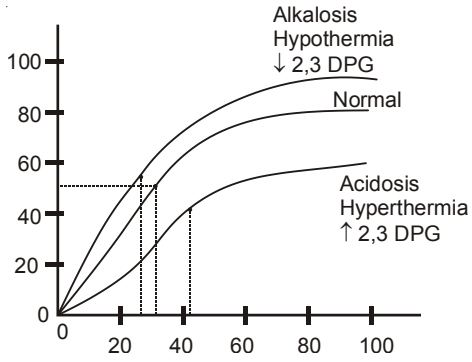

Fig. 1.2: X-axis - PO_2 (mmHg) Y-axis - oxygen saturation (%)

P_{50} less than 27 mmHg describes a left–shift of oxy–Hb curve meaning, at any given PO_2 , Hb has a highest affinity for oxygen and therefore more saturated that normal, i.e. shift of ODC to left. This requires a highest tissue perfusion than normal to produce a normal oxygen delivery to tissues.

Causes are

- Alkalosis (metabolic and respiratory)
- Hypothermia
- Abnormal and foetal Hb
- Carboxy Hb
- Met Hb
- Decrease in RBC 2,3 DPG content–occurs with transfusion of old acid citrate–dextrose (ACD) stored blood (more than 6 to 7 days). This persists for up to 24 hr, after transfusion. Storage of blood DPG with time. Hence to minimise the effects:
  - Should warm all blood.
  - Avoid excessive bicarbonate administration.
  - Use frozen blood if available.
  - Avoid infusing blood that is more than 5-7 days old.

P_{50} more than 27 mmHg describes a right ward shift of oxy-Hb curve which means that at any given PO_2, Hb has a affinity for oxygen and is less saturated than normal.

CAUSES

- Acidosis (metabolic and respiratory)
- Hyperthermia
- Abnormal Hb
- Increased RBC 2,3 DPG
- Anaemia
- Exercise
- Propranolol
- Sickle cell anaemia
- Deficiency of pyruvate kinase
- Drugs like digoxin, testosterone

FACTORS AFFECTING THE POSITION OF THE OXY-Hb DISSOCIATION CURVE

Temperature

Increase in temperature decrease Hb–O_2 affinity and ODC is shifted to right. Conversely, when body is cooled, O_2 demand decreases, P_{50} falls and ODC is shifted to left.

Hydrogen Ions

P_{50} is inversely proportionate to pH. Acute acidosis shifts the ODC curve to the right. Acute alkalosis shifts the ODC curve to the left. Acute changes in pH of 0.1 unit will change of P_{50} by approximately 3 mmHg. The effect of chronic pH changes (lasting longer than 2 to 3 hr) depends largely on compensatory change in organic phosphate synthesis.

CO_2

Modifies the position of ODC by altering pH (Bohr effect). P_{50} is directly proportional to the partial pressure of CO_2. Acute respiratory acidosis will shift the curve to the right; acute respiratory alkalosis will shift the curve to the left.

Organic Phosphates–2,3 DPG and ATP

These bind to the oxy Hb–The normal intra erythrocytic concentration of 2,3 DPG is about 4 m mol/L of RBC. Increase in concentration of 2,3 DPG or ATP or both will shift ODC to the right, and decrease will shift ODC to the left. 2,3 DPG is produced in red cells by hexose monophosphate shunt pathway of glycolysis (The Rapoport-Luebering Shunt).

The production of 2,3 DPG is potentiated through enzymatic response to anaemia, alkalosis and hypoxaemia. Alteration of 2,3 DPG production is suppressed by polycythaemia, acidosis and hyperoxaemia.

Alteration of 2,3 DPG requires several hours to become evident. In chronic acidosis 2,3 DPG is diminished (left shift). In chronic alkalosis 2,3 DPG is increased (right shift).

Congenital Abnormalities of ODC

- Haemoglobinopathies shift to right or left depending on the affinity of abnormal Hb to O_2
- Disorders of red cell metabolism–pyruvate kinase deficiency shifts the ODC to right with elevated 2,3 DPG levels. Hexokinase deficiency shifts the ODC to left with low 2,3 DPG levels.

ODC and Environmental Factors

Physiological adaptation to high altitudes. At high altitudes oxygen tension is markedly reduced. Compensatory mechanisms are—Hyperventilation, polycythaemia, increased breathing capacity (since air is less dense) and pulmonary vasoconstriction.

Hypoxaemia and hypocarbia stimulate 2,3 DPG production and results in a right ward shift of ODC and improved oxygen extraction.

Carbon Monoxide

CO has an affinity for Hb over 200 times greater than that of oxygen and readily displaces oxygen from Hb. It has a direct effect on P_{50} and shifts the ODC to the left, further reducing available oxygen to the tissues.

ODC and Chronic Disease States

- Cardiopulmonary disease–mostly there is compensatory increase in 2,3 DPG. In low cardiac output states as in CCF, tissue compensates by extracting more oxygen. Increase in de-oxy Hb stimulates phosphofructokinase and 2,3 DPG production.

- Anaemia— most important compensatory mechanisms are:
  - Increased cardiac output and oxygen delivery.
  - Right shift of ODC—mostly by increased 2,3 DPG level.

Uraemia and cirrhosis and thyroid disease—increased 2,3 DPG levels and right shift of the ODC.

ODC in Acute Disease State

Hypophosphataemia—results in lowered P_{50} and increased Hb-O_2 affinity. Some of the causes are—parenteral nutrition, alkalosis, starvation, vomiting, malabsorption, antacids, hyperphosphaturia, hypokalaemia, haemodialysis.

Shock—effects on ODC involves interaction of pH, $PaCO_2$, and temperature. Studies showed that 2,3 DPG and P_{50} were lower in patients with septic shock and may be a reason for lower oxygen extraction. Factors contributing to increased oxygen affinity in shock are massive blood transfusion, acute alkalosis (hyperventilation, bicarbonate administration) metabolic acidosis, hypophosphataemia and hypothermia.

Acute Myocardial Ischaemia

P_{50} was found to be elevated and ODC shifted to right after documented myocardial infarction.

Blood Storage and Transfusion

Mentioned earlier.

ODC and Anaesthesiologist

Volatile anaesthetic agents—in general all inhalational agents including N_2O causes a shift to right in the ODC.

Intravenous anaesthetic agents—have no demonstrable effect on ODC.

ODC and Cardiopulmonary Bypass

ODC and cardiopulmonary bypass influenced by the state of hypothermia, type of anticoagulated blood (ACD or CPD) used for blood transfusion, etc.

Therapeutic Manipulation of ODC

- Infusion of inosine, pyruvate and phosphate–caused significant rise in 2, 3 DPG. But there was no difference in P_{50} or oxygen extraction.
- Steroid therapy–Methyl prednisolone was found to induce a transient moderate increase in cardiac indices and a sustained significant increase in P_{50}. The use of steroids, particularly in septic shock has been claimed to be beneficial, by causing membrane stability, haemodynamic improvements and improvements in tissue oxygenation.

- Propranolol was found to decrease oxygen affinity–by releasing the bound 2,3 DPG in red cells. This effect is blocked by epinephrine. Its beneficial effect in angina may be in part due to increased myocardial oxygenation by causing a decreased Hb–C affinity.

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2 Bohr Effect

The shift of the oxygen haemoglobin dissociation curve to the right in response to respiratory or metabolic acidosis is important to enhance oxygenation of the blood in the lungs and also to enhance release of oxygen from the blood in to the tissues. This is called Bohr effect.

The oxygen affinity of blood decreases as the pH of the blood falls. This is closely related to the fact that deoxy–Hb binds with H–more actively than oxyhaemoglobin. The pH of blood decreases as CO_2 content increases, so that when PCO_2—rises, the curve shifts to the right and the P_{50} rises. Most of the unsaturation of haemoglobin that occurs in tissues is secondary to the decline in the PO_2, but an extra 1 to 2% unsaturation is due to the rise in PCO_2 and the consequent shift of the association curve to the right.

As the blood passes through the lungs, carbon dioxide diffuses from the blood to the alveoli. This reduces the blood $PaCO_2$ and also decreases the hydrogen — concentration, because of the resulting decrease in blood carbonic acid. Both these effects shift the oxyhaemoglobin dissociation curve to the left and wards. Therefore, the quantity of oxygen binding with haemoglobin at any given alveolar PO_2 increases, thus allowing greater transfer of oxygen from the alveolar air the pulmonary capillary blood.

Almost exactly the same principles are applicable of diffusion of oxygen through the placental membrane, as through the pulmonary alveolo-capillary. The dissolved oxygen of the large maternal sinuses passes into the foetal blood because of an oxygen pressure gradient existing between mother's and foetal blood. The mean PO_2 in the mother's blood in the maternal sinuses is approximately 50 mmHg, towards the end of pregnancy, and the mean PO_2 in the foetal blood after it becomes oxygenated is about 28 mmHg, the mean pressure gradient being about 20 mmHg. Even this low PO_2 allows the blood to carry enough O_2 due to 3 reasons.

- Foetal Hb has greater affinity for O_2.
- Higher Hb concentration of foetal blood.
- Double Bohr effect.

$PaCO_2$ in maternal blood sinuses is about 33 mmHg (arterial). The $PaCO_2$ of foetal blood is 55 mmHg. Much of the CO_2 and acids from foetal blood diffuses into maternal blood. Loss of CO_2 makes foetal blood more alkaline, while the increased CO_2 in the maternal blood makes this more acidic, shifting the ODC to right releasing O_2, making it available for foetus. Loss of CO_2 from foetal blood shifts the ODC to the left, enhancing the oxygen affinity of foetal Hb. Thus, the Bohr effect operates in one direction in the maternal blood and in the other direction in foetal blood. The addition of these two effects makes the Bohr shift twice as important here, as it is for O_2 exchange in the lungs. Therefore, it is called the 'Double Bohr effect'.

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3 Gate Control Theory of Pain

This theory which was put forward by Melzack and Wall in 1965 tries to correlate the physiological and psychological data regarding pain.

Briefing the physiology, we can see that pain is carried mainly by two types of fibres:

- C–fibres—These are nonmyelinated fibres which carry the sensation of diffuse pain.
- A delta–fibres—These are myelinated non-nociceptive fibres which carry the sensation of sharp localised pain.

The large myelinated A beta fibres which ascend mainly in the dorsal column carry the sensation of fine touch, the importance of which will be discussed later.

For a better understanding of the theory, the following simplified schematic diagram may be referred to (Fig. 3.1).

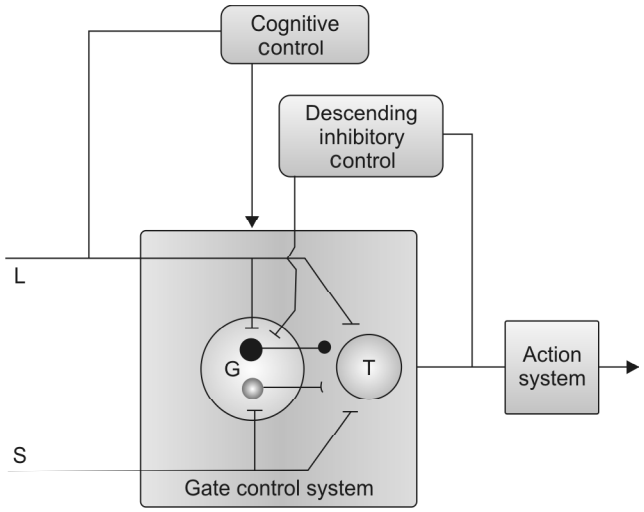

Fig. 3.1: The circuit diagram of the gate control theory of pain perception*
(as proposed by Melzack and Wall)

* The new model includes excitatory (white circle) and inhibitory (black circle) links from G cell or gate cell (originally thought to be cells of substantia gelatinosa) to the transmission (T) cells, as well as descending inhibitory control from brainstem. The round knob at the end of inhibitory link implies that its action may be presynaptic, postsynaptic or both. All connections are excitatory, except the inhibitory link from G cell to T cell.

Interpretation of the Figure 3.1 can be made with the help of the following table.

Afferent input	*I–cell effect*	*T–cell effect*	*T–cell output (T-I)*
M	+	+	0
U	•	+	+ +
M+U	0	+	+

A stimulus that activates only the M afferents has both a direct excitatory and an indirect inhibitory effect on the T–cell. There is no net increase in T–cell activity and no pain. A stimulus that activates only the U–afferents produce a very large increase in T–cell firing because there are both direct and indirect excitatory actions and no inhibition. But most stimuli activate both M and U afferents, producing intermediate levels of pain intensity.

So the inhibitory interneurons act here as a gate, between the M and U fibres and the transmission cell 'T'.

The above said explanations hold good for the fact that slowly rubbing an area (which stimulates A beta fibres and there would not be any T–cell output), after a painful stimuli, reduces the intensity of pain or rather gives a soothening effect. So also does the TENS (Transcutaneous Electrical Nerve Stimulator) which stimulates the A beta fibres.

In summary, it seems likely that, a relative loss of myelinated primary afferents contributes to the painfulness' of some nerve injuries. The loss of their inhibitory effect is associated with spontaneous pain, summation, due to the unopposed action of unmyelinated differents; and allodynia in which, surviving non-nociceptive myelinated axons, which usually inhibit main transmission cells now excite them, thus permitting solid stimuli to produce pain.

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4 Opiate Receptors

The concept of opioid receptors was developed in 1960s. Opiate receptors are found in various regions in the CNS. These include cerebral cortex, the limbic cortex (anterior and posterior amygdala and hippocampus), hypothalamus, medial thalamus, midbrain (periaqueductal grey matter), extra-pyramidal area (caudate, striatum and putamen), substantia gelatinosa and sympathetic preganglionic neurons. Gray matter has more receptors than white matter. Structures and pathways involved with pain contain the highest concentration of opiate receptors.

MECHANISM OF OPIOID ACTION

- Stimulation of stereospecific receptors on or near sodium channels in excitable cell membranes; which result in depression of active sodium conductance. This is the main mechanism.
- Opioid agonists produce a local anaesthetic like effect on the surface of excitable cell membranes that does not involve a stereospecific receptor.
- Opioid agonists block neuron excitability by a mechanism involving increased membrane potassium conductance or by blocking the opening of voltage.

These changes hyperpolarize cell membrane.

- Serotoninergic pathways may also in part modulate opioid mediated analgesia.
- Opioid effects may be elicited at GABA receptors which are closely associated with benzodiazepine receptors.

Tolerance is explained by the fact that it represents an uncoupling of the usual drug–receptor effect. This is probably by down regulation of the number of receptors and/or their affinity for agonists or by an uncoupling between the receptor and intracelluar second messenger.

The long duration of action of certain opioids beyond the production by plasma half-life may be due to the high affinity for the opiate receptor.

The cardiovascular system possesses opioid receptors (Table 4.1). They are situated on the heart, the cardiac branches of the vagus sympathetic nerves and central cardiovascular regulatory centers. Some of them are situated in adrenal medulla also.

Table 4.1: Characteristics of opioid receptors

Receptor		*Tissue bioassay*	*Agonist*	*Major actions*
MU	Mu4	Guinea pig ileum	Morphine, Mep-tazinol, phenyl piperidines.	Analgesia, Bradycardia, sedation.
	Mu2	Guinea pig ileum	Morphine, phenyl Piperedines	Respiratory depression,

Contd...

Contd...

Receptor	*Tissue bioassay*	*Agonist*	*Major actions*
			Euphoria, physical dependence.
Delta	Mouse vas deferens	Enkephalins de-Ala-d-Leu	Analgesic (weak), respiratory depression
Kappa	Rabbit vas deferens	Ketocyclazocine, Butorphanol	Analgesic (weak), Respiratory, depression, sedation.
Sigma		SKF 10, 047, Pentazocine	Dysphoria, delirium mydriasis, hallucination, tachycardia, hypertension
Epsilon	Rat vas deferens	B endorphin	Stress response acupuncture.

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5 Extracellular Fluid

INTRODUCTION

Extracellular fluid (ECF) is part of the internal environment of the body. About 56 to 60% of the adult human body is water. This fluid is distributed 2/3rd inside the cells and is called intracellular fluid (Table 5.1). One-third of the fluid is outside the cell and is called as 'extracellular fluid'.

Extracellular fluid is distributed as:

- Intravascular fluid (IVF)
- Interstitial fluid (ISF): Fluid between the cells and tissue spaces.

In the extracellular fluid are ions and the nutrients needed by the cells for maintenance of cellular life, and since the cells live in the EC fluid, EC fluid is called the internal environment or 'milieu interior' of the body.

Table 5.1: Distribution of body water

Water compartment	*% of body weight*	*Volume in litres*
Plasma water	4 %	2.8
Interstitial water	16 %	11.2
Total extracellular water	20 %	14
Intracellular water	40 %	28
Total average body water in man	60 %	42

RELATIVE SIZES OF BODY FLUID COMPARTMENTS

IVF 5 L
ISF 14 L } ECF
ICF 23 L Intracellular space

CONTENTS OF THE ECF

The ECF is made up of intravascular fluid (IVF) or blood and interstitial fluid (fluid in between cells and tissue spaces). Both ISF and IVF contain electrolytes dissolved in water (Table 5.2). But there are certain differences between ISF and IVF. IVF contain protein molecules, that, because they are contained within that space by the impermeable capillaries, exert a colloid osmotic pressure that tends to keep water within the vessels. Secondly, IVF is more amenable for measurement that ISF.

Table 5.2: The concentration of electrolytes in ECF

Cation	*mEq / L*
Na^+	142
K^+	5
Ca^{++}	5
Mg^{++}	3
	155 mEq / L
Anion	*mEq / L*
HCO_3^-	27
Cl	103
HPO_4^{3-}	2
SO_4^{2-}	1
Organic acid	6
	155 mEq / L

Osmotic pressure of the ECF is 280 to 310 Mosmols/L

DETERMINATION OF IVF

Intravascular fluid and circulating volume is readily amenable to clinical estimation but the ISF and the ICF cannot be measured directly. The parameters to assess the IVF are:

- PR—The pulse rate under the influence of ANS rises with hypovolaemia. A pulse rate of more than 100 beats/min. bears close correlation with significant intraoperative bleeding.
- BP
- Peripheral perfusion
- CVP
- Urine output
- PCWP

DETERMINATION OF INTERSTITIAL AND INTRACELLULAR SPACE

Clinical estimation of ISF and ICF is difficult and is not amenable to measurement and hence measurement is indirect and is estimated roughly by

- History: History of fluid losses in the form of GIT losses, lack of oral intake, fever.
- Assessment of fluid balance charts.
- Skin turgor: Skin turgor supposedly correlates with total body water. Whether loss of skin turgor indicates interstitial or intracellular water loss is less certain.
- Chest X-ray: Extravascular lung water (EVLW) probably representing the ISF of the lung correlates well with the degree of opacification of the chest X-ray. However, the origin of EVLW cannot always be determined by the chest X-ray appearance alone.

- Serum Na^+ : Assuming blood glucose is normal, the determinants of serum Na^+ are the exchangeable stores of sodium (Na_eL), the exchangeable stores of potassium (Ke) and TWB.

$$NaS \propto \frac{Na^+ + K^+}{TBW}$$

The most clinically significant of these is TBW. The serum Na^+ is inversely proportional to TBW. Most sodium disturbances in clinical situation are related to excessive or diminished water, rather than the absolute deficiency or excess of sodium. Serum Na is the most important clinical determination of TBW.

PRINCIPLES OF FLUID REPLACEMENT

Knowledge about the three body fluid compartment is important, because administration of fluid is based on clinical assessment of the body compartment.

Non-ionic Solutions

Water which has a solute that can be metabolised added to achieve iso-osmolality, is distributed proportionately over the three body spaces. Examples are dextrose containing solutions and amino acid solutions. Dextrose gets metabolised and water is freely permeable to all compartments. A very small percentage of these solutions will be distributed to the IVS and mostly will go into the ICS and a smaller volume to the ISS. Hence, they are not preferred for intraoperative use, because intracellular dehydration is not usually the problem. But TBW depletion may be considered in patients with long term fluid problems such a GI tract loss.

Whenever necessary, 5% dextrose solution is used to replenish the ICS and is titrated against the serum Na^+ in the absence of hyperglycaemia or factitious hyponatraemia. Intracellular dehydration is rarely a life threatening problem, and unless hypernatraemia is acute, it should be slowly corrected, enabling osmolar gradients between different fluid spaces to get adequate time to equilibrate different.

Ionic Solutions

They are the mainstay of perioperative fluid therapy. They are used for perioperative, intraoperative, postoperative fluid maintenance.

The Na^+ ion is confined mainly to the ECF by nature of the cell membrane and Na/K pump. Hence NaCl solutions and Ringer's lactate is confined to the ECF. But the ISF is 3 times the size of IVS and the 3/4th of the fluid administered is confined to the interstitial space and 1/3rd to the IVS. Thus isotonic saline is more efficient in resuscitation of the IVS than 5% dextrose which is confined to the ICF.

Blood Products and Colloids

Colloids or plasma substitutes are fluids and because they contain oncotic particles, they are confined mainly to the IVS.

Blood and blood products exert their large oncotic pressure through large protein molecules. Artificial colloid solutions contain large molecules, and as because they have an oncotic pressure greater than blood, ICF is mobilised from the ISS to the IVS, and plasma expands by a volume greater than infused fluid.

COMPLICATIONS OF OVEREXPANSION OF THE INTERSTITIAL SPACE

Since crystalloid solutions have become the mainstay of fluid in perioperative / intra/ postoperative period, it is common in the intraoperative phase to titrate a crystalloid solution which is essentially distributed to the ISS against measurements made of the IVS. The expansion of IVS may be exacerbated by further crystalloid solution, which is essentially distributed to the ISS in the face of neuroendocrine stress response which results in salt and water retention and over expansion of the ISF results.

This can results in:

- Interference of the lymphatic drainage by over expanded ISF and peripheral oedema.
- Decreased lung compliance and pulmonary oedema.
- Peripheral oedema can lead to decreased oxygen delivery to the tissue because the distance between the vessels and cell increases and also increased pressure in the ISS causes shutdown of capillaries.

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6 Effect of Hypothermia on Cardiorespiratory Physiology

Hypothermia is defined as core body temperature less than 35°C. Changes in the cardiorespiratory physiology varies with the degree of hypothermia. Okamura has classified hypothermia in Table 6.1.

Table 6.1: Classification of hypothermia

States	*Core temp. down to*
Mild	30° C
Moderate	30-25° C
Deep	25-20° C
Profound	20-10° C
Severe	less than 10° C

Changes in the Cardiovascular System

Change in Myocardial Function

A biphasic response is seen secondary to the level of circulating catecholamines; from 37° C to 29°C. There is an increase in the circulating catecholamine level. In the unanaesthetised heart, the sympathetic stimulation produces tachycardia, hypertension, increased stroke volume and cardiac output with cutaneous vasoconstriction.

Systemic vascular resistance increases up to 26° C. Anaesthesia can prevent this response. Below 26° C, blood pressure falls due to decrease in cardiac output which is due to fall in heart rate rather than a fall in stroke volume but later contractility of heart decreases and there is peripheral vasodilatation, the circulatory reflexes become inactive leading to a cardiovascular collapse at 20° C.

ECG shows bradycardia, prolonged PR, QRS, QT, intervals. At 30° C, positive deflection is seen on the down stroke of the R wave. This is best seen in lead V_2 . These are called J waves or 'Osborn waves'. There is irritability due to catecholamines. So that the chance of ventricular fibrillation must be below 28° C, and below 24° C there may be asystole. Ventricular fibrillation is possible with a temperatures less than 32° C.

The effect of hypothermia is augmented in the presence of acid-base imbalance, electrolyte imbalance, drugs, coronary, blood flow changes. ST and T wave changes are more below 25° C . The ventricular fibrillation which occurs may not respond to atropine, countershock or pacing and cardiopulmonary bypass is needed to resuscitate.

Changes in Peripheral Circulation

Above 25°C—Systemic vascular resistance increases due to increased catecholamine level, and cardiac output is maintained by increased contractility.

Below 25° C—Cardiac output decreases, leading to fall in BP.

Below 20° C

- Peripheral vasodilatation
- Circulatory reflexes inactive.
- Cardiovascular collapse.

The plasma volume decreases by 25% for 11°C drop in body temperature, while plasma protein concentration is unchanged. This is because:

- There is intravascular sequestration of plasma.
- Increase in transcapillary leak into the extracellular fluid, so there is haemoconcentration leading to increase in blood viscosity.

As the temperature falls below 30°C, there is decrease in velocity of cells and intravascular aggregate formation. Affection of microvasculature produces areas of oxygen debt.

Changes in Respiratory System

If the patient is on spontaneous respiration, minute volume decreases and ventilation ceases at 23°C. Bronchodilatation produces increase in anatomical and physiological dead space. Suppression of cough reflex produces aspiration.

Controlled ventilation results in respiratory alkalosis due to reduced production of carbon dioxide and increased solubility of carbon dioxide. Hypoxic pulmonary vasoconstriction produces increase pulmonary vascular resistance. Hypothermia reduces the MAC of volatile agents, the degree of reduction varies for each agent. Though oxygen solubility is increased, oxygen delivery to tissues is reduced due to shift of ODC to the left and increased blood viscosity. Oxygenation can be improved by:

- Controlled ventilation
- Haemodilution
- Controlled hypothermia.

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7 Regulation of Cerebral Blood Flow

INTRODUCTION

The arterial supply to the brain is derived from the internal carotid and vertebral arteries on each side; two-thirds of the supply come from the carotid vessels. The left common carotid artery arises directly from the aorta, whereas the right is a branch of the innominate artery. The vertebral vessels arise from the subclavian artery on their respective sides and join together at the lower border of the pons to form the basilar artery. The Circle of Willis is formed by the anastomosis between the terminal branches of the basilar and two internal carotid arteries.

Adult human brain receives 12 to 15% of the cardiac output 750 ml/min. This high flow is a reflection of the brain's high metabolic rate.

Whole brain oxygen consumption is about 20% of the total body oxygen utilization.

Normal CBF: 50 to 55 ml/100 gm/min.

Physiological control of cerebral circulation is maintained by a precise interaction of myogenic, metabolic or chemical, thermoregulatory and neurogenic vascular response.

CHEMICAL REGULATION OF CBF

Factors that cause changes in the cerebral biochemical environment that result in adjustments of CBF are:

- Cerebral metabolic rate.
- $PaCO_2$ and
- PaO_2

Cerebral Metabolic Rate

Is usually expressed in terms of oxygen consumption ($CMRO_2$), which averages 3-3.5 ml/100 gm/min. CMR is influenced by:

- Functional state of the nervous system. CMR decreases during sleep, coma and deep levels of barbiturates and althesin hypnosis. CMR increases during sensory stimulation, mental tasks, arousal from any cause or during epileptoid activity.
- Anaesthetic drugs—Intravenous anaesthetics, in general, with exception of ketamine, suppress CMR inhalational anaesthetics increase CBF, but reduces CMR.
- Temperature—CMR and CBF are temperature dependant and change by about 7% per degree celsius change. CBF falls as temperature decreases and is related to increased blood viscosity and decreased cardiac output.

  CMR O_2 is reduced and tissue PCO_2 reduces which also lowers CBF.

Hyperthermia does not increase CBF until the temperature exceeds 39.5°C. Further increases in temperature increase CBF by 30 to 50% at 42°C. At higher levels both CBF and CMR decline rapidly.

$PaCO_2$

Arterial carbon dioxide tension is the most important single factor controlling CBF. CBF varies directly with $PaCO_2$ variation. CBF changes 1 to 2 ml / 100 gm/min for each 1 mmHg change in PCO_2 values. The response is attenuated below a PCO_2 of 25 mmHg and PCO_2 above 60 mmHg.

Increasing arterial CO_2 tension increase flow whilst reduction in arterial CO_2 tension reduced CBF. Response of CBF to CO_2 is diminished in presence of hypotension and arterial hypoxaemia and abolished when system blood pressure reaches 50 mmHg. Prolonged maintenance of arterial CO_2 at abnormal levels is associated with a return of CBF to normal levels.

Mechanism: The changes in CBF caused by $PaCO_2$ depend on the pH alterations in the ECF of the brain. If arterial CO_2 tension is kept constant, marked changes in arterial pH produced by infusing acids or alkalies do not affect CBF. Then the blood-brain barrier protects CBF from the effects of metabolic acidosis and alkalosis.

PaO_2

The effect of alterations of PaO_2 on CBF are less when compared to that of $PaCO_2$ changes. The effects of changes in arterial oxygenation on CBF are opposite to those of carbon dioxide. However, the cerebral circulation does not respond to PaO_2 changes around the normal values. When PaO_2 falls below 50 Hg CBF increases and is doubled at a PaO_2 of about 30 mmHg. Cerebral vasodilatation is due to local tissue acidosis causing fall in tissue pH and affecting smooth muscles of cerebral arterioles.

At higher oxygen tension, CBF decreases. At 1 atmosphere of oxygen, CBF is reduced by 15%. The fall in CBF due to hyperoxia is not associated with fall in cerebral available oxygen.

MYOGENIC REGULATION (AUTOREGULATION)

Definition

Intrinsic capacity of the cerebral adjust its resistance to maintain CBF constant over a wide range of mean arterial pressure. Normally, the limits of autoregulation are approximately 50 to 150 mmHg. Mean arterial pressure above and below this level, there is a passive relationship between CBF and perfusion pressure. Even within the range over which autoregulation normally occurs, a rapid change in arterial pressure will result in a transient alteration of CBF.

$$\left.\begin{array}{l}\text{Cerebral perfusion pressure (CPP)}\\ \text{in persons with normal ICT}\end{array}\right\} = \text{Mean arterial pressure} - \text{Central venous pressure.}$$

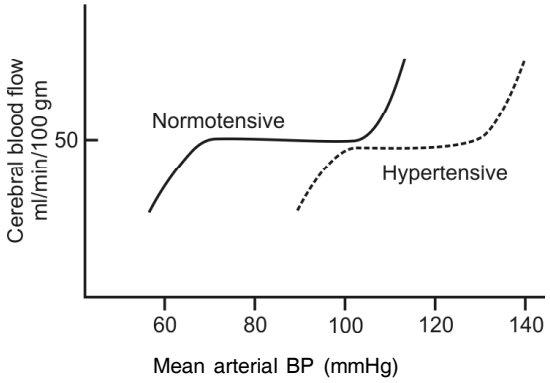

Fig. 7.1: Autoregulation of CBF

Mechanism

Stretch induced myogenic response of arteriolar smooth muscles. Autoregulation is influenced by various pathologic processes (e.g. trauma, arterial hypoxaemia, hypercapnia and deep general anaesthesia) and by the time course over which CPP changes occur.

Abrupt increase in arterial pressure above the upper limit of autoregulation will cause a considerable increase in CBF (Fig. 7.1). Chronic arterial hypertension is associated with a shift to the right of the autoregulation curve. The chronic hypertensives may be able to maintain a normal CBF with mean arterial pressure in a hypertensive subject will cause ischaemic symptoms at higher levels of arterial pressures than occurs in normotensive subjects. Adaptation of autoregulation to hypertension takes one or two months to become established. Effective antihypertensive therapy results in the autoregulatory limits returning to normal values.

CPP in patients with raised ICT = MAP–ICT

ICT below 50 cm H_2O does not reduce CPP below the lower limit of autoregulation and CBF is usually unaffected. Above 50 cm H_2O, marked fall in CBF will occur. Autoregulation may be grossly upset in pathological brain tissue; e.g. tumour, infarct, oedema, head injury, etc. Here flow becomes "pressure dependent".

NEUROGENIC REGULATION

The cerebral vasculature is well supplied with both sympathetic and parasympathetic nerves. The density of innervation declines with vessel size and the greatest neurogenic influence is exerted upon larger cerebral vessels. Therefore the neurogenic regulation is more important in the CBF regulation of large brain areas than in precise local CBF modulation. Maximal sympathetic stimulation decreases CBF by 5 to 10% and parasympathetic stimulation increases CBF. Sympathetic stimulation shift the autoregulation curve to the right and this offers some protection against hypertensive breakthrough of the blood-brain barrier (BBB). In haemorrhagic hypotension, CBF is reduced.

BLOOD VISCOSITY

Blood viscosity can influence CBF. In healthy persons, haematocrit variation within the normal range results in only trivial alteration of CBF. Beyond this range, changes may be more obvious. In polycythaemia vera, increase in cerebrovascular resistance due to increased blood volume can reduce CBF to half the normal values.

In anaemia cerebrovascular resistance is reduced and CBF increases. This is due to direct reduction in viscosity and compensatory increase to match decreased oxygen carrying capacity of blood. In the absence of cerebral complications due to altered blood viscosity, CMR probably does not change.

EFFECTS OF ANAESTHETIC DRUGS ON CBF AND CMR

- **Intravenous induction agents:**
  - Barbiturates, althesin, propofol, etomidate—reduction in CBF
  - Ketamine—rise in CBF
- **Inhalational anaesthetics:** Inhalational anaesthetics increase CBF.
  This is associated with a reduced cerebral oxygen uptake and an increase in cerebral venous oxygen content.
  - Halothane, methoxy flurane, trichloroethylene, enflurane Isoflurane all increase CBF and CMR
  - Cyclopropane Low concentration—decrease in CBF
    High concentration—increase in CBF
- **N_2O:** In normal persons reduces cerebral oxygen uptake without affecting CBF. In patients with increased ICT it increases ICT due to cerebral vasodilatation.
- **Narcotics:** Narcotics have very little effect on CBF and CMR in the normal unstimulated nervous system. When changes occur, generally there will be reduced CBF and CMR.
- **Benzodiazepines:** Decreased CBF and CMR.
- **Muscle relaxants:** Succinyl choline–increase in ICP–transient and easily attenuated; not clinically problematic.
  - Nondepolarising muscle relaxants–the direct effect of these drugs on cerebral vasculature occurs via the release of histamine.
  - Histamine can result in a reduction in CPP.
  - D–Tubocurarine–Most potent histamine release.
  - Analeptic drugs–Increases CBDF.
  - Cerebral vasodilators–increases CBF.

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8 Cerebral Steal

The adult brain weighs about 1350 gm, which is 2% of total body weight. It receives 12 to 15% of cardiac output. At rest $CMRO_2$ is 3.5 ml O_2 / 100 gm of brain tissue.

Autoregulation refers to the intrinsic capacity of the cerebral circulation to adjust its resistance to maintain CBF constant over a side range of mean arterial pressures (MAP). In normal subjects, the limits of autoregulation are approximately 50 and 150 mmHg. Above and below this range, CBF is pressure dependent (pressure passive) and varies linearly with cerebral perfusion pressure. Autoregulation is influenced by various pathological processes and in addition by the time course over which CPP changes occur. The precise mechanism by which autoregulation is accomplished is unknown. It appears to be an intrinsic characteristic of cerebral vascular smooth muscle. $PaCO_2$ and regulation of cerebral blood flow.

CBF varies directly with $PaCO_2$. The effect is greatest within the range of physiologic $PaCO_2$ variation (Fig. 8.1). CBF changes 1 to 2 ml/100 gm/min, for each 1 mmHg change in $PaCO_2$ around normal $PaCO_2$ values. The response is attenuated below a $PaCO_2$ of 25 mmHg.

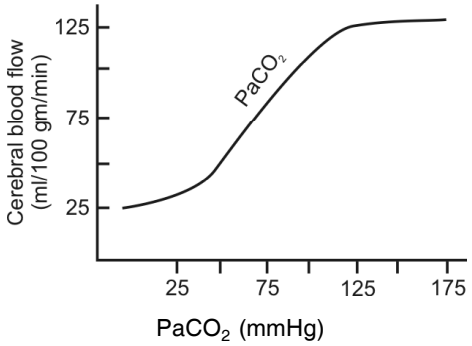

Fig. 8.1: The effect of $PaCO_2$ on CBF

Under normal circumstances, CBF sensitivity to changes in $PaCO_2$ appears to be positively correlated with resting levels of CBF. Accordingly, anaesthetics that alter resting CBF cause change in the response of the cerebral circulation. The changes in CBF depend on pH alterations on the ECF of the brain. The changes in ECF pH and CBF occur rapidly after $PaCO_2$ adjustment because CO_2 diffuses freely across the cerebrovascular endothelium. In contrast to a respiratory acidosis, acute systemic metabolic acidosis has little immediate effect on CBF because BBB excludes H^+ ion from the perivascular space. Although the CBF change in response to a $PaCO_2$ alterations occur rapidly, they are not sustained. CBF returns to normal over 6 to 8 hr, because CSF pH gradually normalizes as a result of the extrusion of bicarbonate.

As $PaCO_2$ is reduced by hyperventilation, CBF is decreased by cerebral vasoconstriction as a direct response to the reduction in H^+ ion concentration in CSF. When $PaCO_2$ is reduced below 30 mmHg, CBF continues to decrease but not so markedly, at levels below 26 mmHg, EEG slow wave actually increases and below 23 mmHg, the vasoconstriction may be so intense that cerebral hypoxic may follow. Hypoxia may act as a greater stimulus and lead to vasodilatation.

Opposite effects occur when $PaCO_2$ increases during hypoventilation with a reduction in this response. When $PaCO_2$ levels exceed 50 mmHg the cerebral blood volume increases and formation of oedema may occur.

Over the central part of the response graph, CBF increases or decreases by 10 ml/100 gm/min for each 1 ka (7.5 mmHg) change in $PaCO_2$.

The normal responsiveness of cerebral vessel to $PaCO_2$ is lost in areas of brain where autoregulation has ceased. The vessels in these areas behave passively and so, when vasoconstriction produced in normally responding cerebral vessels by hyperventilation, the majority of the CBF is channelled through the unresponsive vessels resulting in and increase of CBF through this area. This is known as the inverse steal effect when blood is channelled from the healthy areas of brain to pathological zones. The opposite effect occur in hypoventilation, resulting in a decrease in CBF through pathological areas of the brain–cerebral steal syndrome.

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9 Microcirculation in Shock

Shock may be defined as an abnormal state of the circulation in which cardiac output is reduced to the extend that the tissues of the body are damaged for lack of adequate tissue blood flow.

CLASSIFICATION OF SHOCK

- Cardiogenic
  - Myopathic (reduced systolic function)
    i. Acute myocardial infarction
    ii. Dilated cardiomyopathy
    iii. Myocardial depression in septic shock
  - Mechanical
    i. Mitral regurgitation
    ii. VSD
    iii. Ventricular aneurysm
    iv. Left ventricular out flow obstruction.
  - Arrhythmia
- Extracardiac obstruction
  - Pericardial tamponade
  - Constrictive pericarditis
  - Pulmonary embolism (massive)
  - Severe pulmonary hypertension (primary or Eisenmenger)
  - Coarctation of the aorta.
- Oligaemic
  - Haemorrhagic
  - Fluid depletion.
- Distribution
  - Septic shock
  - Toxic products
  - Anaphylaxis
  - Neurogenic
  - Endocrinologic.

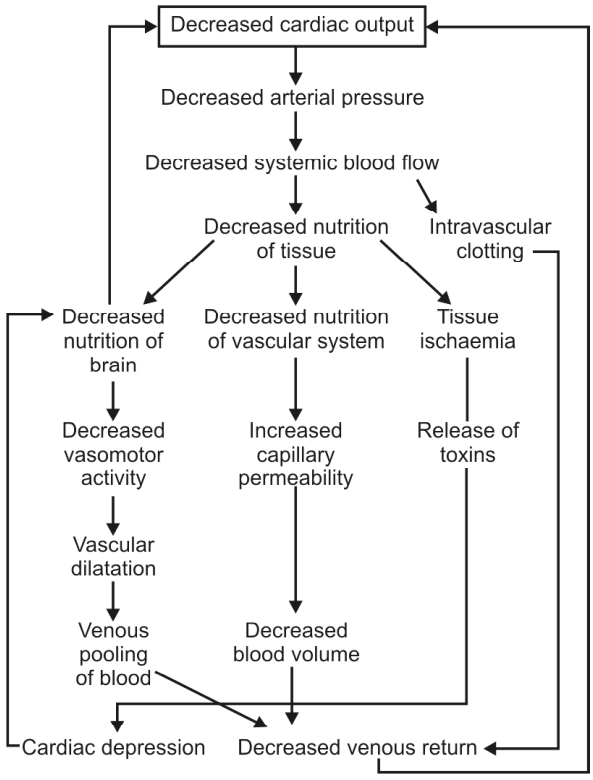
THE STAGES OF SHOCK

- Non-progressive stage (Compensated stage): Compensatory mechanism will eventually cause full recovery without any help from outside therapy.

- Progressive stage: Shock become steadily worse until death.
- Irreversible stage: Shock has progressed to such an extent that all forms of known therapy will be inadequate to save the life of the person eventhough for the moment the person is still alive.

The different types of feedback that can be lead to progression of shock as shown in Flow chart 9.1.

Flow chart 9.1: Different types of feedback that can lead to progression of shock



MICROCIRCULATION

Structure

The transport of nutrients to the tissue and removal of cellular excreta occurs in the microcirculation. The microcirculation of each organ is specifically organized to serve, that organ's own special needs. In general, each nutrient artery entering an organ, branches six to eight times before the arteries become small enough (20 μm diameter) to be called arterioles. Arterioles themselves branch two to five times (5-9 μm diameter) at their ends where they supply blood to the capillaries.

Blood does not flow continuously through the capillaries. Instead it flows intermittently turning on and off every few seconds or minutes. Intermittent contraction of the metarterioles and precapillary sphincter occurs. The most important factors found to affect the degree of opening and closing of the metarterioles and precapillary sphincter is the concentration of oxygen in the tissue. When the rate of oxygen usage is great, the intermittent periods of blood flow occurs more

often and duration of each period of flow lasts for a longer time; thereby allowing the blood to carry increased quantities of oxygen to the tissues.

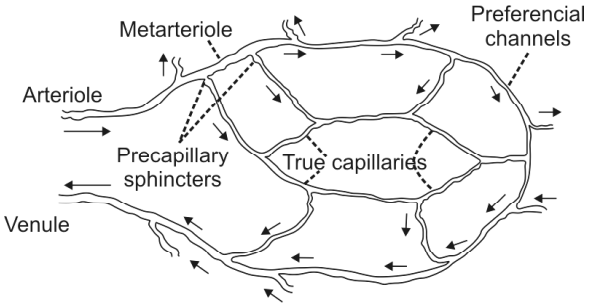


Fig. 9.1: Structure of mesenteric capillary bed

MICROVASCULATURE IN SHOCK

The disruption of microvasculature appears to be a final common path in all shock states. The resistance that various vessels offers to the blood flow resides in three sites.

- The arterioles
- Precapillary sphincter
- Postcapillary sphincter.

The sphincters perform a further function. By their activity relative to each other. They govern the hydrostatic pressure within the capillary itself, hence, they govern the direction of net flow across the capillary wall between the plasma and the extracellular fluid.

The sphincters are influenced both by sympathetic activity and by local metabolites, pH and hypoxia. For the same catecholamine concentrations, precapillary sphincter tone is greater than postcapillary. In this way capillary pressure is reduced and this facilitates the inflow of fluid into the intravascular space with augmentation of blood volume. This response is of value in early shock where circulating volume is maintained although the haematocrit is reduced. Metabolic acidosis, and hypoxia relax the precapillary more than the postcapillary sphincter. In the late stages of haemorrhagic shock, the resistance of the post capillary sphincter exceeds that of the precapillary sphincter—the direction of fluid flow is reversed and interstitial oedema occurs. This may explain the change in fluid shift seen in irreversible shock and the capillary pooling and stagnation seen particularly in the splanchnic beds of the experimental animals. It also explains the observation that arterial pressure cannot be maintained in irreversible shock despite retransfusion and overtransfusion.

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10 Supine Hypotensive Syndrome

In the majority of pregnant women assumption of the supine position results in a reduction in the cardiac output without a reduction in arterial blood pressure. This fall in cardiac output is due to decreased venous return to the heart, which is inturn caused by the compression of inferior vena cava by the gravid uterus.

Compensatory mechanisms that act to maintain arterial blood pressure are as follows:

- Diversion through the paravertebral venous plexus of some amount of blood from the venous system caudad to the site of compression
- Reflex peripheral vasoconstriction.

Though the arterial BP is maintained, uterine blood flow decreases. The mother seldom experiences any symptoms and this is called concealed caval occlusion.

However, in 8–15% pregnant women compensatory mechanisms fail and these women experience symptoms of caval compression. This is termed *Overt caval compression* or *supine hypotension syndrome*. The cause of this syndrome is the total or near total obstruction of IVC with a marked reduction in cardiac output and blood flow to the vital centres in the medulla.

CLINICAL FEATURES

Cardiac output may fall by 50%. The syndrome is characterised by nausea, vomiting, sweating, pallor, bradycardia, hypotension and shock. Hypotension may occur immediately in severe cases with the potential for cardiovascular collapse, but usually hypotension does not develop for 5 to 10 minutes after gravidas assume the supine position.

Primigravidas with strong abdominal muscles and tightly drawn abdominal skin (billiard board appearance) or gravidas with large uteri (multiple pregnancy, hydramnios) or obesity are particularly susceptible to supine hypotension, and often will not voluntarily assume the supine position. Hypovolaemia, dehydration, bleeding and inhibition of maternal compensatory mechanisms by the vasodilatory effects of narcotics, sedatives, metabolic acidosis, general anaesthesia and especially sympathetic blockade with regional anaesthesia increase susceptibility to supine hypotension.

EFFECTS ON FOETUS

Marked decrease in uterine blood flow which accompanies the hypotension may seriously compromise foetal well-being. Foetal hypoxia or acidosis may occur which will manifest as changes in the rate and rhythm of the foetal heart. Intervillous blood flow decreases by 20% in the supine compared to a patient in 15° left lateral tilt. Foetal PO_2 is lower if the mother is supine during the second stage and effective pelvic tilt will counter this.

PREVENTION

Aortocaval compression can be prevented by a complete avoidance of the unmodified supine position after about 30 weeks of pregnancy. Complete lateral position is preferable. Excessive flexion of the spine around a gravid uterus in the lateral position may result in severe venacaval obstruction.

When complete lateral position is not practical, then lateral uterine displacement (usually to the left) should be achieved by placing wedge under the appropriate block to till the pelvis or by tilting the whole operating table. There are also mechanical devices which have been especially designed to produce uterine displacement. Other methods to prevent supine hypotension syndrome before establishing subarachnoid block for caesarean: oxygenation, pre-load 2000 ml 20 min.

Ephedrine 5 mg IV / mephenteramine 5 mg IV if BP falls by 15% of original value or less than 100 mmHg.

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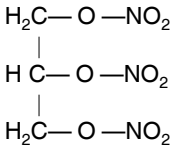
11 Nitroglycerine

INTRODUCTION

Nitroglycerine is an organic nitrate discovered by Sobrero in 1846, that acts principally on venous capacitance vessels to produce peripheral pooling of blood.

Chemical Structure



Pharmacokinetics

Organic nitrates are metabolised by the liver through reductive hydrolysis catalysed by enzyme organic nitrate reductase. This concerts the lipid soluble nitrates esters into water soluble denitrated compounds which are excreted through kidney.

Denitrated compounds have half-life of 40 minutes and have potent 1/10th that of parent compound. Since nitroglycerine given intravenously has half-life of 1 to 2 min. Nitroglycerine IV must be given as infusion.

Pharmacodynamics

Nitroglycerine is principally a venodialator. This causes a decrease in right and left end diastolic pressure and thus reduces the left ventricular wall tension, thereby reducing the myocardial oxygen demand.

Nitroglycerine also produces coronary vasodialation and causes redistribution of coronary blood flow to ischaemic area and relieves coronary spasm. Nitroglycerine thus decreases the myocardial oxygen demand and increases the myocardial oxygen supply.

Mechanism of Action

Organic nitrates acts by producing nitric oxide. Nitric oxide activates the enzyme, guanylate cyclase causing increase in the concentration of cyclic GMP which is a potent vasodialator of both veins and arteries.

Therapeutic Uses

- Treatment and prophylaxis of angina pectoris. Dosage in angina pectoris
  - Nitroglycerine buccal tablet 1 mg every 3 to 5 hours.
  - Nitroglycerine capsule 2.5 mg to 9 mg qid.
  - Nitroglycerine 0.3 mg stat for acute attack of angina pectoris.
  - Nitroglycerine in the dose of 0.5 to 2 mcg/kg/min for prevention of intraoperative myocardial ischaemia.
- Nitroglycerine is also used in the treatment of coronary spasm.
- Nitroglycerine infusion is used to atteruate the pressure response to laryngoscopy and intubation.
- Nitroglycerine infusion is used to treat hypertensive episodes during surgery.
- Used in controlled/deliberate hypotensive anaesthesia.
- Used in acute left heart failure to decrease preload and pulmonary vascular congestion and also to decrease the adrenergic tone and peripheral vascular resistance.
- To relax the uterus during manual removal of placenta.

Disadvantages/Demerits

- Reflex tachycardia
- Headache
- Postural hypotension
- Tolerance.

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12 Beta-blockers and Anaesthetic Implications

INTRODUCTION

In 1906, Dale showed with his work on ergot alkaloids that, sympathetic nervous activity was mediated by two different receptors, and Ahlquist named them alpha and beta receptors. The adrenergic receptors are now identified as alpha, beta, beta 2 and dopamine receptors.

CLASSIFICATION

They are categorised into those with membrane stabilising effect and those with intrinsic sympathetic activity.

The beta-blockers with intrinsic sympathetic activity produces a small agonist effect of their own which provides some protection in patients requiring beta blockade in the presence of bronchospasm.

Propranolol which is a non selective beta-blocker has membrane stabilising effect that is a local anaesthetic like action. It reduces the rate of rise of action potential without affecting the resting potential.

BETA BLOCKING DRUGS

- Nonselective blockers.
  - With ISA (Intrinsic sympathetic activity): Pindolol.
  - With MSE (Membrane stabilising effect): Propranolol, sotolol, timolol.
  - Both ISA and MSE: Alprenolol, oxyprenolol.
  - Neither ISA or MSE: Nadolol.
- Cardioselective blockers.
  - With ISA: Practolol, acebutolol
  - With MSE: Acebutolol
  - Neither ISA or MSE: Atenolol, metoprolol, esmolol

PHARMACODYNAMICS

The pharmacological effect of β blockers, depends both on the degree of block produced and type of receptor blocked.

They produce:

- Reduction in cardiac contractility and excitability. (Hr. so cardiac output)
- Central reduction in vasomotor activity
- Reduction in activity of renin angiotensin system
- Atrio-ventricular conduction defects are worsened, but dysrhythmias are suppressed.

The clinical effects observed depend on the level of sympathetic activity prior to the administration of the drug, for example. In thyrotoxicosis, anxiety states and pheochromocytoma, the circulation can be markedly improved by β receptor blockade.

PHARMACOKINETICS

Drugs with high lipid solubility such as propranolol and alprenolol cross the blood-brain and placental barrier and produce central nervous system effects. Atenolol does not cross blood-brain barrier. So sedative effects are almost nil.

INDICATIONS FOR BETA-BLOCKERS

Cardiovascular

- Hypertension
- Angina
- Sinus tachycardia and paroxysmal atrial tachycardia
- Coronary artery disease
- Dissection aorta
- Hypertrophic obstructive cardiomyopathy
- Digitalis toxicity
- QT–prolongation syndrome
- Cyanotic spells in tetralogy of Fallot.

In Hypertension BP is Reduced by

- Reducing cardiac output
- Reducing release of renin and so reduces sodium and water retention.
- Blocks pre-synaptic β receptors and reduces catecholamine release.
- Resets baroreceptor mechanism
- Reduces vasomotor tone
- Reduces plasma volume
- CNS effect.

Anti-arrhythmic Effect due to

- Reduced sensitivity to catecholamines
- Reduced binding of catecholamines
- Membrane stabilising effect
- Reduces myocardial oxygen requirement.

In Hypertrophic Obstructive Cardiomyopathy

- It reduces contractility and prevents increase in outflow obstruction.
- It reduces myocardial oxygen consumption.

Angina

- Reduces heart rate, so more time for coronary perfusion.
- Decrease exercise induced rise of BP and cardiac contraction.
- Counters tachycardia produced by nitrates.

Cyanotic Spells

Prevents infundibular spasm. Propranalol–dose 0.1 mg / kg I / V during spell followed by 1 mg/kg 4 to 6 hourly orally.

ANAESTHETIC INDICATIONS

- In premedication in a patient on β blocker therapy.
- Whenever induced hypotension is planned blocker gives better control of heart rate.
- Obtund hypertensive response to laryngoscope and intubation.
- Thyrotoxic crisis (Propranolol 1 –0.1 mg/kg I/V maximum up to 2 mg I/V). It reduces peripheral conversion of T4 to T3 and reduces sensitivity of heart to catecholamines.
- Anthiarrhythmic
- Phaeochromocytoma—to counter tachycardia alpha blocker.

NEUROPSYCHIATRIC INDICATION

- Prophylaxis of migraine
- Essential tremor
- Anxiety
- Alcohol withdrawal syndrome.

GLAUCOMA

Example: Timolol

Two new beta-blockers currently used are esmolol which is a B_1 cardioselective and flastolol which is nonselective. Esmolol is effective in quick control of supraventricular tachyarrhythmias in patients with acute myocardial infarction where it reduces heart rate and myocardial oxygen demand. It is also used to control perioperative hypertension with tachycardia. Dose of esmolol 50 to 200 mcg/kg/min. Esmolol has a rapid onset (within 20 seconds) and short duration of action (9 to 10 min).

CONTRAINDICATIONS TO BETA-BLOCKER THERAPY

- Congestive cardiac failure
  - Depends on sympathetic drive which is obtunded by beta blockers, causing—precipitous fall in cardiac output.
- Bronchial asthma

- Heart block
- Insulin dependent diabetes mellitus
  (Masks hypoglycaemic attack)

Abrupt withdrawal of Beta blockers produces rebound increase in heart rate or blood pressure, due to increased concentration of β receptors (upgrading of B receptors due to chronic use).

Adequate Beta-blocker Therapy

- Heart rate 50-60/min.
- Less than 20% increase with stress. (HR)
- No evidence of CCF or AV heart block.

Withdrawal symptoms appear in 24 hr, and so return patient as early as possible to β blocker therapy. It oral intake is not possible continuous infusion of propranolol equal to about 10% of the patients preoperative daily maintenance dose has been recommended.

ANAESTHETIC CONSIDERATIONS IN PATIENTS ON BETA-BLOCKER THERAPY

Beta-blockers do not affect the serum electrolytes, particularly serum potassium/serum uric acid levels, but glucose tolerance deteriorates in some non-insulin dependent diabetics.

DRUG INTERACTIONS

Drug	*Possible effects*
Tubo-curarine	– Enhanced neuromuscular blockade.
Phenothiazines	– Additive hypotensive effects.
Lignocaine	– Propranolol treatment increases blood levels of lignocaine.
Digitals	– Potentiation of bradycardia
Aminophylline	– Mutual inhibition
Antidiabetic agent	– Enhanced hypoglycemia
I/V Heparin	– Increased unbound form of propranolol.

Volatile anaesthetic agents: Causes dose related cardiovascular depression. The additive cardiovascular depression is greatest for enflurane, least with isoflurane and intermediate for halothane. Trichloroethylene, ether and methoxyflurane should be avoided.

Muscle Relaxants

Pancuronium is ideal because the increase in heart rate produced by pancuronium is attenuated.

The muscarinic effect of succinylcholine is exaggerated by beta adrenergic receptor blockade producing bradycardia. So also fentanyl.

Reversing of neuromuscular blockade with neostigmine should be avoided if the intraoperative bradycardia cannot be increased with atropine.

Precautions

Anaesthetic implications in a beta-blocked patient.

- Normal sympathetic response to blood loss, acute hypovolaemia may be obtunded: simultaneous use of calcium channel blockers cause additive myocardial depression (other already mentioned).
- Fluid overload should be borne in mind when large volumes of fluid are infused and hence CVP monitoring essential.
- Bradycardia following acute blood loss should be anticipated.
- Hypercarbia should be avoided as it may cause hypotension because the vasodilatation produced by carbon dioxide is no longer offset by tachycardia due to activation of the adrenergic system.

Reversal of Beta-Blockade

Beta-blocked patient is still capable of cardiovascular stimulation by administering larger dose of sympathomimetic drugs on the grounds that clinically beta blockade is not complete.
The perioperative bradycardia or hypotension can be antagonised with:

- Atropine–dose 0.4 to 0.6 mg/kg up to 2 to 3 mg intravenously.
- Isoprenalin–specific antagonist for beta adrenergic blocking drugs. It is given as an infusion with a dose of 2 to 5 mcg/min adjusting the rate to achieve the desired levels of blood pressure and heart rate.
- Calcium chloride–increases myocardial contractility. Dose: 500 to 100 mg over 10 to 20 min.
- Aminophylline–5 mg/kg/IV, inhibits phosphodiesterase, increases the cyclic AMP course.
- Glucagon 5-10 mg I/V
- Cardiac pacing.

Treatment of Anaesthetic Induced Hypertension

Select alpha + Beta-blocker like:

Labetalol

Beta-blocker with alpha blocking properties. Because of its alpha blocking properties, hypotensive response is more pronounced.
Anaesthetic implications:

- Has additive action with halothane
- Useful in achieving controlled hypotension
- Decreases cardiac output and peripheral vascular resistance
- Used in pregnancy induced hypertension
- Decreases cardiac output and peripheral vascular resistance
- Used in pregnancy induced hypertension
- Effective in controlling blood pressure in hypertensive crisis
- Useful in controlling blood pressure in patients with phaeochromocytoma

Dosage preparation comes as 0.5% solution incremental doses of 5 to 10 mg bolus in an adult produces adequate effect infusion dosage ranges from 25 to 100 mg/hr.

Esmolol

- Intravenous short acting cardiovascular β_1-blocker.
- Reduces HR. Reduction in blood pressure is related to drop in HR.
- Rapid onset of action
- Half-life 9 minutes
- Metabolised by RBC—Cholinesterase
- Safe in patients with hepatic of renal dysfunction.
- Dose–50 to 200 mcg/kg/min.

Uses

- To abolish hypertensive response to laryngoscopy.
- Adjunct to nitroprusside induced hypotension.
- Postoperative hypotension in coronary artery bypass graft patients.
- For by pass surgery on beating heart.
- Supraventricular arrhythmias.

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13 Calcium Channel Blockers

Calcium is involved in the excitation of myocardial cells, genesis of action potential, the coupling of electrical activation to myocardial contraction and constrict vascular smooth muscles (Fig. 13.1).

ROLE OF CALCIUM IN CARDIAC CELL ACTIVITY

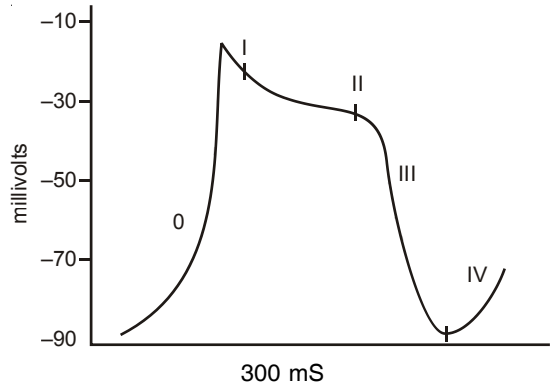

Fig. 13.1: Phases of the action potential of a cardiac muscle fibre

Phase 0: Is the rapid phase of depolarisation during which the resting membrane potential rises from –90 mV to +20 mV. This is produced as a result of rapid influx of Na^+ into the cell which lasts for 2-20 mS. Referred to as fast Na^+ channel.

Phase 1: Continued depolarisation phase. This is due to deactivation of Na^+ current.

Phase 2: Plateau phase when the action potential rise above –40 mV, there is activation of slow calcium channel resulting in calcium influx and K^+ efflux.

Phase 3: Inactivation of slow Ca^{++} channel and outflow of K^+

Phase 4: Resting phase. Due to activation of outflow of Na^+ and inflow of K^+ ions

The cardiac electrical activity is intimately associated with mechanical contraction. Excitation contraction coupling results in calcium influx which triggers off the sarcoplasmic reticulum to bring the intracellular calcium ion concentration to critical level needed to bind sufficient troponin. This allows actin and myosin to interact to shorten the sarcomere. In the vascular smooth muscle this is mediated by calmodulin instead of troponin.

GODFRANID CLASSIFICATION OF CA CHANNEL BLOCKERS

- Selective calcium entry blockers of myocardium
  - Verapamil
  - Nifedipine
  - Diltiazem
  - Nimodipine
  - Nicardipine
- Selective slow channel blockers with its action mainly on arteries.
  - Cinnarizine
  - Flunarizine
- Nonselective blockers
  - Lidoflazine
  - Frenylamine
- Agents having action on calcium channels, but primary action at different sites
  - On sodium channel, e.g. local anaesthetics, phenytoin
  - Catacholamine receptors, e.g. phenoxybenzamine, propranolol
  - Opiate receptors, e.g. loperamide, fleperamides
- Agents act within the cell
  - At sarcoplasmic reticulum, e.g. dantrolene
  - At mitrochondria, e.g. ruthenium red
  - Calmodulin antagonists, e.g. dibucaine

Mechanism of Action

Blockade of the slow Ca^{++} channels

- *Myocardial contractility:* Ca^{++} blockers inhibit the myocardial contractility by direct inhibition of excitation contraction coupling. Verapamil produces more depression of cardiac contractility than nifedipine.
- *Refractory period:* Prolongs absolute refractory period of AV node thereby prevents arrhythmia. Verapamil has more refractory period prolonging effect; hence, heart blocks are common with verapamil.
- *Arteries and arterioles:* Produces dilatation of arteries and arterioles. Nifedipine and diltiazem produce more vasodilatation than verapamil (Table 13.1).

PHARMACOKINETICS

Well absorbed from GIT. Undergoes first pass metabolism in the liver (reason for higher oral dose than parenteral dose) 90% is bound to plasma proteins. Predominantly excreted through the kidneys. Onset of action of IV calcium channel blockers is 2-5 min. After oral dose onset of action occurs within 20 to 30 min and action lasts for 2 to 6 hours.

Intravenous Ca^{++} channel blockers: Nicardipine, nimodipine.

Table 13.1: Comparison of commonly used calcium channel blockers

	Verapamil	*Nifedipine*	*Diltiazem*
Dose (oral)	30–160	30–120	30–90
Half-life	3 –7 hrs	1.5–5 hrs	
Heart-rate	↓↓	↑	↓
Coronary dilatation	↑	↑	↑
Peripheral dilatation	↑	↑↑	↑

INDICATIONS

- *Hypertension:* As drugs like nifedipine has got vasodilatory properties, it is being extensively used for treating hypertension. Recent evidences suggest that nifedipine is not an ideal anti-hypertensive perioperative period.
- *Angina:* Because of its good coronary vasodilator properties.
- *Induced hypotension:* Intravenous verapamil and nifedipine have been used for hypotensive anaesthesia and also for control of intraoperative and postoperative hypertension.
- *Myocardial ischaemia:* Calcium entry blockers produces a reduction in heart rate, enhances coronary vasodilatation and also it prevents acute coronary vasospasm.
- *Myocardial and cerebral protection:* As the pathogenesis of cell damage is intracellular accumulation of Ca^{++} these drugs are being extensively used for myocardial and cerebral protection. Especially drugs like nimodipine, nicardipine, lidoflazine.
  Ca^{++} blockers prevents
  - Calcium sequestration by mitochondria
  - Subsequent alteration in free fatty acid.
  - Arachidonic acid cascade.
- Along with cardioplegic solution to protect the myocardium.
- Anti-arrhythmic in supraventricular arrhythmia, verapamil is used.
- To obtund vasospasm following SAH; IV nimodipine or nicardipine

Advantage over Beta-blockers

- Can be used in patients with bronchial asthma and Raynauds phenomenon.
- Can be used in selected patients with cardiac conduction abnormalities.

ANAESTHESIA AND CALCIUM CHANNEL BLOCKERS

Main anaesthetic implication is because of its interaction with various drugs administered perioperatively.

Drug Interactions

- *Digitals:* Chronic verapamil treatment produce increase in serum digoxin level by 50 to 75% during the first week.

- *Potassium:* Calcium blockers exaggerate the toxic effects of potassium. So reduce the dose of potassium in patients who are on Ca^{++} blockers.
- *Local anaesthetics:* Ca^{++} blockers enhances the local anaesthetic toxicity.
- *Beta blockers:* Enhance the negative chronotropic as well as negative inotropic effect of Ca^{++} blocker.
- *Volatile anaesthetic agents:* The usage of agents like halothane, isoflurane, enflurane along with Ca^{++} blockers produce additive myocardial depressant effect especially with verapamil.

Exaggeration of Hypotension

Exaggeration of hypotension during induction of anaesthesia because of its additive effect with various inducing agents.

Impaired Neuromuscular Activity

Antagonism of neuromuscular blockade may be impaired because of inhibition of presynaptic release of acetylcholine. This effect is predominantly seen when NMBD and volatile agents are used together for maintenance of anaesthesia. This effect is not substantiated in man.

Impaired Cardiac Contractility

Ca^{++} blockers can precipitate cardiac failure in vulnerable patients.

Impaired Outflow

Ca^{++} blockers can exaggerate outflow obstruction in patients with hypertrophic cardiomyopathy.

Treatment of Ca^{++} Blocker Toxicity

- When there is significant hypotension—vasopressors.
- Significant reduction in heart rate—use isoprenaline or dopamine.
- AV block—cardiac pacing.

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14 Propofol (Diprivan)

Propofol is a relatively new entry into the field of IV anaesthetics. It was developed in early 70s. Clinical trial was by Kay and Rolley in 1977.

CHEMICAL CHARACTERISTICS

It is diisopropyl phenol, is water insoluble and was originally formulated with Cremaphor EL (which was discarded due to anaphylactic reactions). Present formulation—propofol 1%, glycerol 2.25%, egg phosphatide 1.2%, pH-7 (Fig. 14.1). It is a viscous milky white solution, stable at room temperature and is compatible with 5% dextrose for dilution.

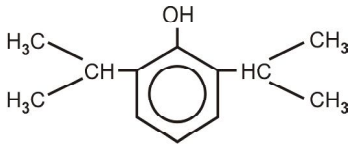

Fig. 14.1: Structural formula of propofol

METABOLISM

Metabolism in the liver–conjugation with sulphates and glucuronides, and excreted in kidney. There are no active metabolities. Less than 1% is excreted unchanged in urine.

PHARMACOKINETICS

Blood levels fall rapidly owing to both redistribution and elimination. Using 2 compartment models, distribution half-life is 2 to 8 min, elimination half-life is 1 to 3 hr in 3-compartment model the values are 30 to 60 min and 4 to 7 hr respectively, indicating a deep compartment with low perfusion vol. of distribution–central 20 to 40 L, steady state 150 to 700 L. Clearance is 1.5 to 2 L/min hepatic BF–suggesting extra-hepatic metabolism (or extrarenal excretion). Elimination is not much affected by mild hepatic or renal impairment.

The quicker clinical recovery to 'street fitness' seen with propofol compared to thiopentone is attributable to it's rapid hepatic metabolism in addition to redistribution.

PHARMACOLOGY

CNS: Good hypnotic, in subhypnotic doses produces sedation, amnesia, mood elevation and hallucinations. Not antanalgesic (e.g. barbiturate) whether it has any analgesic property on it is own is debatable.

A dose of 2.5 mg/kg produces rapid onset of unconsciousness (150-50 sec) and lasts 5 to 10 min, with lower doses, onset is delayed (hysteresis)

- EEG—initial ↑ alpha → ↑ δ wave, ↓ β wave, and ↑ amplitude
- EP–SSEP ↓ amplitude of early components, BAEP–no change.
- Effect on epilepsy–controversial studies, seizure activity following ECT and also associated with grand mal seizures has been demonstrated.
- ICP—by 30%, CPP is also—more so in ICP widely used as induction agent in neurosurgical procedures.
- Intraocular tension, prevent rises following scoline and intubation.
- Blood concentration required for minor surgery: 1.5 to 4.5 mcg/ml, major: 2.5 to 6 mcg/ml, awakening at: 1.6 mcg/ml. Narcotic premedication decreases the requirement.

RESPIRATORY SYSTEM

Action is Similar to Barbiturates

May cause apnea during induction (25% incidence). This is more common with dose, faster injection and narcotic premedication. Both VT and Rate (VT > rate). Reflexes (more than TPS).

Maintenance Infusion

100 μg/min—↓ VT and ↑ rate; on ↑ dose–VT is reduced further, not the rate.

Decreases the ventilatory response to CO_2 and hence increases PCO_2 and reduces pH, but these are not dose dependant.

CVS

- Induction results in—↓ BP (systolic, diastolic and MAP), decrease SVR,
  ↓ Stroke vol. index, pulmonary artery pressure and PCWP–due to cardiac depression and vasodilatation. Heart rate does not change much–resets baroreflex, attenuate sympathetic response to laryngoscopy and intubation.
- Maintenance—↓ BP at low levels and decreases further with high dose. Heart rate is variable, coronary blood flow / O_2 consumption ratio is preserved.
  If breathing air—SVR decrease is more than with $N_2O + O_2$ hypotension more in elderly and is easily correctable with IV fluids.
  In general, depresses CVS more than thiopentone.

Other Effects

No effect on steroid synthesis, hepatic, renal, fibrinolytic and haemopoietic systems. Does not trigger malignant hyperpyrexia.

Uses

- *Induction:* 1.5 to 2.5 mgm/kg (↓ dose with premedication and in old). Rapid administration is better. The duration with this bolus dose is 3 min with 1 mg/kg and 8 min with 3 mg/kg. This may be enough for short procedures and the recovery is symptom free and street fitness results in 1-2 hours.
- *Maintenance:* It is superior to barbiturates and is comparable to inhalational agents. Intermittent boluses of 10 to 40 mg can be given every few minutes.
  Infusion of 100 to 200 mcg/kg/hr; combination with morphine and fentanyl decreases dose requirement. In this case, postoperative–recovery time and side effects ae similar to TPS / isoflurance anaesthesia.
- *Sedation:* Provides titrable sedation for the surgical and ICU patient prolonged sedation does not increase recovery time or tolerance. 50-60 mcg/kg (decreased in elderly and sick).

SIDE EFFECTS

- Pain on injection and thrombophlebitis, decreased by injecting into a large vein and by anaesthetising the vein with lignocaine prior to injection of the drug.
- Myoclonus—incidence is more than TPS, but less than methohexitone or etomidate
- Apnoea
- Hypotension.

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15 Midazolam

Midazolam is a water soluble benzodiazepine with an imidazole ring in its structure, that accounts for stability in aqueous solutions and rapid metabolism.

The parenteral solution of midazolam used clinically is buffered to an acidic pH of 3.5. This is important, because, midazolam is characterised by a pH dependent ring opening phenomenon in which the ring remains open at pH values below 4, thus maintaining water solubility of the drug. The ring closes above pH values of 4, converting midazolam to a highly lipid soluble drug.

PHARMACOKINETICS

Bioavailability of this drug following IM injection is greater than 90%. Only about 50% of an orally administered dose of midazolam reaches the circulation. It is 97% plasma protein bound. It crosses BBB, placental barrier and can reach foetal circulation.

EFFECTS ON ORGAN SYSTEMS

- *Cerebral blood flow:* IV administration of midazolam 0.15 mg/kg induces sleep, reduces CBF (39%) increases cerebral vascular resistance (52%) and reduces $CMRO_2$. This drug is an acceptable alternative to barbiturates for induction of anaesthesia in patients with intracranial pathology. (TPS > midazolam > diazepam) (Table 15.1).
- *Respiration:* Respiratory depressant, similar to diazepam. Patients with COPD are more prone to the depressive effects.
- *CVS:* Induction dose (0.2 mg/kg) produces ↑ HR,↓ BP than TPS. Cardiac output is not altered by midazolam. This drug does not prevent BP and HR responses evoked by tracheal intubation.

Table 15.1: Comparison of diazepam and midazolam

Characteristics	*Diazepam*	*Midazolam*
Equipotent dose (mg/kg)	0.3–0.5 mg	0.15–0.3 mg
Volume of distribution (L/kg)	1–1.5	1–1.5
Protein binding	96–98%	96–98%
Elimination half time	21–37 hr	1–4 hr
Pain on injection	Yes	Rare
Solubility	Nonwater soluble	Water soluble
Potency		2-3 times more potent than diazepam.
Metabolite	Desmethyl Diazepam (potent.)	1 and 4 –OH methyl midazolam (less potent)

CLINICAL USES

- Preoperative medication: potent sedative, amnesic and anti anxiety effects.
- IV Sedation: (1-2.5 mg total dose)
  More rapid onset, greater amnesia, less postoperative diazepam; suitable in day care surgery, cardioversion, etc.
- Induction of anaesthesia: dose 0.2 to 0.3 mg/kg given over 30 to 60 seconds midazolam can produce induction of anaesthesia; but less effective than TPS.

Drug Interactions

Concomitant uses of barbiturates, alcohol and other CNS depressants may potentiate the respiratory depressant action.

- Decreased requirement of inhalational agents during anaesthesia.
- No interaction with nondepolarising muscle relaxants.

Adverse Reactions

Resp. depression (↓ TV and RR) headache, hiccoughs, occasional dysrhythmias, prolonged amnesia.

Over dosage: Sedation, somnolence, confusion and coma.

TREATMENT

Monitor vital signs, maintain a patent airway and support ventilation, and maintain an infusion with suitable IV fluid.

Specific antagonist: FLUMAZENIL–0.2–1 mg IV.

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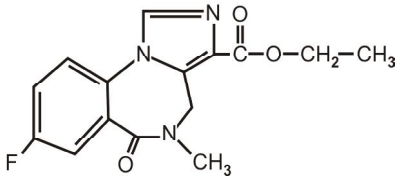
16 Flumazenil

It is the first benzodiazepine antagonist to be introduced for clinical use.

STRUCTURE



8-fluoro–5, 6–dihydro–5 Methyl–6 oxo–4H–Imadazo (1,5) (1,4)–benzodiazepine–3–carboxylic acid ethyl ester.

PHYSIOCHEMICAL CHARACTERISTICS

- Flumazenil was synthesised in 1979, is a colourless crystalline powder with pK of 1.7.
- It has a weak, but sufficient water solubility to prepare an aqueous solution.
  The octano/aqueous buffer partition coefficient is 14, demonstrating lipid solubility at pH 7.4.

MECHANISM OF ACTION

In CNS there are 2 receptors $GABA_A$ and $GABA_B$. Benzodiazepine agonists combine at $GABA_A$ and promote chloride conductance.

Flumazenil which is a competitive antagonist bind equally to these A and B receptors without disturbing the conformational equilibrium, and prevent binding of both benzodiazepine agonists and inverse agonists. It has high affinity, great specificity and minimal intrinsic effect, and its effect depends upon concentration gradient.

Flumazenil has short half-life and hence when used to antagonise the effect of long acting benzodiazepine agonists like diazepam, there is resedation, unlike for midazolam. If high dose of benzodiazepine agonist is given only low dose of flumazenil is given to counter the effect of CNS depression but sedation and amnesia persists.

PHARMACOKINETICS

Metabolised in the liver and 3 metabolites are known, namely:

- N–desmethyl flumazenil
- N–desmethyl flumazenil acid
- Flumazenil acid.

The glucuronides of these metabolites are excreted in urine.

Flumazenil has highest clearance and shortest elimination half-life. The plasma half-life is one hour. To maintain a constant therapeutic blood level, continuous infusion at a rate of 30 to 60 mcg/min (0.5 to 1 mcg/kg/min) is used.

The protein binding is low and hence free fraction of the drug ranges from 54 to 64%.

PHARMACOLOGY

- In low doses, flumazenil is CNS stimulant but high doses, CNS depressant when given alone.
- When administered in patients who benzodiazepine agonist, it reverses unconsciousness respiratory depression, sedation, amnesia, and psychomotor dysfunction.
- The onset of its action is rapid with its pH effect in 1 to 3 minutes.
- Predicted therapeutic plasma level is 20 mg/ml. Effect is expected for 40 to 90 minutes after a dose of 3 IV.

Uses and Doses

- Diagnostic and therapeutic reversal of benzodiazepine receptor agonists. For diagnostic use it is given incremental dose of 0.1 to 0.2 mg IV up to 3 mg. There is no change in CNS depression it is unlikely be solely of benzodiazepines overdose. Flumazenil also used as continuous infusion to prevent resedation with longer lasting benzodiazepine agonists in a dose of 30 to 60 mcg/min.
- Diagnosis in coma with a dose 0.5 mg to 1.0 mg only also counteracts drowsiness and associated severe liver disease as hepatic encephalopathy.
- The CNS depressant effect of alcohol intoxication countered by flumazenil.
- For reversing sedation in ICU patients to monitor the functional status.
- Diazepam toxicity.

Side Effects and Contraindications

- It has high safety margin but produces withdrawal reactions including seizures in a person who is exposed to benzodiazepine agonists for a long time.
- Resedation occurs when used against long acting of benzodiazepine agonists.

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17 Narcotic Antagonists

HISTORY

The first recorded specific opioid antagonist-N-allyl nor codeine-was developed in 1914 by Pohl. Nalorphine was synthesised by Weighland and Erickson in 1914 and was found to be strongly antagonistic to almost all the properties of morphine.

Antagonist

A drug or compound which inhibits or prevent receptor mediated agonist effects by competing for receptor occupancy.
Two types—Competitive and noncompetitive.

Competitive: Can usually be displaced from the receptor complex by the administration of a receptor agonist if given in a sufficient concentration.

Noncompetitive: When bound to the receptor complex, will produce a loss of the expected effect, that can not be reproduced by the concurrent administration of the receptor agonist.

CLINICALLY USEFUL OPIOID ANTAGONISTS

Naloxone Nalorphine Naltrexone Nalmefene	Commonly used
Cypridine Naltridole Norbinaltorphimine	Experimental

Uses of Narcotic Antagonists: General

- Restore spontaneous ventilation in patients who breath in adequately after opioid overdose / opioid anaesthesia.
- Used to antagonise opiod induced nausea, vomiting, pruritus, urinary retention and biliary spasm.
- Essential tool in the scientific evaluation of opiates and opiate receptors.
- Treatment of septic shock and haemorrhagic shock.

Relatively, minor changes in the structure of an opioid can convert a drug that is primarily an agonist into one with an antagonistic action at one or more receptors (μ, κ, δ, ε).

For example, Morphine → Nalorphine / Levorphanol / Levallorphan.
Oxymorphone → Naloxone/Naltrexone.

NALOXONE

Specific narcotic antagonist, at all 3 opioid receptors (μ, κ, δ)

Dose: 0.4 mg to 0.8 mg 1M or IV.

Duration of action: 1 to 4 hr.

Neonatal dose: 10 to 15 mcg /kg.

Preparation: Naloxone HCl–'NARCAN'.

- Drug of choice where potent narcotic antagonist is required.
- An injectable preparation is also available for use in neonates.
- Naloxone in dose of 0.4 to 0.8 mg (IM / IV) given in man prevent or promptly reverse the effects of opioid agonists.

Uses of Naloxone

During opioid administration:

- Respiratory depression reversed in 1 to 2 minutes.
- Sedation reversed.
- BP if depressed return to normal.
- Diagnosis of physical dependence on opioids.
- Therapeutic agents in the treatment of compulsive use of opioids.
- This drug has also been used to decrease neonatal respiratory depression secondary to administration in the mother. (10 to 15 mcg / kg IV IM or SC)

Other Uses of Naloxone

- Treatment of post anaesthetic apnoea in infants when exogenous narcotics have not been used.
- Treatment of primary apnoea
- Benzodiazepine reversal (rarely used)
- Barbiturate and alcohol reversal.
- Partial antagonism of ketamine and N_2O analgesia.

Special Features

- High doses of naloxone are required to antagonise respiratory depressant effects of buprenorphine.
- Naloxone reverses the psychomimetic and dysphoric effects of agonist antagonists such as pentazocine, much higher doses are required (10–15 mg).
- Naloxone and natrexone have little or no potential abuse. Prolonged high doses produce recognisable withdrawal symptoms.

Absorption, Fate and Excretion

Absorbed readily from GIT. Naloxone is completely metabolised by liver before reaching the systemic circulation, hence must be parenterally administered.

Side Effects and Toxicity

- Hypertensive crisis.
- Cardiac dysrrhythmias in patients with previously existing cardiac problems and pulmonary oedema.
- Cardiac arrest and death.
- Sometimes may precipitate a withdrawal syndrome patients dependent on pentazocine, butorphanol, nalbuphine.

NALTREXONE HYDROCHLORIDE (TREXAN)

New opioid antagonist with actions at μ, α, and κ receptors. Has two advantages over naloxone– longer half-life, oral availability. Can be effectively used to treat the frequency and severity of pruritus, nausea and vomiting associated with epidural morphine, without decrease in analgesia.

Side Effects

- Nausea and vomiting
- Anxiety, lassitude and headache
- No potential for abuse and hardly any withdrawal symptoms. This drug is much more potent than naloxone
- Available as 50 mg tablets for oral use.

NALMEFENE

This is a new pure opioid antagonist.

- Greater preference for μ than κ and α.
- Long acting, both parenterally and orally effective.
- Half-life is 8 to 10 hours.
- Excreted via liver and kidney.

Side Effects

- Very few side effects butorphanol, nalbuphine.
- Levallorphan and Nalorphine are hardly used nowadays.

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18 Magnesium Sulfate

INTRODUCTION

Magnesium is an intracellular cation and second most plentiful cation after potassium. Magnesium is important for number of enzyme systems and neuromuscular function. Magnesium protects the heart against myocardial necrosis and dysarrhythmias and hence it is included in cold cardioplegic solution used during cardiac surgery.

Normal plasma concentration is 0.71 to 1.2 mmol/L. An average daily diet contains 10 to 20 mmol of magnesium. The factors regulating magnesium balance have been reviewed and they include the alteration between gastrointestinal absorption and renal excretion.

Magnesium sulfate is a drug used extensively in the management of eclampsia.

MECHANISM OF ACTION

- Magnesium decreases the irritability of the CNS and reduces hyperreflexia and is convulsion threshold.
- Severe vasospasm of the cerebral vessels resulting in cerebral ischaemia is postulated to be the cause of convulsions. Another mode of action of magnesium sulfate is said to be cerebral vasodilatory effect.
- Magnesium is also a neuromuscular blocking drug since it prevents the release of acetyl choline from the presynaptic neuron, and also causes receptor insensitivity of the postsynaptic neuron to acetyl choline, and hence also prevents the external manifestation of convulsions. It also depresses excitability of muscle membrane.
- Magnesium causes decrease in uterine activity and increase in uterine blood flow.
- Magnesium produces bronchial smooth muscle relaxation and effectively used in treating acute severe bronchial asthma.

DOSAGE

Magnesium sulfate in given in a loading dose of 40 to 80 mg/kg/IV followed by infusion of 2 gm/hr to maintain the therapeutic plasma level of 4 to 8 mEq/L. the loading dose is given over a period of 5 min.

The plasma level of magnesium is the accurate indicator of the therapeutic effects of magnesium, and toxicity of magnesium occurs with high plasma levels (Table 18.1).

Table 18.1: Effects of increasing plasma magnesium level

Plasma Mg (mEq / L)	*Effects*
1.5–2	Normal plasma level
4–6	Therapeutic range
5–10	ECG changes (QT interval prolonged, QRS complex shortens)
10	Loss of deep tendon reflexes
15	Sinoatrial, atrioventricular block
20	Respiratory paralysis
25	Cardiac arrest

- Magnesium reduces irritability of heart and it is widely used in treating atrial and ventricular fibrillation.
- Magnesium improves neuronal survival rate after ischaemic injury.

SIDE EFFECTS OF MAGNESIUM THERAPY

- Magnesium therapy is associated with both maternal and neonatal side effects. Overdose can lead to maternal muscle weakness, respiratory insufficiency and even cardiac failure. But these complications are seen only after deep Tendon reflexes are suppressed.
- In the neonate magnesium can also produce marked hypotonia and decreased muscle tone, and high plasma levels of magnesium are associated with apnoea and respiratory depression.
- Magnesium is excreted by the kidneys and should be used with precautions for those with renal failure.
- Magnesium will potentiate the effects of both nondepolarising and depolarising muscle relaxants by its action on postsynaptic acetylcholine receptor and the dose of the relaxants should be decreased in the event of general anaesthesia and be carefully titrated with neuromuscular monitoring.
- Eclampsia by itself is associated with decrease it plasma pseudocholinesterase activity and magnesium also causes inhibition of pseudocholinesterase activity and action of succinyl choline will be prolonged.
- Since magnesium is a central nervous system depressant it potentiates the sedative and opiates used and hence these should be given in decreased dosage and with extreme caution.

MONITORING DURING MAGNESIUM THERAPY

- Estimation of the plasma magnesium level.
- Estimation of the plasma level of magnesium may not be practical and it is also expensive, so a patient on magnesium therapy should be frequently assessed regarding "Loss of deep tendon reflexes" and this is an important sign regarding impending magnesium toxicity.

Antidote: The antidote for magnesium toxicity is intravenous calcium (10 ml of 10% calcium chloride).

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19 Steroids in Anaesthetic Practice

Everyday, during routine anaesthetic practice we come across patients who are or were on steroids. These patients form the majority, who need periop/preop steroid support, in order to avoid an untoward Addisonian crisis during anaesthesia and surgery, although anaesthesia as such causes only a mild stress reaction.

Other situations requiring steroid coverage are:

- Steroids to decrease ICP
- Steroids in the management of malignant hyperthermia
- Management of acute anaphylactoid reaction
- Acute adrenal crisis (Addisonian crisis).

The adrenal cortex secretes 3 groups of hormones from 3 layers:

- Zona glomerulosa/mineralocorticoid–aldosterone 50 to 250 mcg/day
- Zona Fasiculata/Glucocorticoids–cortisol/cortisone 15 to 30 mcg/day
- Zona Reticulosa/sex hormones–dihydroepiandrosterone 15 to 30 mcg/day testosterone (minimal).

Steroid therapy suppresses ACTH from pituitary with resultant adrenocortical atrophy. As short a course as 5 to 7 days may produce depression of the adrenal cortex. Depression may last as long as 1 to 2 years, and in some cases, the adrenal cortex may never regain its full functional reserve. In such cases the adrenal cortex is unable to secrete extra hormones in response to anaesthesia, surgery or trauma.

Commonly used steroids differ in their potency with respect to glucosteroid and mineralocorticoid activity as shown in Table 19.1.

Table 19.1: Comparison of commonly used steroids

Commonly used name	*Glucosteroid potency*	*Mineralocorticoid potency*
Short acting		
Cortisol	1	1
Cortisone	0.8	0.8
Intermediate acting		
Prednisone	4	0.25
Prednisolone	4	0.25
Methyl prednisolone	5	0.25
Triamcinolone	5	0.25
Long acting		
Paramethasone	10	0.25
Betamethasone	25	0.25
Dexamethasone	30–40	0.25

ANAESTHETIC CONSIDERATIONS

- **The patient taking steroids:** Perioperative stress and the need for corticosteroid supplementation.
  - Deep GA / RA causes the normal intra–OP glucocorticoid surge to be suppressed.
  - Few patients who have suppressed adrenal function have perioperative CVS problems, if they do not receive supplemental steroids intraoperative (SVR LVSWI).
  - Perioperative hypotension can occur in a steroid dependent patient.
  - Life threatening Addisonian crisis occurs rarely. High dose steroid coverage perioperatively

  The rise in cortisol output in normal patients occur at the start of surgery and if the surgery is a major one, it may remain elevated for 2 to 3 days. This may vary from 115 to 185 mg / day to as high as 300 to 500 mg/day. There is no hard and fast rule regarding steroid supplement but it is *safer to give steroids than to omit it.*
  - For major surgery, hydrocortisone hemisuccinate 100 mg qid should be administered.
  - This can commence at the time of premedication and can be continued for 3 days. The dose is tapered off slowly.

  Or
  - Hydrocortisone 200 mg/70 kg/day, the dose being gradually tapered off in 2 to 3 days till oral substitution is possible.
  - In minor surgery 100 mg/70 kg/day–for 1 day.
  - This dose is reduced by 25% day till oral dose can be resumed.
  - For endoscopy or other brief procedures 100 mg single shot IM, prior to procedure.
- *Steroids to ICP / treatment and prophylaxis of postoperative cerebral oedema:* Patients coming for neurosurgery with ICP may already be on steroids. Dexamethasone is preferred to other substitutes since it has (i) maximum antioedema property. (ii) minimal mineralocorticoid action. Start at least 24 hr preoperative, preferably 2 to 4 days before surgery and taper over a few days postoperative.

  Dose: Dexona 10 mg IV / IM followed by 4 mg 6 hourly. Treatment takes 12 to 24 hr to be effective.
- *Steroids in management of malignant hyperthermia:* In addition to other specific/general supportive measures, dexamethasone 20 mg IV C other steroids are given.
- *Steroids in status asthmaticus/bronchospasm:* The effect of corticosteroids may not be seen for 12 hr. Even cortisol may take 2 to 4 hr for action. Massive doses are given–especially in patient already on steroids. Besides, perioperative steroids may be required if these patients were on steroids 1 to 1½ years ago.

  Dose: Hydrocotisone 500 mg IV single dose.
- *Steroids in a/c anaphylaxis*: Steroids especially hydrocortisone are given as a supportive measure; it has to be remembered that this will take some time to act. (Hydrocortisone 200 to 300 mg IV)
- *Steroids in a/c adrenal crisis:* Since these patients cannot respond to stressful situations a maximum stress dose of glucocorticoids have to be given perioperatively.

  Dose: 250 to 300 mg/70/kg/day IM.

or 25 mg hydrocortisone phosphate IV immediate preoperative followed by 100 mg IV over next 24 hrs or 100 mg hydrocortisone phosphate IV/12 hrs.

- *Steroids in septic shock*: Massive doses of prednisolone has to be given as early as possible–20–30 mg/kg–This is controversial, since many authorities argue that steroids have no active role in septic shock.
- *Other miscellaneous conditions*
  - Acute epiglottitis/laryngeal oedema.
  - Mendelson's syndrome
  - Cardiac resuscitation—role of steroid is controversial
  - Thyroid crisis.

MINERALOCORTICOID DEFICIENCY

Some patients can have congenital or acquired mineralocorticoid deficiency which has to be treated pre/intraoperative using 9 fluorocortisol 0.05–1 mg/day.

SIDE EFFECTS OF STEROIDS

- Susceptibility to infection
- Delayed healing
- GIT bleeding and perforation
- Electrolytic imbalance.

CONTRAINDICATIONS

- PTB/GIT ulcers/DM/hypertension/CCF
- Pregnancy/glaucoma/renal dysfunction.

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20 Neuroleptanalgesia

Neuroleptanalgesia is a term used to describe the state of a patient following the administration of a combination of a major tranquilizer (butyrophenones–droperidol or haloperidol) and a potent opioid analgesic (fentanyl or phenoperidine). This was introduced by J.A. De Castro and Mundeleer in 1959 and is characterised by analgesia, absence of clinically apparent motor activity, suppression of autonomic reflexes, maintenance of cardiovascular stability and amnesia. The addition of an inhaled anaesthetic, usually nitrous oxide, improves amnesia and analgesia and has been called neuroleptanaesthesia.

The commercial preparation of droperidol–fentanyl (Innovar) is the combination used to produce neuroleptanalgesia although many other combinations are possible. It contains droperidol 2.5 mg/ml and fentanyl 50 mcg/ml (50:1). Lactic acid is added for adjustment of pH to 3.5. The pharmacological effects are additive effect of the two component drugs. Droperidol produces hypnosis, sedation and antiemetic effects by occupying GABA, receptors on the postsynaptic membrane thereby reducing synaptic transmission and resulting in a build up of dopamine in the intersynaptic cleft which alters the balance of dopamine and acetylcholine in certain brain sites. Fentanyl acts through opioid receptors and produces analgesia.

USES OF NEUROLEPTANALGESIA

- As premedication–0.5 to 2 ml of innovar.
- In certain neurosurgical operations when the patient's conscious co-operation is required during surgery, e.g. stereotactic surgery and anterolateral tractotomy.
- In diagnostic procedures like aortography, angiocardiography, bronchoscopy, oesophagoscopy, fibreoptic gastroscopy, etc.
- As a supplement to thiopentone, relaxant and gas-oxygen anaesthesia.
- As an alternative to conventional anaesthesia.
- To provide sedation during regional analgesia.
- During bypass in cardiovascular surgery in order to avoid the cardiac depressant and dysrhythmic effects of volatile agents.
- In ophthalmology to provide sedation in patients undergoing intraocular operation under LA.
- For dressing of burns.
- In ICU to facilitate intubation and IPPV.
- To increase afferent block during halothane anaesthesia.

DOSAGE

Induction of GA: If the components are given separately, droperidol should be administered first in a dose of about 5 to 10 mg (5 to 15 mcg/kg) and followed by incremental doses of fentanyl in 50 to 100 mcg increments. The usual induction dose of Innovar is 0.1 to 0.15 ml/kg administered with nitrous oxide and a muscle relaxant. A test dose of 1 to 2 ml may be administered prior to induction because of possible vasodilatation and hypotension. 200 to 300 ml of balanced salt solution may also be administered prior to induction.

Maintenance of GA: Supplementation of anaesthesia during NLAN should be with fentanyl and not Innovar (prolonged duration of action of droperidol will produce postoperative somnolence if it is repeated).

Sedation and analgesia: 0.5 to 1.0 ml of Innovar IV repeated and titrated to desired effect. IM dose is 1-2 ml.

SIDE EFFECTS

- Muscle rigidity due to large doses of fentanyl managed with muscle relaxants.
- Respiratory depression—due to fentanyl—can be reversed with naloxone.
- Prolonged somnolence—due to droperidol.
- Hypotension due to alpha blockade of droperidol can be treated with blood volume expansion and alpha agonist (phenylephrine).
- Extrapyramidal complications—may be treated with diphenhydramine or benztropine
- Unusual psychological reaction.
- Hallucinations, sensation of weightlessness—can be treated with benzodiazepine.
- Malignant neuroleptic syndrome (hyperthermia, muscular rigidity, autonomic instability). Can be treated with dantrolent and bromocriptine.

CONTRAINDICATIONS

- First trimester of pregnancy
- Patients receiving MAO inhibitors
- Parkinson's disease.

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21 Acute Barbiturate Poisoning

Poisoning and drug over dosage are among the most common causes for hospital admission. Till recently barbiturates were the most common drugs used for suicide. Accidental poisoning is seen in epileptics, psychiatric patients and children who get regular prescription for these drugs.

Barbiturates are classified into four groups depending on the duration of their action as long, intermediate, short and ultrasound acting barbiturates. Of these, phenobarbitone, (a commonly used anticonvulsant) which is a long acting barbiturate is the most commonly used.

CLINICAL FEATURES

Barbiturates exert their effects mainly through suppression of CNS. It has got effects on other systems as well.

Low over-dose-produces confusion, excitement, delirium and hallucinations. Large over-dose shows stupor, progressing through deepening coma with loss of superficial and deep reflexes and gradual loss of response of painful stimuli. Babinski's sign may become positive. Pupils are generally constricted, but dilate in terminal stage.

Cardiac output falls, capillary permeability increases (due to peripheral vasodilatation) and progressive cardiovascular collapse evidenced by cyanosis, hypotension, rapid pulse and cold clammy skin occurs.

Respiratory system: Respirations become irregular, sometimes Cheyne-Stokes in character. Minute volume is usually reduced.

Skin: Necrosis of sweat glands and bullous lesions over the skin develop in a few, as a hypersensitive reaction. Such lesions are commonly found in sites where pressure has been exerted between two skin surfaces like interdigital clefts and popliteal region. This finding gives a positive clue to barbiturate poisoning.

TEMPERATURE

Hypothermia is usually the feature, but fever implies impending bronchopneumonia.

DIAGNOSIS

Poisoning or drug overdose should be suspected in any unconscious patient. Other causes of coma must be excluded. But often the circumstances under which the patient is found will suggest

that poisoning or drug overdose are likely. History of epilepsy or psychiatric illness should be asked for. Areas of erythema and needle marks should also be searched for.

Samples of blood, urine and gastric contents should be taken for toxicological analysis and may help in the diagnosis.

Concomitant ingestion of alcohol potentiates the toxic effects of acute barbiturate over dose (Table 21.1).

Table 21.1: Toxic dose and blood levels

	Fatal dose	*Toxic plasma level*
Long acting	3–4 gm	> 7 mcg / 100 ml
Medium acting	2–3 gm	> 3 mcg / 100 ml
Short acting	1–2 gm	> 1 mcg / 100 ml

MANAGEMENT

The first steps are:

1. Ensure a clear airway
2. Breathing adequately
3. Maintenance of adequate BP.

- After securing an airway, cannulate the patient with large bore IV cannula. Get a sample of blood which may be sent for grouping, cross matching and toxicological studies.
- Warmed fluids are infused through the line to maintain BP. If necessary, a central venous line may be put through which volume can be infused and CVP measured.
- In cases, where airway cannot be maintained an ETT should be introduced especially when ventilation is inadequate, patient is drowsy and is to have a gastric lavage. The most common indications for ventilation are respiratory failure, pulmonary aspiration.
- Hypothermia managed by passive rewarming with a space blanket, warmed infusion fluids and warmed humidified gases.
- Care of skin, mouth and bladder and regular physiotherapy, like in any comatose patient.

SPECIFIC THERAPY

- Gastric lavage should be carried out with warm water mixed with $KMnO_4$ and suspension of activated animal charcoal of 50 gm or tannic acid. A concentrated solution of magnesium sulphate should be left in the stomach to ensure purgation and minimise intestinal absorption. Gastric lavage is usually unnecessary, if the patient is seen 4 hr after ingestion of the drug.
- Other methods to reduce the drug absorption like induced emesis (using ipecacuanha syrup) and faecuresis (using 20% mannitol orally) are less commonly used now a days.
- Excretion of barbiturates can be increased by alkalinising the urine to a pH of 7.5 to 8.5 by giving bicarbonate (forced alkaline diuresis).
- Dialysis can be used in patients with renal failure.

- Haemoperfusion through activated charcoal columns (charcoal coated with acrylic hydrogel to avoid its inherent complications like embolism, coagulopathy leukopenia and fever) is considered as the best method available for increasing elimination of barbiturates.

MONITORING

- Pulse rate.
- Blood pressure
- EKG through a cardioscope
- CVP
- PCWP (esp. useful in patients developing pulmonary oedema)
- Urine output
- Temperature.

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22 Rationale for Premedication

The first use of the word premedication is attributed to Memechan, 1920. Management of anaesthesia begins with the preoperative psychological preparation of the patient and administration of a drug or drugs selected to elicit specific pharmacologic responses.

Psychological premedication is provided by the anaesthesiologists preoperative visit and interview with the patient and family members to reduce their anxiety.

Pharmacologic premedication: It is typically administered orally or intramuscularly in the patients hospital room 1 hour to 2 hours before the anticipated induction of anaesthesia. For outpatient surgery, premedication is usually administered intravenously in the immediate preoperative period.

CONSIDERATIONS FOR PHARMACOLOGIC PREMEDICATION

- Anxiolysis
- Amnesia
- Analgesia
- Sedation
- Antisialagogue
- Antithrombotic
- Antibiotic
- Attention to pre-existing disease
- Antacid
- Antiemetic
- Adjunct to anaesthesia
- Antivagal
- Antihistaminic

SELECTED GOALS OF PREMEDICATION

- Relief of apprehension
- Analgesia
- Reduction of gastric fluid volume
- Antisialagogue effect
- Decrease MAC
- Prophylaxis against allergic reaction.
- Sedation
- Amnesia
- Prevention of nausea and vomiting
- Prevention of autonomic reflex responses
- Facilitation of anaesthetic induction

DETERMINANTS OF DRUG CHOICE AND DOSE

- Patient age and weight
- Physical status
- Tolerance to depressant drugs
- Previous adverse experience with drugs used for preoperative medication
- Allergies

- Elective or emergency surgery
- Inpatient or outpatient surgery.

DEPRESSANT PHARMACOLOGIC PREMEDICATION

Not indicated/or minimal use in:

- Less than 1 year of age
- Elderly
- Decreased level of consciousness
- Intracranial pathology
- Severe pulmonary disease
- Hypovolaemia

Indicated in:

- Cardiac surgery
- Cancer surgery
- Coexisting pain
- Regional analgesia.

Amnesia may be desirable for an especially upset or for patients having frequent general anaesthetics. Amnestic drugs also reduces the risk of awareness. Analgesia is desirable if the patient has pain during preoperative period. It also reduces dose of anaesthetic agents. Antisialagogues are indicated in infants, and also for airway and intraoral surgery.

Antiemesis is required especially in patients with full stomach, with compromised airway and if an opioid is given. Antacid is advisable in cases with increased risk of regurgitation of gastric contents, e.g. pregnancy, hiatus hernia, obese patients.

Anticholinergics are used prophylactically to prevent bradycardia due to vagal reflex following noxious stimuli. (Antihistamines if given, protect from the effects of endogenously released histamines may be important in certain atopic individuals). (Antibiotics are included in the premedication principally for prophylaxis against bacterial endocarditis in susceptible individuals e.g.: patients with valvular heart disease, congenital cardiac abnormalities, or artificial valve prosthesis). (Antithrombotic drugs are to be considered for patients who have increased risk of developing postoperative intravascular thrombosis, e.g. polycythemia, obesity, malignancy, postoperative immobility, pelvis and lower limb surgery. Heparin 5000 units SC 8th hourly is given prophylactically.

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23 Placental Transfer of Drugs

Perinatal pharmacology is concerned with the classic pharmacologic processes of drug absorption, distribution, biotransformation and excretion in the mother and the foetus.

DETERMINANTS OF PLACENTAL TRANSFER

To achieve a physiological effect, a critical concentration of free drug (nonionized and nonprotein bound) must arrive at and react with a given tissue receptor site.

It is the lipid soluble free from of drug that readily passes biologic membranes such as placenta.

Maternal Factors

Factors that determine the concentration of free drug in uterine arterial blood (Cm)

- Total dose
- Route of administration
- Presence of adrenaline in local anaesthetic solution decreases peak level of local anaesthetic.
- Maternal metabolism and excretion—decreases drug concentration in the intervillous space.
- Maternal protein binding.
- Maternal pH and pKa of the drug.

Placental Factors (Fig. 23.1)

Once a given drug has reached the IVS, the quantity transferred per unit time is described by Fick's equation of passive diffusion. The law assumes that the membrane characteristics remain constant.

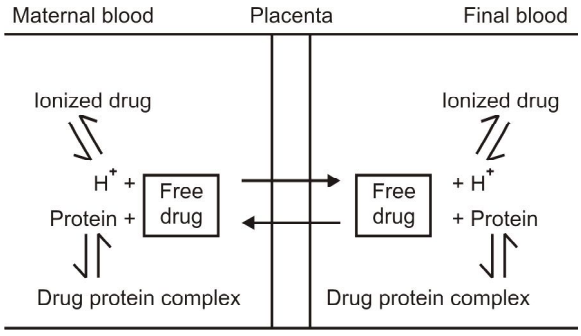

Fig. 23.1: Factors influencing the movement of drugs across placental barrier

$$Q / t = \frac{KA\ (Cm–Cf)}{X}$$

Q / t	rate of diffusion	K	diffusion constant of drug and membrane
A	surface available for transfer.	Cm	drug concentration in maternal blood
Cf	drug concentration in foetal blood	X	thickness of membrane.

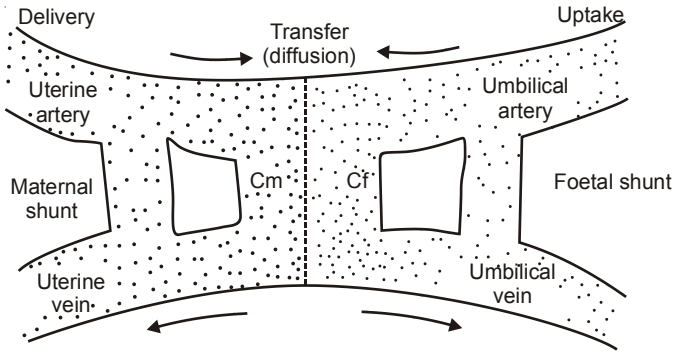

Fig. 23.2: A schematic diagram of the placental exchange site and maternal, placental and foetal factors that influence drug transfer and foetal drug uptake

Foetal Factors (Fig. 23.2)

Foetal Uptake of Drugs

Foetal uptake is determined by

- Solubility of the drug in foetal blood.
- Quantity and distribution of foetal blood flow to the IVS.
- Concentration of drug in foetal blood returning to the placenta.

Foetal Distribution of Drugs

Factors that determine the concentration of free drug in foetal umbilical arterial blood (cf):

- Umbilical venous blood concentration input.
- Uptake by foetal tissues.
- Foetal pH.
- Nonplacental routes of foetal drug excretion.
- Foetal hepatic metabolism.
- Foetal protein binding.
- Foetal renal excretion.

Foetal Metabolism and Excretion of Drugs

Foetal hepatic enzyme activity is less when compared to the adult.

PLACENTAL TRANSFER OF INDIVIDUAL DRUGS

Inhalational Anaesthetics

Inhalational anaesthetics diffuse rapidly across the placenta. The incidence of depressed neonates increases with duration of anaesthesia before delivery.

Barbiturates

Barbiturates are weak acids whose pKa values are near or above the physiological pH range. They are therefore largely nonionized and pass readily across the placenta by lipid diffusion. However, the foetal brain will not be exposed to high concentration of barbiturate if the induction dose is less than or equal to 4 mg/kg. narcotic analgesics generally cause neonatal respiratory depression. The foetal concentration of pethidine reaches the peak plateau within 1 to 5 hours after the maternal dose. In equianalgesic doses. Morphine produces more repiratory depression in newborn than does pethidine.

Tranquilizers

Phenothiazine crosses the placenta rapidly. Diazepam causes loss of beat to beat variation of foetal heart rate and if given in dose of 30 mg or more, hypothermia and hypotonia in the neonate occurs. In smaller doses of diazepam there is only minimum effect on the foetus. If a 5 mg bolus of midazolam is given shortly before caesarean, the newborn may be depressed.

Local Anaesthetics

Local anaesthetics readily cross the placental barrier.

These drugs may produce changes in neurobehavioural patten of the newborn.

Neuromuscular Blocking Drugs

All are quarternary ammonium compounds, which are fully ionised and therefore would be expected to diffuse across the placenta only very slowly. Gallamine crosses the placenta comparatively faster.

Atropine and Neostigmine

Atropine is a weak base. It crosses the placenta. Neostigmine a quarternary ammonium compound is unlikely to cross the placenta.

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24 Anaphylactic Reactions during Anaesthesia

DEFINITION

Anaphylaxis is acute, severe, potentially life threatening allergic reaction characterised by respiratory distress, cardiovascular collapse and angioneurotic oedema in a sensitised person on exposure to an offending antigen.

The first reported death from anaphylaxis occurred in the year 2600 BC from bee sting! Richet coined the term anaphylaxis from the Greek words *ana* (contrary to) and *phylaxis* (protection).

INCIDENCE

Anaphylactic deaths occur between 1 in 5000 to 1 in 25,000 anoesthetic cases.

PATHOPHYSIOLOGY OF ANAPHYLACTIC REACTION

- The term anaphylaxis is used to denote an Ig E antibody mediated reaction, Hypersensitivity Reaction Type I.
- Initially antigenic molecules (usually proteins) capable of stimulating Ig E are introduced into the body during the first exposure.
- Once produced, Ig E antibody become fixed to tissue mast cells or circulating basophil both of which contain Ig E receptors.
- Re-exposure to Ag (antigen) causes direct bridging of the cell surface Ig E receptor molecules.
- This causes activation of membrane associated enzymes causing complex biochemical cascades that lead to efflux of Ca^{++} ion with release of granule associated mediators and generation of new mediators from cell membrane phospholipids.

Mediators released are:

- Histamine
- Prostaglandins
- ECF–A (Eosinophil Chemotactic Factor–A).
- NCF (Neutrophil Chemotactic Factor).

Histamine dilate terminal arterioles and increase the permeability of venules, causing increased extravasation of fluid. Histamine also causes increased airway resistance. In the skin histamine produces vasodilatation (flushing) pruritus and urticaria.

Other mediators: Slow Reacting Substance of Anaphylaxis (SRS–A), Leukotriens and Platelet Activating Factor (PAF).

CLINICAL MANIFESTATIONS

Individuals vary in onset and manifestations of anaphylaxis, but most often the clinical hallmark is appearance of symptoms within seconds to minutes after exposure of the offending antigen most commonly by injection and less commonly by the ingestion route.

Primary anaphylactic target organs in human beings are:

- Cutaneous
- Respiratory system
- Cardiovascular system
- GI tract.

Evaluation and treatment of patients who develop anophylaxis in the operating room are challenging to anaesthetist. This is because of multiple medication. Patients are frequently unconscious and in deep level masking symptoms and signs of anaphylaxis.

During anaesthesia symptoms are often limited to cardiovascular, respiratory and cutaneous systems. Of these cardiovascular signs predominate during anaesthesia, (Sometimes this may be the only manifestation of an intraoperative anaphylaxis).

CVS Manifestations

- Tachycardia
- Hypotension
- Dysrrhythmias

If the patient is being monitored, it is characterised by decrease in MAP, DPB and SBP.

Respiratory System Manifestations

- Wheeze
- Cyanosis
- Laryngeal oedema

Sudden decrease in pulmonary compliance is manifested by an increase in airway pressure during positive pressure ventilation. Cyanosis or PaO_2 may be noted.

Cutaneous Manifestations

- Flushing
- Erythema
- Urticaria and angioedema.

DIFFERENTIAL DIAGNOSIS IN ANAESTHETISED PATIENT

Confused with other catastrophies producing CVS collapse or bronchospasm.

- Myocardial infarction
- Irritant induced bronchospasm

- Pulmonary embolism
- Gastric content aspiration
- Acute pulmonary oedema.

MANAGEMENT OF ANAPHYLAXIS

Initial Therapy

- Stop administration and / or reduce absorption of offending agent. If Ag is given subcutaneously, 0.5 ml of Epinephrine 1 in 1000 Soln. Injected subcutaneously near the previous injection site.
- Mainstay of initial pharmacological treatment is adrenaline (epinephrine).
  a. 0.01 m/kg increments of 1 : 1000 solution IV.
  b. 10 ml of 1 : 10,000 soln. For endotracheal administration.
- Maintain airway and administer 100% O_2
  If there is risk of laryngeal oedema present, as evidenced by stridor and dyspnoea, the patient should be intubated immediately and ventilated with 100% O_2. Another alternative is needle cricothyroidotomy.
- Rapid intravascular volume expansion.
  25 to 50 ml/kg (total 2-4 lit) of colloid or crystalloid may be needed for correcting hypotension.
- Discontinue all anaesthetic agents, because they have negative ionotropic effects and may interefere with reflex response to hypotension.
- Consider use of MAST for hypotension.

Secondary Treatment

- Administer antihistamine
  – Diphenhydramine 1 mg/kg IM or IV
  – Ranitidine 1 mg/kg IV
  Diphenhydramine is helpful to relieve postoperative itching. Ranitidine is helpful for persistent hypotension
- Glucocorticoids may be useful in preventing potential late phase reactions, but they have no immediate effects. Hydrocortisone 5 mg/kg up to 200 mg initial dose and then 2.5 mg/kg every 6 hours OR methyl prednisolone 1 mg/kg IV every 6 hr.
- For persistent bronchospasms, it is prudent to use aminophylline 5 mg/kg IV in loading dose followed by 0.5 mg/kg/hr by IV infusion.
- For persistent hypotension: catecholamine infusion may be needed.
  Epinephrine may be helpful for both hypotension and bronchospasm
  It is given as 0.05 mcg/kg/min infusion. Dopamine in the dose of 5 to 10 mcg/kg/min helps to maintain cardiac output, thereby maintaining cerebral, coronary and mesenteric blood flow.
- If acidosis is suspected, $NaHCO_3$ (1 mEq/kg) should be administered.

IMMUNODIAGNOSTIC TEST

Intradermal skin test is the most readily available and generally useful diagnostic test for drug allergy.

Skin test must be done in the absence of medications that affect the skin test response like antihistamines, TGA, etc.

SPECIFIC ALLERGIC REACTIONS SEEN BY ANAESTHESIOLOGIST

Narcotics

Narcotics cause nonimmunologically mediated histamine release from mast cells. Skin mast cells are uniquely sensitive to narcotics where as GIT and lung mast cells and circulating basophils do not release histamine when exposed to narcotics.

Most opioid induced reactions are self-limiting and restricted to cutaneous reactions and hypotension which respond to IV fluids. However the pharmacological release of mediators induced by opiates is a very common clinical occurrence and routinely cause positive skin test secondary to non-immunological skin mast cell histamine release.

Barbiturates

Acute allergic reactions have been reported with administration of thiobarbiturates. Proposed mechanics for these reactions include non-immunologically induced mediator release.

Local Anaesthetics

Despite patients commonly reporting adverse reactions to local anaesthetics, true allergic reactions are rare, seen only with the benzoic acid esters, e.g. benzocaine, chlorprocaine, procaine, tetracaine. Very rarely anaphylactic reactions are also reported to amide group due to the preservative methyl paraben.

Radio-contrast Media

- Incidence of allergic reactions to radio-contrast media is between 5 and 8%.
- Anaphylactic reactions occur in 2% to 3% of patients receiving IV infusion
- Most reactions begin 1 to 3 min after intravascular administration.
- There is no evidence that IgE play a role in the anaphylactic reaction.
- It is said to be due to non-immunological mediator release from mast cell and basophil due to complement activation.
- A patient who requires radio-contrast media (RCM) administration and who has had previous exposure to RCM has an increased (35%-60%) risk for re-exposure.
- Pretreatment of these high-risk patients with prednisolone (50 mg) one hour before RCM administration, reduces risk to 9% and the reactions if they occur are mild and of not much clinical importance. Pretreatment with ephedrine (25 mg) 1 hr before RCM administration is also helpful.

Protamine

Protamine sulfate, an extract from salmon fish is used intraoperatively to reverse heparin anticoagulation.

The use of IV protamine has increased in the last decade with the advent of cardiopulmonary bypass, cardiac catheterisation and haemodialysis.

The patients who are predisposed to protamine are:

- Diabetics who receive subcutaneous injections of insulin.
- Men who have undergone vasectomies. 20 to 30% of such men develop haemagglutinating antibodies.
- Patients who are allergic to fish.

  In case of protamine sensitivity, heparin antagonists such as hexadimethrine is used.

Streptokinase

It is a protein derived from group C haemolytic streptococci. It is used as a thrombolytic agent in myocardial infarction due to coronary embolism and in deep vein thrombosis. Anaphylactic reactions have been reported.

Mannitol

The administration of mannitol may cause direct non-immunologic histamine release from circulating basophils and mast cells. It is said that, slow infusion helps to solve this problem.

Methyl Methacrylate

Bone cement (methyl methacrylate) is used during orthopaedic surgery to attach a prosthetic joint to raw bone. Cardiopulmonary complications to methyl methacrylate include hypotension, hypoxaemia, non cardiogenic pulmonary oedema and cardiac arrest.

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25 Assessment of Blood Loss during Surgery

Normal blood volume in an adult is 75 ml/kg or 7.7% of body weight. Total blood volume can be measured as the sum of the red cell volume and plasma volume or from formulae depending on height and weight.

Blood volume is variable at birth and in the neonate ranges from 85 to 100 ml/kg. By 2 years it is 75 ml/kg.

ASSESSMENT OF BLOOD LOSS DURING SURGERY

Blood loss can be assessed by measuring the amount lost from the patient or the amount remaining in the patient. Disadvantage of the latter is that it is more difficult and normal blood volume of patient before the blood loss may not have been measured.

METHODS TO MEASURE BLOOD LOSS

- Visual observation of degree of bleeding.
- Clinical signs: Give indirect information concerning blood loss. These include fall in BP, fall in CVP tachycardia, sweating and pallor of skin. It is advantageous to replace blood loss before these signs become evident.

  These methods give a reasonable estimate in the case of minimal blood loss. Errors are likely to be cumulative if blood loss continues over a prolonged period.
- Gravimetric method: Blood loss is estimated by measurement of the gain in weight of swabs and towels and measurement of the contents of suction bottles. 1 ml of blood weighs 1 gm.

  Weighing of swabs underestimate blood loss by 25%. Evaporation will underestimate blood loss. Blood lost on the floor, into the drapes and into body cavities also cannot be measured.

  Overestimation may result if swabs also collect urine, pus or other fluids.
- Dilution methods
  - *Colorimetric:* All swabs and packs from the patient are mixed and vigorously stirred in a large volume of water and the resulting change in the optical density of water at the isosbestic absorption wavelength of Hb is measured and related to the Hb concentration of the patients blood. Alternatively the change in the electrical resistance due to the blood electrolytes in the bath can be measured. Errors may occur due to incomplete extraction or contamination with bile. This method can be used to wash out the bladder in TUR of prostate.

$$\text{Blood loss (mL)} = \frac{\text{Colorimeter reading} \times \text{volume of soln.}}{200 \times \text{patient's Hb (g\%)}}$$

– *Use of radioactive tracer dilution methods:* When measuring the volume of any body compartment by this method, it is important that the tracer used remain within that compartment.

In the case of blood volume, either the patients own red blood cells (labelled with Cr ^{51} following incubation with the isotope) or pooled human albumin (labelled with I ^{125} or I ^{131}) are used. All 3 isotopes are gamma emitters, but I^{125} is the isotope of choice because it emits less energy. The activity of the tracer is first measured and then injected intravenously.

The activity remaining in the empty syringe is measured and deducted from the amount of the isotope injected. After 10 to 15 minutes, a sample of blood is withdrawn from the opposite arm. This is to ensure there is no contamination of the sample from any isotope remaining at the injection site. The activity of this sample is then measured and the dilution volume calculated from the result. Repeated measurements can be made to estimate the change in blood volume with allowance made for residual radioactivity from the previous measurements.

In a shocked patient, the time taken for mixing throughout the total blood volume may be in excess of the 10 to 15 minutes usually allowed. So the measurement is a better indication of the effective circulating volume than it is of the total blood volume, errors may also arise from loss of the injected isotope by vigorous haemorrhage before mixing is complete.

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26 Massive Blood Transfusion

The rapid transfusion of large volumes of blood is common place now, as the blood banking services are available universally. Over and above the routine transfusion, it has certain special implications. Many definitions have been put forward by different groups.

- Transfusion which involve 10% blood volume replaced in 10 min or less (50 ml/min in an average adult)
- Any transfusion in which half the initial blood volume is replaced in 1 hour
- Any transfusion in which 1 unit of blood is given in 5 min
- Transfusion of more than 1 blood volume in 24 hours or less

The physiological effects of massive blood transfusion are two-fold. One is the usual implications of routine transfusion which won't be considered here. More important is the effect of rate and volume of transfusion and the associated changes.

APPARATUS

Microfilters, warming devices, equipments for transfusion under pressure. Two sets connected with 3-way connectors ensure that time is not lost when units or filters are changed.

INDICATIONS

- Trauma, crush injury
- Orthopaedic surgery—hip and spine
- Liver surgery—transplantation
- Exchange transfusion, etc.

COMPLICATIONS

Complications can be rate and volume dependant factors.

Rate Dependent Complications

- Hypothermia—stored blood at 4°C if infused, causes ventricular irritability and arrhythmias. Moreover shivering increases oxygen consumption several fold. So warmers are a must. Simplest is to pass through plastic tube immersed in warm water (37–38°C). microwaves warmers can cause haemolysis. Radiowave warmers of low frequency avoid risk of over heating and haemolysis.
- Citrate toxicity, calcium and potassium levels—citrate can bind Ca^{++} and cause hypocalcaemia, especially if 1 unit/5 min is given. Since Ca^{++} is a physiological antagonist of K^{+}, its levels are

important. Still Ca^{++} levels return to normal immediately on stoppage of transfusion and the hypocalcaemia is not low to cause bleeding disorders. But still the K^+ levels of blood may be as high as 20 to 30 mEq/L, if stored for 21 days, which is evidently dangerous. So calcium administration is practiced in massive B.T. 10% $CaCl_2$, though more irritant gives 3 times the Ca^{++} ions and may have to be given 1 to 1.5 mg/kg/min. Rapid infusion can precipitate arrhythmias especially under halothane anaesthesia. Calcium glucanate 10% is an alternative.

Hypothermia, liver disease, hyperventilation etc. decrease citrate metabolism and increase requirement of calcium administration, e.g. liver transplantation. Low flow states–though hypocalcaemia is doubly dangerous, the improvement of haemodynamics may actually mobilise more calcium from stores. So indications of Ca^{++} administration is just as in other cases.

- *Acid-base abnormalities:* pH of citrated blood is 7 to 7.1 and a temporary acidosis is likely. But 1 mmol citrate on metabolism give 3 mmol bicarbonate and hence a progressive metabolic alkalosis ensues. So there is no need for bicarbonate administration.

Volume Dependent Complications

- *Dilutional coagulopathy:* Stored blood has almost no platelets and little or no factors V, VIII and II. When a recipient has a transfusion equal to his blood volume, he will have 25 to 30% of his original blood circulating and after 2 exchanges only 10%.
  - Dilutional thrombocytopenia is evident if blood stored 24 to 48 hr is given and warrants platelet transfusion, though release from stores will partly compensate for this. The acute deficiency (< 75000) is likely to cause spontaneous bleeds and in patients undergoing surgery even at higher levels. About 10 units of platelet concentrates will raise the count by 1 lakh in an adult.
  - Labile clotting factors V, VIII and II have to be replaced in the form of FFP–one unit for every 10 units of blood given.
  - DIC–is likely if 80% of blood volume is replaced, but is an uncommon entity practically. Still it has to be watched for.

    The laboratory findings, in massive BT are increased PT and PTT, decreased platelets, Normal thrombin clotting time, fibrinogens and FDP levels.
- *Volume overload:* Massive BT. If used injudiciously can cause volume overload and pulmonary oedema.
- *Microaggregates:* Clots and debris are said to increase with storage and may contribute to lung injury leading even to ARDS. So instead of usual 170 μm filters microfilters of 40 μm are suggested. But this is controversial, since only large fibrin–WBC–platelet clots only are damaging for which microfilters are not needed and there is no objective data supporting the causation of lung injury. Anyway microfilters are not needed if storage is < 5 days. Also, microfilters result in high pressures being required for infusion, which may produce damage to RBC. Consensus of opinion is that microfilters are not required for blood transfusion.

Other things to be remembered are the implications of underlying condition warranting massive BT. Another is immunological alterations produced especially in patients for transplantations. This is said to benefit marrow transplantations and is deleterious in renal transplantations.

In short, though massive BT is a life saving procedure, it should not be taken too lightly.

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27 Blood Component Therapy

The basic concept of blood component therapy is based on the fact that patients are best treated by administration of the specific fraction of blood that they lack.

General principle of separation of blood components are as shown in Figure 27.1.

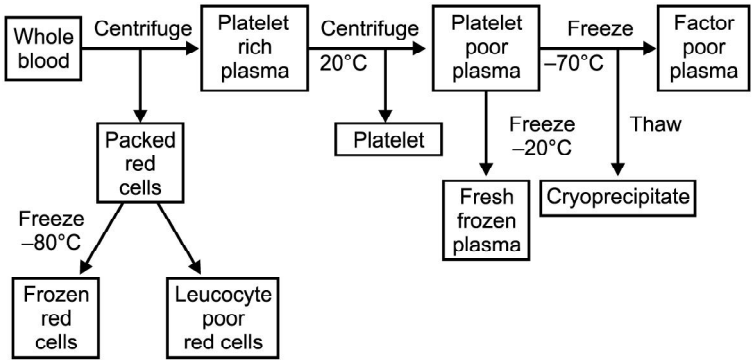

Fig. 27.1: Diagrammatic scheme of how whole blood is separated for component therapy

PACKED RED BLOOD CELLS

One unit contains 300 ml volume, erythrocyte mass 200 ml, haematocrit 70% donor/recipient ratio is 1 unit every 4 to 6 patients. Whole blood provide both oxygen carrying capacity and blood volume expansion. Packed red cells provide only oxygen carrying capacity.

ADVANTAGES OF USING PACKED CELLS

- Retain plasma and the components for other patients.
- Lesser incidence of transfusion reactions if washed cells are used.
- Higher incidence of cancer survival rate but no proved decrease in risk of hepatitis.
- Decreased potassium load if red cells are packed immediately before infusion.

Packed cells are used for losses of blood less than 1500 to 2500 ml/70 kg. With greater losses, whole blood probably should be used. The administration of red cells is facilitated by reconstituting them with a crystalloid'or colloid. Calcium containing crystalloids, e.g. Ringer lactate if used can produce clotting. If hypotonic diluent is used it can cause hemolysis. Solution used as diluents are 5% dextrose in 0.4% saline, 5% dextrose in 0.9% saline, Normosal - R with a pH 7.4. Recently adenine saline dextrose solution is also used as diluent.

PLATELET CONCENTRATES

If platelets are stored at room temperature, they can be used 5 days after collection with constant and gentle agitation. If they are stored at 4° C, they should not be used longer than 24 or possibly 48 hr after collection.

Indications for platelet therapy: Patients with severe thrombocytopenia (<20,000/m^3) and clinical signs of bleeding require platelet transfusion. Individuals when require surgery may need a platelet count of >100,000 cells/m^3 to maintain adequate haemostasis. ABO compatible platelets have improved survival rate. Rh -ve individuals requiring Rh^+ platelet should be treated with Rh immunoglobulin. 170 mm filters are used for platelet transfusion. One platelet concentrate produce an increase of 7,000 to 10,000 platelets/m^3, one hour after transfusion to the 70 kg adult.

FRESH FROZEN PLASMA

Fresh frozen plasma (FFP) contains plasma proteins, particularly FV and FVIII. Risk of infection and sensitization are present.

Indications for FFP

- Replacement of isolated factor deficiency.
- Reversal of warfarin.
- Antithrombin III deficiency
- Treatment of immune deficiency.
- Treatment of ITP.
- Massive blood transfusion.

CRYOPRECIPITATE

It contains factor VIII, and fibrinogen. It contains von Willebrand factor and fibronectin. Risk of hepatitis is present. ABO compatible cryoprecipitate advisable as it may contain red cell pigments. Paradoxical bleeding can occur. It should be administered through filter as rapidly as possible. Rate— 200 ml/hr. Infusion should be completed within 6 hours of thawing.

Use

Factor VIII deficiency or haemophilia.

PROTHROMBIN COMPLEX

Recovered from plasma or plasma fractions absorption with ion exchanges or inorganic chemicals products are all complexes of FII, VII, IX and X. Preparation (1) Koyne (2) Proplex.

Indication

Treatment of F IX deficiency; Sodium warfarin overdosage.

SINGLE DONOR PLASMA

Used as a volume expander. It is plasma removed from stored blood without any effort being made to preserve coagulation factors. Risks involved are hepatitis, AIDS and sensitization.

ALBUMIN AND PLASMA PROTEIN PREPARATIONS

5% or 25% solution is isotonic saline. No need of grouping and cross matching. It is very expensive. It should be administered within 4 hours of starting the infusion because of potential contamination after entering the bottle. Plasma protein fraction but not albumin can cause hypotension due to decreased systemic vascular resistance probably caused by generation of bradykinin.

Indication

Hypoproteinaemia, burns, peritonitis.

GRANULOCYTE CONCENTRATE

Obtained by continuous or intermittent flow leukopheresis or by filtration leukopheresis. It is difficult to obtain and it remains in the recepients body for a short period of time.

Indications

Severe leukopenia–(WBC $< 500/mm^3$) with evidence of septicaemia and fever. It should be ABO and Rh compatible. Granulocyte concentrate should be administered through a filter, over 2 to 4 hours. More rapid infusion can cause pulmonary reaction. Fever and chills are very common. Granulocyte concentrate is to be given for at least 4 to 5 days or until a satisfactory clinical response is observed.

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28 Autologous Blood Transfusion

DEFINITION

Autologous blood transfusion is one in which both the donor as well as the recipient are the same.

HISTORY

Autologous transfusion was done for the first time in the year 1818. Since then blood banks have come into existence. In the beginning, it did not gain much popularity but in course of time, due to the non-availability of blood in times of need and as the disadvantages of homologous transfusion became more apparent, autologous transfusion gained importance. It may be imperative in Jehovah's witness patients. The following complications of homologous transfusion can be eliminated by autologous transfusion:

- Infections.
- Haemolytic and immunological reactions.
- Immune response.
- Non-availability of suitable blood.
- Non-acceptability of banked blood.

TYPES OF AUTOLOGOUS TRANSFUSION

- Preoperative phlebotomy
- Intraoperative blood salvage
- Postoperative blood salvage
- Isovolaemic haemodilution.

Of these four methods, isovolaemic haemodilution is extensively discussed as a separate topic.

Preoperative Phlebotomy (POP)

Patients of all age groups are suitable for preoperative phlebotomy. Body weight is not a criterion. Usually, patients with haemoglobin above 11 gm% and haematocrit above 33% are suitable for preoperative phlebotomy (POP).

The volume of blood collected at one time is usually less than 15% of the estimated blood volume. Blood can be removed as often as once in every six days. Normally, 4 or 5 phlebotomies can be done before the elective surgery. If for any reason the surgery is postponed, the red cells can be separated and frozen for any length of time. Following phlebotomy, the intravascular

volume is replenished by protein poor fluid. It takes 48 to 72 hours for complete restoration of extracellular fluid volume. To increase RBC production, marrow can be stimulated by oral iron or recombinant human erythropoietin.

Uses

Orthopaedic surgeries, e.g. spine surgery, total hip replacement, etc. Cardiac surgery, liver transplant, major vascular surgery, pregnant women where excessive bleeding is anticipated. In these situations, it reduces requirements of homologous blood.

Complications

- Risk of contracting infection during phlebotomy.
- Vasovagal attack during blood donation.
- Problems associated with the transfusion of stored homologous blood are also seen in autologous transfusion. So as a safety measure, it is prudent to test autologous blood for ABO and Rh type to avoid human errors in transfusion reaction.

Cross-over Usage

The amount of blood salvaged preoperatively may exceed the actual usage subsequently. In such cases, the remaining blood meant for autologous transfusion, if used for other patients, is called cross-over usage.

Intraoperative Blood Salvage

The main steps of this technique are:
- Salvaging the blood from the operative sites.
- Anticoagulating the collected blood.
- Reinfusing the whole blood immediately as such, after washing and red cells filtering through 40 μm filters.

The important methods of collection are:
- Using semicontinuous flow devices.
- Canister collection.
- Single use reservoir.

Characteristics of Salvaged Blood

- Red cell survival is very similar to that of the homologous red cells.
- Salvaged blood is more alkaline than homologous stored blood and hence better buffer.
- 2, 3 DPG level is more than stored homologous blood platelets are deficient.
- Coagulation factors are deficient.

- FDP and complement activation is seen.
- Free haemoglobin level of 200 to 500 mg% are common.

Indications

- Open heart surgery
- Vascular surgery
- Orthopaedic surgery
- Liver transplant.

Contraindications

- Blood taken from infective sites.
- Blood containing significant amount of amniotic fluid.
- Blood containing would irrigant.
- Blood that may possibly contain malignant cells.

Complications

- Haemolysis
- Renal failure
- Coagulopathies
- Air embolism, fat embolism, amniotic fluid embolism, etc.

Postoperative Blood Salvage

After cardiopulmonary bypass, blood can be collected from the heart-lung machine and can be transfused after processing.

Blood shed from mediastinum after cardiac surgery can be salvaged and reused.

Post-traumatic Salvage

Following chest or abdominal trauma (haemothorax, haemoperitoneum as in ruptured liver, spleen, ruptured ectopic) blood collected in serosal cavities is devoid of fibrinogen and hence does not clot. This can be salvaged and autotransfused.

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29 Acute Normovolaemic Haemodilution

DEFINITION

Acute normovolaemic haemodilution refers to rapid removal of blood and simultaneous replacement with acellular fluid which is employed in surgical patients.

Haemodilution can occur spontaneously after haemorrhage and can result from fluid therapy.

The aims of normovolaemic haemodilution are to:

- Reduce the red cell loss when haemorrhage occurs.
- It provides a practical means of obtaining fresh whole blood for transfusion.
- Tissue perfusion is usually improved with haemodilution because blood viscosity is reduced.

PHYSIOLOGICAL EFFECTS

Oxygen delivery, the produce of cardiac output and arterial oxygen content is usually not affected by haemodilution, even when the haematocrit is markedly reduced to 20%.

- When haematocrit decreases to 21%
- Cardiac output increases by 16 to 36%.

  The increased cardiac output is due to:
  - Decreased peripheral resistance.
  - Increased stroke volume.
  - Increase in venous return.
  - Increase in myocardial contractility.

Although increase in total and local blood flow rates are sufficient to maintain oxygenation in the resting normovolemic moderately haemodiluted individuals, other mechanisms may be involved with severe degrees of haemodilution which include increased oxygen extraction and reduction of haemoglobin oxygen affinity and shift of the oxygen dissociation curve to the right.

But the margin of safety is decreased in patients with coronary artery disease.

PATIENT SELECTION

Haemodilution should be considered in patients with adequate haematocrit who are expected to lose more than 2 units of blood intraoperatively.

Haemodilution can be considered in healthy adults, small children and in elderly. An added advantage is some members of Jehovah's witness faith will agree to haemodilution if blood is maintained in a closed circuit system with continuous flow.

Technique

The amount of blood to be withdrawn depends upon the patients estimated blood volume, preoperative haematocrit and lowest haematocrit desired. As much as 4 units can be withdrawn in some patients.

$$\text{The volume withdrawn} = \frac{EBV \times [H_O - H_F]}{H_{av}}$$

EBV = Estimated blood volume
H_o = Original haematocrit
H_F = Minimum allowable haemotocrit
H_{av} = Average haematocrit.

When blood is withdrawn, the normoavolemia is maintained by either crystalloid or colloid infusion. When crystalloid is used, the amount must be three times the blood withdrawn and in case of colloid the amount administered equals the amount of blood withdrawn.

The blood withdrawn should be kept in the same operating room as the patient and maintained at room temperature to preserve maximum platelet function. If it is anticipated that more than 6 hours will elapse prior to reinfusion, refrigeration of blood is necessary. The blood is reinfused after major blood loss has occurred or sooner, if indicated. Estimation of blood loss and serial haematocrit determinations are used to guide transfusion therapy.

Complications

Myocardial ischaemia and cerebral hypoxia are the major potential complications of haemodilution. The augmented cardiac output increases the myocardial oxygen consumption and hence tachycardia and hypovolaemia must be avoided.

Contraindications

- Anaemia is a major contraindication to haemodilution. Hb reduces 1 gm/dl for each unit of blood removed, it is usually inappropriate to employ the technique when Hb is less than 11 gm/dl.
- Patients with coronary artery disease are not ideal candidates because an increase in cardiac output is not desirable.
- Severe carotid artery disease is also a relative contraindication.

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30 Regional Anaesthesia in Children

Regional anaesthesia produces profound pain relief without narcosis in the immediate postoperative phase.

Why is it Better than GA?

- Provides good perioperative analgesia thus reducing general anaesthesia requirement.
- Children return to the alert state earlier, and parents feel happy that, they can feed them earlier; children can be discharged earlier which reduces emotional trauma.
- The chance of respiratory complications like sleep apnoea are less after regional techniques.
- It provides optimal postoperative pain management.
- Complications of parenteral medication like vomiting is less. Out of the various regional techniques, infiltration anaesthesia still holds prime of place followed by caudal block and various types of other blocks (dorsal nerve of penis, ilioinguinal N block, brachial block, wound instillation with LA, etc). The studies regarding the safety of spinal block are not yet complete.

Caudal block: This is very commonly practised in children because (i) sacral hiatus is wide and easily identifiable in children, (ii) sacral cornua are more prominent, (iii) epidural fat in children has more space unlike—in adults. These factors make caudal block much easier compared to adults, and (iv) spread of local anaesthetics is also more predictable.

Technique: Induce light general anaesthesia, place the child in right lateral position and do skin preparation and draping. The index finger of left hand is moved around from the tip of coccyx, till the coruna are felt which defines the hiatus. Introduce a 23 SWG needle 60° to the coronal plane, when a distinct "pop" is felt needle is advanced at an angle of 20° to pierce the membrane 2-3 mm more.

Agents: Two commonly used local anaesthetics are 1% xylocaine and 0.25% bupivacaine.

Two accepted formulae for the drugs are:

Schulte-Steingerg and Rahlfs– 0.1 ml/dermatome/year of age.

Armitage

- 0.5 ml/kg for procedures over perineum and lower limb
- l.0 ml/kg for procedures over lower limb and lower abdomen
- 1.5 ml/kg for upper abdominal procedures.

Indications

- To reduce the intraoperative need for potent inhalational agent when combined with GA.
- To produce excellent postoperative analgesia. Local anaesthetics like bupivacaine can produce a motor blockade up to a period of 100 min but offers pain relief for 5 to 6 hours. Combination of local anaesthetic along with narcotic can extend the duration of analgesia even up to 24 hours.

- For excellent intraoperative analgesia for procedures like circumcision, inguinal hernia, orchidopexy, anoplasty, etc.

Complications

- Dural puncture, especially in infants where dural sac can extend up to 4th sacral vertebra. It can be avoided by limiting the advance of the needle 2-3 mm inside the canal.
- Intravascular injection
- Subperiosteal injection results in severe pain.
- Cardiovascular collapse/CNS toxicity: from intravascular injection or a toxic dose
- Perforation of the rectum.

BLOCK OF DORSAL NERVE OF PENIS

Principle

Mainly used for circumcision and repair of hypospadiasis. The penis is supplied by ilioinguinal nerve and genitofemoral nerve at the base while distal 2/3 is supplied by branches of pudendal nerves (S2-S4 segment). The nerves emerge through Buck's fascia at the base.

Technique

Two dorsal nerves are situated at the 10.30 and 1.30 position with reference to the root of penis. This is approached through a midline technique and inject 2 ml of 0.5% bupivacaine without adrenaline for each nerve into the Buck's fascia. This will provide sufficient pain relief well into the postoperative period without the need for narcotic. Two separate injection at 10.30 and 1.30 positions can also be made deep to the Buck's fascia.

NERVE BLOCK FOR HERNIA REPAIR

This is used for postoperative as well as intraoperative pain relief for patients undergoing hernia repair, hydrocoelectomy or orchiopexy. Achieved by blocking ilioinguinal and iliohypogastric nerve which are branches of 1st lumbar segment. Ilioinguinal nerve runs between transversus abdominis and internal oblique muscle while iliohypogastric traverses more superficially in between external and internal oblique muscle.

Technique

A point 1.25 cm medial and inferior to the anterior superior iliac spine is chosen. A 24 SWG needle is used to puncture the skin, external oblique and internal oblique characterised by three distinct pops. Inject 0.5%, bupivacaine with 1 in 200000 adrenaline in a dose of 0.4 ml/kg. This block is being commonly practised for day care surgery procedures. Provides excellent post- operative analgesia as well.

SUBARACHNOID BLOCK

Recent studies by Abajian and Rice found spinal anaesthesia extremely safe when they administered it to high-risk infants and ex-prematures who had been treated for respiratory distress. This technique

is of great interest in the sense that prematures are more prone to life threatening apnoea, bradycardia as well as periodic breathing after GA. The suggested dosage regimen is 'heavy' Bupivacaine (1%) in a dose of 0.3 ml for neonate and 0.4 ml to 0.6 ml for "older children". A midline approach at L_5–S_1 interspace is the preferred site for the puncture. It has been observed that heart rate, blood pressure and blood gases usually remain steady throughout the block period. Respiratory depression leading to apnoea is common. This may necessitate intubation and controlled ventilation which may obviate many of the advantages claimed.

IVRA

It is an accepted technique in children because it is easily performed. It provides rapid onset of action, excellent muscle relaxation followed by early resolution of action. The duration of analgesia is related to the tourniquet time.

Procedure

With patient in supine position two pneumatic tourniquet are applied on the extremity. A 20 or 22 SWG cannula is inserted into a vein close to the site of operation. The limb is elevated and exsanguinated using Esmarch bandage, proximal cuff is inflated to about 50 mmHg above measured systolic BP. 0.5% lignocaine without adrenaline in a dose of 0.8 to 1 ml/kg is injected. Action starts within 3 to 5 min. The distal tourniquet can be inflated, and the proximal is deflated to avoid tourniquet pain.

The problem with this technique is premature or accidental deflation of cuff, can result in systemic toxicity of the drug. If the procedure lasts for less than 30 min, the cuff deflation should be done in steps. Any way this block is good for upper limb fracture reductions in children especially in the child with full stomach.

BRACHIAL PLEXUS BLOCK

The axillary route is preferred to the supraclavicular route.

Technique: Child is placed in the supine position with arms abducted to 90°. Arterial pulsation is located as high as possible in the axilla. A short needle is introduced at an angle of 45° to skin and parallel to the artery and the needle is slowly advanced to axilla. A distinct pop can be felt when it pierces the neurovascular bundle. During his time the unsupported needle should pulsate. Recommended volume of local anaesthetic is 0.3 ml/kg 1% lignocaine or 0.5% bupivacaine with 1 in 400,000 adrenaline.

This technique is being widely used for closed reduction of fractures, microvascular surgeries and cardiac catheterization, etc.

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31 Trigeminal Neuralgia (Tic Douloureux)

Trigeminal neuralgia is characterised by sudden attacks of brief but intense unilateral facial pain triggered by local sensory stimuli to the affected side of the face. The pain is precipitated by touching the "trigger zones" of the affected side of the face. The various stimuli which may be sufficient to bring an attack are a cold wind blowing across the face, washing the face, chewing or even talking. The 2nd and 3rd divisions of the trigeminal nerve are usually involved.

Trigeminal neuralgia most often manifests after 50 years of age. Appearance before 50 should arouse the suspicion of multiple sclerosis. This is consistent with histologic evidence of degeneration or absence of myelin along the trigeminal nerve. The aetiology of these changes is unknown, but proposed mechanisms include viral infection and vascular compression of the nerve.

TREATMENT

Medical

Trigeminal neuralgia is an example of the central pain state and stimuli producing pain is believed to arise from within the CNS. Pain is probably caused by some defect in the transmission system whereby intermediate cells generate aversive, afferent stimuli. This condition resembles abnormal epileptic activity on the sensory input side of the nervous system and requires specific drug therapy. Conventional analgesics and sedatives provide little relief for the condition.

The most appropriate medications are phenytoin and carbamazepine (drug of choice).

Phenytoin: 100 ng tds. Most patients responds favourably to this treatment alone.

Carbamazepine: Refratory cases respond to carbamazepine starting with 200 mg a day, increasing by 200 mg increments to a maximum of 1500 mg a day.

Medical therapy is usually effective for 85% of patients with trigeminal neuralgia.

Thermocoagulation

Thermocoagulation of neural elements has been most effective in patients with trigeminal neuralgia who do not respond to medical therapy.

Thermocoagulation of gasserian ganglion is accomplished by percutaneous passage of a probe through the infratemporal fossa and foramen ovale using the standard clinical approach for injection of the gasserian ganglion. Light intravenous analgesia and sedation are usually necessary (e.g. fentanyl 50 mg and droperidol 2.5 mg). By means of a stimulating current, the electrode is localised in the part of the ganglion corresponding to the tic pain, patient confirming the location. The patient is then given a short acting general anaesthetic (usually with inhalation of nitrous oxide) to

relieve pain from creation of the thermal lesion. Because of its effectiveness and low incidence of side effects, this technique is now preferable to alcohol block of gasserian ganglion. The patient is spared hemifacial numbness with its conjunctival inflammatory problems.

A new device cryoprobe, uses expansion of nitrous oxide to produce an ice ball at the tip of the probe. These instruments disrupt sensory nerve function for several weeks and do not seem to be associated with the denervation neuralgia that often follows destruction of nerve with neurolytic agents.

Trigeminal Nerve Block

Trigeminal nerve divides into 3 main branches in the middle cranial fossa. These divisions, the ophthalmic, maxillary and mandibular nerves, provide sensation to the eye and forehead, midface and upper jaw and lower jaw respectively. With the exception of motor fibers to the muscles of mastication carried by mandibular nerve these nerves are wholly sensory.

Gasserian Ganglion Block

Approached classically through the foramen ovale. In the past, was primarily applied to the diagnosis and treatment of trigeminal neuralgia. However, the increasing popularity and safety of thermocoagulation have rendered neurolytic block obsolete.

Advantages: Blockade of 2nd and 3rd divisions of trigeminal nerve or its peripheral branches are still used in management or diagnosis of trigeminal neuralgia.

Disadvantages: 1. Protective corneal sensation may be lost. 2. Block of maxillary nerve can be associated with haematoma formation as well as spread of local anaesthetic solution to involve gasserian ganglion. 3. Spread of local anaesthetic solution to the optic nerve can cause temporary blindness. 4. In mandibular nerve block pharynx may be entered, increasing the risk of contamination of the infratemporal fossa. 5. Rarely subarachnoid spread of local anaesthetic producing brain stem anaesthesia.

Technique: Mandibular and maxillary nerves are blocked through the same needle site entry.

Maxillary Nerve

The coronoid notch of the mandible is located and with the mouth closed, a 22G - 8 cm needle is inserted at the inferior edge of the coronoid notch perpendicular to the skin. The needle will contact the lateral pterygoid plate at a depth of about 5 cm. It is then withdrawn and redirected anteriorly and superiorly to 'walk off the plate and is advanced 0.5 cm into the pterygopalatine fossa. 3-5 ml of local anaesthetic solution is injected.

Mandibular Nerve

Blocked via the same entry site. Bone of lateral pterygoid plate is contacted. The needle is withdrawn and redirected along the posterior border of the pterygoid plate. The needle should not be inserted more than 0.5 cm past the plate. 3-5 ml of solution is injected.

Other Surgical Approaches

- Selective radiofrequency destruction of trigeminal nerve fibres
- Microsurgical decompression of the trigeminal nerve.

Neurolytic Blockade: Considerations

Because peripheral nerve destruction with alcohol or phenol is frequently followed by denervation dysaesthetic pain that is often as bad, if not worse than the original pain, this type of therapy has now been almost abandoned, incidence of post-neurolytic neuralgia is least after the neurolytic therapy of branches of trigeminal nerve.

Alcohol and phenol are the most widely used agents. Injection of alcohol is very painful, lasting for a few seconds until neurolysis occurs. Injection of phenol which is usually mixed with saline or glycerine is painless. Neurolytic effect of alcohol is more intensive than phenol which has both local anaesthetic and neurolytic action. No permanent neurolytic block is truly permanent and sensation and pain returns within weeks or months. Diagnostic nerve blocks should always precede the use of neurolytic agents for the patient to evaluate the final outcome of therapy.

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32 Coeliac Plexus Block

ANATOMY

Coeliac plexus lies anterior to aorta at the level of 1st lumbar vertebra. Nerve fibres reach the plexus from:

- Preganglionic—sympathetic fibres from splanchnic nerves.
- Preganglionic parasympathetic from vagus.
- Some sensory fibres from phrenic and vagus.
- Afferent fibres concerned with nociception.

There are three pairs of splanchnic nerves which descend to the coeliac plexus. They are (i) greater splanchnic nerves arises from T5-T9 (sometime T4 and T10 also) spinal segments, (ii) lesser splanchnic nerves arises from T10 and T11 spinal segments and (iii) least splanchnic nerve arises from T12 spinal segment. These nerve fibre synapse in the ganglia of coeliac plexus. There are 3 pairs of ganglia exist within the plexus. They are:

- Coeliac ganglia.
- Superior mesenteric ganglia.
- Aorticorenal ganglia.

The nociceptive afferent fibres travel from viscera along the sympathetic fibres, through the ganglia, splanchnic nerve, sympathetic chain, white rami communicants and then synapse in the dorsal root ganglia. The proximal axon of these cell bodies synapse in the dorsal horn of spinal cord.

PHYSIOLOGY OF COELIAC PLEXUS BLOCK

Blockade of Afferent Nociceptive Fibres

Any pain originating from visceral structures innervated by coeliac plexus can be effectively alleviated by this blockade. This include pancreas, liver, gallbladder, omentum, mesentery, alimentary tract (from the stomach to transverse colon).

Blockade of Sympathetic Fibres to GIT

The complete sympathetic denervation of GIT allows unopposed parasympathetic activity and increased peristalsis, increase in gastric motility and relaxed spincters lead to increased gastric emptying. Diarrhoea may develop. Nausea and vomiting seen in GIT malignancy may be abolished after coeliac plexus block.

Blockade of Sympathetic Fibres to Splanchnic Vessels

Secondary to vasodilatation of large splanchnic bed, hypotension may develop after well placed coeliac plexus block.

DRUGS USED FOR COELIAC PLEXUS BLOCKADE

- Local anaesthetic alone
  - 0.5%, sensorcaine with 1:200,000 dilution of adrenaline
  - 15 ml on each side (Total 30 ml)
  - Used in non-malignant conditions.
- Local anaesthetic + steroid
  - Commonly used steroid is beta methazone
  - In chronic abdominal pain following multiple surgeries it will give good results.
- Neurolytic blockade
  Most commonly used compounds are alcohol or phenol.

Alcohol

- 50-100% used
- *Disadvantage:* Immediate severe pain.

  This can be abolished by using 0.5% bupivacaine and absolute alcohol at a ratio of 1:1 (Total amount of 50 ml to be used 25 ml on each side).

Phenol

Used as 6% aqueous solution or 12% phenol prepared in renografin.

INDICATIONS

- Pain relief in upper abdominal malignancies originating from stomach, pancreas, gallbladder and liver. Neurolytic block is indicated.
- Pain after multiple abdominal surgeries. Local anaesthetic and steroid is usually indicated.
- For evaluation of upper abdominal pain local anaesthetic alone is indicated.
- Non-malignant pain of upper abdomen, e.g. chronic pancreatitis.

  Here also neurolytic block is indicated. But its long-term effect is variable. Regeneration of new pathways result in development of different pain syndromes in these patients after 6 to 12 months.

TECHNIQUE

- Preparation
  - Informed consent to be obtained
  - I/V cannula to be introduced and start an I/V infusion
  - Resuscitation equipments and drugs must be readily available.

- Position
  Prone position with a pillow under the lower abdomen to remove lumbar lordosis. Block can be performed in lateral position also, but needle placement is difficult.
- Premedication
  I/V premedication for patients who are anxious and patients with severe pain.
  Avoid premedication if the block is preferred for diagnostic purposes.

PROCEDURE

Sterility has vital importance.

Land marks to be marked are:

- Spin us process of T12, and T11 vertebrae
- Inferior border of 12th rib
- Site of needle entry is 7-8 cm lateral to the spinous processes in the inferior border of 12th rib.

Needle used: 12-18 cm long 20-22 G needle with an attached skin marker.

Infiltrate the skin and muscle with local anaesthetic solution. Introduce the needle at the point of needle entry and advance. The direction is towards the L1 spine and proceeds to hit on the 11 vertebral body. After the vertebral body has reached, a skin marker is placed on the needle, 2 to 3 cm from the skin.

The needle is then withdrawn to the subcutaneous plane and make minor adjustments and reintroduce lateraly until it just slips from the lateral surface of the vertebral body. After the lateral aspect of vertebral C body has passed a "POP" is often felt when the needle passes through the psoas fascia. At this point needle approaches the great vessels and should advance slowly. The needle should pass 2 to 3 cm beyond the lateral part of vertebral body.

After proper placement of the needle a careful aspiration is performed. If blood is encountered, the needle is slowly withdrawn until negative aspiration is achieved. Alternatively, on the aortic side the needle can be advanced through the aorta until no blood is returned, and injection is made at this location.

Same procedure can be repeated on the other side also. After proper needle-placement, 2 to 3 ml of local anaesthetic containing epinephrine is injected for further test of either I/V or intrathecal placement. If this is negative full dose of local anaesthetic or neurolytic drug can be injected.

Image intensification techniques like radiography, fluoroscopy and CT scan increase the accuracy of coeliac plexus block.

Catheter placement can be done in non-malignant conditions and can give local anaesthetic infusion for prolonged blockade.

Single needle approach using left side needle and injecting the drug posterior and anterior to aorta is also described.

COMPLICATIONS

- Due to blockade of sympathetic fibres:
  Hypotension can be avoided or minimised by giving I/V infusion of 500 to 1000 ml of RL before the block. Orthostatic hypotension may persist after a neurolytic block. But in most cases, it is self-limiting with in a week.
- Complications due to the technique
  - Backache
  - IVD damage
  - Injury to blood vessels → Retroperitoneal haematoma
  - Injury to spinal nerves
  - Injury to kidney → Renal haemorrhage.
    → Nephrocutaneous fistula.
- Complications due to local anaesthetic/neurolytic agent
  - I/V injection can lead to toxicity
  - Intrathecal injection can produce total spinal block.

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33 Patient Controlled Analgesia

INTRODUCTION

Patient controlled analgesia (PCA) is an intermittent I/V administration of analgesic under direct patient control with use of a special device.

Intramuscular administration of opioids on demand is the method used more commonly for postoperative analgesia. Most of time the pain relief is inadequate. Almost 60% of patients report dissatisfaction with the quality of postoperative analgesic administered in this way.

To counter this drawback bolus I/V administration and continuous I/V infusion came into practice. But the problems of overdosage, fatal respiratory depression and cannot be undertaken by nursing staff etc. are major disadvantages.

Now with the introduction of PCA, most of its drawbacks due to I/M or I/V bolus dose of opioids can be prevented. The analgesia produced by this method is excellent without any fear of over dosage.

PRINCIPLE

To understand the principle of PCA we must know the relation between opioid concentration in blood and analgesia. The analgesia is obtained when the plasma opioid concentration, reaches a particular level depending on the individual patient. After that a small increase in plasma opioid concentration causes a rapid decreases in pain. At a particular minimum opioid concentration patient cannot appreciate pain. This is called the minimum effective analgesic concentration (MEAC). By increasing concentration, above this MEAC there is no further increase in intensity of analgesia. But for a small decrease in concentration below MEAC there is rapid perception of pain.

There is considerable interpatient variability of MEAC. This will explain the large interpatient variability in analgesic requirement. So, only patient can determine when and how much analgesia is required. This is the basis of PCA.

Figure 33.1 gives a good idea regarding the difference in conventional intermittent 4th hourly I /M opioid and PCA.

Originally, PCA was introduced for intermittent I/V administration of opioids only. Now PCA is used for opioids, local anaesthetic, benzodiazepines, etc. Similarly, apart from I/V route, transbuccal, subcutaneous and epidural routes of administration have been used for PCA.

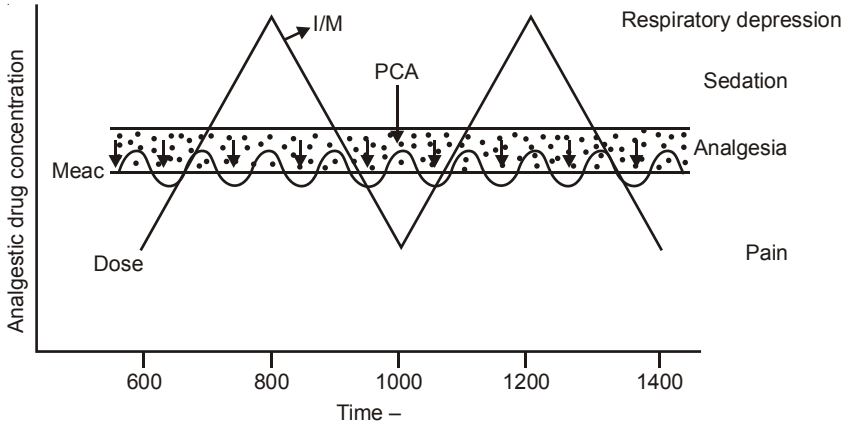

Fig. 33.1: Recommended demand dose and lockout interval for some commonly used PCA regimes

PCA DEVICES

The PCA device basicaly comprises a microprocessor controlled pump triggered by a depressing button. When triggered a pre set amount of narcotic is delivered into patient's intravenous line. At times the pump prevents the administration of an additional bolus until a specific period has elapsed. Thus, individual patient, titrate narcotics to their own needs with in safe clinical parameters.

Cardiff palliator was the first commercially available PCA device. But now so many PCA devices are available with different sophisticated modes and basic variables. We must have a general idea regarding the different modes available and different basic variables before using a PCA devise.

PCA MODES

- Demand dosing mode: Where a dose of fixed size is self administered. This is the most common mode of PCA administration.
- Constant rate infusion plus demand dosing mode: This implies administration of a minimum background infusion determined by a physician which can be supplemented by patient demand. When this mode is used it is important to maintain the background infusion at a minimum, in order to not ablate the necessity of patient's demand; otherwise the risk of respiratory depression is enhanced.
- Infusion demand mode: In this the patient's demand are granted as an infusion. This type of mode is less commonly available.
- Variable rate infusion plus demand dosing mode: This is the most sophisticated node and this to be studied in detail. In this a microprocessor monitor demand and controls the infusion rate accordingly.

  For each mode described above have basic variables.

BASIC VARIABLES IN PCA

- *Loading dose:* This is the dose of opioids required to reach the plasma opioid concentration to the minimum effective analgesic concentration (MEAC) for that individual. For postoperative patient, because most of the patients are on opioid during intraoperative period, there is no need of loading dose. When the patient sufficiently recover from anaesthesia he may initiate patient demand.
- *Demand dose:* This is the quantity of analgesic given to the patient on activation of the PCA infuser's demand button. This is also called incremental dose or PCA dose.
- *Lockout interval:* The lockout interval is the length of time between patient demands as which the infuser will not administer analgesic. To prevent over dosage by continuous demand, all PCA systems use a lockout interval (Table 33.1).
- *1 and 4 hours limit:* Some modern PCA devices allow determination of total dose at 1 and 4 hr and limit the further dosage.

Table 33.1: Recommended demand dose and lockout interval for some common opioids used in PCA

Drug and concentration	*Demand dose (mg)*	*Lock out interval (min)*
Morphine 1 mg/ml	0.5-3	5-12
Fentanyl 0.01 mg/ml	0.01-0.02	3-10
Pethidine 10 mg/ml	5-30	5-12

ADVANTAGES OF PCA

- Dose matches patients requirements and is also well compensated for pharmacodynamic variation.
- Dosage given are small and so fluctuation in plasma concentration is reduced.
- Reduces work load on nursing staff.
- Less pain.
- Placebo effect from patient autonomy.

DISADVANTAGES OF PCA

- Technical error may.be fatal.
- Expensive equipment.
- Requires cooperation and understanding from the part of the patient.

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34 Noninvasive Methods to Assess Ventricular Function

Over the past decade, the number of new techniques available to assess and monitor the patient with circulatory failure has increased dramatically. Some of these techniques are widely available, inexpensive, portable and simple to use, others are available in a few centres, require expensive stationary equipments and trained technicians or present special hazards like radiation.

With recent developments in echocardiography and computing power, non-invasive methods offer hope of continuous monitoring of ventricular function with minimum danger or complications.

CARDIAC RHYTHM

Electrocardiogram: ECG is the device routinely used to detect arrhythmias. Bipolar lead II and unipolar leads afford easy detection of arrhythmias. Precordial or oesophageal stethoscope also can be used for this purpose.

MECHANICAL FUNCTION OF THE VENTRICLES AND PERIPHERAL VASCULAR SYSTEM

Heart Sounds

Monitored continuously using a precordial or oesophageal stethoscope, we can get some crude estimation of cardiac output from such monitoring. Loudness of Ist heart sound is correlated with the force of contraction of the ventricles. So it gives information regarding the stroke volume. IInd heart sound gives some information about the after load.

Arterial Pressure

Many methods are there for measurement of arterial blood pressure non- invasively.

- Auscultation of 1st and 4th Korotkoff sounds.
- Palpatory method.
- Doppler ultrasound technique
- Oscillometry technique.

Provided there is no mechanical obstruction to ventricular outflow, arterial systolic pressure is same as the peak systolic pressure in the ventricle and is an index of the force of contraction and hence stroke volume.

Arterial diastolic BP gives an estimate of the after load or impedence to ventricular ejection. Since myocardial blood flow occurs mainly during diastole, diastolic pressure, gives idea about myocardial perfusion pressure.

The product of systolic pressure (in mmHg) and heart rate (beats/min) known as rate pressure product correlates with myocardial O_2 consumption.

CVP

CVP is a reflection of the ability of the right heart to cope with venous return at the moment of examination. For non invasive measurement of CVP an ultrasound probe is placed over the external or internal jugular vein. The venous flow is usually heard as a "hum", where as the arterial flow signal has a pulsatile nature to it. The airway pressure is then raised either by asking the patient to blow into a manometer or in case of the anaesthetised patient, by squeezing the reservoir bag of circuit. The CVP roughly corresponds to the airway pressure at which jugular venous flow ceases.

A sudden elevation of CVP associated with a decreasing cardiac output or a decreasing arterial pressure is a sign of acute ventricular failure or tamponade.

CARDIAC OUTPUT

Noninvasive techniques to measure cardiac output includes:

- Doppler cardiac output measurement: Out of the non-invasive techniques, this is the most practical and reproducible method. A probe which emits as well as receives sound waves of frequencies between 2 MHz and 10 MHz is directed towards the descending aorta either across the oesophagus or through the suprasternal notch. The emitted sound waves are reflected by red cells, which are moving away from the receiver. Frequency of the reflected sound is less than the frequency, of the emitted sound. This frequency shift is a function of the velocity of the red cells' and the blood flow velocity is easily measured.

  $$\text{Flow (ml/s)} = \text{Flow velocity (cm/s)} \times \text{Cross-sectional area of aorta (cm}^2\text{)}$$

  Cross-sectional area of aorta is usually derived from the patient's height, weight and body surface area using a nomogram or formula. Currently devices are available for continuous monitoring of cardiac output which works on the Doppler principle.

  Instead of descending aorta, flow velocities can be measured at the mitral annulas or left ventricular outflow tracts by a transoesophageal ultrasound probe.
- Cardiac output by gas exchange analysis.
- Roentgen densitometry cardiac output.
- Echocardiographic (M mode echo) estimation of cardiac output.

ECHOCARDIOGRAPHIC ESTIMATION OF LEFT VENTRICULAR SIZE, FUNCTION AND REGIONAL WALL MOTION

The major uses of echocardiography are:

- Measurement of left ventricular volume and function.
- Analysis of left ventricular regional wall motion abnormalities.

- Determination of stroke volume and cardiac output.
- Estimation of left ventricular filling pressures.
- Determination of intracardiac shunts.
- Detection of pericardial effusion.
- Estimation of pulmonary artery pressure.

M mode echocardiography: M mode (Motion) echocardiography (MME) is the study of cardiac structures moving in one plane, forward and away from the transducer with time. A Piezo electric crystal is used for the production of a beam of sound waves of high frequency. The time taken for the sound waves to come back after reflection is a measure of the depth of the structure.

The most important limitation of MME is its singular 'ice-berg' view of the heart, which permits visualisation of only a small part of any chamber at a time.

Two-dimensional (2D) echocardiography: It overcomes most of the limitations of MME, instead of emitting sound waves the 2DE transducer emits a wedge shaped 30 to 90 degree plane of sound waves. The motion of structures is recorded at 30 frames per second as movement away and toward the transducer as well as one dimension perpendicular to it.

Left ventricular cavity usually assumes the approximate shape of a polate ellipse, with 3 planes one long axis and two short axes. A transoesophageal probe enable measurement of left ventricular diameters, at various stages of cardiac cycle (left ventricular internal dimension systole (LVIDs) and left ventricular internal dimension diastole (LVIDd).

The fractional shortening of left ventricle measures the extent of left ventricular contraction along the micro axes.

$$\% \text{ SF} = \frac{100 \times (\text{LVIDd-LVIDs})}{\text{LVIDd}}$$

The mean V_{CF} is the ratio of LV fractional shortening to ejection time (ET).

$$V_{CF} = \% \text{ SF/ET}$$

Left ventricular volume can be calculated using 2D echocardiography. Using a 4 chamber image the LV endocardial surface may be traced and the area obtained by planimetry. The length of the LV major axis is measured and a regression equation applied to calculate LV volume.

Ejection fraction (EF) is the fraction of the end-diastolic volume (EDV) that is ejected during each systole.

$$\text{EF} = \frac{\text{EDV-ESV}}{\text{EDV}}$$

This is a very good index of left ventricular function. Regional wall motion abnormalities are usually, seen after myocardial infarction. Abnormal wall movements indicate poor ventricular function.

FLOW PATTERN USING COLOUR DOPPLER

Direction of flow of blood can be detected using Doppler principle. By noting the frequency shift, the device detects whether the flow is towards the receiver (displayed in red colour) or away from the receiver (blue). Such colour flow imaging is very useful for quantitative assessment, of abnormal flow patterns that occur across incompetent valves or septal defects.

RADIONUCLEIDE IMAGING

Radio-isotope evaluation of ventricular function includes:

- Analysis of ventricular function by labelling the blood pool within a chamber. Technetium 99m pertechnetate (Tc 99m) is the commonly used radio-isotope, which is given intravenously and radioactivity within the cardiac chambers detected using a scintillation camera. The intensity of radioactivity is proportional to the volume of blood in the chamber.
  Nuclear cardiac probe (nuclear stethoscope): This is a new tool in radionucleide cardiac imaging, provides a continuous beat by beat analysis of LV function.
- *Myocardial imaging*: Using Thallium 201, helps diagnosis and sizing of acute myocardial infarction and diagnosis of coronary artery disease on the basis of perfusion defects which appear during resting studies or following exercise.
  99m Tc concentrate in zones of ischaemic or infarcted myocardium and display "hot spots".

MYOCARDIAL METABOLIC STATE

Metabolic derangements such as ischaemia and electrolyte abnormalities affect the mechanical function of the heart. ECG has been used to detect these derangements. A two lead system consisting of lead 11 and V chest lead has been advocated for detecting ischaemia. Elevation or depression of ST segment greater than 1 mm and lasting longer than one minute is indicative of ischaemia.

Computer-aided ST segment analyser facilitates detection of ischaemia easier.

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35 Advanced Life Support

Advanced life support (ALS) is the second phase of the cardiopulmonary resuscitation, which involves the restoration of circulation. After the 'A' 'B' and 'C' of resuscitation, ALS measures are designated 'D', 'E' and 'F'. Which stands for:
D—Drugs and fluids
E— ECG monitoring and endotracheal intubation (if not already done)
F—Fibrillation treatment.

DRUGS AND FLUIDS

An essential part of ALS is prompt establishment of an intravenous infusion for reliable delivery of drugs and fluids into the circulation. Whenever possible it is advised to administer the drugs through a centrally placed catheter, to avoid the delay in reaching heart.

Drugs Used

Adrenaline

Action of adrenaline that helps in cardiac arrest is:

- Alpha-adrenergic stimulation-increases peripheral vascular resistance which will raise systolic and diastolic pressures during external cardiac compression. The raised diastolic BP increases coronary blood flow (CPP= Aortic diastolic pressure – LVEDP). This will facilitate return of spontaneous cardiac contractions.
- In cardiac asystole and in electromechanical dissociation adrenaline may restore spontaneous cardiacaction.

Dose: 5 to 10 mcg/kg intravenously
This is given diluted in 5 to 10 ml every 5 minutes of arrest time 4 dosage schedule.
Standard – 1 mg IV push every 3-5 minutes
Intermediate – 2-5 mg IV push every 3-5 minutes
Escalating – 1 mg, 3 mg, 5 mg IV push every 3-5 minutes
High dose – 7 mg, 100 mg/g IV push every 3-5 minutes
Disadvantage of IV route: Since the circulation will be slow, it will take some time to reach the heart to exert action (if given through peripheral vein). But a bolus dose of fluid can be used to 'flush' the drug into the circulation.

Other Routes

Intracardiac injection: Although it is the most effective way of achieving an adequate blood concentration of adrenaline, its use is not favoured because of complications which include:

- Accidental injury to coronary vessels - hemo-pericardium which if go undetected will be the reason for failed resuscitation.
- Accidental injection into the cardiac muscle. Avascular area which will behave like an infarcted muscle.
- Pneumothorax
- Cardiac tamponade.

Because of these complications, intracardiac injection is recommended only as a last resort when the IV or tracheal route is not available. *Site*: 4th Left intercostal space close to sternum.

Intratracheal: If an endotracheal tube is in place adrenaline may be instilled into the endotracheal tube. It is supposed to get absorbed fast from the lungs. Recent studies have shown that endotracheal instillation may not be very effective; twice the IV dose is recommended.

Sodium Bicarbonate

There is considerable controversy concerning the routine use of sodium bicarbonate. The administration of sodium bicarbonate in the absence of pH measurement is not recommended. These are specific indications:

- Known pre-existing hyperkalaemia (Class I)
- Known pre-existing bicarbonate responsive acidosis (e.g. diabetic ketosis) or overdose of tricyclic antidepressants cocaine, aspirin, etc. (Class II)
- Prolonged resuscitation with effective ventilation. (Class IIb)
- Not useful or effective in hypercarbic acidosis (e.g. cardiac arrest and CPR without intubation) (Class III)
- IV infusion 1 mEq/kg/IV bolus, repeat half this dose every ten minutes thereafter.

Vasopressin

- May be used as an alternative to adrenaline in the treatment of shock refractory VF
- May be used for haemodynamic support in vasodilatory shock.
- Dose 40 μ IV push is the only route recommended. Tracheal route not recommended.
- Not indicated in patients with CAD.
- Potent vasoconstrictor and increase in peripheral vasoconstriction may provoke ischaemia/ angina.

Calcium

Calcium should not be used routinely in treatment of cardiac arrest.

Indications

- Known suspected hyperkalaemia (Renal failure)
- Toxicity of calcium channel blockers
- Hypocalcaemia, after multiple blood transfusions.
- Some patients with electromechanical dissociation (doubtful value).

Dose

Calcium chloride 2 ml of 10% solution repeated as necessary. Calcium gluconate 5 to 8 ml of 10% solution repeated as necessary.

Other Drugs

- Lidocaine for recurrent or refractory VF 1 mg/kg IV. Then continuous IV infusion of 15 mcg/kg/min to 60 mcg/kg/min. Lidocaine may increase the defibrillation current required, but it increase the fibrillation threshold.
- Procainamide (when lidocaine is not effective) 0.75 mg/kg/over 5 min. not to exceed 1 gm in an adult.
- Bretylium
  - When lidocaine and procainamide are not effective.
  - Persistant VF despite multiple attempts at external defibrillation. 5 mg/kg IV every 5 min, not to exceed 30 mg/kg.
- Atropine—Bradycardia, 3rd degree AV block, cardiac asystole—70 mcg/kg/IV not to exceed 3 mg.
- Isoproterenol—(when atropine not effective) 0.03 mcg/kg/min. to 0.3 mcg/kg/min.—generally not recommended; it increases myocardial O_2 consumption and lowers the fibrillation threshold.
- Verapamil—paroxysmal SV T-70 mcg/kg IV initially followed in 15 min by 140/mcg/kg if cardiac dysrrhythmias persist.

ECG Monitoring

'E' for ECG monitoring and endotracheal intubation. When a patient is clinically in cardiac arrest, electrically the heart may be in one of the following states:

- Ventricular fibrillation
- Asystole
- Electromechanical dissociation
- Pulseless ventricular tachycardia.

Ventricular Fibrillation

Commonest form of cardiac arrest, coordinated heart beats breaks down totally. Individual muscle fibres depolarise and contract independent of each other.

- ECG - coarse wave form (waves irregular in rate, rhythm and amplitude) or asystole.
- Only definitive R_x is electrical defibrillation.

Asystole

Less frequent form of cardiac arrest than VF. Prognosis for resuscitation is poor. Ventricular standstill due to suppression of pacemaker. Extensive myocardial ischaemia can occur from prolonged periods of inadequate coronary perfusion.

Precipitating factors include myocardial disease

- Electrolyte abnormalities
- Anoxia
- Drugs like β-blockers.

Treatment

- Basic life support.
- Inj. atropine 70 mcg/kg/IV.
- Inj. Adrenaline 0.5-1 mg IV.
- Inj. sodium bicarbonate .

Intracardiac adrenaline or pacing can be considered if there is any evidence of electrical activity. If the cardiac rhythm is unclear, it is advised to electrically defibrillate the patient.

Electromechanical Dissociation

Normal or near—normal electrical excitation. But the electrical activity does not result in cardiac contraction.

A normal ECG in the absence of pulse and BP.

Causes

- Cardiac tamponade
- Pulmonary embolism
- Tension pneumothorax
- Hypovolaemia
- Prognosis is grave. Treat correctable causes.

Pulseless Ventricular Tachycardia

Is treated like VF cardioversion requires less energy than defibrillation (50 joules) but must be synchronised to avoid delivering the shock during the relative refractory period of cardiac cycle.

Fibrillation Treatment

Definitive, effective treatment for VF. Here, a large electrical impulse is delivered to the heart through the chest wall externally or directly if internal. The entire myocardium gets depolarised followed by spontaneous electrical activity.

Most important factor in the success of external defibrillation and the survival of the victim is the length of the interval from cardiac arrest to application of shock. Current recommendations are to apply external defibrillation as soon as VF is identified and a defibrillator if available.

An initial defibrillation setting of approximately 200 J is recommended for adult victim regardless of body weight. If this initial attempt is unsuccessful, a second attempt should be made using energy setting of 200 to 300 J. If the first 2 shocks fail to defibrillate, a third shock not to exceed 360 J should be delivered immediately. If VF recurs, defibrillation should be reinitiated at the energy level that had previously resulted in successful defibrillation.

Paddle electrodes used to deliver current from the defibrillator must be placed in positions that will maximise current flow through the myocardium. Standard placement is with one electrode to the right of upper sternum and below the clavicle, and the other electrode at the level of the apex of the heart in the midaxillary line. The paddles for the electrodes should be applied to the chest with firm pressure equivalent to about 10 kg. Electrodes 8 to 10 cm in diameter are appropriate for infants and children.

Failure of defibrillation — cause may be due to faulty technique, hypoxia, or acidosis.

Endotracheal intubation and hyper ventilation will correct these.

The pulse and cardiac rhythm should be checked after each defibrillation attempt. Epinephrine is repeated every 5 minutes during the resuscitation.

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36 Pulmonary Capillary Wedge Pressure

Pulmonary capillary wedge pressure (PCWP) is one of the most advanced and accurate among the haemodynamic monitoring parameters available at present. Still the highly invasive technique and expertise needed had limited its widespread use.

The importance of PCWP is that it gives a reasonable estimate of left ventricular preload, i.e. Left ventricular end diastolic volume (LVEDV). When ventricular compliance is normal LVEDP can be used as an indicator of LVEDV. Unless there is a pressure gradient across mitral valve (e.g. MS) LAP and hence PCWP is used for the same purpose. Wedging the catheter into a small pulmonary artery causes the blood flow and pressure in the distal branches to cease leaving a static column of blood between catheter tip and LA (acting as conduit, extending the catheter into LA).

For the PCWP to be true reflection of LVEDP certain factors have to be ensured.

- The tip should be in zone III of lung (dependant areas)
- There should not be any mass or embolus in between
- PEEP 10 cm H_2O converts large areas into zone I, and LAP to PCWP correlation may be lost.

CONDITIONS WHERE PCWP DOES NOT REFLECT LVEDP

MS, LA myxoma, ↑ intrathoracic pressure - PCWP > LVEDP Pulmonary venous obstruction, severe LVF - PCWP < LVEDP.

Pulmonary artery diastolic pressure is an indicator of PCWP. But it is influenced by PVR which in turn is altered in hypoxia, hypercarbia, etc. and hence is not always useful. Also with tachycardia, PA diastolic pressure may be >PCWP.

Normal PCWP is 8 to 12 mmHg. Volume infusion to correct a low PCWP will increase CO. But there is an optimum PCWP (18 mmHg) beyond which volume infusion is useless. PCWP of 25 mmHg or more is associated with pulmonary oedema but in chronic LVF patients the value may be higher.

Indications for Measurement

Depends on individual patient.

- LV dysfuction—dilated cardiomyopathy, valvular heart disease, ventricular aneurysm, severe IHD, idiopathic hypertrophic subaortic stenosis.
- Aortic surgery, cardiac surgery.
- Severe pulmonary disease - pulmonary HT, pulmonary emboli.
- Shock—fluid challenge test.

Measurement

Swan-Ganz Catheter—developed in 1970 by Jereony Swan and Henry Ganz. There are 4 lumen:

- For balloon
- For right atrium
- For the tip and
- For thermistor, can also be used for measuring CO, PA pressure, pacemaker insertion.

Sizes are 7 to 7.5 for adult and 5 for children. Balloon capacity is 1.5 ml. Right IJV or left subclavian is the route and is similar to CVP. An introducer kit containing 1.5 cm teflon sheath with a dilator stylet is passed over guide wire into the vessel. The catheter flushed with heparinised saline is introduced upto 20 cm so that curve is slightly leftward, balloon inflated (when in the RA) and further advanced RV 40 cm; pulmonary artery 50 cm and pulmonary capillary 55 cm each showing the characteristic tracing. Balloon deflated to check pulmonary artery wave restoration. X-ray to confirm the position is ideal. Pressure waveforms will change according to position at catheter tip as shown in Figure 36.1.

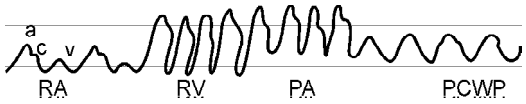

Fig. 36.1: Pressure wave tracings during pulmonary catheter insertion

Complications—due to insertion of catheter presence later.

- Arrhythmias—45 to 79% incidence of .tachyarrhythmias. Heart blocks occur, especially in pre-existing β-blockade.
- Infections—more with increased durations and pre-existing sepsis.
- Catheter knotting—excessive insertions, incomplete inflations, etc. increase risk. It is untied with intravascular snares under flouroscopy or surgically removed.
- Pulmonary infarction—due to persistant wedging, IV caused by excessive insertion.
- Hypotension, $\downarrow PaO_2$
- Pulmonary artery rupture—0.2 to 0.5%. High mortality (45%); erosion by tip, eccentrically inflated balloon driving the tip, etc. cause this. Age>60 predispose, but not atheromas. Haemoptysis, hypoxia, HT, bronchospasm etc. occur. Confirmed by radiography. Treatment include ventilatory support, lateral position with ruptured side up, PEEP to tamponade the bleeding or surgical intervention.

Table 36.1: Fluid challenge test in hypovolaemia using PCWP

Time	*PCWP*	*Action*
Before infusion	< 12	200 ml in 10 min.
	12-15	100 ml in 10 min.
	> 16	50 ml in 10 min.
During infusion	> 7	Stop infusion and monitor
10 min. after infusion	> 3	Stop infusion and monitor
	< 3	Rpt challenge

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37 Intra-Aortic Balloon Pump

Intra-aortic balloon counterpulsation is the simplest and most commonly used form of cardiac assist device. The concept of mechanical support of the failing left ventricle was first proposed by Clause (1961), but it was Moulopoulos in 1962, who devised the modern intra-aortic balloon pump (IABP).

This device produces diastolic counterpulsation with compressed gas driven volume displacement of an elongated catheter mounted balloon which is electronically gated to the patient's ECG. It is introduced from the common femoral artery into the descending thoracic aorta. The inflation is timed with the dicrotic notch of the arterial pressure and deflation is done just before isometric left ventricular contraction thus reducing the afterload.

Previously, it used to be introduced surgically via the common femoral artery. Another route of insertion is the transthoracic approach where it is inserted through a tetrafluoroethylene graft stitched to ascending aorta. The device has become popular with the advent of the percutaneous approach but it has been shown that the incidence of complications is more with the percutaneous approach than the surgical approach.

ADVANTAGES OF IABP

- Physiologically effective.
- Relative ease and safety to use.
- Scope of clinical applicability .
- Useful when rapid reversibility of cardiac damage is expected. Unloading of left ventricle also assists the right ventricular function.

CRITERIA FOR MECHANICAL SUPPORT OF CIRCULATION

Severe left ventricular dysfunction (cardiac index less than 1.7 L/min/m^2, systolic BP less than 100 mmHg, systemic vascular resistance more than 1200 dynes, sec.cm^{-5} mean atrial pressure more than 20 mmHg, heart rate more than 80 beats/min) despite maximum inotropic support. Maximum inotropic support means a combination of two or more of the following:

- 10 mcg/kg/min of dopamine
- 10 mcg/kg/min of dobutamine
- 10 mcg/kg/min of amrinone
- 0.2 mcg/kg/min of adrenaline.

INDICATIONS FOR IABP

- Post-cardiotomy cardiogenic shock
- Acute myocardial infarction with:
  - Cardiogenic shock.
  - VSD with progressive LV failure and shock.
  - Acute papillary dysfunction causing acute mitral regurgitation, LV failure and cardiogenic shock.
- Myocardial ischaemia refractory to medical therapy:
  - Unstable pre infarction angina.
  - Unstable post infarction angina.
  - Refractory ventricular tachyarrhythmias.
- Postoperative cardiogenic shock
- Preopprophylaxis: (i) severe LV dysfunction, (ii) critical left main coronary—stenosis with occlusion of dominant right coronary or unstable angina, (iii) Combined severe valvular and coronary artery disease with severe LV dysfunction or unstable angina.
- Failed coronary angioplasty with unstable myocardial ischaemia.

PHYSIOLOGICAL CHANGES WITH IABP

- Increases aortic diastolic pressure thus improving the coronary perfusion, producing an increase in cardiac output.
- Increases coronary perfusion pressure by:
  - Reducing afterload with increased forward flow.
  - Reducing intramyocardial wall tension.
  - Reducing myocardial oxygen consumption.

Diastolic pressure time index, which is a measure of myocardial oxygen supply is increased so the endocardial viability ratio is improved thus useful in myocardial salvage after acute myocardial infarction.

APPLICATIONS

- Complicated myocardial ischaemia.
- Cardiogenic shock.
- In cardiac surgery.
  - Standby
  - Elective balloon pumping.
- In postoperative cardiac critical care unit.
- In cardiac catheterisation.
- In septic shock.
- In paediatrics.
- Pulmonary artery balloon pumping.
- Trauma and haemorrhagic shock.

COMPLICATIONS

8-30% incidence

- Limb ischaemia.
- Infection.
- Coagulopathy.
- Haemorrhage.
- Aortic dissection.
- Aortic or iliac artery perforation.
- Renal artery embolism or thrombosis.
- Mesenteric infarction.
- Spinal cord ischaemic injury.
- Balloon rupture with gas emboli.
- Air embolism.
- Cerebrovascular accident.

To reduce the incidence of vascular complications

- Heparinise the patient within 2-4 hours and maintain ACT at 1-1½ times the normal.
- Use longer introducer sheath 16 inches.
- Abdominal aortogram to delineate aorto-ilio-femoral artery anatomy to select the preferred-side.
- Use central lumen wire guided IABP.

LIMITATIONS OF IABP

- Not useful when the left ventricle is not able to eject blood into aorta.
- Not very effective in irregular and fast cardiac rhythms, because there is insufficient time for the gas to fill and empty the balloon.

CONTRAINDICATIONS

- Aortic insufficiency—the incompetent aortic valve allows the ventricle to distend during diastole thus reducing coronary perfusion pressure.
- Sepsis.
- Severe vascular disease-technically difficult, and prone to thrombosis. Risk of rupture of abdominal aneurysm.

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38 Cardiopulmonary Cerebral Resuscitation Controversies

Cardiopulmonary cerebral resuscitation (CPCR) is a clinical situation which produces so dramatic results, that a few minutes of treatment makes the difference between life and death. There is no role for a single improper step or delay. So also, any modification which offers a very minute advantage may be important. Thus, it is natural that the intricate details of the procedure are the subject of intense research and arguments.
Let us now discuss certain controversial points:

CARDIAC PUMP VERSUS THORACIC PUMP

It works as mechanism of external cardiac massage. It has long been held that a closed chest massage compressed the heart between the sternum and the spine, thereby increasing ventricular pressure ; which is transmitted to vessels and not backwards due to a closed mitral valve, on release, the ventricle refills passively. This cardiac pump mechanism was questioned on several grounds. In 1976, Criley found during angiography that if the patients who developed an arrest coughed immediately, the peripheral perfusion was maintained. This led the John Hopkin's Researchers to investigate the letter, and they found that an increase in intrathoracic pressure increased peripheral perfusion. Ineffectiveness of ECM in flail chest patients was also an argument in favour of this thoracic pump mechanism.

Here heart acts as a passive conduit between two vascular beds and an increase intrathoracic pressure drives blood from a low pressure pulmonary circulation to a high pressure systemic circulation. This increased intrathoracic pressure compresses systemic veins more than arteries (being more collapsible) and since they have valves at thoracic inlet there is no back pressure along them. This creates an arterio-venous pressure- gradient which is essential for organ perfusion (since IVC has no valves, the back pressure reduces the perfusion of lower half of body during ECM). This has been supported by experimental evidence of open mitral valve on cine angiography and equal pressures in all 4 chambers. More time spent in compression was assumed to increase intrathoracic pressure and hence a low rate with increased compression duration was adopted (60/min). But this increased intrathoracic pressure is likely to decrease coronary and cerebral perfusion. So also various techniques like SCV-CPR, IAC-CPR, abdominal binding, etc. were not found to give superior results in arrested humans.

During manual CPR in dogs it was founds that LV and aortic pressures were higher than the rest and mitral valve was closed during compressions. This rekindled interest in cardiac pump mechanisms. Echo evidence showing deformation of heart on ECM (being more anteriorly) brought forth the argument that a High Impulse (increased force and velocity). CPR was needed to compress

the chamber, rather than the displacement which occurs at low impulse. Moreover increased rate naturally increased the CO and rate of 120 or more was suggested and was supported by experimental evidence of better prognosis.

Ultimately which of these mechanism is active in a person is decided by heart size, thoracic size and compliance, force and rate of compression, compression/relaxation ratio, etc. In order to strike a compromise AHA recommends a rate of 80-100/min with 50:50 ratio. Alternation of compression and ventilation (5:1 or 15:2) is still the method of choice.

Paediatrics—more of heart being behind sternum, cardiac pump may be more important.

Site of Compression

The site of compression in children for ECM has been subjected to change recently. Midsternum was earlier used assuming that in children, heart is higher and lower compression will cause liver injury. Recent experimental data show that there is no such variation in position of heart in children. Both these positions have their own advocates.

CAB or ABC?

CAB sequence for basic life support has been put forward by a Dutch group in cases of witnessed cardiac arrest. They argue that the partially oxygenated blood (patient may even be gasping) could be delivered to vital organs, buying precious time. But this has not been universally accepted, though a thumb on the chest is practiced widely.

The Demise of Polypharmacy

An area where changes are made every now and then is DRUGS. There is a trend towards fewer drugs. The parts of disagreement are:

- *Route of administration:* Intracardiac has been and is still the choice with many practitioners. High rate of complications have made the AHA to supercede this with IV route. Transpulmonary instillation is now thought to be less effective even with higher doses.
- *Sodabicarbonate:* Countless are the pages that are written, both for and against $NaHCO_3$ but is still used wide as the last resort. It is used on the ground that it neutralized the metabolic acidosis occuring as a result of anaerobic metabolism. But it leads to following side effects hypernatraemia, hyperosmolarity (ventricular haemorrhage in infants), liberate CO_2, paradoxical CSF acidosis, alkalosis, shift of ODC to left neutralise adrenaline in the IV line. After lot of arguments AHA has finally discontinued its routine use in CPCR (as per '85 guidelines). Its use is reserved for prolonged arrest, pre-existing acidosis, hyperkalaemia, etc.

  Tris-hydroxymethyl aminomethane (THAM) and carbicarb ($NaHCO_3$ + Na_2CO_3) are prescribed on the ground of fewer side effects, but many do not favour any buffers in arrest.
- Ca^{++}—was a universal cardio tonic used, but has been found to cause muscle necrosis and promote cellular autolysis in ischaemic organs. AHA has limited its use to situations like hypo

Ca^{++}, hyper Mg^{++}, Ca channel blocker toxicity, some electromechnical dissociation (EMD) cases, etc.

- Adrenaline—Its role as the ultimate drug in cardiac arrest has been questioned by countless studies compairing it with other stimulants and noradrenaline. But still none has been proved superior to it. A higher dose (0.1-0.2 mcg/kg as against classical 0.01 μgm/kg schedule) was found to give superior results in several studies, but is still to get unanimous approval.
  5-10 mcg/kg for every 5 minute arrest time is the accepted regimen now.

COLLOID VERSUS CRYSTALLOID

Controversy has been discussed too many times. Suffice is to say that a crystalloid may be sufficient as the 1st time of management and colloids. Hypertonic dextran is advocated by several groups as it suddenly expands intravascular volume.

Defibrillation

There is a trend towards early usage of electrical defibrillation. Three rapid shocks without pause has been claimed to be more effective than the classical method; though not proved beyond doubt.

Early usuage of bretylium (a clinical difibrillation) routinely is being advocated. Though it increases defibrillation threshold, it decreases post- resuscitation arrhythmias.

Early Intubation

It is the choice of WFSA, but AHA says it will intefere with external cardiac massage (ECM) and should be reserved for prolonged arrests.

Role of Laymen

Role of laymen or trained paramedic has been often disputed whether to allow them to do intubation and defibrillation. Now they are being increasingly involved in the early management. The development- of EGTA, EOTA, oesophageal combitude and automatic defibrillators are evidence of recognition of this role.

Cerebral Protection

It is an area of intense research and conflicting reports. The use of high dose barbiturates is now condemned and even the low dose regimen is controversial, though many drugs offer theoretical, advantage and have their proponents—none have been recognised for routine clinical use.

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39 Aetiology and Management of Intraoperative Cardiac Arrhythmias

No arrhythmia is unique to anaesthetic practice, indeed cardiac arrhythmias are a relatively frequent occurrence during anaesthesia. There is an overall incidence of significant arrhythmias in 60% of anaesthetised patients.

The situations during anaesthesia where cardiac arrhythmias are commonly seen are:

- Metabolic disturbances, metabolic acidosis, hypercapnia, serum calcium or potassium abnormalities, hypoxia, hypomagnesaemia, succinylcholine, intubation in light plane, laryngoscopy etc.
- Cardiac surgery, pulmonary surgery, other surgeries involving manipulation of heart or lungs.
- Associated cardiac lesion, e.g. valvular HD.
- Coronary artery diseases
- Use of volatile anaesthetic agents, local anaesthetic drugs, e.g. bupivacaine.
- Ingestion of potential cardiotoxic drugs quinidine, digoxin, L-dopa, sympathomimetics, lithium, etc.
- Other miscellaneous conditions like cardiomyopathy, pericardial disease, left ventricular aneurysm, electrical microshocks, etc.

Though the volatile anaesthetic agents have been implicated as arrhythmiogenic, current thinking favours that, in the absence of sinus dysfunction, impaired conduction, beta or Ca channel blockers, none of the currently used vapours at clinically useful levels appear to be a likely cause of sinus arrest or significant AV conduction block.

Any preoperative EKG abnormalities should put the anaesthesiologist on alert to the possible intraoperative development of cardiac arrhythmias.

Which Leads to Monitor

Ideally it is necessary to monitor leads which amplify P + wave (Lead, II, avF, V_1) so that the relation of p wave to ventricular complexes can be ascertained. The other lead necessary is V_5 lead to monitor any ischaemia, especially of anterior wall.

INDIVIDUAL ARRHYTHMIAS

Supraventricular Arrhythmias

- Sinus tachycardia
- Sinus bradycardia

- Sinus dysrrhythmia
- Sinus pause or arrest
- Sick sinus syndrome
- Premature atrial beats
- Atrial flutter
- Atrial fibrillation
- PAT with or without block
- Paroxysmal supraventricular tachycardia
- Pre excitation syndrome
- AV junctional rhythm and tachycardia.

VENTRICULAR ARRHYTHMIAS

- Premature ventricular beats
- Ventricular tachycardia
- Torsades de pointes
- Ventricular fibrillation.

Heart Block

I° Block	
II° Block	Wenkebach - (Mobitz type 1)
	Mobitz type II.
III° Block	Complete heart block

Sinus Tachycardia

- Rate >100/min.
- Regular rhythm p : QRS =1:1
- Normal QRS
- Upright P in L_{II} biphasic in MCL_I
- Common condition
- Usual causes: Hypovolaemia, pain, anxiety, light anaesthesia, pyrexia and administration of adrenaline.
- Sinus tachycardia, by decreasing diastolic filling time, may decrease cardiac output especially in fixed output states. It also adversely affects myocardial oxygen balance.
- Treatment should be directed at the cause.
  However in patients at risk (e.g. IHD), a beta adrenergic blocker has to be considered.

Sinus Bradycardia

- Rate < 60/min
- Regular rhythm. At very slow rates, ventricular escape beats may be seen

- Normal QRS
- Upright Pin L_{II}, and biphasic in MCL_{II}
- Commonly seen in athletes. It may be secondary to an increase in vagal tone (as in tracheal intubation in children), hypoxaemia, due to drugs like beta blockers, digitalis, repeat doses of scoline, halothane, etc.
- Identify and remove the cause. Treatment of bradycardia is necessary only if escape beats occur or there is hypotension. IV atropine usually restores the rate (0.5 mg). Rarely isoprenaline or temporary pacing may be necessary.

Premature Complexes: Atrial vs Ventricular

ECG characteristics of PAC and PVC differ as shown in Table 39.1.

Table 39.1: Differences between atrial and ventricular premature beats

Features	*PAC*	*PVC*
QRS	Usually normal	Wide and bizarre
P	Usually abnormal	Usually absent
Pause	Noncompensatory	Compensatory
Sinus Rhythm	Reset	Not reset.

Occassional PAC's are haemodynamically insignificant and need no treatment. All PVCs are potentially dangerous because they may precipitate VF.

Indications for treating PVCs

- > 6 PVC s/min
- 3 or more sequential PVCs
- Coupled PVCs
- Multifocal PVCs
- The 'R on T' phenomenon.

When PACs require treatment, the underlying cause should be treated. PVCs can be usually suppressed promptly by IV lignocaine 1 to 1.5 mg/kg bolus. Recurrent PVCs can be treated with a lignocaine infusion 15 to 50 mcg/kg/min. Occassionally treatment of refractory PVCs may require B adrenergic blockade, bretylium, disopyramide, procainamide, atropine (when the basic rate is slow) or overdrive pacing.

ATRIAL FLUTTER

This condition represents a very rapid discharge from an ectopic atrial focus. The rate of discharge is much faster than that in PSVT. It is usually associated with AV block.

- Rate—125 to 200/mt.
- Rhythm—normally regular may be irregular in the presence of an AV block.
- Normal QRS.
- 'Saw tooth' or picket fence appearance of baseline.

- P:QRS is usually 2:1
- Atrial flutter almost always indicates organic heart disease.
- The initial treatment of choice is cardioversion. Overdrive pacing also usually terminates atrial flutter.
  Useful drugs include verapamil and digitalis with or without propranolol.

ATRIAL FIBRILLATION (AF)

A dysrhythmia characterised by extremely rapid, totally irregular and ineffective atrial activity.

- Rate 160 to 180/min.
- Irregularly irregular rhythm
- Normal QRS
- No P waves—since there is no organised atrial activity, the chaotic electrical activity of the atria produces the 'f' waves characteristic of atrial fibrillation.
- Atrial fibrillation almost always indicates severe organic heart disease.
- Cardioversion is usually successful in atrial fibrillation of recent onset. Long standing AF needs both drug therapy and treatment of the cause. Digitalis and verapamil are helpful to decrease the ventricular rate. Propranolol can be used when the dysrrhythmia is refractory to digitalis alone. Quinidine and procainamide are other drugs used.

VENTRICULAR TACHYCARDIA

This is the occurrence of more than 3 sequential ventricular extra systoles with a cardiac rate of more than 120 to 150.

- Rate 100 to 220/min.
- Rhythm usually regular, sometimes irregular

"Wide and bizarre QRS complexes"

- P waves are not seen with fast rates of ventricular tachycardia (VT)
- They may be seen when AV dissociation occur in a slow VT
- VT is a grave life threatening dysrrhythmia which greatly reduces cardiac output and lead on to VF.
- Cardioversion is the treatment of choice. If the patient is hemodynamically stable, IV lignocaine can be given, procainamide and bretylium are also useful. Overdrive pacing may have to be used, if drugs are not effective.

VENTRICULAR FIBRILLATION (VF)

It is chaotic, incoordinated ventricular depolarization where the biventricular chamber is electrophysiologically fragmented into a 'mosaic' of milling tissue islets in various stages of recovery and excitation.

- Rate—too rapid and disorganised to count totally irregular rhythm.
- No P, QRS or T waves.

Ventricular Fibrillation is Cardiac Arrest

Management of VF

The definitive treatment of VF is DC counter-shock (or electrical defibrillation); fine VF is usually refractory. It should be converted to coarse VF by the intravenous administration of 0.3 to 0.5 mg of adrenaline before giving counter shock.

- DC defibrillation a 200 to 360 J.
- Adrenaline 1 mg IV early during cardiac arrest and then every 5 min of arrest time.
- Further DC shock of 360 J after first dose of adrenaline.
- If VF persists lignocaine 100 mg IV followed by DC shock of 360 J.
- If unsuccessful—use different paddle position/different defibrillator/other anti arrhythmic drugs.

Simultaneous CPR with 100% oxygen via ETT, IV adrenaline 1 mg, external cardiac massage and sodium bicarbonate 0.5 to 1 mg/kg bolus dose-has to be continued. If the heart does not respond to a lower shock, the DC shock strength can be increased step by step (200-360 J).

Management of Electromechanical Dissociation

Rule out:

- Pneumothorax
- Cardiac tamponade
- Pulmonary embolus
- Hypovolaemia.

Adrenaline 1 mg is given IV immediately, calcium chloride is also indicated in this condition, especially in association with hyperkalaemia, hypocalcaemia and previous administration of Ca^{2+} antagonists.

Ventricular Asystoles

Asystole is cardiac standstill. It is the second most frequent EKG finding after VF, in cardiac arrest.

- No electrical activity—only a straight line is seen
- Rarely a few 'P' waves may be seen
- Asystole is more difficult to treat than VF. Treatment is continued by maintaining BLS (Basic life support — airway, breathing and circulation) while administering drugs such as sodium bicarbonate, calcium chloride, adrenaline and isoprenaline. Transvenous pacemaker may be helpful.

General Points

External cardiac massage with artificial ventilation must continue for at least 2 minutes after the bolus administration of each drug. CPR should not be interrupted for 10 sec except for the administration of DC shocks. As per the new guidelines of the resuscitation council, 1 mg of adrenaline is administered every 5 min of cardiac arrest time. This is an attempt to maintain cerebral and myocardial perfusion pressure.

Treatment with sodium bicarbonate, although unfashionable now, 50 ml of 8.4% may be useful in the management of a severe metabolic acidosis, provided "elimination of CO_2 is maintained by artificial ventilation". Sodabicarb is indicated only if the ABG shows metabolic acidosis pH < 7.2.

The endotracheal route in drug administration is no longer considered reliable and should only used if intravenous access cannot be achieved-double the normal dose of the drug (adenaline, atropine, lignocaine) has to be administered.

Pre-excitation syndrome: The commonest pre excitation syndrome is the WPW (Wolf Parkinson White) syndrome. The ECG is characteristic, with a short PR interval (0.1-0.125 sec), a delta wave, with widening of QRS complex due to incorporation of the delta wave. This is due to an abnormal accessory pathway called the 'Bundle of Kent' which causes premature -activation of part of the ventricular myocardium. There are 2 types (Type A and type B) with left ventricular, and right ventricular predominance respectively.

Other association: MVP/Ebsteins anomaly /HOCM (hypertrophic obstructive cardiomyopathy).

Significance of the WPW Syndrome

- Individuals with the WPW 'syndrome are prone to attacks of supraventricular tachyarrhythmias.
- The WPW may mimic other EKG manifestations and lead to erroneous diagnosis, e.g. MI, RVH, BBB.

Other rarer pre-excitation syndromes are Mahaim fibres pre-excitation, the lown-ganong-levine syndrome (short PR internal and normal QRS complex).

Heart blocks

I° Prolongation of PR interval (>0.2 mm)

II° (type I)–gradual prolongation of PR interval till one QRS complex is dropped—(Type II)—PR interval is fixed, but the QRS complexes are dropped at random (more dangerous type).

III° A state of complete HB where the atrial and ventricular beats are completely disorganised and uncoordinated.

Heart blocks form some of the most common indications for temporary or permanent pacing—following are the indications for pacing.

- Complete heart block
- II° HB—symptomatic mobitz type I, mobitz type II (all)
- Symptomatic 1° AV block
- Trifasicular block
  - Alternating LBBB/RBBB
  - Long PR Interval + LAHB + RBBB
  - RBBB + LPHB
  - Long PR Interval and LBBB
- Sick sinus syndrome unresponsive to drugs.

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40 Air Embolism

The introduction of air bubbles in to systemic circulation can result in significant morbidity. Monitoring for massive venous air embolism is an important part of anaesthesia care in patients undergoing surgery in sitting position. Moreover open chamber cardiac surgical procedures necessarily result in intra cardiac air introduction.

VENOUS AIR EMBOLISM

Conditions promoting venous air embolism are (i) an open vein and (ii) negative intravenous pressure relative to atmospheric pressure. This conditions frequently coexist in neurosurgery when the head is positioned above heart level. During neurosurgery, peak occurrence may be expected during skin-muscle incision and when bone-venous sinusoids are initially exposed. The most severe episode of venous air embolism (VAE) occur when a major dural sinus is opened.

Volume of intravascular gas, its rate of entrainment, presence of a patent foramen ovale, elevated right heart pressure, the presence of nitrous oxide, anaesthetic depression of cardiovascular function of the patient's inherent cardiopulmonary compensatory capacity determine the clinical outcome.

Small bubbles of air entrainment slowly are usually of little significance. Gas bubbles are removed by the lung at a rate that mainly depends on a compensatory increase in pulmonary artery pressure (PAP). Overload of lung excretion results in further increase in PAP and ultimately to reduced cardiac output and circulatory failure. Hypoxaemia develops only late and mostly due to massive shunting of pulmonary blood flow. Paradoxical air embolism is another potentially serious complication of VAE.

MONITORING

- *Central venous pressure (CVP):* A CVP tip positioned near the superior vena caval—right atrial junction provides a measure of intravascular volume status and can be used to confirm the diagnosis of air embolism as well as to evacuate the intravascular gas.
- *Doppler ultrasound:* It is a sensitive method for detection of intracardiac gas. It can detect as little as 0.5 ml of air flowing through the right heart. High pitched swishing sounds, represent the flowing blood. Air is an excellent acoustic reflector; its passage through the detection field is heralded by a change over to an erratic roaring noise.
- *Transoesophageal echocardiography (TEE):* TEE is 5 to 10 times more sensitive than Doppler. Because TEE provides a visual representation of location of air in the right and left heart it can diagnose paradoxical VAE. Major disadvantage of TEE is lack of specificity as it detects fat emboli and blood micro emboli. Less than 0.25 ml of air can be detected using TEE.

- *Pulmonary artery pressure (PAP):* Increase in PAP can provide a semi-quantitative estimate of the volume of VAE. Changes in PAP and end tidal CO_2 tend to occur prior to changes in arterial blood pressure and cardiac output.
- *End-tidal carbon dioxide ($ETCO_2$):* As volume of air entrained per unit time increases, it enters the pulmonary arterial system, ultimately lodging in pulmonary micro circulation. This leads to a reduction in lung perfusion relative to ventilation and development of an increased physiological dead space and results in a reduction of end expiratory concentration of CO_2. Degree and duration of the $ETCO_2$ depression provide a semi-quantitative estimate of volume of intravascular gas.

End-tidal nitrogen (ETN_2): Mass spectrographic analysis of end-tidal gases permits detection of nitrogen. Studies have found ETN_2, to be slightly more sensitive or equal to $ETCO_2$ monitoring of VAE.

Other Methods

- Oesophageal stethoscope—for a change in heart sounds or a 'mill-wheel murmur'—depends on a large amount of intracardiac air.
- Dramatic fall in arterial pressure, elevations in CVP and ECG changes may indicate severe pulmonary gas embolism.
- VAE may elevate airway pressures during mechanical ventilation due to bronchoconstriction and reduced pulmonary compliance.

Other Causes of Air Embolism

- Head and neck surgeries—open neck veins create the possibility of air embolism during head and neck surgeries.
- Orthopaedic surgery—air embolism is possible to occur when the operating field is above the level of the heart. Especially in surgery of cervical spine or shoulder in sitting position, in total hip replacement in lateral decubitus position,or in lumbar spine surgery in prone position.
- Intravenous hyperalimentation.
- Liver transplantation—massive venous air embolism can occur at the time of graft reperfusion. Adequate flushing of donor liver prevents air from entering the recipient circulation.
- Venous catheter insertions.
- Haemodialysis.
- Autotransfusion.

Treatment: On suspicion that an embolus has occurred surgeon is informed:

- Ventilation with 100% O_2 is initiated.
- Attempts are made to aspirate air through right heart catheter.
- Trendelenberg tilt is given.
- In case of cardiovascular collapse active cardiopulmonary resuscitation is started.
- Left lateral position to dislodge any major emboli from the root of pulmonary artery.
- Preventing additional air entry at the surgical site by packing the wound and applying unilateral or bilateral jugular vein compression.

- Cardiovascular drugs are administered as required. Rapid inflation of an antigravity venous compression device may provide transient cardiovascular support by increasing venous pressure and return.
- Hyperbaric oxygen therapy.

Occurrence of Air Embolism during Cardiopulmonary Bypass

After cardiopulmonary bypass is terminated occurrence of air embolism is a major problem. Sudden embolization of air lodged in one of the left chambers of the heart or in the aorta can occur, when the orientation of the heart and great vessels is altered by manipulation of the heart, closure of the sternum or even by movement of the patient. Embolization to the brain or to the coronaries can occur. Temporary CNS functional deficits resembling a cerebrovascular accident can occur. They usually resolve in days as air is absorbed. Air embolism in the coronary arteries may be manifested by ST segment elevation, bradycardia, conduction abnormalities, arrhythmias, ventricular fibrillations or impaired contractility. Treatment includes resuscitative measures as indicated, maintenance of a high perfusion pressure in hopes of flushing the emboli out of the arterial circulation and perhaps administration of corticosteroids.

Following has been suggested to prevent arterial air embolism during CPB:

- Fine gauge needle puncture at the highest level of saphenous vein graft and massage to the graft to express air before aortocoronary blood flow.
- Large bore needle puncture at the highest level of aorta with the head at a slightly lower level than ascending aorta.
- Positive pressure ventilation before and passive Valsalva manoeuvre maintained during the removal of left ventricular cannula.

For open heart surgery massage of the left atrium and ventricle by the surgeon, lateral rotation of the patient, prevention of nitrous oxide administration after CPB.

Hyperbaric oxygen therapy: Involves administration of high inspired oxygen concentration thus increasing arterial PO_2 resulting in increased rate of resolution of gas bubble.

Effect of N_2O on air emboli: The entrance of N_2O into the air bubble results in an increase in volume. An alveolar concentration of 50% might double the gas volume, while a 75% concentration produce a four-fold increase occuring in seconds. This suggest caution in the use of N_2O for procedures in which air embolization is a risk.

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41 Oxygen Toxicity

Oxygen toxicity is a complex phenomenon which could affect many organ systems. Certain systems are more susceptible.

The common manifestations of O_2 toxicity are:

- Pulmonary toxicity
- Retrolental fibroplasia
- Hypoventilation
- Central nervous system toxicity
- Circulatory system

PULMONARY TOXICITY

Lorraine Smith, a pathologist showed that oxygen at a partial pressure less than that required for convulsions was a lung irritant and produced inflammation, known as the "Lorraine Smith Effect". The toxicity depends on dose and duration of exposure to oxygen.

- The earliest symptom is substernal distress which begins as irritation in carina and cough.
- Reduction in vital capacity.
- Changes in capillary endothelium with an increase in its permeability which leads to accumulation of fluid in interstitial spaces and widens diffusion pathway between alveolar gas and alveolar capillary blood.
- Depression of mucociliary transport.
- Inhibition of phagocytosis by alveolar macrophage.
- Changes in surfactant- activity and its production.
- Due to high concentration of oxygen in alveolar gas of zones of lungs with low V/Q ratios, the oxygen diffuses in the pulmonary capillary blood, which leads to alveolar collapse.

Highest safe concentration of oxygen for lung ventilation is not known as it varies depending up on individual, the pre-existing pulmonary pathology and duration of exposure.

Pathological Changes in Lungs

- The first change, after few days of oxygen exposure to lung is slight swelling of endothelial cells and interstitial oedema.
- After 4 days exposure, there is destruction of type I alveolar cells, and increase in thickness of air-blood barrier.
- After 7 days hyperplasia of type II cells and complete destruction of type I cells occurs.
- After 12 days proliferative changes in type II cells and further reduction of alveolar spaces occurs.

Hypoventilation

Seen in patients with chronic hypoxaemia and hypercarbia (as in chronic bronchitis) who depend on hypoxic drive. When oxygen is given in high concentrations, the hypoxic drive is abolished. Ventilatory depression and increase in PCO_2 leading to respiratory arrest can occur. They are managed by repeated blood gas measurements and endotracheal intubation and IPPV.

Retrolental Fibroplasia

O_2 to premature babies given in a concentration of more than 40% and $PaCO_2$ more than 13 KPa will cause development of a fibrovascular membrane posterior to the lens. The chance of retrolental fibroplasia is more when O_2 saturation exceeds 80 to 85%.

CNS TOXICITY

Paul-Bert Effect: When oxygen is given in high concentrations convulsions can ensue. This occurs when oxygen is given at 3 ATA. This is due to increased cerebral PO_2. This effects begins as twitching around eye lids, facial muscle and progresses to the diaphragm and is characterised by a tonic phase followed by a clonic phase. It is diagnosed early by EEG and EMG of lip muscles.

The treatment is:

- High pressure O_2 is immediately withdrawn and patient is allowed to breathe air.
- If a patient lies in a single chamber rapid decompression is not allowed as it produces laryngeal spasm and glottic closure, trapping oxygen under high pressures leading to the risk of rupture of lungs which could be avoided by an endotracheal tube *in situ*.

PROPOSED MECHANISMS OF O_2 TOXICITY

Absorption Collapse

Simple atelectasis due to blockage of small airways and with resultant absorption of gases trapped peripheral to the obstruction. This can be prevented by

- Chest physiotherapy.
- Ensure patency with high tidal volume.
- PEEP to increase FRC.

Lung Surfactant

Due to decrease in production of lung surfactant, the phospholipid, dipalmityl lecithin by type II cells.

Metabolic Upset

The enzymes vital to the metabolism are inhibited mainly those with sulphydryl group, several of which are involved in tricarboxylic acid cycle.

- Glyceraldehyde phosphate dehydrogenase in glycolysis.
- Flavoproteins in cellular respiration and electron transport system and
- Enzymes in the metabolism of glutamate and GABA.

Myocardial Failure

Myocardial metabolism may be depressed by high levels of oxygen, which cause pulmonary oedema.

Role of Endocrine System

Thyroxine and thyroid extract hasten the onset of both convulsions and pulmonary damage and cortisone and adrenal corticoids cause pulmonary damage and sympathomimetic agent augmenting its onset.

Healthy patient can inhale 40% oxygen indefinitely.

- 100% O_2 is non-toxic for a period of upto 24 hours.
- The crucial factor is arterial PO_2.
- O_2 free radicals are said to inactivate the antiprotease- antitrypsin of alveolar cells.

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42 Flail Chest

DEFINITION

It is an abnormal movement of the chest wall occurring as a result of fractures of 3 or more ribs in 2 places at the same side.

On inspiration there is encroachment of the injured segment upon the lung due to paradoxical movement leading to impairment of ventilation and oxygenation.

- It is common with blunt trauma of chest wall and involves anterolateral aspect; and posterior wall is rarely involved as it is covered with muscle mass. Injury to the upper ribs results in serious trauma, and is usually associated with injury to intrathoracic or intra-abdominal organs.
- The older age group are more susceptible due to more calcification and brittleness of their chest wall and hence serious injuries result; but in contrast, in the paediatric age group the chest wall is extremely elastic and resilent and hence affords greater protection to underlying structures.

DIAGNOSIS

- History of blunt trauma in the chest or upper abdomen. It is difficult to diagnose in upper thoracic cage injury.
- Radiological signs of parenchymal damage is not evident in early stages, but presence of mediastinal air will signify bronchial or tracheal injury.
- Serial blood gas measurements are helpful in establishing diagnosis.
- Repetitive clinical assessment is necessary.

Pendelluft phenomenon: It is the to and fro movement of air from the damaged to the normal lung. This was considered to be the major cause of respiratory dysfunction associated with flail chest previously but recent reports indicate that this theory is not entirely true. The primary pathologic defect is inadequate oxygenation.

MANAGEMENT

Aims

- To stabilise the injured segment
- Maintenance of adequate ventilation and
- Effective pain relief.
  - Patient is admitted in surgical ICU, and temporary stabilisation of flail segment with sand bags and/or pillows is done until specific decision is taken.

- Treatment varies from simple supportive therapy as giving oxygen, physiotherapy and pain management to full ventilatory support. Ventilatory support is considered as most effective treatment for patients who develop respiratory failure.
- External stabilisation of the flail segment by traction on the injured segment is done. Moore and his colleagues stabilised the chest wall by intramedullary pinning of the fractured ribs.
- Before intubating the patient, he should be clinically observed and serial blood gas analysis done and vital capacity measured.

Criteria for intubation

- PaO_2 less than 70 mmHg with Oxygen enrichment
- $PaCO_2$ more than 50 mmHg
- pH less than 7.25
- Tachypnoea 30/min or more
- Vital capacity less than 15 ml/kg
- Negative inspiratory force less than - 20 cm H_2O

These criteria are guides to therapy. Two or more fractured ribs should be present before endotracheal intubation is considered. If it is decided to intubate, endotracheal intubation with sedation and topical anaesthesia is done, preferrably nasal intubation, which is more comfortable but may cause maxillary sinus infection.

- Tracheostomy is avoided, unless there is extreme difficulty in clearing secretions and is deferred upto 2 weeks. The LANZ tracheostomy or ETT has the advantage that the cuff of this tube is fitted with special pressure relieving valve which prevents the application of excessive presssures.to the tracheal wall.

All gases delivered should be humidified, as dry gas for long time cause disruption of tracheal mucosa and erosion and formation of mucosal plugs. In the initial stage of ventilatory therapy patient is put in intermittent mandatory ventilation (IMV) with a rate of 6/mt, initially. Arterial CO_2 and patient's intrinsic rate act as guides to therapy. About 5 cm H_2O of distending airway pressure is selected and the inspired oxygen concentration is given to maintain $PaCO_2$ less than 80 mmHg. Weaning is attempted when arterial blood gases are within acceptable range, and vital capacity approaches 15 ml/kg and the inspiratory force is about - 20 cm H_2O. Extubation is carried out when the patient maintains normal blood gases on room air and is capable of generating negative pressure of -30 to -40 cm H_2O and has vital capacity 20 ml/kg body weight.

MONITORING

- ECG—continuously as dysrrhythmia is a problem in case of cardiac contusion.
- Frequent arterial blood gas and other laboratory data
- Inspired oxygen tension with oxygen analyser.
- Pulmonary artery pressure to calculate degree of pulmonary shunting.
- Other cardiovascular parameters.

TREATMENT

- Adequate pain relief.

  In the early stages 3 to 4 mg morphin for 70 k patient every 2 to 3 hours, and is followed by intercostal block or continuous thoracic epidural. The latter is preferred and is the choice in respiratory failure secondary to chest injury.
- Appropriate antibiotics after frequent cultures.
- Adequate oxygenation.
- Ventilatory support if necessary.

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43 Acid Aspiration Syndrome

Pulmonary aspiration of stomach contents is one of the most serious complications of the obtunded or unconscious state (including anaesthesia). This is also responsible for the morbidity and mortality among the obstetrical, surgical and ICU patients.

HISTORY

In 1946, Curtis Mendelson described 66 cases in which gastric contents had been regurgitated and initiated the syndrome, which now bears his name.

INCIDENCE

Aspiration accounts for 1 to 20% of all anaesthetic deaths. Anaesthesia accounts for 10% of maternal deaths and with aspiration for 52% of the maternal deaths.

Mortality

Thirty percent (3 to 70%).

Morbidity

Consist of serious complications ranging from pneumonitis, lung abscess, renal failure, etc.

PATHOPHYSIOLOGY

Morbidity and mortality following aspiration depends on the volume, nature, acidity and distribution of aspirated material.

Gastric contents reaches the pharynx as a result of i) vomiting ii) regurgitation which will be aspirated into the airway when protective laryngeal reflexes are depressed.

ASPIRATION DUE TO VOMITING OCCURS

- Stormy induction.
- Light plane of anaesthesia.
- During emergence from anaesthesia.

Regurgitation is more common than vomiting under anaesthesia. The important causes of regurgitation are: (i) raised intragastric pressure, (ii) incompetence of gastroesophageal sphincter, and (iii) delayed gastric emptying.

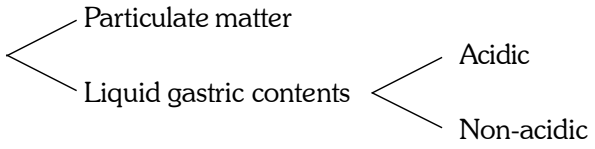
FACTORS WHICH CAN DELAY GASTRIC EMPTYING TIME

- Pain, anxiety and trauma.
- Narcotics, increased ICP, intestinal and pyloric obstruction.
- Acid in duodenun.

FACTORS CONTRIBUTING INCOMPETENCE TO GASTROESOPHAGEAL SPHINCTER

- Increased intragastric pressure above 20 cm H_2O.
- Coughing, straining, etc.
- Drugs like atropine, hyoscine, morphine, etc.
- Alkaline pH.
- Hiatus hernia.
- Nasogastric tube.

Types of Pulmonary Aspirate



Liquid aspiration is more common.

FACTORS DETERMINING THE EXTENT OF PULMONARY INJURY

- Volume of aspirated material—Volume as small as 25 ml can produce widespread pulmonary damage.
- pH of the aspirate—When the pH decreases to less than 2.5, the complications are more.

CLINICAL PICTURE

Four phases have been described.

Phase I: Profound dyspnoea and tachypnoea. Bronchospasm is often present. Chest X-ray may be normal.

Phase II: Increasing cyanosis and hypoxemia. Minor chest X-ray abnormalities present.

Phase III: Respiratory failure, profound hypoxemia with wide alveolar - arterial O_2 tension gradient, reduced compliance, diffuse bilateral infiltration.

Phase IV: Hypoxemia unresponsive to oxygen. Metabolic and respiratory acidosis.

DIAGNOSIS

- Severe aspiration—immediate collapse
- Moderate aspiration—symptoms after 6-8 hrs.
- Bile-stained fluid in endotracheal suction.

MANAGEMENT

- Solid gastric contents require urgent removal by bronchoscopy.
- Aspiration of liquid contents should be managed in the following way. Early diagnosis and appropriate management is essential inorder to reduce mortality. The ultimate degree of pulmonary destruction cannot be judged at the time of aspiration.
  - Suctioning of the aspirate by bronchial lavage is contraindicated. Because it may aggravate injury by removing or aspersing surfactant.
  - IPPV with PEEP.
  - Correction of blood and fluid volume and acid-base balance at the same time, avoid overloading.
  - Monitoring of CVP, PAWP and blood gases.
  - Use of membrane oxygenator may be indicated if PaO_2 continues to fall below 50 mmHg.
  - Antibiotics, antispasmodics and steroids are often used if indicated.

PREVENTION OF ACID ASPIRATION SYNDROME

- Decrease gastric fluid volume by
  - Restriction of oral intake.
  - Empty the stomach by physical means like passing a wide bore nasogastric or stomach tube.
  - Empty the stomach by pharmacological means.
- Decreasing gastric fluid pH:
  - Use nonparticulate antacid like 0.3 molar sodium citrate - 30 ml orally.
  - Cemetidine 400 mg followed by 200 mg 2 hourly - orally.
  - Ranitidine 150 mg evening prior to and 150 mg on the morning of operation - orally.
- Preventing regurgitation;
  - Increase tone of lower oesophageal sphincter metoclopramide can increase the tone of the lower oesophageal sphincter. It can promote gastric emptying and also possess antiemetic property (5 to 10 mg IV 30 min before anaesthesia).
  - Avoid increase in pressure pressure.
  - Induction in upright position.
- Prevention of inhalation:
  Apply cricoid pressure (Sellick's technique)
  (All anaesthetists should be familiar with failed intubation, drill).

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44 Tension Pneumothorax

INTRODUCTION

Pneumothorax is an accumulation of air outside the lung but within the pleural cavity or mediastinum, which occupies space needed for cardiac filling and full lung inflation. Air may remain in the chest following any intrathoracic operation or it may accumulate post-operatively from air leak in the lung or tracheal tree.

There are three types of pneumothorax.

OPEN PNEUMOTHORAX

Open pneumothorax occurs as a result of penetrating injuries of the chest wall and here intrapleural pressure equalises with atmospheric pressure. This is characterised by diminished air entry on the affected side, decreased venous return due to positive pressure and mediastinal shift. Conditions that mimic pneumothorax include haemothorax or atelectasis.

CLOSED PNEUMOTHORAX

Here the communication of the pneumothorax space to the airway is closed. Air can neither get in or go out.

TENSION (VALVULAR) PNEUMOTHORAX

Occurs when air enters the pleural cavity during inspiration but, owing to ball valve action, cannot escape during expiration.

Causes

A tension pneumothorax can occur from

- Rupture of the lung surface
- Rupture of a small bullae due to excessive positive pressure, e.g. coughing
- Intraoperative trauma, or agitation in a patient receiving mechanical ventilation with high airway pressure or PEEP. This may also occur due to malfunction of a ventilator.
- As a result of accidental opening of the pleural cavity during operation or a bronchopleural fistula may be present.
- Attempted central venous cannulation.
- Supraclavicular brachial plexus block.

Pathophysiology

Air enters the pleural space as if through the one-way valve and causes the intrapleural pressure to rise sometimes as high as + 20 mm of mercury thus displacing the heart and compressing the other lung, heart and great vessels.

Clinical Features

It may occur in a conscious patient spontaneously breathing or in an anaesthetised patient on intermittent positive pressure ventilation. In a conscious patient rapid deterioration occurs, which is characterised by inaudible breath sounds, marked tracheal deviation, cyanosis and cardiovascular collapse. Tension pneumothorax should be suspected during anaesthesia if inflation becomes increasingly difficult and the patients condition worsens rapidly.

Cardinal Signs of Tension Pneumothorax

- Cyanosis
- Marked decrease in pulmonary compliance.
  (Manifest as increased tension in the reservoir bag during anaesthesia and excessively high airway pressures).
- Rapid deterioration in vital signs characterised by hypertension followed by hypotension, tachycardia followed by bradycardia and respiratory distress.
- Diminished or absent breath sounds, displacement of cardiac point of maximal impulse.
- Hypoxemia, hypercarbia, acidosis as reflected in arterial blood gas analysis.

  This condition is usually confused with pericardial tamponade and pneumo-mediastinum. Subcutaneous emphysema may or maynot be present. The onset of marked wheezing leads to the mistaken diagnosis of bronchospasm. Rarely a tension pneumothorax may follow rupture of a bullae during an attack of bronchospasm. It can be confirmed by clinical findings, reduced chest movement on the side of pneumothorax, deviation of trachea to the other side, hyper resonance to percussion and reduced breath sounds on the affected side.

Radiographic Evidence

Radiographic evidence of developing tension pneumothorax reflects the over expansion and compression. If the lung is not significantly consolidated, it will collapse, ribs on the ipsilateral side will spread and ipsilateral diaphragm will be depressed, mediastinum shifted to opposite side and contralateral lung and trachea compressed. Of these, depression of ipsilateral diaphragm is confirmatory. The ipsilateral hemithorax will look unusually translucent due to presence of air.

Occasionally, a tension pneumothorax may be loculated usually in an inferior subpulmonary or paracardiac location and only radiographic evidence of tension may be a slight flattening of cardiac border or a contour change in the ipsilateral diaphragm.

ANAESTHETIC AND PERIOPERATIVE CONSIDERATIONS

During general anaesthesia a rapid deterioration in compliance should alert the anaesthesiologist to the problem. Nitrous oxide should be discontinued as soon as possible as it accentuates the size of the pneumothorax. This is a surgical emergency and valuable time should not be wasted seeking radiological confirmation.

As soon as it is suspected a large bore (16 G) intravenous cannula is inserted into the chest in the second intercostal space anteriorly in the mid clavicular line. The needle is withdrawn leaving the cannula *in situ* so that the lung is not damaged as it expands. This converts the tension pneumothorax to an open pneumothorax. A chest drain can then be reinserted in due course and connected to an under water seal.

Very frequently a tension pneumothorax may be bilateral, so that this possibility must be kept in mind and confirmed with a chest X-ray as soon as possible.

Occassionally with re-expansion of a pneumothorax or rapid removal of large effusion, the re-expanded lung will develop unilateral alveolar pulmonary oedema which usually resolves rapidly.

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45 Hyperbaric Oxygen Therapy

The modern clinical application of hyperbaric oxygen (HBO) therapy began in late 1950s.
Goal : To ↑ tissue oxygen tension.

LIST OF CONDITIONS FOR WHICH HBO IS USED

- Gas bubble disease—Air embolism, decompression sickness
- Poisoning—Carbon monoxide, cyanide
- Infections —Clostridial myonecrosis, other soft tissue necrotising infections, refractory chronic osteomyelitis, mucomycosis.
- Acute ischaemia—Crush injury, compromised skin flaps.
- Chronic ischaemia—Radiation necrosis (soft tissue, radiation cystitis, and osteoradionecrosis), ischaemic ulcers.
- Central nervous system oedema
- Cancer therapy—The radiosensitivity of the tumour cells are improved by 30%.
- Acute hypoxia—Support of oxygenation during therapeutic lung lavage.
- Blood loss, anaemia (when transfusion is delayed or unavailable)
- Thermal injury (burns)

PHYSIOLOGIC EFFECTS OF INCREASED GAS PRESSURE

- Increased barometric pressure causes increased heat production whereas decompression generates cooling. Also pockets of trapped gas within the body, for e.g., middle ear, paranasal sinuses, intestinal gas, pneumothorax, etc. contract on compression and expand on decompression. (Boyle's law: $P \times V$ is a constant)
- Increased partial pressure of oxygen. Breathing oxygen at increased ambient pressure will lead to elevation of alveolar PO_2 (PaO_2) and therefore arterial PO_2 will remain high. With elevated PaO_2 substantial quantities of oxygen may exist in dissolved form. Increased PaO_2 has 3 major effects:

  Henry's Law : Exposure to increased atmospheric pressure will increase concentration of gas dissolved in liquid phase.
  - increased blood oxygen content
  - vasoconstriction
  - Antibacterial action particularly against anaerobic bacteria.

  Vasoconstriction is the rationale for the treatment of oedema (e.g. cerebral oedema and peripheral compartment syndrome) with HBO.

  Increased PaO_2 causes inhibition of toxin production and growth of certain anaerobic bacteria. Increased PaO_2 has shown to return phagocytic activity and ability of aminoglycosides to kill aerobic bacteria in ischaemic tissue back to normal.
- Elevation of inert gas pressure and pressure reversal of anaesthesia.

Elevation of partial pressure of inert gas that is present in a breathing mixture (usually N_2) is associated with a narcotic effect. N_2 has 0.03 to 0.05 times the narcotic potency of nitrous oxide.

Another effect is the tendency of high pressure to reverse general anaesthesia especially the effect of both intravenous and inhalational anaesthetics.

RATIONALE FOR TREATMENT OF SPECIFIC SYNDROMES

Carbon Monoxide Poisoning

CO has about 200 times the affinity for Hb than oxygen. This has two major effects:

- Proportion of Hb that is bound with CO is unavailable for oxygen transport.
- Avidity with which remaining Hb binds with oxygen is increased (shift to left of Oxy-Hb dissociation curve).

  Recently in addition to this, binding of CO to intracellular pigments has also been discovered as the cause of CO toxicity. This results in toxicity to multiple organ systems including brain and the heart.

  Mechanism by which HBO acts: (1) High arterial PO_2 hastens the removal of CO from blood. (2) Increased dissolved oxygen in plasma may support tissue oxygenation. HBO treatment thus may decrease both early and late morbidity in CO poisoning. (3) HBO shifts ODC to (right).

  Guidelines to application HBO therapy in CO poisoning:
  - Carboxy Hb greater than 20 to 25%
  - History of neurological impairment including loss of consciousness.
  - Patients presently symptomatic or with evidence of cardiac abnormality (ischaemia, arrhythmia or ventricular failure).
  - Pregnancy with COHB > 15-20%

Gas Embolism

- As in scuba divers, due to pulmonary barotrauma during ascent from dive, breathing compressed gas.
- Iatrogenic - which occurs:
  - During cardiopulmonary bypass
  - Due to inadvertant injection during diagnostic arteriogram or haemodialysis,
  - Due to introduction of air in venous system (during central venous catheterisation, haemodialysis, introduction of air into a cranial venous sinus during operation while in sitting position).

  Formation of gas bubbles in decompression sickness as seen in aviators and deep sea divers.

  Due to sudden decrease in pressure, bubbles of inert gas (N_2 or He) are formed in tissues and in blood.

  Role of HBO therapy: (1) High inspired oxygen concentration results in high partial pressure gradient for diffusion of N_2 from gas bubble into blood. (2) HIGH ambient pressure will cause reduction in size of the bubbles as explained by the Boyle's law.

Acute Infection

Anaerobic bacteria are sensitive to high tissue PO_2, probably because they lack antioxidant defenses such as superoxide dismutase. HBO therapy exerts a (a) Bacteriostatic effect (b) inhibits exotoxin production especially the alpha toxin by clostridia.

Support of Arterial Oxygenation During Anaesthesia

In patients with extremely low PaO_2 due to cyanotic heart disease, hyperbaric oxygenation offers a substantial margin of safety during anaesthesia. For example, patients having a PaO_2 of around 25 mmHg while breathing 100% O_2 at 1 ATA, the arterial PO_2 values increases to 50 mmHg or greater at pressures upto 4 ATA. Halothane anaesthesia was used in most cases. Nitrous oxide when used as a primary anaesthetic agent, along with the difficulties, associated with its use, may produce tissue gas bubbles (decompression sickness) on decompression from elevated pressure.

Maintenance of Oxygen Transport in Severe Anaemia

HBO therapy has been used for temporary support of severely anaemic patients.

THERAPEUTIC SYSTEMS

Multiple Chambers

Multiple chambers can accommodate 2-3 people. Ambient pressure is kept at about 3 ATA with compressed air and patient breathes oxygen with a head tent, face mask or endotracheal tube. Attenders are allowed inside the chambers and hence patient monitoring can be done at close hand. They are costly.

Monoplace Chamber

Large enough to accommodate only 1 patient. Chamber is compressed with 100% oxygen—Low cost. But patients cannot be evaluated 'hands on'. Monitoring is more remote and emergency care of airway cannot be provided.

TREATMENT MODE

Several factors limit the dose and duration of hyperbaric oxygen therapy

- Oxygen toxicity
- Decompression obligation of nursing staff accompany patients
- Difficulty of monitoring
- Patient discomfort

For decompression sickness, main stay of treatment consists of exposure to 2.8 ATA followed by slow decompression to 1.9 ATA. Periods of oxygen breathing is interspersed with 5 to 15 periods of breathing air to decrease oxygen toxicity.

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46 Adult Respiratory Distress Syndrome

This syndrome, since its introduction by Asbaugh and Petty and others in 1967, despite a lot of scientific investigations thereafter, still eludes clear understanding though we have learned a lot about it.

DEFINITION

This is a type of acute respiratory failure and is a descriptive term which encompasses acute diffuse lung lesions of diverse aetiologies

- Noncardiogenic pulmonary oedema
- Severe hypoxaemia
- Radiographic evidence of diffuse pulmonary infiltration
- Decrease in pulmonary compliance and also associated multiorgan dysfunction.

PREDISPOSING FACTORS

The conditions which may lead to ARDS are:

- Diffuse pulmonary infections, e.g. viral, bacterial, fungal and pneumocystic
- Aspiration of gastric contents or water in near drowning
- Inhalation of toxins and irritant, e.g. chlorine gas, N_2O, smoke, ozone, high concentrations of oxygen.
- Narcotic overdose pulmonary oedena, e.g. heroin, methadone, morphine, dextropropoxyphene.
- Non-narcotic drug effects, e.g. nitrofurantoin.
- Immunologic response to host antigens, e.g. Good-pasteur's syndrome, SLE.
- Effects of nonthoracic trauma with prolonged hypotension.
- In association with systemic reactions to processes initiated outside the lung
  - Gram-negative septicemia
  - Haemorrhagic pancreatitis
  - Amniotic fluid embolism
  - Fat embolism following long bones/pelvis fracture.
- Postcardiopulmonary bypass (Pump lung/Postperfusion lung).
- Other causes like
  - Disseminated intravascular coagulation
  - Multiple and massive blood transfusions
  - Burns.

AETIOLOGY AND PATHOPHYSIOLOGY

There are no clearcut aetiological factors and hence the pathophysiology of the syndrome can not be listed down in correlation to the aetiology.

The proposed mechanisms are as follows:

Lungs: Changes in the lungs can be divided into 3 phases:

- *Exudative phase* (1 to 6 days) characterised by:
  - Alveolar oedema, the oedema fluid having a high protein content and large number of inflammatory cells indicating increased capillary permeability.
  - Pleural effusion as an important route of clearance of oedema fluid.
  - Airway obstruction which appears to be mediated by thromboxane.
- *Proliferative phase:* Where there is infiltration of the interstitium by fibroblasts and collagen. Also there is proliferation of alveolar type-II epithelial cells. This is a subacute phase of lung injury and lasts from 6 to 10 days.
- *Fibrotic phase* (10 to 14 days onwards) where there is large destruction resulting in emphysema and the dead space increases. Functionally the oxygenation improves; but there is pulmonary vascular obliteration due to the fibrosis. In survivors beyond 3 to 4 weeks, the lungs are completely remodelled by sparsely cellular collagenous tissue.

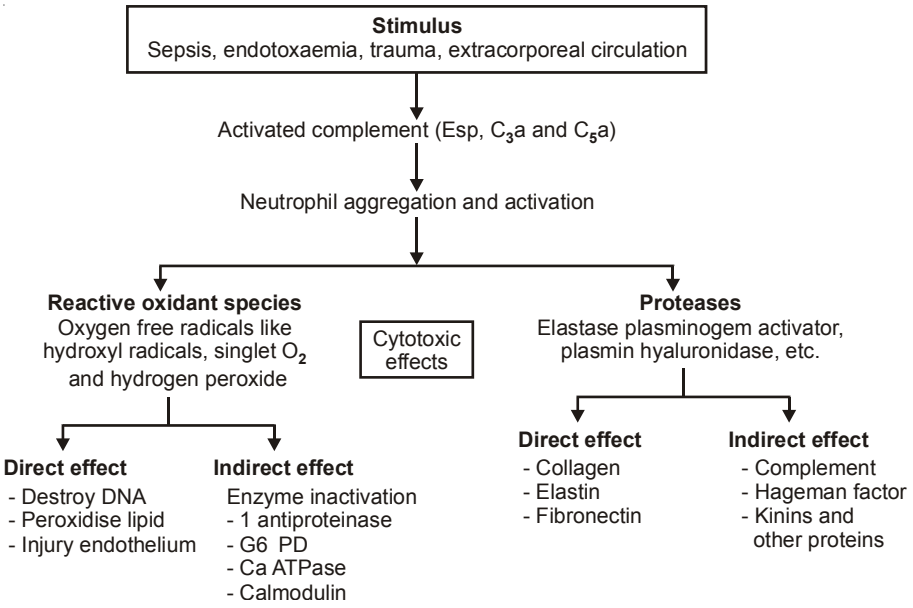
The causative factors for pulmonary pathology are:

- Physical obstruction to pulmonary circulation by fat emboli, microthrombi from transfused blood and from DIC. AS a result increase in dead space and further ischaemia to lung results.
- Circulating cells like:
  - Neutrophils
  - Platelets—A level of 100,000/mm^3 favours development of ARDS
  - Mononuclear cells—Because of release of cytokines like tumour necrosis pactor (TNF)

  The mechanism by which neutrophils inflict the cytotoxic effects as summarised by the Flow chart 46.1.

  The neutrophil aggregation is particularly increased in the presence of sluggish circulation, the failure to produce normal phagocytosis produces toxic materials like oxygen free radicals and proteases. This is referred to as "Frustrated Phagocytosis".
- Resident lung cells
  - *Endothelial cells:* Undergoes structural and functional changes in response to endotoxin and also release prostacyclin.
  - *Type II epithelial cells:* Production of surfactant and active removal of excess fluid in the air spaces by active Na^+ pump mechanism is affected.
  - *Intravascular lung macrophage:* A recently discovered cell , may release intracellular products which increase endothelial and epithelial permeability.
- Mediators:
  - O_2 free radicals, which in normal conditions are dealt with by the body with the action of the enzymes 'cytochrome oxidase, superoxide dismutase (SOD), catalase (CAT) and

Flow chart 46.1: Cytotoxic effects of neutrophil activation



glutathione peroxidase and also vitamin A and E, If free radicals accumulate, they can cause injury practically to all tissue components—lipids, proteins, collagen, etc.

– Proteases
– Complement system
– Prostaglandins and leukotrienes
– Tumour necrosis factor (TNF)

MULTIPLE ORGAN FAILURE

CVS: Occurs in 10 to 23% of patients with ARDS. The important cause is sepsis and the depression of myocardial contractility by TNF produced in sepsis.

Cardiac failure occurs when

- Cardiac index is < 2 L/mt. m^2
- Mean arterial pressure < 60 mmHg
- VF or asystole

Renal: Occurs in 40 to 55%, with ARDS, caused mainly because of hypotension, nephrotoxic drugs and sepsis. Renal failure is suspected when

- Rise in serum creatine>2 mg/100 ml

  or
- Urine output less than 600 ml/24 hrs.

Liver: Fulminant hepatic failure occurs only in 10%, of patients with ARDS. But reversible enzyme changes occur in almost 95% of cases. Criteria for diagnosing significant hepatic dysfunction are:

- Serum bilirubin >4 mg/100 ml
- Prothrombin time>1.5 times the normal
- Serum albumin <2 g/100 ml.

Hepatic failure causes altered host defence, and also blood borne substances not being detoxified in the liver can cause damage to the pulmonary endothelium.

CNS: Manifested as contusion, agitation, seizures and coma. Occurs in 7 to 30% of cases with ARDS. Thought to be present when the Glasgow coma scale is less than 6 to 8.

Causes of CNS dysfunction are:

- Production of neurotransmitters
- Direct microvascular injury
- Cerebral ischaemia

Blood: Haematological dysfunction occurs in 26% of cases. Diagnosed when

- Platelet count less than 50,000/mm^3
- White blood cell count <1000/mm^3
- Fibrinogen levels<100 mg/100 mls

GIT: Occurs in 7 to 30%, of cases and is manifested as

- Haemorrhage
- Ileus
- Malabsorption
- Cholecystitis Occasionally
- Pancreatitis

These abnormalities occur because of mucosal ischaemia and alterations in microvascular permeability.

The multiple organ failure occurs because of:

- **Altered blood flow:** Oxygenated blood bypasses nutrient capillary beds. This occurs because of:
  - Redistribution of blood to skeletal muscles.
  - Bypassing capillary bed through anatomic precapillary arteriovenous channels.
  - Reduction in recruitable capillary reserves.
- Endothelial or parenchymal injury can be seen in clinical picture as well as biochemical abnormalities and other associated disorders.
  - *Clinical picture:* ARDS is suspected and provisionally diagnosed if there is acute respiratory failure following one of the predisposing factors. An arterial O_2 tension below 50 mmHg when breathing air can be taken as a diagnostic index. The clinical picture being described here occurs during the early phase of development of the syndrome and are divided into 4 stages:
    Stage I: Hyperventilation is the only clinical abnormality. No cyanosis and chest X-ray is normal
    Stage II: Hyperventilation persists. But cyanosis ensues

Stage III: Increasing respiratory distress (with hyperventilation and cyanosis) and CXR shows diffuse consolidations.

Stage IV: Hypercarbia, metabolic acidosis and multiple organ failure manifest in this stage.

– *Biochemical and other investigation abnormalities:* A four point scoring system for lung injury has been formulated and is as follows:

1. Chest radiograph score	Score
No alveolar consolidation	0
Alveolar consolidation in one quadrant	1
Alveolar consolidation in two quadrants	2
Alveolar consolidation in three quadrants	3
Alveolar consolidation in all four quadrants	4

2. Hypoxaemia score	Value	Score
PaO_2/FIO_2	>300	0
PaO_2/FIO_2	225-299	1
PaO_2/FIO_2	175-224	2
PaO_2/FIO_2	100-174	3
PaO_2/FIO_2	<100	4

3. Respiratory system Compliance score (When ventilated)	Value	Score
Compliance	> 80 ml/cm. H_2O	0
Compliance	60-79 ml/cm. H_2O	1
Compliance	40-59 ml/cm. H_2O	2
Compliance	20-39/cm. H_2O	3
Compliance	< 19 ml/cm. H_2O	4

4. PEEP Score (when ventilated)	Value	Score
PEEP	< 5 cm. H_2O	0
PEEP	6-8 cm. H_2O	1
PEEP	9-11 cm. H_2O	2
PEEP	12-14 cm. H_2O	3
PEEP	> 15 cm. H_2O	4

The final value is obtained by dividing the aggregate sum by the number of components that were used:

– No lung injury	0
– Mild to moderate lung injury	0.1-2.5
– Severe lung injury (ARDS)	2.5

MANAGEMENT

Aims primarily at prevention and when the condition is established, treatment of the condition.

PREVENTION

- Injudicious administration and hence overloading of the circulation by crystalloids should be avoided while resuscitating the patient, especially if the CVS is compromised.
- Use of proper filters during blood transfusion to prevent microthromboembolism.
- Inotropic support - Manipulation of the circulation with inotropes and vasodilators could lead to avoidance of many of the irritating events in ARDS.
- Good aseptic precautions (especially while doing invasive monitoring) and broad spectrum antibiotic cover are mandatory).
- Controlled early O_2 therapy
- Avoid hyperoxia and barotrauma.

TREATMENT

This can be put mainly under five headings:

- *Control of infections:* Early detection and treatment of infections according to the culture and sensitivity with appropriate antibiotics. The days to come may prove other forms of therapies effective as well
  - Immunotherapy administration of polyclonal antibodies against the core lipid of endotoxin
  - Increasing the immunological surveillance by the use of muramyl dipeptides.
- *Improving gas exchange:* In potential victims, controlled O_2 therapy should be started initially. But once the condition has progressed, endotracheal intubation, IPPV with PEEP is thought to be a better choice. PEEP is advantageous in such situations because it increases the arterial oxygenation with as tittle FIO_2 as possible.
  Ventilation Strategy (see Chapter 42).
- *Limiting or decreasing alveolar oedema:* May be done by:
  - *Decrease in hydrostatic pressure*—Suggested agents include; prostacyclins, prostaglandin E_1, nitrates, Sodium nitroprusside, Ca-channel blockers, diuretics the last four being used when the patient is haemodynamically stable.
  - Increase colloid osmotic pressure. No effective method till date and whatever colloids we give in the phase of capillary leak, pass into the interstitium, thus increasing the oedema and worsening the problem.

  - Reduce capillary leak—No convincingly efficacious modalities of therapy are available. Recently tried ones are
    i. PGE and thromboxane synthetase antagonist DAZOXIBEN
    ii. N-acetyl cysteine (supposed to increase lung lymph flow).
    iii. Cryoprecipitate rich in fibronectin for reversal of opsonic deficiencies.
  - Increase the lymphatic drainage—Here also no specific measures are there. Currently experimented ones are
    i. agonist infusion
    ii. Airway instillation of amiloride (which inhibits Na-transport).
- *Improving Systemic O_2 delivery:* This may be achieved by:
  - Optimising the blood volume keeping a watch on the CVP, PAWP
  - Inotropes like dopamine

  Recent trends in this field are the use of vasodilators like sodium nitroprusside (SNP) and Glyceryl trinitrate (GTN).
- *Attenuation of pulmonary or systemic injury*

  Apart from preventive measures like keeping low FIO_2 and preventing barotrauma described earlier, some currently tried modes of'treatment in this perspective are
  - Extracorporeal membrane oxygenation (ECMO)
  - Oxygen free radical scavengers
  - Anti TNF antibodies
  - Pentoxifylline—As it is a rheological agent and may improve, the microcirculation by altering the moulding capacity of RBC's.

Attention to other modalities of care (like that of eyes, bladder, nutrition, and frequent change of position to avoid bed sores and chest physiotherapy) should be given as in any other patient admitted in ICU.

MONITORING

- Heart rate
- Blood pressure
- CVP
- PAWP
- Urine output
- Blood gas
- Pulse oximetry
- Temperature
- Serum electrolytes
- Cardiac output
- Lung compliance

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47 Hyperkalaemia

Potassium is the chief intracellular cation in the body. Normal intracellular potassium is 150 mmol/L and normal plasma K^+ is 3.5-5 mmol/L. Total body K^+ = 50 m Eq/kg. Less than 2% of the body's potassium content is in the ECF. Intracellular potassium may be considerably depleted in the presence of a normal or even a high plasma concentration. Resting membrane potential

$$E_k = 61.5 \log \frac{[K_i]}{[K_e]}$$

K_i = intracellular K^+

K_e = extracellular K^+

Hyperkalaemia is a potentially life-threatening clinical problem in which potassium concentration exceeds the upper limit of normal range. This is dangerous chiefly because of its effects on the heart.

CAUSES

- Renal failure—acute and chronic
- Respiratory/metabolic acidosis
- Steroid deficiency—Addison's disease
  Hypoaldosteronism
- Iatrogenic
  - Excessive rate of infusion of potassium salts.
  - Excessive oral or intravenous supplements.
  - Potassium sparing diuretics
- Crush syndrome
- Muscle wasting diseases—myopathies, muscular dystrophies, paralysis.
- Muscle trauma, spinal cord lesions
- Burns
- Suxamethonium
- Massive haemolysis caused by:
  - Mismatched blood transfusion
  - Massive transfusion of old stored blood
  - Malignant hyperthermia.
- Thrombocytosis
- Haemolysis
- DIC

SIGNS AND SYMPTOMS

Hyperkalaemia causes partial depolarisation of cell membranes. The effects of hyperkalaemia are on the neuromuscular system and the heart; but most patients are asymptomatic until there is marked rise in plasma potassium that is sufficient to cause cardiac arrest (greater than 7 mmol/L). It is unusual for neuromuscular effects to occur until the concentration is greater than 8 mmol/L.

Cardiac disturbances are often the first only manifestation. Pulse becomes irregular and heart block of varying degree can occur. ECG is the best indicator (Discussed later).

Neuromuscular effects are: Muscle weakness, parasthesia, decreased or loss of tendon reflexes and rarely paralysis. Mental confusion, tingling of the face, hands and feet, abdominal distension Oaqa due to ileus. Respiratory muscle weakness may cause respiratory embarassment.

ECG CHANGES

Potassium level when increases above 5.5 mmol/L:

Peaking of T waves—Earliest change (not an invariable feature)

As the potassium concentration rises further—QRS complex widens, p wave becomes lower and wider with prolonged PR interval ultimately becoming unidentifiable.

Later 'sine wave' ECG

K^+ concentration 7.5 mmol/L—Ventricular fibrillation + cardiac arrest.

TREATMENT

Treatment should be started immediately if the plasma potassium concentration is greater than 6-6.5 mmol/L.

Bl. urea, electrolytes, blood sugar and acid base balance should be checked.

- Identify and remove and/or treat the cause
- Calcium gluconate—10 ml of 10% calcium gluconate given over 2 to 5 minutes intravenously. This antagonises the cardiotoxic effects of hyperkalemia and reduces the risk of life-threatening dysarrhythmias. But it does not alter the potassium concentration.
- Correction of systemic acidosis with intravenous sodium bicarbonate. This is the fastest, safest and most reliable way of correcting hyperkalaemia.
- Glucose and insulin administration—50 ml of 50% dextrose with 5 units of soluble insulin injected over 10 mts. 'This will cause shift of potassium into cells.
- Methods to increase potassium excretion include:
  - Ion exchange resins in the sodium or calcium phase - given orally or in the form of enemas.
  - Induced diuresis if fluid balance permits-
  - Peritoneal or haemodialysis.

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48 Aetiology and Management of Intraoperative Bronchospasm

DEFINITION

The term bronchospasms denotes spasms of the bronchi, but a better term would be 'bronchiolar spasm' as this is the area of the respiratory tree which is mainly involved.

The term 'bronchospasm' was first used by T Willis (1621-1675). The commonest incidence of 'bronchospasm' intraoperatively is in a patient with COPD. But, not all wheezing that occurs intraoperative can be attributable to asthma, even in a patient with known bronchospastic disease.

DIFFERENTIAL DIAGNOSIS

- Pulmonary oedema.
- Intubation in a light plane/laryngospasm
- Endobronchial intubation
- Aspiration of gastric contents
- Mechanical obstruction of the ETT or chest wall
- Tension pneumothorax/severe atelectasis
- Anaphylaxis (drugs, mismatched blood, etc.)
- Use of a negative phase during positive pressure respiration.
- Drug induced (Beta-blockers).

DIAGNOSIS OF INTRAOPERATIVE BRONCHOSPASM

Bronchospasm during anaesthesia is detected by:

- Sudden increase in airway resistance - difficulty in ventilating the patient - airway pressure readings become high.
- Audible wheeze over lung fields detected by a precordial/ oesophageal stethoscope.
- In a spontaneously breathing patient, it is evidenced by progressively increasing difficulty in breathing with audible wheeze. All these are much more common in a person with a bronchospastic disorder.

Inspiratory and expiratory wheezes can usually be heard on auscultation, although, if the bronchospasm is very severe, there may be insufficient airflow to make any noise.

Bronchospasm is a diagnosis that should be made only when all other causes of ventilatory difficulty have been excluded.

It is very important to be sure that the difficulty in ventilating the patient and the high airway pressure are not due to mechanical obstruction of the tube - either due to kinking, mucous plugs, secretions, herniation of the cuff, etc. If there is any doubt about this, change the tube immediately. Foreign material in the tube can be suctioned out using gentle suction. Similarly endobronchial intubation; pneumothorax, possible anaphylactoid reactions, light plane of anaesthesia, etc. have to be ruled out rapidly.

A suggested protocol would be:

If wheezing/high inflating pressure/difficulty in ventilation present

↓

1. Check patency of ETT/Ensure cuff inflation/Try deflation of cuff.
   - Rule out β-blocker administration
   - ABO/Rh incompatibility
   - Over infusion (IV fluids).
2. Suction of ETT
   - Better
   - Wheeze persisting
3. Check air entry
   - Bilateral basal creps
     ↓
     could be pulmonary oedema

   Re-check and manage accordingly
4. Check air entry bilaterally-

   ↓

   ↓ air entry unilaterally
   - Endobronchial-intubation
     ↓
     Ascertain ETT
   - Pneumothorax

     Continue IPPV/Chest tube and intercostal drainage.
     Cut N_2O/ ventilation with 100% alone
     Have postoperative X-ray
     Maintain analgesia with vapour
     better
5. Wheezing persists – Deepen Anaesthesia
   - Better
   - No

   Possibly bronchospasm, especially if the patient is an asthmatic/COPD

   ↓

   Manage as outlined below.

MANAGEMENT OF INTRAOPERATIVE BRONCHOSPASM

- Deepen anaesthesia using volatile anaesthetic agent. Cut off N_2O (Halothane isoflurane or enflurane can be used to reduce surgical stimulus).
- Residual bronchospasm is treated using immediately available bronchodialators.
  - *Aminophylline:* 5 to 6 mg/kg over 10 to 15 minutes and maintained with a 0.5 to 1 mg/kg/hr infusion. Plasma theophyllin levels must be ideally within 10 to 20 mcg/ml.
    Caution—Higher incidence of dysrhythmia, convulsions and cardiac arrest, especially if used along with halothane.
  - *Salbutamol infusion*—5 mcg/kg/min (5 ml of 1 mg/ml solution added to 500 ml of 0.9% saline to give a final concentration of 10 mcg/ml).
  - When bronchospasm is severe and protracted, hydrocortisone 4 to 5 mg/kg or its equivalent to be given.

Other drugs:	**B_2 adrenergic agonists**	**Anticholinergic**
	• Albuterol	atropine
	• Terbutaline	scopolamine
	• Tenoterol	glycopyrolate
	• Salbutamol	ipratropium bromide
	• Isoprenaline	atropine methyl nitrate

Miscellaneous
Adrenaline—0.3 to 0.5 ml of 1/1000 solution given subcutaneously.

Beta Adrenergic Agents

These drugs can be given I/V, S/C, I/M or inhalational using metered dose inhalers in the breathing circuit.

Terbutaline 0.25 mg S/C or slow IV.
0.25 mg by inhalation.

Aerosol administeration has more advantages since the drugs are deposited in high concentration at the site of action thereby increasing therapeutic effects and minimising side effects. The canister can be kept in the anaesthesia circuit close to the ETT as possible using a T-piece.

Anticholinergics

The newer anticholinergics atropine methyl nitrate and ipratropium bromide are available in the aerosol form and are proven to be of lesser side effects than the parent compound.

Other Measures

- Ensure adequate depth of anaesthesia
- Maintain oxygenation

- Avoid high inflating pressures to prevent barotrauma.
- Maintain adequate humidification and hydration.
- Continue β dilator therapy to postoperative period.
- Extubate in deep plane of anaesthesia
- Use lignocaine 0.5 to 1 mg/kg IV prior to extubation.

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49 American Society of Anaesthesiologists Grading

For the assessment of anaesthetic risk there are several systems in use; the most extensively used is the American Society of Anesthaesiologists (ASA) system.

AMERICAN SOCIETY OF ANAESTHESIOLOGISTS PHYSICAL STATUS

The American Society of Anaesthesiologists classified patients into a number of grades according to their general condition. Originally there were grades, later in 1961 Dripps et al described the following categories:

ASA I: Healthy patient, i.e. the patient has no organic, physiological, biochemical or psychiatric disturbance. The pathological process for which operation is to be performed is localised and does not entail a systemic disturbance.

ASA II: Mild systemic disease – no functional limitation, i.e. mild to moderate systemic disturbance caused either by the condition to be treated surgically or by other pathophysiological process. Mild organic heart disease, diabetes, mild hypertension, anaemia, old age, obesity, mild chronic bronchitis.

ASA III: Severe systemic disease - Definite functional limitation, i.e. severe systemic disturbance or disease from whatever cause., eventhough it may not be possible to define the degree of disability with any finality.
e.g. Angina, healed myocardial infarction, severe diabetes, cardiac failure.

ASA IV: Severe systemic disease that is incapacitating and a constant threat to life, i.e. severe systemic disorders that are already life threatening, not always correctable by operation.
e.g. Marked Cardiac insufficiency, persistent angina, active myocarditis, advanced pulmonary, renal endocrine or hepatic insufficiency.

ASA V: Moribund patients unlikely to survive 24 hrs with or without operation.

ASA VI: Brain dead patient—A brain dead patient whose organs are being removed for donor purposes.

If the operation is an emergency, the letter E is placed beside the numerical classification, and the patient is considered to be in poorer physical condition.

This ASA physical status rating is applied to patients before anaesthesia and surgery. Though it is the most comprehensive system it is only a predictor of overall outcome and is most certainly not a predictor of "anaesthetic risk". The operative risk also depends upon the skill of the surgeon.

The physical status was never intended to be a nultifactorial index or predictor of outcome and it only facilitates communication among anaesthesiologists.

Marx et al ASA physical status and anaesthetic deaths (Table 49.1).

Table 49.1: Anaesthetic deaths recording as per ASA grades

ASA grades	*No. of cases*	*No. of anaesthetic deaths*	*Incidence*
I	18,320	2	1:10000
II	10,609	1	1:10000
III	3,820	11	28:10000
IV	1,073	8	74:10000
V	323	5	155:10000
VI	—	—	—

17.8% of all patients experienced one or more anaesthetic complications other than death of which:

- Nausea and vomiting was 50% of the total postoperative complications
- Sore throat was the second most but minor.

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50 Post-dural Puncture Headache

Post-dural puncture headache is a low pressure headache usually experienced over the forehead or occipital region due to continuous CSF leakage, through the dural puncture hole.

INCIDENCE

The incidence is about 3-30% in inadvertent dural puncture during extradural block with Tuohy-needle, has its maximum incidence of headache. The incidence will vary according to size of needle used.

- 16 G needle – 24%
- 20 G needle – 14%
- 24 G needle – 6%
- 25 G needle – 3.5%
- 32 G needle – 1.4%

AETIOLOGY

- *Low CSF pressure theory:* The pressure of CSF in subdural space is 150 mm of H_2O. But the pressure in the lumbar epidural space is sub-atmospheric. So some escape of CSF will invariably occur after LP and will continue until the hole becomes occluded. It is calculated that average, CSF leakage is about 10 ml/hr. This may lead to decreased CSF pressure as low as 50 mm of H_2O. The healing of the dural puncture hole may take up to 3 weeks. So a large gauge needle and any thing which increases CSF pressure like pregnancy increases the leakage and so incidence of postpuncture headache. When the rate of leakage exceed the rate of formation it results in change in hydrodynamics of the CSF. This lead to loss of cushioning of the brain and pressure or traction on vessels and pain sensitive structure like basal dura and tentorium, producing headache.
- *High CSF pressure theory:* Due to bacterial or chemical due to bacterial or chemical meningitis.

DIAGNOSIS

Diagnosis is purely clinical. Typical postdural puncture headache has certain well defined characteristics.

- The onset of headache is likely to be 12 to 24 hrs after LP but may delay/upto 2 to 7 days and this may last for a week but occasionaly it may last for several weeks.
- It is a diffuse, some time throbbing headache, and is different from any other previously; experienced pain.
- The pain may spread across the whole of frontal area, or be localised behind the eyeball or on the occipital and nuchal region. When it is localised in the nuchal region it is often associated with neck stiffness.

- Pain is made worse by sitting up and relieved by lying supine or by abdominal compression.
- Patient may also have difficulty for focusing his eyes.
- Tinnitus or deafness may occur due to fall in intralabyrinthine pressure.

PREVENTION

- Avoid spinal analgesia in patients with history of frequent headache and neurotic patients.
- Prophylactic use of smallest possible needle.
- Needle should be introduced so that the bevel is parallel to the longitudinal fibres of dura.
- Use spinal needle which are designed to spread the dural fibres rather than cutting.
- Coughing and straining should be avoided.
- Encourage the patient to remain recumbant for 12 to 24 hrs after intradural analgesia.
- Adoption of prone position is also to be encouraged.
- Avoid dehydration.
- Do not flex the back excessively during LP; this would stretch the dural hole wide open.

TREATMENT

- Conservative measures for prevention should be continued.
- Frequent- long drinks to be taken for good hydration.
- A tight abdominal binder may be used.
- Simple analgesics like aspirin or paracetamol may be helpful.
- Antidiuretic drug (promotes fluid retention and increased formation of CSF) was used in some centres, but the results are not encouraging.
- Injection or infusion of saline or Hartmann's solution into the epidural space by putting a catheter has been recommended, but sometimes the relief may be temporary.
- In intractable cases, an epidural blood patch may be considered. Its success rate is 92%. The firm clot will seal the dural hole and prevent the CSF leakage. There is no role for prophylactic blood patch.

Two sterile, gowned and gloved operators are required, one to carry out the epidural and other to collect the patient's own blood, with a sterile anticoagulant free syringe simultaneously. A volume of 10-20 ml of blood is used in the same interface where LP was done. Epidural blood patch frequently produce mild backache, neckache and paraesthesia and causes introduction of infection also. So it is a procedure which should only be used in severe refractory post dural puncture headache.

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51 Role of Anaesthesiologist in Eclampsia

INTRODUCTION

Pre-eclampsia and eclampsia remains a major cause of maternal mortality and morbidity and a cause of foetal death around the world.

The role of the anaesthesiologist in eclampsia includes :

- Management of pain of labour
- Provide anaesthesia for caesarean section.
- Management of convulsions, and hypertensive crisis and other life-threatening complications of eclampsia.
- Resuscitation of the small for gestational age/preterm neonate.
- Airway management.

DEFINITIONS

To understand the role of the anaesthesiologist in eclampsia we need to understand what eclampsia is and the pathophysiological changes in eclampsia.

Pre-eclampsia is a disorder which manifest usually after 20th week of pregnancy characterised by the triad of (1) hypertension (2) generalised oedema (3) proteinuria.

Hypertension: The BP should be more than 160 mmHg systolic and more than 90 mmHg diastolic. Atleast two readings should be obtained 6 hours apart.

Eclampsia is defined as pre-eclampsia with convulsions not related to any other cerebral conditions, with any degree of hypertension.

Pre-eclampsia becomes *Imminent eclampsia* when following features are seen:

- Diastolic BP more than 110 mmHg.
- Visual disturbances/headache.
- Epigastric pain.
- Oliguria (Less than 400 ml/24 hrs)
- Pulmonary oedema.

TARGET ORGAN CHANGES IN SEVERE PET/ECLAMPSIA

- *CNS:* The cause of convulsions in severe PET is due to severe vasospastic ischaemic injury resulting in cerebral ischaemia and focal cerebral oedema. The effects of severe PET in CNS are:

  - Convulsions
  - Intracranial haemorrhage
  - Cerebral oedema
- *CVS:* In the CVS main changes seen are:
  - Decreased intravascular volume because of the chronic hypertensive state.
  - Increased SVR commonly occurs, but SVR may remain unchanged
  - Decreased cardiac output.
  - Pulmonary oedema and left ventricular failure can occur in severe PET/eclampsia.
- *Haematological:* There is decreased perfusion of all organs systems in the body because, of: (a) decreased intravascular volume, (b) platelet aggregation, (c) hypertension, (d) increased viscosity.

Coagulation Abnormalities

- Thrombocytopenia.
- HELLP syndrome (Disordered liver function resulting in haemolysis, elevated liver enzyme and low platelet.
- *DIC:* This occurs because of release of thromboplastin from the placenta and results in increase in fibrin degradation products.
- *Respiratory system:* The most serious problem in the respiratory system from the anaesthesiologist's point of view in eclampsia is laryngeal oedema which can result in difficult intubation and cause postoperative stridor.
- *Liver:* Subcapsular haemorrhages and hepatic necrosis results in impaired liver function. A lethal complication is spontaneous liver rupture.
- *Kidneys:* Kidneys are affected because of decreased renal blood flow due to renal artery vasospasm and increased glomerular permeability because of fibrin degradation products and glomerular swelling.
  This results in:
  - Proteinuria
  - Acute renal failure.

Anaesthetic Considerations and Management of Severe PET/Eclampsia

The only definitive cure for severe PET is delivery of the foetus and most important of the placenta. Until this occurs, the obstetric objective is control of the disease process to provide a uteroplacental environment favourable for the growth and maturity of the foetus without endangering the mother.

THE AIMS OF THERAPY IN SEVERE PET/ECLAMPSIA

- To minimise vasospasm.
- To improve circulation to uterus, placenta, kidneys.

- To improve intravascular volume.
- To correct electrolyte and acid base imbalances.
- To decrease both CNS and reflex hyperactivity.

TREATMENT OF PET

- Hospitalisation and bedrest.
- Rest in left lateral position to improve uteroplacental circulation by preventing aortocaval compression.
- Adequate fluid intake to improve intravascular volume.
- Antihypertensive Drugs: Drugs most commonly used are: (a) Hydralazine, (b) Methyldopa, (c) Nifedipine

Eclampsia: The first and foremost treatment modality in eclampsia is *stop convulsions and control of convulsions*. The next step is *termination of pregnancy*, since maternal health and life is at jeopardy. This can be accomplished by vaginal delivery/LSCS.

Control of convulsions: The drugs given to control convulsions in eclampsia are:
- $MgSO_4$
- Diazepam
- Phenylhydantoin
- Chlormethiazole.

Magnesium sulfate: IV loading dose of 20 to 40 mg/kg body weight infused over 5 minutes, followed by infusion of 1 to 2 gm/hr to maintain therapeutic plasma level of 2 to 4 mmol/L.

Monitoring during Magnesium Therapy

Since magnesium causes neuromuscular blockade, it is important to keep in mind that magnesium causes severe muscular weakness and ventilatory failure, and depression of deep tendon reflex is indication of impending magnesium toxicity.

Diazepam: Given in 5 to 10 mg IV increments until effective. Disadvantages: (1) Excessive sedation with consequent risks to airway. (2) Marked foetal depression may occur and has been a major factor in decline in its use.

Phenytoin: Phenytoin is given in loading dose of 10 mg/kg in 100 ml saline is infused at a rate of 50 mg/min, followed 2 hours. later by second bolus of 5 mg/kg. In convulsions, simultaneously, the airway of the patient must be maintained and protected.

Control of hypertension: Antihypertensive therapy is started if diastolic BP remains above 110 mgHg.

Aims of Antihypertensive Therapy

To cause a gradual reduction in blood pressure so that the diastolic BP is below 110 but above near normal range, since sudden fall in BP can be disastrous and cause further uteroplacental ischaemia and renal failure.

DRUGS USED TO TREAT HYPERTENSION

- *Hydralazine:* Administered IV in 5 mg increments followed by 5 mg/hr titrated against BP. This drug is most widely used for control of hypertension in eclampsia. It is preferred, because of its rapid onset of action and it is principally an arterial vasodilator and hence improves vasospasm.
- *Nitroglycerine:* Nitroglycerine infusion of 2 to 5 mcg/kg body weight/min.
- *Nitroprusside:* Nitroprusside infusion in dose of 2 to 5 mcg/kg body weight/min.
- *Trimethaphan:* The principle advantage claimed for this agent is absence of significant cerebral vasodialatory affect.

Prerequisites to be fulfilled in eclampsia before delivery:

- Control of convulsions
- Control of hypertensive crisis
- Prehydration of patient
- Improvement of renal function

Monitoring of the patient

- *Blood pressure:* Should be frequent and is by:
  - Indirect BP monitoring by automated oscillometric method.
  - Direct arterial cannulation and direct BP monitoring for beat-to-beat assessment of BP.
- *CVP monitoring*
  - To know the amount of fluid to be infused.
  - To understand the likelihood of CCF.
- *Pulmonary artery catheterisation by Swan Ganz cathetar:* This is to ascertain the presence of left ventricular failure, but since the presence of LVF in severe PET/Eclampsia is rare, it is not used frequently. Indicated in the following situations.
  - Cases of refractory oliguria.
  - Cardiac lesions
  - Signs of CCF.
- *Haematological profile study:* Since DIC is one of the risks involved in eclampsia, a coagulation profile must be carried out and it includes:
  - Platelet count
  - Total clotting time
  - Bleeding time
  - Activated partial thromboplastin time
  - Prothrombin time

  - Demonstration of fibrinogen degradation products.
  - Serum fibrinogen level.
- *Urinary catheterisation*
  - To assess the renal status
  - To assess the prognosis of the patient.

Anaesthesia for Caesarean Section

In severe PET/Eclampsia emergency caesarean section is most often required because of deteriorating: (a) Condition of the mother, (b) Foetal distress caused by stress of labour, when uteroplacental perfusion is compromised.

The two options available are:
- Regional anaesthesia
- General anaesthesia.

In regional anaesthesia, it has been proved beyond doubt that "Lumbar continuous Epidural Anaesthesia" is a superior technique if there is no disturbance in the coagulation profile.

Advantages
- Excellent pain relief and muscular relaxation.
- Avoidance of narcotics and sedatives which have a depressant action on the neonates.
- Gradual reduction of BP (sudden hypotension avoided) with adequate prehydration.
- Risk of pulmonary aspiration minimised.
- The maternal circulating level of epinephrine is decreased and hence improves uteroplacental blood flow and also renal blood flow.
- Sudden hypertensive crisis especially ocurring during intubation is avoided.

Spinal anaesthesia is not preferred because of the very sudden fall in BP associated with it, which compromises the circulation.

Epidural analgesia is instituted by placement of epidural catheter and a dose of local anaesthetic sufficient to produce sensory block upto T10. (8-10 ml of 3% chloroprocaine/0.5%, Bupivacaine/ 1.5% Xylocaine). After the initial block is obtained and the central venous pressure and maternal blood pressure stabilised,the sensory level is raised to minimum T4 level by titrated doses through epidural cathetar. After delivery and closure, additional doses may be given for postoperative pain relief. If hypotension occur, ephedrine in small dose of 5 to 10 mg IV can be given.

GENERAL ANAESTHESIA

Indicated in cases of:
- Failed epidural analgesia
- Haematological profile disturbances
- Rapid delivery required as due to haemorrhage or foetal distress.

The important considerations to keep in mind are:

- Crash induction.
- Avoidance of severe maternal hypertension on endotracheal intubation.
- Prehydrating the mother to improve the intravascular volume status.
- Because of risk of laryngeal oedema in few cases, it is safer to use a small size endotracheal tube.
- Likelihood of muscle paralysis being prolonged because of interaction of magnesium, with muscle relaxants and titrated dose of muscle relaxants to be given.
- Maintain and ensure optimal foetal oxygenation.
- Possibility of stridor after, extubation because of oedema to be watched for.

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52 Myxoedema and Anaesthetic Indications

The word myxoedema was introduced by Ord in 1878. It is the generic term for exposure of the body to subnormal amounts of physiologically active thyroid gland hormone.

AETIOLOGY OF HYPOTHYROIDISM

Decreased function of thyroid gland may be due to secondary or primary causes.

Secondary hypothyroidism	*Aetiology*
• Hypothalamic dysfunction	• Deficiency of thyroid releasing hormone.
• Anterior pituitary dysfunction	• Deficiency of thyroid stimulating hormone.
Primary hypothyroidism	
• Thyroid gland destruction	Previous subtotal thyroidectomy, previous therapy, irradiation of the neck.
• Thyroid gland hormone deficiency	• Chronic inflammation Hashimoto's • Dietary iodine deficiency • Excess iodides (inhibits release) • Antithyroid drugs

Subclinical hypothyroidism, present in about 5%, of population; is manifested solely by elevated plasma concentration of thyroid stimulation and hormones. Chronic thyroiditis (Hashimoto's thyroiditis) is assumed to be the cause of dysfunction.

DIAGNOSIS

- Clinical signs and symptoms.
- Decreased thyroid gland function demonstrated by appropriate tests.

SIGNS AND SYMPTOMS

Depend on the age of onset of thyroid gland dysfunction.

- In neonatal period, can result in cretinism which is characterised by decreased physical development and mental retardation.
- In adult, development of hypothyroidism is insidious and may go unrecognized for several years.

Physical Examination

- Delayed ankle jerks
- Husky voice

- Presence of dry skin
- Generalised reduction in metabolic activity.
- Lethargy is prominent and intolerance to cold.

Cardiovascular System

- Bradycardia and decreased stroke volume.
- Decreased cardiac output, increased systemic vascular resistance and decreased blood volume, diastolic hypertension result in prolongation of circulation time.
- Narrowed pulse pressure.
- Peripheral vasoconstriction leads to cool and dry skin.
- Many manifestations mimic cardiac failure, cardiomegaly, pleural effusion, peripheral oedema and ascitis.
- Increased circulating catecholamines.

Endocrine System

- Associated atrophy of adrenal cortex.
- Associated atrophy of pituitary.

Unrecognised hypoadrenocorticism could lead to cardiovascular collapse during anaesthesia- and surgery.

TREATMENT

- Exogenous replacement of the hormones.
- Thyroid hormone concentration in circulation must be restored slowly because of the danger of precipitating angina pectoris, cardiac dysrhythmia and congestive cardiac failure.
  - Thyroxine takes 10 days to exert a physiological effect and hence not useful.
  - Triiodothyronine acts within six hours and reaches peak with in forty eight to seventy two hours.
  - Exogenous administration of cortisol.
- Digitalis for congestive cardiac failure may adversely affect the hypothyroid heart which cannot perform increased myocardial contractility.

  Adequacy of thyroid replacement confirmed by normalisation of serum TSH.

MANAGEMENT OF ANAESTHESIA

Elective surgery: Postponed until the patient is euthyroid.

Emergency: In emergency situation, the following items that affect anaesthetic management should be noted and corrected.

- Hypothyroid patients exhibit marked sensitivity to depressant drugs.
- Cardiovascular system is characterised by a low cardiac output, high systemic vascular resistance and low heart rate.

- Drug metabolism reduced.
- Baroreceptors tend to be unresponsive and beta receptors down regulated.
- Intravascular volume is decreased.
- Ventilating response to hypoxaemia and hypercarbia are blunted.
- Free water clearance, is impaired resulting in hyponatraemia.
- Gastric emptying is delayed.
- Baseline hypothermia is common.
- Normocytic anaemia is common.
- Primary adrenal insufficiency and consequent hypoglycaemia.

Premedication

- Cortisol support.
- Reduced doses of sedatives or anticholinergic drugs can be administered intravenously on arrival in the operating room if essential.

Preinduction Monitoring

- Pulse oximetry.
- Invasive blood pressure monitoring.
- Central venous pressure monitoring for guiding the rate of I/V fluids infusion.
- Core temperature recording for early detection of onset of hypothermia.
- Continuous electrocardiogram monitoring
- Pulmonary artery catheterisation and transoesophageal echocardiography for left ventricular function monitoring.

INDUCTION OF ANAESTHESIA

This is accomplished with slow intravenous ketamine. Short acting muscle relaxants like scoline are used to facilitate intubation. However, both propofol and thiopental can be used in reduced doses.

MAINTENANCE OF ANAESTHESIA

This is achieved with inhalation of nitrous oxide and oxygen. Supplementation if necessary with minimal dose of short acting opioids (alfentanil), benzodiazepine, (midazolam) or ketamine.

Precautions

- Volatile anaesthetic agents if used, the sensitivity to myocardial depression should be borne in mind. Temperature below 37° C and slow hepatic metabolism and .renal elimination would reduce the anaesthetic requirements of these drugs.
- Nondepolarising muscle relaxants like pancuronium with mild sympathomimetic effect is beneficial.

- Controlled ventilation is necessary as these patients have tendency to hypoventilate - Avoid hyperventilation.
- Reversal of nondepolarising neuromuscular blockade with anticholinesterase drugs combined with anticholinergics poses no known hazard.
- Recovery from sedative effects of anaesthetic drugs may be delayed resulting in prolonged post- operative sedation.
- Extubation should be done only after patient responds and body temperature is near 37° C.
- Postoperative analgesia only with nonopioids.
- Postoperative hypotension treated with hydrocortisone.

REGIONAL ANAESTHESIA

Doses of local anaesthetic necessary might be reduced. The metabolism of amide local anaesthetics seen to be slowed. This could predispose to development of systemic toxicity.

MYXOEDEMA COMA

Mild undiagnosed hypothyroidism in patients undergoing surgery may postoperatively result in myxoedema coma and respiratory obstruction requiring intravenous T3 thyroid replacement, and airway support, and hydrocortisone hemisuccinate and temperature maintenance.

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53 Anaesthetic Considerations in Ophthalmic Surgery

Ophthalmic surgery may be classified into

- Extraocular—Operations for retinal detachment, squint correction, orbital surgery.
- Intraocular—Cataract extraction, peripheral iridectomy, trabeculectomy, etc.
- Mixed.

LOCAL ANAESTHESIA

Many ophthalmic operations can be performed under local anaesthesia. Local anaesthesia is feasible for short procedures, but general anaesthesia permits the surgeon to operate without undue hurry and eliminates the apprehension of the patient.

Advantages

- Safer, especially in patients with heart disease.
- Less postoperative nausea and vomiting.
- Less bleeding.
- Less risk of pulmonary embolism.
- Less upset of biochemical processes.
- Less postoperative restlessness.
- Less postoperative coronary or cerebral thrombosis.

Disadvantages

- Lack of control of the patient.
- Difficulty in controlling intraocular pressure.

Complications of retrobulbar block, which include haemorrhage, local anaesthetic toxicity, due to accidental intravascular injection, accidental brainstem anaesthesia (unconsciousness, apnoea).

Facial nerve block is necessary before all intraocular procedures under local analgesia to prevent blepharospasm.

Infraorbital block from the infraorbital canal below the eye, produces analgesia of the central part of the lower lid; lacrimal block its lateral part; supra and infra trochlear block its medial part—these can all be reached as they emerge from the skull.

Retro-ocular block must be done before operation under local analgesia on the globe of the eye.

GENERAL ANAESTHESIA

The goals of the general 'anaesthesia in ophthalmic surgery' include maintaining an immobile eye with a stable intraocular pressure, preventing the oculocardiac reflex and achieving a smooth induction and emergence from anaesthesia with minimal nausea and vomiting.

Anaesthetic Considerations

- *Intraocular pressure:* Normal IOP is 15 to 25 mmHg. It is kept remarkably constant by a dynamic balance between the production and drainage of aqueous humor. IOP is lowered by:
  - Physiological factors—Hypocapnia, hypotension, reduction of central venous pressure, hyperoxemia.
  - Pharmacological factors—Intravenous anaesthetics except ketamine and narcotic analgesics, volatile anaesthetic agents, carbonic anhydrase inhibitors—Acetazolamide, osmotic diuretics—mannitol, urea, glycerin, sucrose (50% solution).

  Non-depolarising relaxants, Barbiturates, benzo-diazepines, opioids.

  IOP is raised by overhydration -(increased venous pressure), hypoxaemia, hypercapnia, drugs like suxamethonium, ketamine, topical steroids, laryngoscopy and tracheal intubation, vomiting, coughing, sneezing, straining and other causes of raised central venous pressure. Atropine raises IOP only in narrow angle glaucoma.
- *Oculocardiac reflex:* OCR is a trigeminovagal reflex in which a variety of stimuli arising in or near the eye may cause abnormalities of the rate and rhythm of the heart, manifested by arrhythmias such as bradycardia, nodal rhythm, ectopic beats, ventricular fibrillation or asystole. It occurs most often during strabismus surgery in children and retinal surgery. This may be seen in operations on the empty orbit also. Hypoventilation, hypercarbia, hypoxaemia increase the incidence of bradycardia during strabismus surgery.

  Drugs to prevent OCR
  - Intravenous atropine—15 mcg/kg
  - Intravenous glycopyrrolate 7.5 mcg/kg
  - Intravenous gallanine
  - Alcuronium
  - Hyosine butyl bromide.

  If an arrhythmia develops and persists, the surgeon should be asked to temporarily suspend all manipulations and allow the heart to recover. Atropine 10 to 15 mcg/kg may be given intravenously and repeated if necessary. Recurrent episodes of OCR may be treated by infiltration of the extraocular muscles with a local anaesthetic and deepening anaesthesia. Normocarbia should be maintained.
- All disturbances causing movement, cough or contraction of the orbicularis oculi must be avoided as they may increase the intraocular tension with resulting iris prolapse or vitreous prolapse.
- For the same reason, postoperative vomiting and coughing should be minimised.

- Congenital cataract may be associated with dystrophia myotonia or with diabetics.
- Ophthalmic medication applied topically to the cornea may undergo sufficient absorption to produce systemic effects. Unexpected drug interaction during or after surgery may reflect systemic effects of these drugs, e.g. timolol local application - associated with bradycardia and bronchospasm, exacerbation of myaesthenia gravis postoperative apnoea in neonates. Chronic treatment with acetazolamide causes metabolic acidosis with hypokalaemia.

Anaesthetic Management of Ophthalmic Surgery

The eye is a highly innervated and sensitive organ. Ophthalmic surgery therefore requires a fairly deep level of general anaesthesia (stage III, plane 3). Light anaesthesia may result in hypertension and patient movement. Bucking and straining may cause orbital venous congestion, bleeding, increased IOP and loss of vitreous.

Premedication: Goals of premedication are to allay anxiety and minimise nausea, vomiting and coughing. Narcotic analgesics are better avoided for this reason. Suitable agents include phenothiazine derivatives, diazepam and lorazepam. Atropine 0.01 mg/kg may be given (IV) to children prior to strabismus surgery. In adults, atropine is best reserved for treating an established OCR.

Anaesthetic technique: An (IV) induction sequence using thiopentone and succinyl choline can be performed in elective surgery. Suxamethonium increases IOP, but the rise is probably transient; confined to the period of apnoea. It should not be used during an operation when the eye is already open or in patient with recent eye surgery for fear of precipitating vitreous prolapse. Intravenous injection of acetazolamide 500 mg immediately before the induction of anaesthesia largely prevents rise in IOP due to suxamethonium. The trachea is intubated after achieving an adequate depth of anaesthesia. The use of a topical lignocaine spray to the larynx and trachea may facilitate smooth emergence at the end of surgery and prevent coughing.

Anaesthesia is maintained with 0.65 MAC of the volatile agent in 30% oxygen with N_2O. N_2O must be used with caution if an intravitreal injection of sulfahexaflouride is planned. Controlled ventilation with IPPV to maintain $PaCO_2$ at normal levels. Non depolarising muscle relaxants like pancuronium or vecuronium can be used.

Gallamine can be used to block OCR.

Emergence from anaesthesia should not be associated with any reaction to the tracheal tube. IV lignocaine—1.5 mg/kg can temporarily suppress the cough reflex during emergence. Neostigmine or edrophonium is used along with atropine to reverse residual neuromuscular blockade without altering IOP. All efforts should be made to avoid coughing and vomiting postoperatively.

Monitoring: Close monitoring of these patients is mandatory because of the lack of access to the patients airway, the method of draping and the position of the anaesthesiologist at the patients side. Standard monitoring include exposing one hand of the patient to check colour and radial artery pulsations, blood pressure, electrocardiography, end tidal carbon dioxide and neuromuscular monitoring.

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54 Transurethral Prostatectomy Syndrome

Transurethral resection of prostate is one of the most commonly performed surgical procedures in men over 60 years of age. The operation is performed through a modified cystoscope, and with an electrically energized wire loop. The hypertrophied lobes are resected, bleeding being controlled with a coagulation current. Continuous irrigation is needed for distending the bladder and to wash away blood and dissected prostatic tissue.

Absorption of irrigation fluid into blood stream is inevitable, and at the time of use of distilled — water as irrigation fluid, was one of the foremost causes of iatrogenic water intoxication. Absorption of fluids occurs through the open venous sinuses.

Simple principles to determine the amount of absorption fluid are:

- The height of the container of irrigating solution above the surgical table determines the hydrostatic pressure driving fluid into prostatic veins and sinuses. When the height exceeds 60 cm, significant volume absorption can occur.
- Time of resection is proportional to the quantity of fluid absorbed. An average 10 to 30 ml of fluid is absorbed per minute of resection time. Time is not an important factor unless the duration of surgery exceeds 150 m.
- Number of venous sinuses opened during resection.
- Skill of the surgeon.

Complications as a consequence of absorption of irrigating fluids depend on: (1) Amount of fluid, (2) type of fluid.

Earlier, distilled water was used for bladder irrigation as it interferes least with visibility. However absorption of large quantities of water led to dilutional hyponatraemia which resulted in haemolysis of RBCs and CNS symptoms due to cerebral oedema. Iso-osmotic or near to iso-osmotic fluids are now being used. Normal saline and Ringer lactate would be well tolerated if absorbed intravascularly, but their highly ionised charges can facilitate dispersion of high frequency current from the resectoscope. So non-electrolyte solutions like glucose, urea (1%), glycine (1.2%), mannitol (20.54%) or sorbitol (2.7%) are now used. Of these crystal (sorbitol + mannitol) and glycine, are the two most commonly used solutions. Complications are very much reduced but CNS symptoms, overhydration caused by circulatory overload and decreased plasma osmolarity still remain as complications.

CLINICAL FEATURES

Symptoms

- Dizziness, headache, nausea, vomiting
- Tight feeling in the chest and throat

- Shortness of breath
- Restlessness, confusion, tonic clonic seizures leading to coma in some cases.

Cardiac symptoms like frank pulmonary oedema, dysrhythmia and conduction abnormalities can also result from hyponatraemia.

Signs

- Initial rise in blood pressure with reduction in heart rate which is followed by hypotension and tachycardia.
- In some cases presents as refractory bradycardia.
- EKG-ST segment changes, U waves, widening of QRS complexes.
- Serum Na—clinical findings usually appear when sodium falls below 120 mEq/L; convulsion occurs when the sodium level is less than 100 mEq/L.

Concentration of extracellular sodium must be in the physiological range for depolarisation of excitable cells and for the production of action potential.

Absorption of glycine, a non-essential aminoacid has been attributed to some of CNS symptoms associated with TURP, transient blindness being the commonest manifestation. Glycine has a distribution similar to gamma aminobutyric acid, which is an inhibitory transmiter in the brain. Also absorption of glycine results in its oxidative biotransformation to ammonia. Delayed awakening after TURP was found to be associated with elevated levels of blood ammonia.

Bladder perforation with a resectoscope can occur leading to extra or less often intraperitoneal leak of irrigating fluid leading to increased absorption and similar symptoms.

Some other common accompaniments of TURP syndrome are:

- Hypovolaemia due to excessive blood loss. Average loss is 280 ml.
- Coagulopathies—Because of dilution of coagulation factors.

Management of anaesthesia: Spinal/epidural anaesthesia has been recommended for TURP since awake patients may demonstrate early signs of excessive intravascular absorption of irrigating fluids or accidental urinary bladder perforation. General anaesthesia may mask these signs. Regional anaesthesia also produces sympathetic block which increases venous capacitance and to a level, mitigates intraoperative fluid overload. But when the block dissipates, venous capacity decreases and can precipitate circulatory overload.

Monitoring: Intraoperative monitoring of haematocrit, plasma sodium concentration or osmolarity may be useful in detecting excessive haemodilution. Central venous pressure elevations are likely to accompany hypervolaemia. Plasma sodium concentration below 120 mEq/L signifies excessive haemodilution.

TREATMENT

- Terminate the surgical procedure as soon as possible.
- Administer Frusemide IV 20 mg bolus dose and repeated.

- Administer oxygen by nasal cannula or face mask.
  If the patient is in pulmonary oedema, consider the possibility of ET intubation and ventilation with 100% oxygen.
- Arterial blood gas estimation for blood gas as well as sodium level estimation.
- If sodium level is below 120 mEq/L, administer hypertonic saline 3-5% at rate not faster than 100 ml/hr.
- If the patient develops convulsion use diazepam intravenously. Other drugs which can be used are dilantin sodium and barbiturates.
- If the patient has already developed pulmonary oedema, or shock, go for complete invasive monitoring with pulmonary and systemic arterial lines.

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55 Sickle Cell Anaemia and its Anaesthetic Implications

Sickle cell anaemia was first noted clinically by JB Herrick in 1904. In 1927 Hah and Gellespie induced hypoxia and acidotic sickling and incriminated haemoglobin as the cause, by demonstrating them in ghost erythrocytes.

Sickle cell disease represents an inherited group of disorders, ranging in severity from benign sickle cell trait to the debilitating sickle cell anaemia. All the variants possess varying quantities of haemoglobin-S which differs from normal haemoglobin-A by substitution of valine for glutamic acid at the sixth position on the beta chain of haemoglobin molecules. Confirmation of haemoglobin-S is by electrophoretic studies.

Sickle cell anaemia: This is present when patients are homozygous for Hb S. In this state, 70% to 98% of haemoglobin is of the S type. Approximately 0.3% to 1%, of black population of United States suffer from it.

Sickle cell trait: This is the heterozygote manifestation of sickle cell disease containing genotype AS. 20-40% of haemoglobin are of S type and remainder of haemoglobin

A. Incidence in black population is 10%.

PATHOPHYSIOLOGY

Deoxygenated forms of haemoglobin S result in deformation of erythrocytes into sickle shapes. Tactoids of sickle cells increase viscosity of blood, leading to stasis of blood flow and infarctive crisis. In addition, low oxygen concentration causes haemoglobin to precipitate into long crystals inside the RBCs. Thus damaging erythrocyte membranes leading to their rupture and chronic haemolytic anaemia.

Factors Promoting Formation of Sickle Cells

- Low arterial oxygen partial pressures below 40 mmHg in sickle cell anaemia and in sickle cell trait, below 20 mmHg.
- Presence of acidosis.
- Reduction in body temperature results in vasoconstriction, stasis of blood flow and sickling.
- Dehydration and resulting stasis of blood flow favour formation of sickle cells.

CLINICAL MANIFESTATIONS

- Those due to infarctive events due to occlusion of blood vessels with sickle cells.
- Those due to chronic haemolytic anaemia (Hb 6-8 gm/dl).

Infarctive events are responsible for widespread organ damage.

Cardiovascular system: Corpulmonale due to repeated pulmonary emboli, and also, secondary to high output failure.

Central nervous system: Cerebrovascular accidents are common, especially in children. Stroke is significantly reduced by transfusion programme of 2 units every fortnight. Storke may be induced by hyperventilation, severe anaemia, infection, sickle cell crisis.

Respiratory system: Total lung capacity (TLC) and vital capacity (VC) are frequently decreased. Pulmonary embolism and respiratory infection are common in postoperative period.

Genitourinary system: Renal abnormalities are established by the age of five to eight years. The hypertonic medulla concentrates Hb and with its low oxygen partial pressure promotes sickling. This produce papillary necrosis, haematuria and inability to concentrate urine and renal failure. Priapism is a common occurrence.

Hepatic and splenic infarcts: May be focal or diffuse. Severe liver dysfunction can result in pseudo-cholinesterase deficiency.

Skeletal system: Aseptic necrosis of femoral head and osteomyelitis are common. Salmonella infection of small bones of the hand.

Chronic haemolysis of erythrocytes is reflected by:
- Elevated levels of plasma bilirubin leading to cholelithiasis and cholecystitis.
- Periodic transfusion increases risk of viral hepatitis.
- Haemochromatosis ensues with iron overload following repeated transfusions. It leads to cirrhosis of liver and left ventricular dysfunction also.
- White cell function is depressed with increased susceptibility to infection.

Infarctive Crisis

This is trigerred off by infection, trauma or associated elevations in temperature. It is characterised by acute onset of pain usually abdominal with fever and vomiting.

TREATMENT

- Adequate hydration
- Partial alkalisation of blood
- Partial exchange transfusion with erythrocyte containing haemoglobin A.
- Antibiotics.

MANAGEMENT OF ANAESTHESIA

Preanaesthetic Transfusion

Transfusion of normal (Haemoglobin A) RBC's may be single multiple or exchange transfusion if haemoglobin is below 7 to 8 gm/dl. Packed cell transfusion is desirable. For major thoracic or haemorrhagic surgery exchange transfusion is indicated.

Preoperative Assessment and Preparation

- Existing organ dysfunction should be assessed.
- Aggressive preoperative hydration is essential.
- Prophylactic antibiotic cover.
- Systemic peroperative alkalisation which on one hand confers an antisickling effect and on the other hand shifts the oxyhaemoglobin curve to left.
- Correction of co-existing infection.

Premedication

Avoid drugs that causes respiratory depression.

Preoxygenate well; induction with thiopentonesodium and succinyl choline followed by tracheal intubation and controlled moderate hyperventilation with nitrous oxide and oxygen. 30% oxygen is adequate and judicious doses of halothane to promote vasodilatation.

Intraoperative and Postoperative Precautions

- Strict asepsis during placement of all invasive catheters.
- Supplemental O_2 should be provided with high FIO_2 to enhance dissolved O_2 delivery and avoid hypoxaemia.
- Aggressive monitoring of central venous pressure, intra-arterial pressure and pulse oximetry and restoration of intravascular volume.
- Avoid use of tourniquets. Moderate hyperventilation to induce respiratory alkalosis.
- Avoid hypothermia.
- Maintain normal pH.

Postoperative Period

- Controlled oxygenation upto 8 hrs.
- Early mobilisation of patient
- Chest physiotherapy.
- Adequate antibiotic cover to control chest infection.
- Careful watch for infarction crisis bone pain usually heralds bone infarcts. Heparin and magnesium sulfate should be given immediately.
- Maintenance of intravascular volume.

Role of Regional Anaesthesia

Regional anaesthetic techniques have been advocated in preference to general anaesthesia but the same precautions regarding ventilation, oxygenation, hypotension and stasis of blood flow must be appreciated.

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56 Anaesthetic Technique and Problems in Total Hip Replacement

Total hip replacement (THR) is a major surgery, the anaesthetic management of which varies according to complexity and complications arising during surgery and the medical status of the patient. Complex procedures, such as, those involving acetabular bone grafting, removal of prosthesis, revision surgery or surgery in patients with acetabular protrusions complicate the management of anaesthesia. If the patient is elderly or suffers from coexisting systemic disease and anatomic distortion of airway e.g. rheumatoid arthritis or ankylosing spondylitis, then the management of anaesthesia is even more complicated.

ANAESTHETIC MANAGEMENT

Monitoring

THR is a long procedure which takes 2 to 4 hours and there is severe blood loss added to the other systemic disturbances. The patients have limited ability to exercise and their cardiopulmonary function is difficult to assess. The elderly may have underlying systemic diseases. General anaesthesia with induced hypotension may be preferable in such cases.

- Fluid management is done carefully by CVP monitoring
- ECG is mandatory for the aged
- Foley's catheter to monitor urine output
- Arterial cannulation for continuous BP monitoring and blood gas analysis.

Hypoxaemia and/or pulmonary oedema occur due to pulmonary endothelial injury from fat or bone marrow emboli and from V/Q mismatching. Hence invasive hemodynamic monitoring is done perioperatively in the elderly undergoing THR, especially when it involves complex revision surgery.

BLOOD LOSS

- Hypotensive or regional technique reduces blood loss approximately by 30 to 50%.
- Blood loss during THR is significantly greater during revision surgery and when noncemented components are used.
- Preoperative autologous blood donation and cell saver techniques may also reduce transfusion requirements.

POSITIONING

- Most THR is done in lateral decubitus position. This creates a potential V/Q mismatch with resultant hypoxaemia which is 'common in patients underlying lung disease. It also occurs when SNP infusions are used.
- The lateral decubitus position create neurovascular problems involving the axillary artery and brachial plexus and the structures in the femoral triangle.
- In lateral decubitus position sometimes the following nerves will be compressed:
  - Lateral femoral cutaneous nerve of thigh: Due to pressure over anterior iliac crest and post-operatively numbness of lateral aspect of thigh and knee occurs.
  - Femoral nerve: Pressure over groin of dependent limb. Numbness over anterior thigh and medial aspect of lower leg.
  - Common peroneal nerve. Below head of fibula it is compressed. Foot drop can occur.

CHECKLIST

- Preoperative evaluation of nerve dysfunction.
- If a tourniquet is applied the duration and pressure used is noted.
- Postoperative positioning, splint application and bandage should be carefully done.
- Neuropraxia occurs in case of prolonged surgery.

INTRAOPERATIVE HYPOTENSION

Profound hypotension following insertion of cemented femoral prostheses often occurs which may lead to cardiac arrest.

To Minimise these Complications

- Use of a plug in the femoral shaft to limit the distal spread of a cement in the femur.
- Venting of entrapped air and
- Waiting for cement to become more viscous before its insertion.

The cause of hypotension may be due to (possibly)

- Caused by direct vasodilatation and/or cardiac depression from methyl methacrylate, or
- May be due to the forced entry of air, fat or bone marrow into the venous system with resultant pulmonary emboli.

Hypoxia is present immediately following insertion of a cemented femoral prosthesis and for upto 5 days into the postoperative period. Rule out any specific cause such as atelectasis of the dependent lung, hypoventilation or fluid overload. Even with no specific cause hypoxaemia persists for some days after surgery and is thought to be secondary to embolic effects of femoral shaft cement or fat embolism. Pulmonary ernboli with cement or bone marrow increase pulmonary artery pressure.

POSTOPERATIVE MANAGEMENT

- Nasal oxygen (if necessary for several days)
- Judicious use of narcotics to provide analgesia but to avoid hypoventilaltion or airway obstruction and appropriate fluid management.
- Hypoxia and fluid overload further increase pulmonary pressure and hence predispose to pulmonary oedema or right heart failure.
- Continuous epidural for pain relief.

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57 Positioning in Anaesthesia

SUPINE POSITION

This is the commonest position in anaesthesia. The head elevated, and extended and the neck is flexed slightly. This is called the 'sniffing' position. Arms may be placed alongside the trunk or extended and placed on arm boards. The hips, knees and elbows are flexed slightly. Padding may be used to maintain the normal vertebral curve.

Complications

Injuries from pressure or stretching of nerves vessels, joints or skin can occur. Arms should be protected, if abducted, should be at an angle of 90° or less. The elbows should be rotated externally to prevent pressure on the ulnar nerve, legs should be supported and there should not be undue pressure on the heels.

Head should be supported and a pad distributes pressure on the occiput. Bony prominence need special care. The most frequent nerve injuries involve the ulnar, brachial plexus, sciatic, femoral, common peroneal, saphenous, anterior tibial, obturator and lateral femoral cutaneous nerves.

PRONE POSITION

"Classic" prone position is when the ventral surface of the body lies on some surface and supports the body weight.

Indications

- Lumbar laminectomy and lumbar spinal fusion.
- Posterior cervical and occipital surgery.
- Rectal and perineal surgery and sigmoidoscopy
- The posterior approach to adrenal explorations and renal biopsy.
- Thoracotomy by posterior approach.

POSITIONING

Care must be taken to ensure circulatory stability (light anaesthetic level, leg wraps, etc) and to avoid overextension of the patients arms or neck during positioning. The head should be maintained in a neutral position during and after prone positioning.

Before turning the anaesthesiologist should be assured that the intravenous catheter and endotracheal tube are securely attached. The blood pressure cuffs should be place high on the arm to prevent neurovascular compression in the antecubital fossa if the arm is flexed. ECG contact pads should be positioned on the patients back or side so that the patient will not be on them after positioning.

Effect on the CVS

- IVC compression → ↓ led venous drainage, hypotension distension of vertebral veins.
- Carotid sinus pressure hypotension, arrhythmias.
- Obstruction of venous return from the head engorged eye vessels, eyelid oedema, postoperative headache, subglottic oedema.
- ↓ ed blood flow through the vertebral arteries due to lateral twisting or severe extension of neck.
- Engorged veins due to venous drainage obstruction increased intraoperative blood loss.

Effect on Respiratory System

Restriction of diaphragmatic movement and decreased tidal volume, if abdominal wall is not free to move. It will also cause a moving field over the entire back. So the upper thorax and pelvis should be raised to get a free movement of abdominal wall.

Neurologic Injuries

- Pressure against bones
  - Brachial plexus
  - Lateral cutaneous nerve of thigh
  - Facial
- Eyes—contusion, penetration, ischaemia due to compression
- Spine injury
- Injury to the nerves and tendons of the dorsum of the foot.
- Ulnar nerve palsy

  The pressure areas should be protected with padds as and when needed.

Other Injuries

Injuries can occur on male genitalia, female breasts, ears, neck, skin overlying bony prominences etc., shoulder dislocation, persistent pain due to pressure on the sternum or infraclavicular fossae, air embolism, injuries due to nasotracheal tube pressing the nasal cartilage.

HEAD ELEVATED POSITIONS

The Sitting Positions

Physiologic Effects

CVS: ↑HR
↑SV
↑CO
↑PVR

Respiratory system: Finer diaphragmatic movements are affected. There is lesser rise in intrathoracic pressure and the shunt fraction is lessor.

Neurological aspects: CSF pressure at the vertex is negative.

Complications of the sitting position: Hypotension, endotracheal tube migration, air embolism, respiratory acid base imbalance, arrhythmias, other complication are tension pneumoencephalus, stretch injury to sciatic nerve, trauma to cervical spine swollen tongue, face and neck.

The Trendelenburg Position

No marked change for BP, jugular venous pressure is increased, respiratory embarrassment, masks hypovolaemic symptoms, accumulation of gastric contents in the hypopharynx.

Complications: (1) Hypotension, (2) Increased CVP, retinal detachment, cerebral oedema, venous thrombosis, intubation of a mainstem bronchus, atelectasis. Neuropathy—Brachial plexus injury, sore knees or hips, damage of fingers.

The Lateral Decubitus Position

Physiology (Respiratory system): The hemidiaphragm on the dependent side lies higher in the chest and is more stretched. The hemidiaphragism has better mechanical efficiency. In mechanically ventilated anaesthetised man tidal volume is preferentially distributed to the nondependant lung.

Pulmonary blood flow: Dependent lung is preferentially perfused in spontaneous respiration. During mechanical ventilation, this preferential perfusion persists.

CVS: Venous return is unaffected.

Complications
Atelectasis: Increased incidence in the dependant lung.

Nerve injury: Brachial plexus.

LITHOTOMY POSITION

Complications

- For any patient: (a) venous stasis, (b) nerve damage like obturator nerve, saphenous nerve, femoral nerve, common peroneal nerve.
- Obstetric patients: Supine hypotension syndrome, damage to the HIPS and knees.

Anaesthesiologic Considerations

Respiration—decrease VC

CVS—Sudden lowering of legs can produce circulatory collapse.

Nervous system

- Brachial plexus injury; common peroneal injury
- Cutaneous and musculoskeletal system
- Backache—Torsion injury to the ligamentous structures.

Unusual positions: Urology—Kidney position.

Respiratory system: Tidal volume decreased.

CVS: Cardiovascular collapse while turning from supine to the kidney position.

G/A depresses carotid and aortic sinus mechanism. Mediastinal compression interfering with venous return and decreasing cardiac output.

Nervous, cutaneous and musculoskeletal systems.

- Injury to brachial plexus
- Cervical spine injury
- Pressure on the arm underneath.

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58 Malignant Hyperthermia

DEFINITION

A pharmacogenetic myopathy, manifested in susceptible individuals as a life threatening syndrome of muscle hypercatabolism and rigor, accompanied by rapid and inexorably progressive rise in body temperature, in response to some general anaesthetics.

This syndrome is characterised as "malignant" due to its rapid progression to irreversibility despite withdrawal of the original stimulus. First described in 1960 by Denborough and Loveli.

INCIDENCE

Rare, but worldwide in occurrence. Incidence is 1/5,000 to 1/20,000.

PATHOGENESIS

Malignant hyperthermia (MH) is a myopathy that features an acute loss of intracellular control of calcium. This results in a release of free unbound ionised calcium from storage sites; there is a concomitant increase in both aerobic and anaerobic metabolism to provide more ATP to drive the calcium pumps that maintain calcium homeostasis, subsequently, there is muscle rigidity and hyperpyrexia.

Abnormalities

They are functional rather than structural.

SKELETAL MUSCLE

The histology shows central core, internal nuclei, target fibre, supercontracted fibres, variation in fibre diameter are the primary involvement.

MYOCARDIUM

It is controversial whether the myocardium is primarily involved or whether it is secondarily affected-by hyperthermia, acidosis, hyperkalaemia, and subsequent increased membrane permeability.

CENTRAL NERVOUS SYSTEM

Secondary involvement. There is coma, areflexia, unresponsiveness and fixed dilated pupils suggesting a/c cerebral oedema.

SYMPATHETIC NERVOUS SYSTEM

Probably involved only secondarily.

BLOOD

Variation in ionic permeability, fragility, cholesterol content, deficiency of plasma pseudocholinesterase, DTC may occur following MH probably due to a release of tissue thromboplastin.

THEORIES OF MALIGNANT HYPERTHERMIA

- Abnormalities of sarcoplasmic reticulum proposed in 1977 by Endo.
- Raynodine receptor protein theory—this protein spans the gap between the terminal cisternae of sarcoplasmic reticulum and T- tubules and has been suggested as the site of defect in MH.

CLINICAL PRESENTATION

There are 2 types of clinical presentation of MH-rigid and nonrigid. The manifestation can range from classic cases to those with unusual presentation and mild symptomatology.

Muscle hypertonus or rigor appear in over 80% cases. This is preceded by an exaggerated myotonic response to suxamethonium. This myotonic response is evidenced by masseter muscle spasm (MMS), producing difficulty in tracheal intubation. MMS is now recognised as a reliable evidence of presence of acute MH.

During clinical anaesthesia, there is a surge of sympathetic activity—sudden unexplained tachycardia, tachyarrhythmias, cyanosis despite adequate ventilation hyperpyrexia and an excessively hot sodalime canister. Other features are marked skeletal muscle rigidity, etc.

Incidence of Various Clinical Signs

Tachycardia	96%	Rigidity	80%
Tachypnoea	85%	Profuse sweating	50%
Arrhythmia	70%	Altered BP	85%
Hyperpyrexia (41°C)	30%	Over heated CO_2 absorber	60%
Cyanosis	70%	Skin mottling	20%

CRUCIAL SIGNS

- Muscle rigidity after suxamethonium.
- Precipitous ↑ in end tidal CO_2.
- Unexplained hypertension and tachycardia even with deep plane of anaesthesia.
- Unexplained myoglobinurea in perioperative period.
- Unexplained increase in body temperature in perioperative period.

LAB FINDINGS OF ACUTE MH

ABG	↓ pH	Serum	↑ Lactate
	↓ PO_2		↑ Pyruvate
	↓ PCO_2		↑ CPK/LDH
Electrolytes	↑ K^+		↑ AST/ALT
	↑ Ca^{++}		↑ Aldolase
	↑ Mg^{++}		↑ Myoglobin
	↓ Na^+		↑ Tissue thromboplastin

DIFFERENTIAL DIAGNOSIS

- Severe sepsis
- Thyrotoxicosis
- Pheochromocytoma
- Drug reaction
- Anaesthesia in small children in abnormal environment

MH TRIGGERING FACTORS

- Possession of MH gene
- Triggering agents
- Factors which sensitise the patient to the triggering agents.

Anaesthetic Agents

Safe

Tranquillizers in general (except chlorpromazine), opiates, barbiturates, etomidate, propofol, non-depolarising relaxants, amide and ester anaesthetics, epinephrine and norepinephrine, digitalis, calcium, dantrolene etc.

Unsafe

- Volatile anaesthetics—Halothane, enflurane, isoflurane, sevoflurane.
- Relaxants—Succinyl choline

Genetics

Mostly autosomal dominant.

Associated Disorders

- Duchenne dystrophy
- Periodic paralysis
- Myotonia congenita

- Other myopathies
- King Denborough

Coincidental Association

- Sudden infant death syndrome (SIDS)
- Neuroleptic malignant syndrome
- Other diseases like lymphoma
- Osteogenesis imperfecta
- Glycogen storage disorders.

TREATMENT

- Specific
- Supportive

Specific

Dantrolene sodium 2-4 mg/kg is necessary for effective therapeutic plasma levels. It can be started with initial doses of 1 mg/kg and 0.5 mg to 1 mg/kg increments at 15 to 30 min intervals, until symptoms subside in a total dose of 10 mg/kg body weight is given. It is seldom that doses exceeding 4 mg/kg is necessary. Some advocate a repeat (Prophylactic) dose of 2.4 mg/kg after 10 to 12 hrs to prevent MH recurring in the postanaesthetic period. Dantrolene, for IV use is prepared as a highly alkaline solution in mannitol (3 mg per 20 mg vial).

Other drugs—Ca^{2+} channel blockers, procaine and procainamide

Supportive Therapy

Discontinuation of all anaesthetic agents and hyperventilation with 100% oxygen.

- Correction of metabolic acidosis to a pH of 7.2 to 7.3
- If possible, get another machine with a new circuit.
- Aggressive cooling.
- Treat hyperkalaemia.
- Treat tachyarrhythmias.
- Monitor urine output and induce diuresis if oliguria present.
- Provide calories with 20% or 50% dextrose.
- Monitor the patient—HR, BP, EKG, temperature, pulse oximetry, end tidal CO_2 , ABG.
- Coagulation profile—Get FFP for infusion if doubt of DIC.

The patient must be closely monitored and sedated for 24 hours. Dantrolene should be administered for at least 3 days after successful treatment of MH.

PREANAESTHETIC DIAGNOSIS

This is essential for prevention of the syndrome, in susceptible individuals. Screening should also include near relatives.

Tests for diagnosis: These include blood tests, electrophysiological tests, biochemical tests, phosphorous—31 NMR spectroscopy, muscle histology, contracture test etc.

Contracture test: The most reliable test which is the '*in vitro* contracture test' (IVCT) with halothane and caffeine. The tests employed are:

- Caffeine contracture test
- Halothane contracture test
- Caffeine-Halothane contracture test
- Raynodine contracture test.

ANAESTHESIA IN MH PATIENTS

Four simple guidelines:

- Avoidance of triggering agents.
- Suppression of patients trigger sensitivity.
- Meticulous monitoring of patient.
- Immediate availability of dantrolene.

Avoidance of Triggering Agents

- Local/regional technique used
- GA-avoid all potent volatile inhalational agents and suxamethonium.

Suppression of patient's trigger sensitivity: Proper preanaesthetic control of apprehension and anxiety.

Dantrolene prophylaxis: (Controversial); should be administered IV in a dose of 2.4 mg/kg with induction of anaesthesia.

Precautions

- The patient should carry a warning card for identification of this syndrome.
- The family, and other relatives should be investigated for this syndrome.
- The individual with a history of MH in his family or other relatives should be treated as susceptible to MH until proved otherwise.

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59 Awareness During Anaesthesia

Unconsciousness is an integral part of anaesthesia and hence the patient becoming aware while under anaesthesia is highly unjustifiable. But this problem is as old as the speciality itself and more reports are coming out now a days. This may be either due to increase in reporting or due to the practice of 'Balanced Anaesthesia', whereby a conscious patient can be kept still by muscle paralysis.

George Crile was one of the earliest persons to report about awareness (1908) under N_2O anaesthesia. It was Winterbottom who reported the first case after- the introduction of relaxants. The first systematic investigation into the problem was by Hutchinson (1961).

Awareness is defined as the spontaneous recall of events occurring under GA. The term spontaneous is important, since some auditory inputs occur even in sufficiently deep anaesthesia and some meaningful images are retained. These can be brought out at a deliberate session of hypnosis later. This 'amnestic awareness' had been investigated by Levinson, Blacher and Benette separately. The general consensus is that critical news are registered more, there is no psychological trauma, but still it is dangerously close to awareness. A stimulus may evoke a response or be registered in explicit memory or only in implicit memory with deepening anaesthesia. Thus there are 3 types of true awareness:

- Conscious awareness without amnesia response to verbal command present but no memory afterwards.
- Conscious awareness with amnesia.
- Subconscious awareness with amnesia: No response to verbal command, but retention of verbal information can still occur.

Dreams are another entity which may be confused with awareness. Patients may report them from memory which may be even postoperative. But some hold the view that; awareness provokes them. Hallucination also may be misinterpreted.

Incidence reported from various studies is around 2% in general and 2-17% in obstetrics. Certain difficulties are met with in accurate estimation. A large group is needed since the incidence is very low. Deliberately subjecting the patient to awareness for research is unethical. The assessment is subjective. Many patients do not report spontaneously and clinical assessment depends on the experience of anaesthetist. Prospective studies may be manipulated. A systematic study by Hill et al demonstrated a fall in incidence over last 30 years from 1.5% to 0.2%. More than mere unpleasantness, awareness leads to considerable psychiatric morbidity. Wakefulness in a paralysed patients can lead to the syndrome of traumatic neurosis. Repetitive nightmares, irritability, anxiety, preoccupations with death and suicidal tendencies make their life miserable. There are also intra-

operative problems, since there are haemodynamic, sudomotor, and hormonal responses to noxious stimuli which are harmful. Thus the practice of keeping the patient 'light' to avoid certain damages may be more deleterious.

Predisposing factors: Faulty techniques are the most important cause of awareness though the situation may be unpredictable and in many situations unavoidable. Drug requirement vary considerably according to age, obesity, addictions, previous exposure, systemic disease, etc. Psychiatric illness and certain personality makeups predisposes to awareness. Still some technique are particularly associated with awareness. The N_2O-O_2 anaesthesia following TPS + SC (the classical Liverpool technique) is reported to have higher incidence especially with lower proportion of N_2O and absence of volatile agents. Inadequate premedication, switching on N_2O late and putting off early, keeping O_2 flush on, early reversal all lead to awareness. Some ventilators contribute by leaks and entraining air when gas flows are inadequate. A sudden powerful surgical stimulus may provoke awareness at sufficient depth.

Detection of awareness require utmost vigilance as monitoring equipments are not fool proof. Symptoms of ANS stimulations like tachycardia, HT, pupillary dilations, increased secretions, tear and sweating etc. may point to awareness. Movements of limbs, eyelids and swallowing are also pointers. EEC and evoked potential monitoring may help detection of awareness, but are generally unavailable for clinical use. Attempts to process EEC simultaneously has lead to 'cerebral function monitor' which is the best one available at present. Development of power spectrum analysis offers the best hope for future. In isolated forearm technique (Turnstall) tourniquet applied to one U'L. prevents its paralysis, enabling the patient to indicate awareness, if explained to previously. EMG of frontalis muscle is possible even at clinical neuromuscular blockade.

Prevention of awareness involves:

- Adequate preparation of patient
- Check apparatus before anaesthesia
- Faultless technique and usage of certain adjuvants
- Preoperative discussion with the patient explaining the possibility of dreams and warning about awareness in special situations (wake up test, obstetrics) goes a long way in decreasing morbidity
- Premedication with lorazepam, hyoscine and adequate narcotics, etc. are helpful
- 'Proper' dose of induction agents rather than 'sleep' doses. Ketamine is reported to be a better induction agent in this respect. Using N_2O in adequate proportion and duration, volatile agents judiciously, IV agents in increments, etc. all contribute
- Do not rely entirely on G-O-R technique. Relaxants are better given as infusion
- If intubation is found difficult, add incremental anaesthetic
- At end of surgery maintain N_2O till relaxants are reversed. In a reported case of awareness sympathetic explanation by the anaesthetist and timely psychiatric intervention are important.

Special Situations

- *Obstetrics:* The practice of keeping the patient under light anaesthesia till delivery to avoid neonatal depression leads to decreased uteroplacental blood flow due to the sympathetic stimulation caused by awareness. From induction till delivery a low dose volatile agent (e.g. halothane 0.5%) can help prevent awareness; this does not cause uterine relaxation and haemorrhage.
- *B' Scopy:* Apnoea oxygenation and jet ventilation are associated with high incidence
- *Paediatrics:* It is a faulty concept that small babies do not require much anaesthetics
- *Total IV anaesthesia:* Tailored doses should be given and maintained carefully - monitoring anaesthetic depth is not often possible with total intravenous anaesthesia
- During one lung anaesthesia because of $\uparrow FiO_2$.

In conclusion: In this era of Medical Litigations, awareness during anaesthesia should be avoided at all cost. But it has not only legal sequelae but medical and ethical implications also. The trend towards muscle relaxation and light GA has increased the problems. At the same time a patient should not be denied the benefits of light GA, just for the fear of awareness. A meticulous technique and careful monitoring are all that is needed.

Awareness with analgesia is regrettable, and with pain is unforgivable.

PRST score system for systemic evaluation of the autonomic signs of light anaesthesia.

Index	*Condition*			*Score*
1. Systolic pressure (mmHg)	< Control	+	15	0
	< Control	+	30	1
	> Control	+	30	2
2. Heart rate (Beats/min)	< Control	+	15	0
	< Control	+	15	1
	> Control	+	30	2
3. Sweating	Nil			0
	Skin moist to touch			1
	Visible beads of sweat			2
4. Tears	No excess of tears in open eye			0
	Excess of tears in open eye			1
	Tears overflow in open eye			2

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60 Monitoring of Intracranial Pressure

Intracranial pressure (ICP) is the pressure within the cranium and reflects the CSF pressure. The source of this pressure is the secretory pressure of the choroid plexus. It has to be sufficient to overcome the flow resistance of the microtubules of the arachnoid villi. It is closely related to the venous pressure and oscillates with the arterial pulse and with respiration. Falls with inspiration and rises with expiration.

Normal intraventricular ICP = 5-15 mmHg

ICP greater than 20 mmHg is an indication for intracranial hypotensive therapy.

Factors which increase ICP

- *Physiological*
  - Coughing
  - Sneezing
  - Straining
- *Pathological*
  - Pressure from outside - bony tumour or craniostenosis
  - Presence of a space - occupying lesion neoplasm, abscess, haematoma
  - Hydrocephalus
  - Venous obstruction
  - Arterial dilatation secondary to hypercarbia
  - Cerebral oedema
    i. Inflammation
    ii. Neoplasm
    iii. Trauma
    iv. Hypoxia
    v. Venous obstruction
  - Head-down position.

Factors which decrease ICP

- Hypovolaemia
- Following removal of an intracranial space occupying lesion.
- Head up position -Raising the head 20 cm above horizontal reduces ICP by 20 cm H_2O.

Importance

- Cerebral perfusion pressure CPP = Mean arterial pressure (MAP) – intracranial pressure (ICP). Any rise in ICP causes a reduction in the CPP. So careful monitoring and prompt treatment of

intracranial hypertension is the most important factor in the reduction of mortality following head trauma.

- Control of ICP prevents pressure-gradients between various compartments of intracranial cavity and prevents shifts of brain structures (Brain herniation) (Fig. 60.1).

Monitoring of ICP began way back in 1951.

Lundberg's classic work demonstrated that there are 3 waves in ICP tracing:

- **A waves** (Fig. 60.2) or plateau waves are recurring increases in intracranial pressure to values of 50 to 100 mmHg lasting 5 to 20 min which usually arise with severe decompensation of ICP control mechanism. Seen only when base line ICP is more than 20 mmHg. At 60 mmHg headache, restlessness incontinence, non purposive movements, etc. appear. At 80 mmHg loss of consciousness decerebration, respiratory arrest, etc. occur.
- **B waves** (Fig. 60.3) occur at a rate of 1 or 2 per min, are of small amplitude and may be precursors of plateau waves. Warns against impending intracranial hypertension and impairment of intracranial compliance. They were initially thought to be related to Cheyne-Stokes respiration, but can occur in patients on controlled ventilation. They are particularly prominent during REM sleep, and may occur in normal people.
- **C waves** (Fig. 60.4) occur more frequently (6/min) and are of less amplitude and of little clinical significance except that they only occur in the presence of brain pathology and imply an unstable control of cerebral blood flow.

  There are waves which do not fall into any of the categories (Fig. 60.4) and it may be that the recognized errors are not distinct entities but merely points in a spectrum.

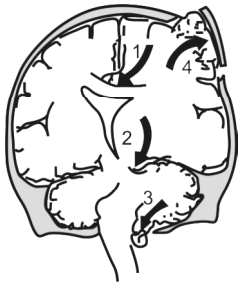

Fig. 60.1: Brain herniations: 1. Cingulate, 2. Temporal (uncal), 3. Cerebellar, 4. Transcalvarial (post-traumatic or postoperative)

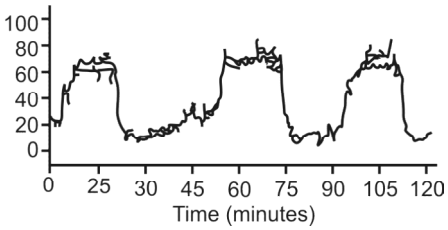

Fig. 60.2: ICP tracing—A waves

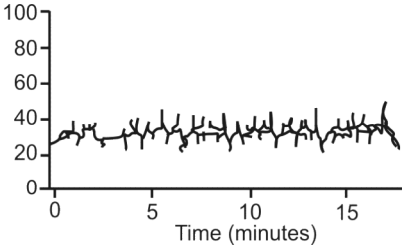

Fig. 60.3: ICP tracing—B waves

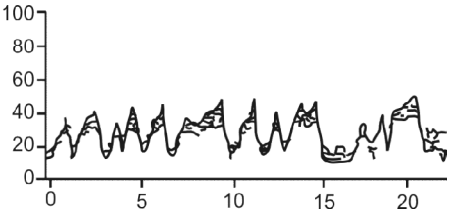

Fig. 60.4: ICP tracing—C waves

THE INTRACRANIAL PRESSURE/VOLUME CURVE

The intracranial pressure volume curve shows that as intracranial volume increases there is very little change in pressure during the phase of compensation (Fig. 60.5). With

further increase in volume pressure begin to rise forming a 'knee'. Still further increase in volume causes marked rise in pressure during the phase of decompensation.

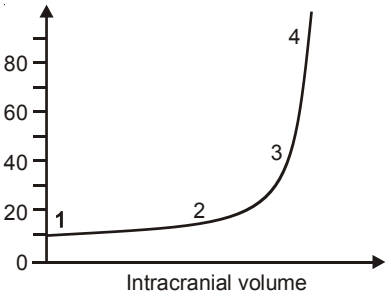

Fig. 60.5: ICP—volume (level y-axis ICP torr)

INDICATIONS FOR ICP MONITORING

- Head injury -When Glasgow Coma Scale is less than 8.
- Hepatic encephalopathy
- Reye's syndrome
- Anoxic encephalopathy
- Large intracranial space-occupying lesion
- Intraoperatively in cases where the ICP is deemed very high and likely to increase further.

USES OF ICP MONITORING

- *Diagnostic:*
  - Early detection of cerebral oedema or other causes of raised ICP.
  - Confirms that neurological deterioration is due to rising ICP and prevents inappropriate therapy.
  - Suggests the need for repeat CT scan in a patient with head injury.
- *Prognostic:* In patients with head injury.

TECHNIQUES OF ICP MONITORING

Intraventricular Catheter

The catheter is placed inside the ventricles through a twist drill hole and is exteriorised through the skin and connected to a fluid coupled system kept on patient's head or at bedside.

Advantages

- High fidelity recording
- Compliance studies possible
  Ventricular pressure response more than 4 mmHg per ml is always associated with a mass lesion.
- Permits emergency tapping of CSF for acute rise in ICP
- Cheap and simple
- Recalibration can be possible at any time.

Disadvantages

- High infection rate
- Intracerebral haemorrhage
- Difficult to insert when ventricles are small or collapsed.
- May not be suitable in head injury
- Zeroed with change in position of the head.

Fluid Filled Surface Monitoring Devices

Balloon Devices

Fluid-filled balloons are placed subdurally or epidurally and connected to external fluid filled system.

Disdvantages

- Small balloons are too sensitive to changes in internal volume
- Large balloons are difficult to introduce
- Rupture of balloon exposes brain to the contents of the balloon.

Hollow Screw Devices

Here a hollow screw is placed through a twist drill hole and this utilises the arachnoid as the sensing membrane. It is then connected to a fluid filled system.

Advantages

- High fidelity traces
- Cheap, simple
- Recalibration possible
- Avoids cerebral trauma.

Disadvantages

- High infection rate
- Can underestimate ICP monitoring
- Produces artefact on CT scan
- Fluid drainage not possible

Cup Catheter

This is a hollow ribbon-shaped device with a cup on the flat surface of the ribbon at its end. The cup utilises the arachnoid as its sensing membrane.

Advantages

- It can be brought out through a scalp incision remote from the area of opening in the skull. So chance of infection is reduced.
- Can be inserted through a craniotomy.

Nonfluid Coupled Devices for Surface Monitoring of ICP

Mechanical (ICP Pressure Switch)

A small balloon containing electrical contacts fastened to its inner walls is introduced into the subdural or intradural space. When the balloon is fastened because of raised ICP the electrical contacts remain in contact, thereby completing a servo circuit. Servo circuit powers an infusion which pressurises the inside of the balloon up to a point which separates the electrodes and breaks the circuit. The applied pressure at this stage represents the ICP.

Optical (Ladd ICP Monitor)

It utilises a fibreoptic system. Distortion of a tiny mirror inside a balloon system by the raised ICP is transmitted through a fine fibreoptic cable, this signal switches on an infusion pump which applies adequate intraluminal pressure to the balloon to restore the position of the mirror.

Electronic Devices

Convert ICP into an electric signal which is transmitted through cables to the monitor. A device which detects changes in capacitance is commonly utilised.

Implantable Devices

Converted into electrical signal which is detected transcutaneously by external sensing antennae.

Disadvantages

- Infection
- Loss of accuracy

Epidural Sensors

Advantages

- Low risk of infection
- Can be used in children
- No CT artefacts

Disadvantages

- Difficult to calibrate
- Expensive
- Compliance studies not possible
- Fibreoptic epidural devices have a high latency and transmit waveforms poorly.

Properties of an ideal transducer

- Recording must be from the intracranial space
- Minimum risk of infection
- Insertion of the device should produce no brain damage
- Procedure for insertion and removal must be simple
- If transducer is to be implantable it must be small.
- It must be drift-free with time and temperature and capable of being calibrated *in situ*.
- It should allow freedom of patient movement.
- It should have adequate sensitivity.
- It should be impervious to the environment.
- It should have extended use.
- It should not be too expensive.

Limitations

- None of the methods are universally useful.
- Errors can occur as a result of technical fault.
- Monitoring of ICP alone does not ensure a good outcome as other factors like regional blow flow distribution and changes in cellular level also influence the outcome.

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61 Brain Preservation: Recent Trends

INTRODUCTION

The term cerebral protection has been used to include measures instituted before or after an insult to prevent or ameliorate the harmful effects on the brain for a known cerebral insult. These measures mainly include pharmacological agents, but simple basic care such as well managed neurosurgical anaesthetic will contribute much to cerebral protection.

CEREBRAL PROTECTION MODALITIES

- *Measures to decrease cerebral metabolism*
  Which includes hypothermia (physiological method) and pharmacological agents like
  - Barbiturates
  - Propranolol
  - Etomidate
  - Isoflurane
- *Measures to increase microcirculatory flow*
  - Induced hypertension
  - Haemodilution
  - Anticoagulants
- *Reduction of secondary injury*
  Agents used for this purpose are:
  - Calcium channel blockers
  - Excitatory amino acid antagonists
  - Free radical scavengers
  - Prostaglandin inhibitors and
  - Steroids

Out of these techniques, this use of calcium channel blockers, free radical scavengers and prostaglandin inhibitors are new in the field of brain preservation. So they will be discussed in detail.

CALCIUM CHANNEL ANTAGONISTS

During ischaemic period, calcium enters through both voltage sensitive channels and receptor controlled channels. It destroys cell membrane by activating proteases and causes receptor dysfunction, and transport mechanisms fail. The calcium influx also activates phospholipases A1, A2 and C leading to hydrolysis of phospholipids which in turn release free fatty acids that destroy the lipid part of the membranes.

Out of the four classes of calcium channel antagonists, dihydropyridines such as nimodipine are the most potent and have preferential effects in the cerebral blood vessels.

There are several potential mechanisms by which calcium channel antagonists may exert potential effects:

- Reduction of calcium induced cell damage, by delaying or preventing the passage of calcium into the cells in postischaemic period.
- Prevention of postischaemic hypoperfusion by inhibiting calcium induced cerebral vasoconstriction.
- Attenuation of free fatty acid liberation.

Other calcium channel blockers that are extensively investigated are lidofasine and nicardipine. All the three drugs selectively act on voltage sensitive calcium channels.

EXCITATORY AMINO ACID ANTAGONISTS

The N Methyl D-Aspartate receptor is predominant in vulnerable areas of cerebral ischaemia. The glutamate can be antagonised competitively or noncompetitively.

Competitive antagonists are SGS 19755, CPP, AP-7 CGP 37849, NPC 12626. They have poor blood-brain barrier penetration.

Noncompetitive antagonists bind within the ion channel, thereby impeding ion flux. They include Dizocipine maleate, (MK 801), Ketamine and dextorphan. Ketamine block the NMDA receptor associated low channel in the closed state. They are lipid soluble and easily cross the BBB. They have predictable half-life also. The side effects of NMDA receptor blockers are: (1) non NMDA mediated excitatory synaptic- transmission, (2) Impairment of learning, (3) Morphological neurological changes, (4) Psychomimetic and adverse behavioural changes.

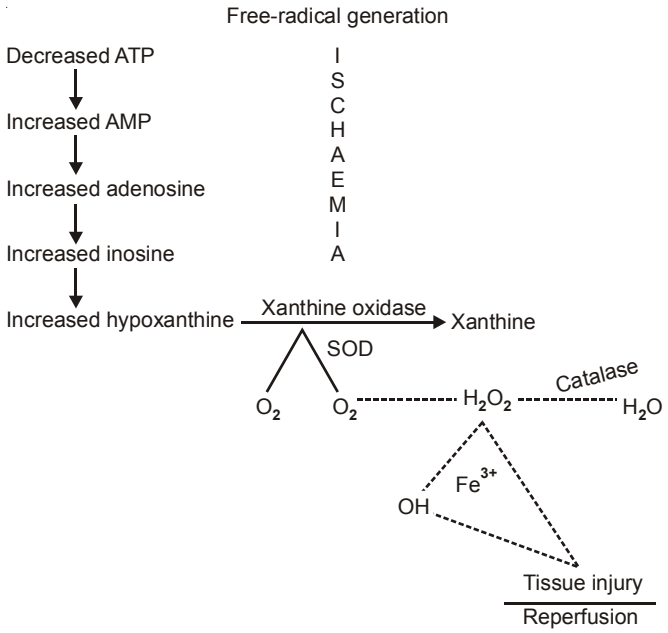
AMPA receptor (Alpha amino 3 hydroxy 5 methyl 4 isoxazole) mediates: (a) Rapid excitatory transmission in the brain, (b) depolarisation of cell membrane - increased Na-K influx, (c) Reversal of $Na^{+}Ca^{++}$ exchange mechanism. AMPA antagonist (NBQX) crosses BBB and is able to decrease ischaemic neuronal injury.

Kainate receptor: Found in stratum incidence of the hippocampus is also acted upon by glutamate and the noncompetitive antagonists acts in this receptor thus reducing postischaemic neuronal injury.

Free radicals like superoxide (O_2) hydroxyl (OH), Fe, etc. are released during ischaemia which can destroy cell membrane and cause irreversible tissue damage. They can damage protein, nucleic acids, lipids, etc. They may promote lipid peroxidation causing cytotoxic and vasogenic oedema, alter vascular endothelial and BBB permeability and produce inflammation.

Free radical scavengers stop free radical generation. They include: (a) Superoxide dismutase directed against superoxide, (b) Catalase and glutathione peroxidase directed against H_2O_2, and (c) Vitamin E and are directed against those radicals in lipid and aqueous phases.

Flow chart 61.1: Pathway free-radical scavenger generations



Therapy is directed to:

- Anoxic perfusion to deliver free radical scavengers before reoxygenation. Superoxide dismutase, catalase, methylthionine.
- Administration of specific free radical scavengers to break the chain reaction.
- Binding of free ion by iron chelators such as desferoxamine to prevent formation of hydroxyl radicals.
- Inhibition of xanthine oxidase by allopurinol.
- Antioxidants vitamin E
- Stabilize membrane—gangliosides.

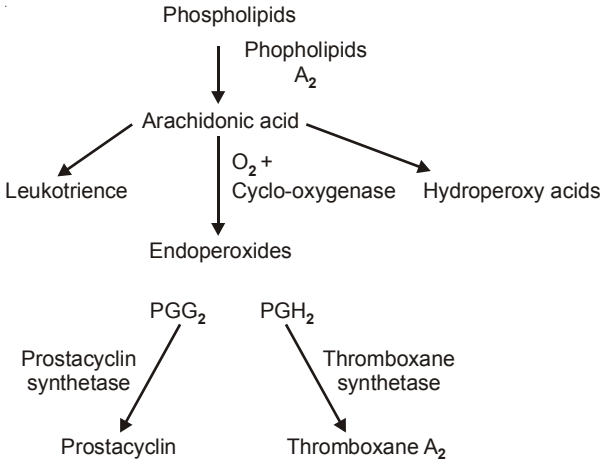
PROSTAGLANDIN INHIBITORS

Indomethacin given before the ischaemic event may protect the brain by preventing thromboxane A 2 formation (formed in arachidonic acid cascade). The endoperoxide may decrease prostacyclin (a potent vasodilator) leading to hypoperfusion. Endoperoxide and thromboxane A2 are potent platelet aggregators contributing to hypoperfusion.

CORTICOSTEROIDS

Corticosteroids in normal doses are of no benefit in attenuating either cytotoxic or vasogenic ischaemic peroxidation of cell membranes via intercalation into cell membranes and protect susceptible fatty

Flow chart 61.2: Pathway for prostaglandin synthesis



acids from peroxidative attack by oxygen free radicals. The 21 aminosteroids readily cross the BBB and are potent inhibitors of iron catalysed lipid peroxidation. Examples are U 74006F and U 74500.

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62 Sensory Evoked Potential

DEFINITION

Sensory evoked potentials are defined as the electrophysiological manifestation of the central nervous system to sensory stimuli.

Sensory evoked potentials are gaining wide popularity because of the ability to monitor the functional integrity of the nervous system intraoperatively, of the anaesthetised patients undergoing surgical procedures that would place the sensory pathways at risk of injury.

CLASSIFICATION OF SENSORY EVOKED POTENTIALS

Sensory evoked potentials can be classified in many ways:

- Depending upon distance between the neural generator and recording electrode, they are classified into far field potentials and near field potentials.
- Depending upon, sensory modalities which are monitored.

Depending upon Distance between Neural Generator and Recording Electrode

Near field potentials: Evoked potentials recorded by electrodes placed close to the neurophysiological generator is called Near field potential.

Example: Cortical potentials recorded by electrodes placed close to the scalp.

Farfield potentials: These potentials are recorded by electrodes placed far from the neurophysiological generator and are conducted to the recording electrode through volume conduction of brain and CSF.

Example: Subcortical potentials recorded from the spinal cord by electrodes placed on the scalp.

Sensory Evoked Potentials

Sensory evoked potentials can be classified depending upon the sensory modality stimulated into:

- Somatosensory evoked potential (SSEP)
- Visual evoked potential (VEP)
- Auditory evoked potentials (AEP)

Poststimulus Latencies

The time interval between the stimulation of sensory pathway and recording of evoked potential is known *as* Post Stimulus Latency.

Poststimulus latency varies from 10-120 msec depending upon the pathway stimulated-short latency SEP are recorded from the subcortical region and long latency SEP are recorded from cortical area.

The conduction, velocity and central conduction time (CCT) are measurements derived from post stimulus latencies that help to assess and classify neurological function. The time between evoked potentials arising from cervical spinal cord and later from the evoked potentials arising from the primary sensory cortex is called as central conduction time which can be diagnostically important in that they can indicate pathophysiological alterations in brain function.

RECORDING OF SENSORY EVOKED POTENTIALS

- The sensory evoked potentials occurs at a constant time after the sensory stimulus whereas the EEC occurs at random intervals after the stimulus.
- SEP differs from EEG in that they are of low amplitude (0.1 to 20 microvolts) and hence to distinguish from background brain wave activity computer signal averaging or summation is required.

Any system to adequately record SEP should contain:

- Sensory stimulation.
- Amplification, acquisition and filtering of electrophysiologic signals.
- Signal processing.
- Display, measurement and storage of SEP waveforms.

For all SEP monitoring recording electrodes are placed on the scalp using the international 10-20 system as for recording the standard EEG (Fig. 62.1).

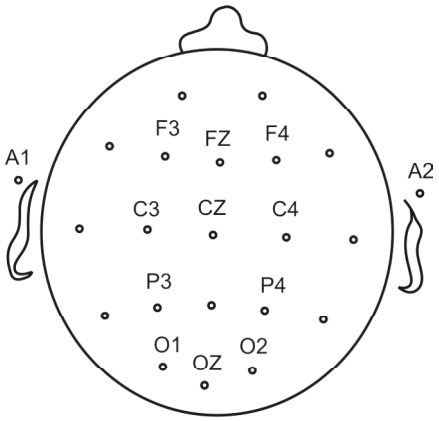

Fig. 62.1: International 10-20 systems for placement of scalp electrodes.

Intraoperative changes in evoked potentials such as:

- Decreased amplitude
- Increased latencies or
- Complete loss of waveform are considered to be indications of surgical trespass or ischaemia.

When these changes are detected or considered to be significant the anaesthesiologist and/or surgeon can make changes to relieve the insult to the monitered pathway.

Interventions by anaesthesiologist are directed at improving the perfusion to nervous system and include increasing the arterial blood pressure especially if controlled hypotensive technique is used. Alteration in evoked potentials may warn the surgeon of excessive retractor pressure or surgical dissection of the pathway at risk.

Somatosensory Evoked Potentials

Somatosensory evoked potentials are recorded after stimulation of a peripheral sensory nerve with electrical stimulation via a surface electrode placed on the skin or by fine needle electrodes.

The current strength used is 10-20 mA and duration of stimulus varies from 0.1-2 milli seconds.

The sites of stimulation of the peripheral nerve are:

- Median nerve at wrist
- Posterior tibial nerve at ankle.
- Peroneal nerve.

Clinical Uses

Somato sensory evoked potentials are used to assess the functional integrity of the sensory pathways. Intraoperative SSEP monitoring has been described for:

- Scoliosis corrective surgery
- Stabilisation and decompression after spinal cord surgery.
- Spinal fusion surgery.
- Brachial plexus exploration.
- Resection of acoustic-neuroma.
- Resection of thalamic tumour.
- Abdominal and thoracic aortic aneurysm repair.
- Decompressive laminectomies.

Factors Affecting SSEP

Apart from the anatomical disruption of the nervous pathway, certain factors can decrease the amplitude and increase the latency of somatosensory evoked potential. They are:

- Volatile anaesthetic agents - halothane, enflurane, isoflurane
- N_2O
- Diazepam
- Hypothermia.

Auditory Evoked Potentials

Auditory evoked potential is monitored when CRANIAL NERVE VIII or Brainstem is at risk of injury. The risk of hearing loss during resection of acoustic tumour and other tumors of the cerebellopontine angle is substantial and hence the need for auditory evoked potential monitoring.

TECHNIQUES

There are three major techniques for monitoring auditory evoked potential.

- Brainstem auditory evoked potential (BAEP).
- Electrocochleography (ECOG)
- Auditory nerve action potential recording (ANAPR).

Brainstem Evoked Potentials

Brainstem evoked potentials are recorded after a brief auditory stimulus is delivered to one ear that produces a sequential activation of the peripheral auditory pathway.

BAEP are recorded by delivering clicks repeatedly (500-1000 stimuli) in one ear with frequency of 10 Hz and duration of 100 microseconds. Recording electrodes are placed on the lobe of stimulated ear and on top of the head (vertex).

A normal BAEP shows 7 peaks or waves.

Wave	*Generator*
Wave I	Acoustic nerve
Wave II	Cochlear nucleus
Wave III	Superior olive (pons)
Wave IV	Lateral leminiscus
Wave V	Inferior colliculus (midbrain)
Wave VI	Medial geniculate (thalamus)
Wave VII	Thalamocortical radiations.

A decrease in the recorded amplitude or increase in latency shows functional derangement of the auditory pathway.

Uses

- Microvascular decompression of cranial nerves
- Resection of acoustic neuroma.
- Posterior fossa exploration for vascular and neoplastic lesions.
- Clipping of basilar artery aneurysm.
- Section of the nerve VIII for intractable tinnitus.

Electrocochleography

With ECOG, a monopolar needle electrode is placed through the tympanic membrane onto the soft tissue covering the bony promontory of the middle ear.

A reference needle electrode is placed over the mastoid. Thus near field action potentials, can be recorded from the cochlear membrane and lateral segment of the auditory nerve analogous to wave I of BAEP.

This technique affords the surgeon rapid feedback on the functional status of the cochlea since ECOG requires only 20 to 50 stimuli.

Auditory Nerve Action Potentials (ANAP)

The recording of ANAP involves placement of an electrode onto the auditory nerve under direct vision after the cerebellopontine angle has been discovered. A click or burst stimulus is delivered to the ipsilateral ear and a well-defined 10 to 30 microvolt potential can be recorded.

Visual Evoked Potentials

Visual evoked potentials are recorded after mono-ocular stimulation with recording electrodes placed over occipital, parietal and central scalp. The stimuli are produced by light emitting diodes placed in a goggle or contact lens over a closed lid. The flash rate is 1 to 3 Hz with duration of 3 to 5 milliseconds. Two positive peaks at approximately 100 and 200 milliseconds are observed.

Visual Evoked Potential are Used in the Following Procedures

- Resection of pituitary tumours.
- Resection of craniopharyngioma, optic glioma orbital pseudotumour, occipital arteriovenous malformation.
- Resection of meningioma impinging on optic chiasma
- Clipping of basilar artery aneurysm.
- Correction of orbital fracture.

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63 Pulse Oximetry

INTRODUCTION

Pulse oximetry is the measurement of oxygen saturation of pulsating arterial blood.

The pulse oximeter was first introduced into clinical practice by Yelderman and New in the year 1983. Pulse oximeter provides continuous, accurate, real time and noninvasive measurement of arterial oxygen saturation.

PRINCIPLE

Basically this instrument works on photospectrometry which uses the principle of Beer Lambert Law. In short the pulse oximeter works on the principle that oxyhaemoglobin and reduced haemoglobin absorb red light and infrared radiation differently. Oxyhaemoglobin is much more transparent to red light (wavelength around 660 nm) while reduced Hb is somewhat more transparent to near infrared radiation (wavelength about 940 nm).

BASIC DESIGN

The pulse oximeter consists of a simple probe which can be attached to a finger, ear lobe or fixed across the nasal bridge or wrapped around a child's digit. The probe carries two LEDs, one emits light at a wavelength of 660 nm (red region of spectrum), and the other emits light at 940 nm (in the infrared region). The probe also contains a photodiode placed to the opposite side of the digit or ear lobe as transmitting and sensing transducer.

INTERPRETATION

O_2 saturation is directly displayed digitally and no interpretation (other than physiological) is required.

ADVANTAGES AND USES

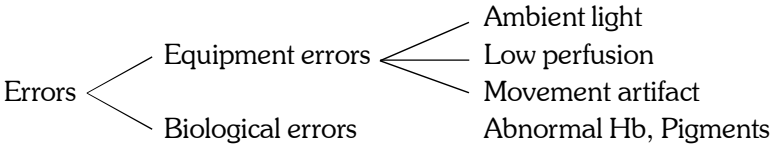
- Gives noninvasive, continuous real time and accurate value of O_2 saturation in arterial blood.
- Simple to use, no previous training of personnel or calibration of instrument is necessary.
- It provides an overall assessment of integrity of all systems involved in the delivery of O_2 to the tissues.
- The function is unaffected by pigmented skin.

Main Uses

- Anaesthesia in infants and children. For example, Bronchoscopy.
- Transportation of critically ill patients.

- One lung ventilation.
- Conditions with reduced lighting. For example, In radiology, in ENT.
- Endoscopic examination.
- In monitoring patients with sleep apnoea.
- Assessment of the adequacy of CPCR.
- Assessment of collateral flow of blood in hand (Allen's test).
- Testing viability of micrograft.
- In ICU.

Limitations and Errors



- *Ambient light:* This can produce errors, but modern pulse oximeters solve this problem by alternating the red and infrared sources.
- *Low perfusion:* Low perfusion states, as in peripheral vascular disease, vasoconstriction, hypothermia, severe hypotension, etc. can produce a false negative or reduced value.
- *Movement artifact:* Signal artifact is abolished in modern machines by increasing the signal averaging time.

Biological Errors

- Carboxy haemoglobin gives a false positive (higher) value.
- Meth haemoglobin also gives false positive value (Lower values).
- Dyes in circulation can produce error.
- Nail polish especially blue.
- Bilirubin, skin pigmentation, etc. will not produce any significant alteration in the values.

Disadvantages

- Inability to detect hyperoxia.
- Cannot sense PaO_2 changes.
- Inaccurate reading in the presence of other Hb like carboxy Hb, Meth Hb, etc.

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64 End Tidal Capnography

Capnography is the technique by which the fluctuations of CO_2 in the end tidal gas drawn from the alveoli is measured and monitored. The principle was developed by Luft in 1943. The clinical practice of this was pioneered by Smallhout and Kalenda in the Netherlands.

This technique provides information regarding:

- Efficacy of the breathing system.
- Identifies the life threatening intraoperative complications like air embolism, pulmonary embolism, etc.
- Help us to find out early, mishaps like oesophageal intubation.
- Alterations in ventilation, cardiac output distribution of pulmonary blood flow and metabolic activity.

Capnograph: Device which record the CO_2 fluctuation.

Capnogram: Tracing of trend of CO_2 fluctuation on the recording paper.

PRINCIPLE

Carbon dioxide in the respired gas may be continuously monitored by mass spectrometry, Raman spectroscopy, or infrared analysis. Infrared analysis is the most cost effective and reliable technique.

Principle involved in this technique is that peak absorption of IR light by CO_2 is around the wave length of 4300 nm. Infrared light of this wave length is passed through the gas column of unknown concentration of CO_2 . Carbon dioxide absorbs some of IR light while the remaining is transmitted. The intensity of transmitted light is compared with a reference beam.

EQUIPMENT

Basic unit consists of light emitting diode (LED) to produce light of the required wavelength with a sold state photodetector to measure the amount of light reaching it, alternatively via the measuring and reference cells, with the beam chopped at 4000/min.

ANALYSER

- Side stream analyser
- Main stream analyser

SIDE STREAM ANALYSER

These devices draw gases continuously from the sampling site via small bore tube of 2 m length and diameter of 2 mm. Ideal is a Teflon tube which will not react with halogenated hydrocarbons. Sampling rate should be 50 to 250 ml/mt. Site of sampling should be closer to the distal end of the endotracheal tube, especially when we are employing higher flows in case of infants and children. Otherwise it can result in dilution of the sample. Disadvantage of this type is delayed response.

MAINSTREAM ANALYSER

These do not draw gases but incorporates the analyser cell with IR source and detector into the specially designed airway adaptor which is interposed into the system. This has got a very fast response. Disadvantages are, water can condense and can offer significant resistance to the flow of gases and also it damages the cells. The added dead space is also a disadvantage.

NORMAL CAPNOGRAM

Normal waveform (Fig. 64.1) of expired CO_2 concentration is a square one and consists of 4 phases.

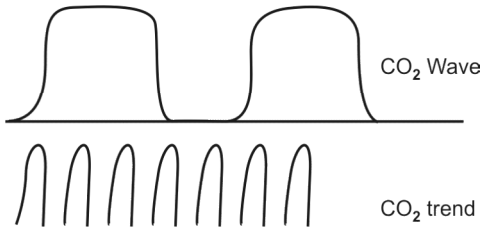

Fig. 64.1: Normal capnogram

- Inspiratory baseline – AB
- Expiratory upstroke – BC (produced by alveolar gas displacing the anatomic dead space)
- Expiratory plateau phase – CD (produced by the alveolar gas)
- Inspiratory downstroke – DE.

Systematic Examination of Capnogram

Presence of Exhaled Gas

If capnograph registers no flow, suspect (Fig. 64.2)

- Dislodged or misplaced ETT
- Disconnection of breathing circuit.
- Apnoea.

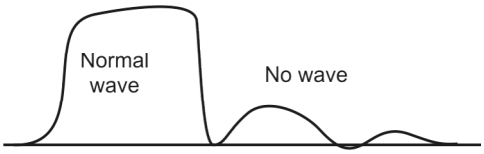

Fig. 64.2: Sudden drop of end-tibial CO_2

Analysis of Phases of Capnogram

- Inspiratory base line: Normal inspiratory CO_2 is zero. It is elevated in
  - Exhaustion of soda lime
  - Incompetence of soda lime
  - Low FGF
  - Hypoventilation
- Expiratory upstroke (Fig. 64.3): Prolonged or less steep upstroke
  - Obstruction of ETT
  - Bronchospasm
  - COPD, uneven alveolar emptying

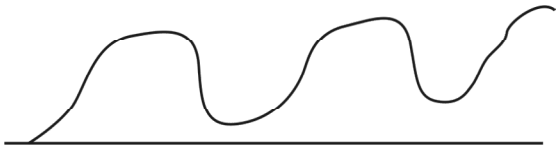

Fig. 64.3: Expiratory upstroke

- Expiratory plateau phase (Fig. 64.4): Normally, it is smooth; irregularities indicate
  - Dislodgement of ETT into upper larynx or lower pharynx
  - Manipulation of the lung
  - Transmission of cardiac impulse "Cardiogenic oscillation"
  - Attempted breathing by a patient on ventilator resulting in a dip "curare cleft".

Fig. 64.4: Irregular expiratory plateau

- Inspiratory downstroke: This becomes less steep with rebreathing in case of
  - Rebreathing
  - Use of Bain's circuit with low EGF
  - Low sampling rate or decreased alveolar ventilation.

END TIDAL CO_2

The $ETCO_2$ may not reflect the $PaCO_2$ if there is a gradient either between. $PACO_2$ and P $ETCO_2$ or between PCO_2 and $PACO_2$. The first one occurs because of low expiratory flow rates, uneven emptying of the lungs and shallow breathing. The second one is because of VA/Q mismatch producing a dead space effect. For example, pulmonary embolism, severe hypovolaemia. The normal gradient between $PACO_2$ and $PaCO_2$ is 5 mmHg.

CLINICAL APPLICATIONS

- Monitoring mechanical ventilation. ET CO_2 monitoring warns sudden changes in breathing system due to disconnection, leaks, obstruction, twisting of tubes exhaustion of soda lime, oesophageal intubation, etc.
- Monitoring pattern of breathing; $ETCO_2$ monitoring provides information about hypoventilation or hyperventilation, apnoea, or periodic breathing.
- Monitoring metabolic activity. For early detection of malignant hyperthermia syndrome, when there is dramatic rise of $ETCO_2$. Also useful in case of conditions associated with increase in metabolism such as shivering, seizure, etc.
- Monitoring the circulation: Factors which produce a reduction in blood flow through lungs lead, to decay of graph towards zero.
  - Pulmonary embolisn
  - Air embolism
  - Hypotension
  - Cardiac arrest
- Monitoring extracorporeal circulation: Carbon dioxide monitoring is of value in CP bypass, because hypocapnoea is an invariable consequence when pure oxygen is used in the oxygenator.
- Finally, the capnogram, requires systematic analysis like EKG to obtain the best result (Monitoring the base line, height, frequency, rhythm, shape, etc.).

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65 Humidifiers

Humidity is the term used to describe the amount of water vapour present in air or in the gases concerned. The amount of water that a gas can carry depends upon its temperature.

For Example: 1 cubicmetre (1 m^3) of air can carry 10 gm of H_2O at 10° C and 30 gm of water at 37° C and 44 gm of H_2O 37° C.

DEFINITIONS

Absolute Humidity

Absolute humidity is defined as mass of water vapour present in a unit volume of air expressed in gm/m^3.

Humidity at Saturation

The maximum mass of water vapour that can be carried in one cubic metre of air is the humidity at saturation at a given temperature.

Relative Humidity

Relative humidity is the ratio between absolute humidity and humidity at saturation.

For air to be fully saturated with water vapour within the body (in the respiratory tract), 36.5 gm of water should be added to each cubic metre of air.

36.5 gm of water will fully saturate (1000 cc) or 1 cubic metre of air.

If a humidifier saturates air at room temperature and then that air is warmed to body temperature the absolute humidity is unchanged but the relative humidity will fall to 30%.

Saturation is achieved by adding to air a mist or fog of minute droplets of water, which vapourise when relative humidity falls.

Thus, by humidification it means addition to air or gas, water in the form of water vapour or nebulised droplets (Fig. 65.1).

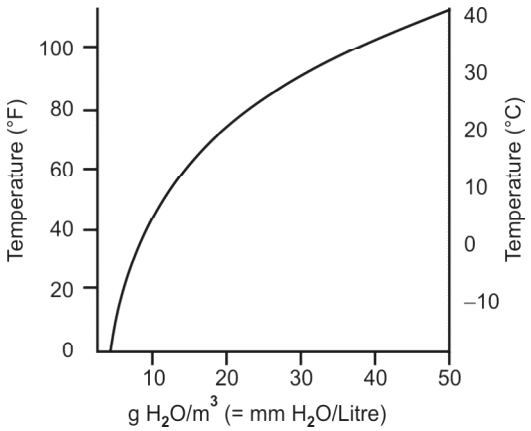


Fig. 65.1: Graph showing mass of water that is carried at saturated vapour pressure in terms of grams of water per cubic metre of air

IMPORTANCE OF HUMIDIFICATION

- The natural humidifier of the body (nose) is bypassed by an endotracheal tube or tracheostomy tube. This can cause drying of the respiratory tract. This can lead to (1) Impaired functioning of the ciliary epithelium and cause tracheitis, bronchitis (2) Respiratory infection.
- Some medicaments, e.g. salbutamol can be administered by being nebulised in the inspired air. The droplet size should be very small, less than 1 micrometre in diametre.
- Nebulisation of liquid may also be employed to disperse sterilising agents for (i) decontamination of breathing systems (2) for disinfection around the entire operating room.

CLASSIFICATION OF HUMIDIFIERS

The various devices may be classified in different ways:

- Active or passive:
  Active: This requires either electric power as in ultrasonic nebuliser, or the power of jet of air or gas as in venturi type, which can humidify oxygen from cylinder or pipeline.
  Passive: In passive humidifier the gases simply bubble through the water as in bubble bottle.
- Secondly, they can be classified according to whether they produce pure vapour e.g. Humidifier, or droplets of water e.g. Nebuliser.
- Humidifiers can be classified as hot or cold, depending upon the temperature of the humidifier. In hot humidifier/warm humidifiers it is important to check against scalding of the patient.

HUMIDIFICATION METHODS

Direct Instillation of Saline

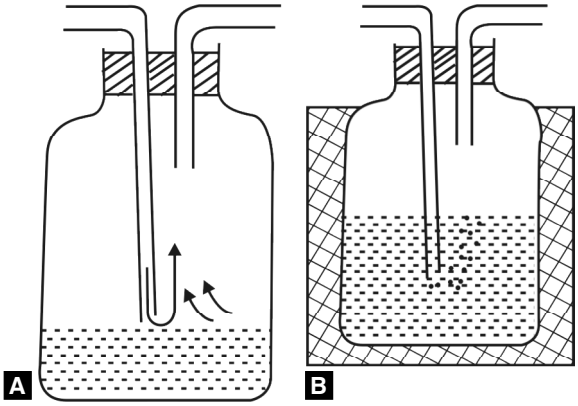
Normal saline can be instilled directly into an endotracheal tube or tracheostomy tube. Though not used, much these days, as a method of humidification an extension of this practice is endobronchial lavage which is carried out to remove thick secretions or mucous plugs.

Simple Bottle Humidifier (Fig. 65.2)

A simple bottle humidifier can be used for humidification. If air passes over the surface of the water, a modest degree of vapourisation will take place. This can be increased by (1) increase in temperature of water by water jacket. (2) bubbling the air through the water.

In cases of heated humidifiers the temperature of the water is thermostatically controlled so that the patient is not scalded.

The humidifier should always be kept at a lower level than the patient, so that, in event of an accident there is no water running down the tubes and scalding or drowning of the patient is avoided.



Figs 65.2A and B: (A) The simple bottle humidifier, (B) Simple bottle with a hot water jacket

The Heat and Moisture Exchanger (HME) or Artificial Nose (Fig. 65.3)

The artificial nose is a passive device and therefore cannot achieve full humidification.

It consists of a chamber containing a screen through which the respiratory gases pass in each direction. The screen may consists of wire mesh, a block of hygroscopic foam or specially formulated paper. Its operation is as follows. When exhalation occurs moist warm gases impinge on the cooler dryer screen, and the vapour condenses, the specific heat of the condensed expired air and the latent heat of water warm the screen.

Thus, at the start of inhalation, relatively cooler and dryer inspired gases are warmed and humidified as they pass through screen.

A relative humidity of 70% can be attained at 33° C.

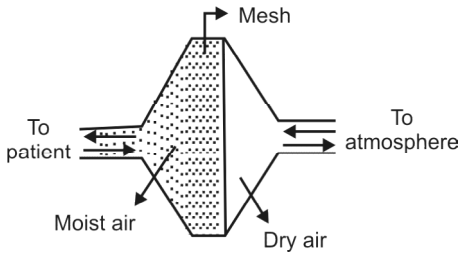


Fig. 65.3: The heat and moisture exchanger (artificial nose)

Advantages

- Simple to use
- In expensive
- Does not depend upon electric power for its use.
- Immune to accidents such as drowing of patients and scalding of patient.

Disadvantages

- 100% humidification cannot be achieved as it is passive, type. Only 70% humidification of inspired air is possible.
- Increases dead space as much as 10 ml to 90 ml depending upon type of HME.
- Increase in airway resistance. Has to be changed once in 3 hrs.

NEBULISER (FIG. 65.4)

A jet of air or gases may be used to entrain water drawn up from a reservoir. As the water enters the jet it is broken into a number of droplets that is nebulised. These droplets can be made to impinge on an anvil so causing them to be broken up into still smaller droplets of 2 to 4 microns which humidify the airway. The large particles fall back into the reservoir.

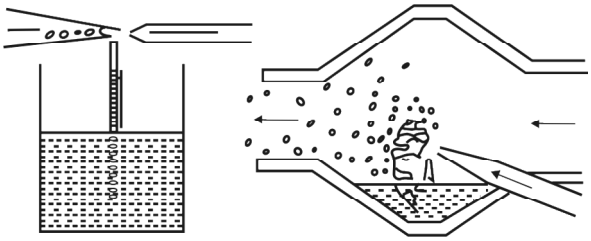


Fig. 65.4: The principle of a nebuliser

Ultrasonic Nebuliser Droplets (Fig. 65.5)

Water is broken up into droplets or fine particles by continuous sonic bombardment generated by high frequency resonator. The ultrasonic nebuliser should produce droplets of adequate size. Since too large droplets will fall out and be deposited on the breathing tube but too small droplet will be deposited on the alveoli and will not humidify the trachea.

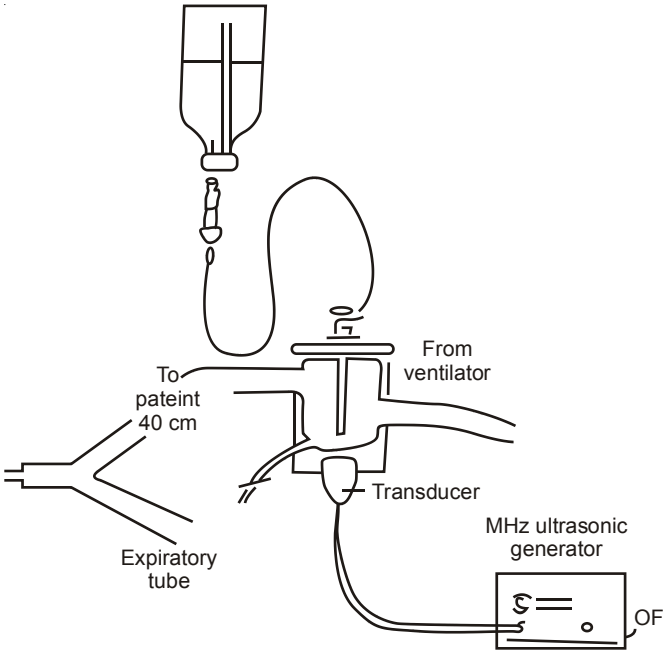


Fig. 65.5: Ultrasonic nebuliser

In 1964, Herzog and his colleagues introduced into clinical practice the ultrasonic nebuliser. Each drop of fluid in ultrasonic nebuliser is nebulised to a size of 0.8 to 1 micron.

Advantage

It is one of the most efficient method of humidification. Humidification is above 100% (nearly 160%).

Disadvantages

- Overhydration in children
- Waterlogging of the patient
- Difficult to sterilise and cross infection can occur
- Expensive.

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66 Mapleson Circuits

Mapleson classified the semiclosed rebreathing anaesthetic breathing systems into 5 types. They are A, B, C, D and E. The Mapleson E was subsequently modified by Rees, but is classified as Mapleson F system.

MAPLESON A SYSTEM (FIG. 66.1)

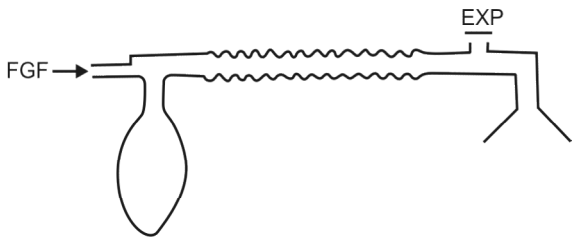


Fig. 66.1: Mapleson A—breathing system

Also known as Magill system (Magill attachment), It consists of the following parts in order: 1. Fresh gas inlet, 2. Reservoir bag, 3. Corrugated tubing, 4. Release valve, 5. Face mask or endotracheal tube adaptor. The corrugated tubing should have adequate length usually 110 cm.

Technique of Use

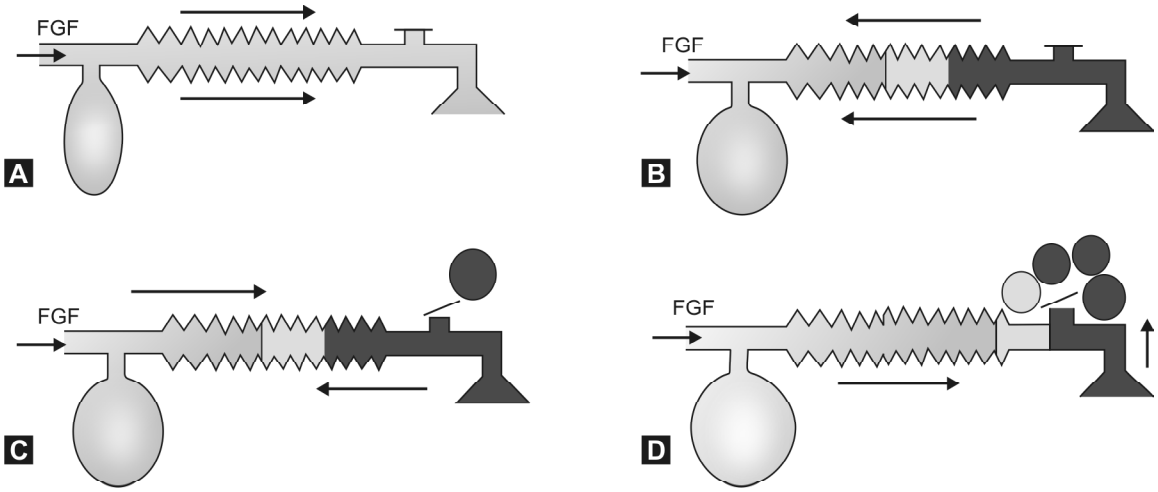
This is the most efficient system on spontaneous respiration and least efficient system during controlled ventilation. For spontaneous ventilation, the relief valve is opened and the fresh gas flow set so that the bag is full when inspiration begins. The excess gas is discharged through relief valve during expiration.

Functional Analysis and FGF Recommendations

This system function very differently during spontaneous and controlled ventilation.

During Spontaneous Ventilation

There are 3 phases in ventilatory cycle. They are inspiration, expiration and expiratory pause. During inspiration, gas is inhaled from system and the reservoir bag collapses. *During expiration*, first the dead space gas in (dotted area) and then the alveolor gas (cross- hached area) passes into the corrugated tubing. Simultaneously, the fresh gas will fill in reservoir bag and passes into the corrugated tube and the pressure in the system increases until the relief valve opens and gas is discharged into the atmosphere. The first gas discharged will be the alveolar gas lying at the patient



Figs 66.2A to D: Mapleson A—functional analysis: Spontaneous ventilation*

end of corrugated tubing. *During Expiratory* pause, continued flow of fresh gas from the machine pushes alveolar gas and dead space gas distally along the corrugated tube to be vented through the spill valve. Provided the FGF rate is sufficiently high to vent all alveolar gas before next inspiration, no rebreathing takes place from the corrugated tube.

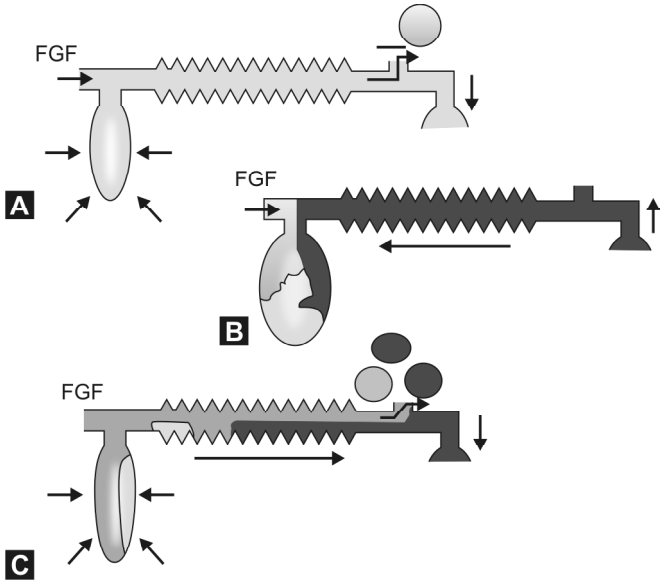
If the system is functioning correctly and no leaks are present a fresh gas flow rate equal to patient's alveolar minute volume is sufficient to prevent rebreathing. In practice in order to compensate leaks, the FGF rate selected usually is equal to total minute volume (6L in 70 kg man). Even with use of pediatric face mask, the dead space remain too high for children, especially below 4 years, so ideally should be avoided in children of less than 4 years age.

During Controlled Ventilation

During expiration, the exhaled gas enter the corrugated tube which become filled with dead space gas and alveolar gas. It may reach upto the reservoir bag. During expiratory pause the reservoir bag is filled with expiration fresh gas. The inspiration is produced by squeezing reservior bag, and the

* The system is filled with fresh gas before connecting to the patient. When the patient inspires, the fresh gas from the machine and the reservoir bag flows to the patient, and as a result the reservoir bag collapses. (Fig. 66.2A). During expiration, the FG continues to flow into the system and fill the reservoir bag. The expired gas, initial part of which is dead space gas, pushes the FG from the corrugated tube into the reservoir bag and collects inside the corrugated tube (Fig. 66.2B).

As soon as the reservoir bag is full, the expiratory valve opens and the alveolar gas is vented into the atmosphere (Fig. 66.2C). During the expiratory pause, alveolar gas that had come into the corrugated tube is also pushed out through the valve, depending on the FGF. The system is filled with only fresh gas and dead space gas at the start of the next inspiration when FGF is equal to the alveolar ventilation (Fig. 66.2D). The entire alveolar gas and dead space gas is vented through the valve and some FG also escapes, if the FGF is higher than the minute ventilations. Some amount of alveolar gas will remain in the system and lead to rebreathing with a FGF less than the alveolar ventilation. This has been confirmed theoretically and experimentally by many investigators. *The system functions at maximum efficiency,* when the FGF equals the alveolar ventilation and the dead space gas (which has not taken part of gas exchange) is allowed to be rebreathed and utilized for alveolar ventilation.



Figs 66.3A to C: Mapleson A—functional analysis: Controlled ventilation*

relief valve open during inspiration. Before the pressure in the system rises sufficiently to open the valve some alveolar gas will be forced into the lung, and when the valve open, fresh gas, dead space gas and alveolar gas are vented through the valve.

Therefore, the rebreathing is marked, but may be decreased by increasing the FGF and increasing the tidal volume. FGF 3 times the estimated minute ventilation must be there to prevent rebreathing. Because of wastage of gas Magill circuit is not recommended for controlled ventilation.

THE LACK CIRCUIT (FIG. 66.4)

This is a coaxial version of Magill (Mapleson-A) circuit in which the expiratory valve is positioned at the anaesthetic machine end of the circuit.

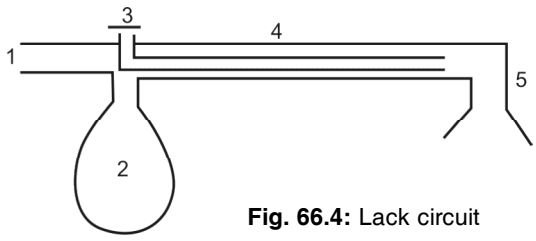


Fig. 66.4: Lack circuit

Equipment

It consists of the following parts in order: (1) Fresh gas inlet, (2) Reservoir bag, (3) Relief valve, (4) Corrugated tube. (5) Adaptor for mask/ETT.

* To facilitate IPPV the expiratory valve has to be partly closed. During inspiration, the patient gets ventilated with FG and part of the FG is vented through the valve (Fig. 66.3A) after sufficient pressure has developed to open the vlave. During expiration, the FG from the machine flows into the reservoir bag and all the expired gas (i.e. dead space gas and alveolar gas) flows back into the corrugated tube till the system is full (Fig. 66.3B). During the next inspiration the alveolar gas is pushed back into the alveoli followed by the FG. When sufficient pressure is developed, part of the expired and part of the FG escpae through the valve (Fig. 66.3C). This leads to considerable rebreathing, as well as excessive waste of fresh gas. Hence, these systems are inefficient for controlled ventilation.

The expired gas vented through the relief valve is collected from the corrugated tube near the patient end through an inner tube with 14 mm internal diameter. It runs through out the length of corrugated tube. Corrugated tube has a length of 1.5 m and an outer diameter of 30 mm and it has a capacity of 570 ml.

Technique of Use

Because this is a modification of Mapleson-A system ideally can use only for spontaneous respiration. The relief valve should be completely opened during spontaneous ventilation.

Functional Analysis and FGF Requirements

During expiration gas flow is through both inner and outer tube. During expiratory pause fresh gas pushes the expired gas from the outer tube towards the patient and to be vented through the inner tube. *During inspiration*, if all expired gas is vented through the inner tube, fresh gas will be inhaled by the patient.

In order to avoid rebreathing FGF of 150% of patients minute volume is recommended when the Lack system is used for spontaneous respiration.

Comparison with other Circuits

- *Bain circuit*
  - Fresh gas flow through the outer tube and expired gas flow through the inner tube in Lack system, but it is just opposite in Bain circuit.
  - Unlike Bain circuit it does not permit the use of anaesthetic ventilator to provide controlled ventilation.
- *Magill circuit*

  The resistance to gas flow is very high in Lack system compared to Magill circuit.

MAPLESON B SYSTEM (FIG. 66.5)

Equipment

It consists of the following parts in order: (1) Reservoir bag, (2) Corrugated tubing, (3) Fresh gas inlet, (4) Relief valve, (5) Face mask.

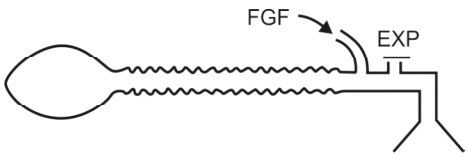


Fig. 66.5: Mapleson B system

Technique of Use

Can be used for spontaneous, assisted or controlled ventilation. Best system for controlled ventilation and 2nd best system for spontaneous ventilation. Technique of use similar to Mapleson A.

MAPLESON C SYSTEM (FIG. 66.6)

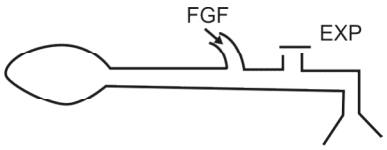


Fig. 66.6: Mapleson C system

Equipment

It consists of the following parts in order: (1) Reservoir bag, (2) FG inlet, (3) Relief valve, (4) Adaptor for facemask/ endotracheal tube.

In both Mapleson B and C system the FG inlet is shifted near the patient to reduce the rebreathing of alveolar gas and to improve the utilization of FG during controlled ventilation. The end result is that these systems are not efficient during spontaneous or during controlled ventilation.

MAPLESON D SYSTEM (FIG. 66.7)

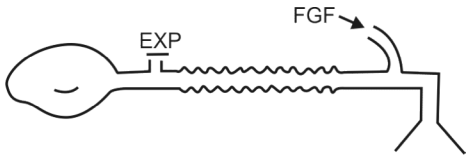


Fig. 66.7: Mapleson D system

The Mapleson D consists, of the following parts namely: (1) Reservoir bag, (2) Relief valve, (3) Corrugated tube, (4) Fresh gas inlet, and (5) Adaptor for face mask or ETT.

For this system to function efficiently and economically during controlled ventilation, the fresh gas entry and the expiratory valve are separated by a volume equivalent to at least one tidal volume of the patient. They are not economical during spontaneous ventilation.

BAIN'S CIRCUIT (FIG. 66.8)

This is a modification of Mapleson D System. It was described by Bain and Spoerel in 1972.

Equipment

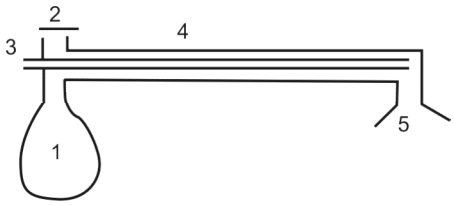


Fig. 66.8: Bain's circuit

Bain's circuit comprises the following parts in order: (1) Reservoir bag, (2) Relief valve, (3) Fresh gas inlet. It delivers fresh gas at the patient end of the circuit by a small bore tube of 7 RAM outer diameter which runs throughout the length of an outer corrugated tube, (4) Corrugated tube, has 1.8 m length and has 22 mm outer diameter. It is made up of transparent, nonconductive plastic material, (5) Face mask or endotracheal tube adaptor, (15 mm) combined adaption (CANAX attachment) can be used to accommodate reservoir bag and expiratory valve.

The Penlon company has introduced a modified version of Bain circuit called Penlon Coaxial Circuit.

In this fresh gas passes straight through the valve unit in a metal tube and connected to the inner tube of coaxial circuit. The expiratory valve is of the shrouded type for connection of scavenging system.

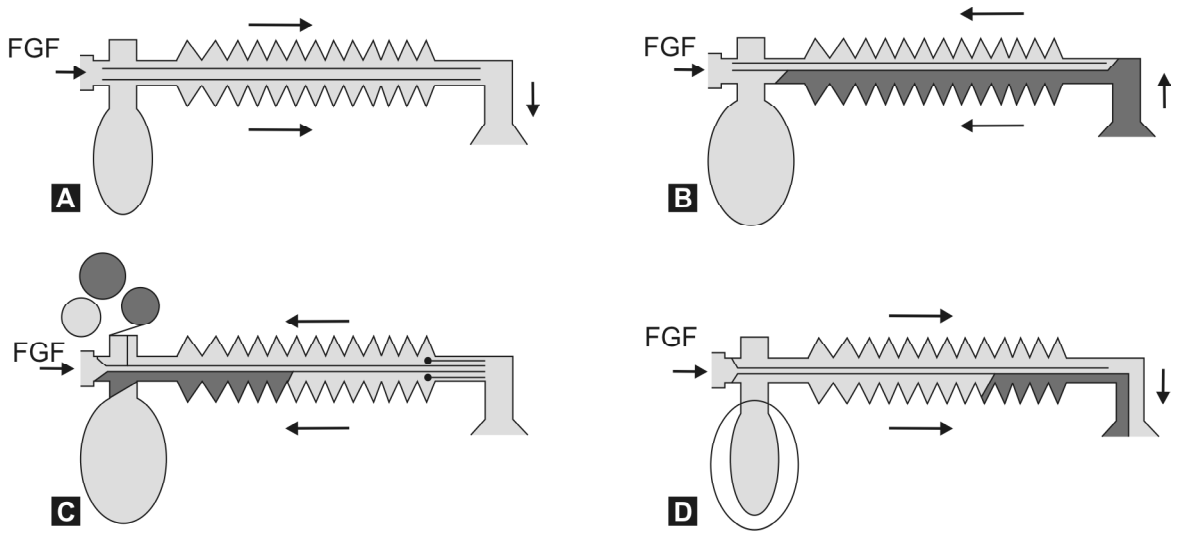
Technique of Use

Because this is a modification of Mapleson D, system can be used for both, spontaneous and controlled ventilation. This system is 1st choice for controlled ventilation and 2nd choice for spontaneous ventilation. For spontaneous ventilation the relief valve, is opened and excess gas will be vented during expiration. For controlled ventilation the resistance of relief valve is to be increased so that excess gas will be vented only during inspiration.

Functional Analysis

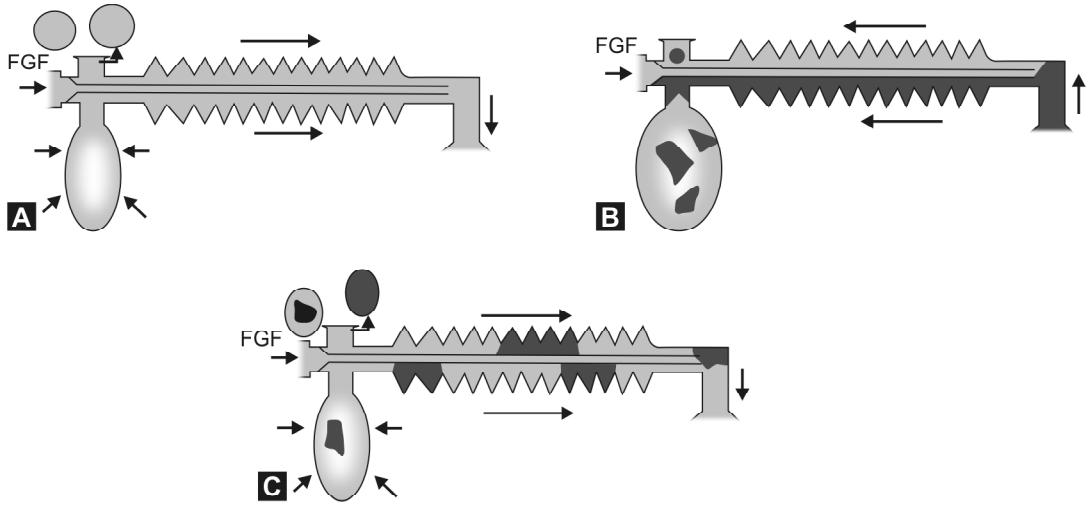
During Spontaneous Ventilation

The breathing system is first filled with fresh gas (FG) and connected to the patient. When the patient inspires, the FG from the machine, the reservoir bag and the corrugated tube flow to the patient (Fig. 66.9A). During expiration the expired gas gets continuously mixed with the FG as it flows back into the corrugated tube (Fig. 66.9B). Once the system is full, the excess gas is vented.



Figs 66.9A to D: Bain's circuit—functional analysis: Spontaneous ventilation*

* The breathing system should be filled with FG before connecting to the patient. When the patient takes an inspiration, the FG from the machine, the reservoir bag and corrugated tube flow to the patient (Fig. 66.9A). During expiration, there is a continuous FGF into the system at the patient end. The expired gas gets continuously mixed with the FG as it flows back into the corrugated tube and the reservoir bag (Fig. 66.9B). Once the system is full the excess gas is vented to the atmosphere through the valve situated at the end of corrugated tube near the reservoir bag. During the expiratory pause the FG continues to flow and fill the proximal portion of the corrugated tube while the mixed as is vented through the valve (Fig. 66.9C). During the next inspiration, the patient breaths FG as well as the mixed gas from the corrugated tube (Fig. 66.9D). Many factors influence the composition of the inspired mixture. They are FGF, respiratory rate, expiratory pause, tidal volume and CO2 production in the body. Factors other than FGF cannot be manipulated in a spontaneously breathing patient. It has been mathematically calculated and clinically proved that the FGF should be at least 1.5 to 2 times the patient's minute ventilation in order to minimise rebreathing to acceptable levels.



Figs 66.10A to C: Bain's circuit—functional analysis: Controlled ventilation*

During expiratory pause the FG flows continuously and, fills the proximal part of the corrugated tube while the mixed gas is vented into the atmosphere (Fig. 66.9C). During next inspiration the patient breaths FG as well as mixed gas from the corrugated tube (Fig. 66.9D). The FG flow should be 1.5 to 2 times the patient's minute ventilation in order to minimise rebreathing.

During controlled ventilation the expiratory valve partially closed. When the system is filled with FG, patient's gets ventilated with FG from machine, the corrugated tube and the reservoir bag (Fig. 66.10A). During expiration the expired gas is continuously gets mixed with FG and during expiratory pause the FG flow persists that pushes the mixed gas towards the reservoir bag (Fig. 66.10B). During next inspiration the patient is ventilated with FG, alveolar and dead space gas mixture (Fig. 66.10C). As the pressure in the system increases the contents of the reservoir bag are discharged into the atmosphere.

FGF during Controlled Ventilation

- Minimum flow 3 L/min is required,
- 70 ml/kg/min. for normocarbia,
- 100 ml/kg/min for hypocarbia.

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* To facilitate intermittent positive pressure ventilation, the expiratory valve has to be partly closed so that it opens only after sufficient pressure has developed in the system. When the system is filled with fresh gas, the patient gets ventilated with the FGF from the machine, the corrugated tube and the reservoir bag (Fig. 66.10A). During expiration, the expired gas continuously gets mixed with the fresh gas that is flowing into the system at the patient end. During the expiratory pause the FG continues to enter the system and Pushes the mixed gas towards the reservoir (Fig. 66.10B). When the next inspiration is initiated, the patient gets ventilated with the gas in the corrugated tube, i.e. a mixture of FG, alveolar gas and dead gas (Fig. 66.10C). As the pressure in the system increases, the expiratory valve opens and the contents of the reservoir bag are discharged into the atmosphere.

Hazards of the Bain Circuit

- Accidental disconnection of inner tube.
- Co-axial attachment malconnection can lead to large apparatus dead space.
- Double back (Z-effect) of inner tube can lead to obstruction.
- Small leaks in the inner tube can lead to decrease FGF reaching the patient end of the circuit.
- Co-axial circuits should not be used with intermittent flow anaesthetic systems.

Testing of Brain and Penlon Coaxial Circuits

Testing is to prove the integrity of inner tube and its connections.

- Because the outer corrugated tube is made up of transparent plastic it allows inspection of inner tube with in the circuit.
- The integrity of inner tube can be assessed by utilising ventury principle with the use of 0_2 flush.
- Pethick's test
- Combined, inner and outer tube occlusion test.

Sterilisation of Bain Circuit

- Ethylene oxide sterilization is effective.
- Low pressure autoclaving is an alternative.
- Activated glutaraldehyde sterilization is unsatisfactory.

Advantages of Bain Circuit

- Brain circuit is considered as a universal circuit it can be useful in both controlled ventilation and spontaneous ventilation and used both in children and adults.
- Light weight,convenient, easily sterilized and reuseable.
- Scavenging of the expiratory gases from the expiratory valve is facilitated.
- Exhaled gases in the outer reservoir tubing add, warmth and humidity to inspired fresh gases.

MAPLESON E SYSTEM (THE T PIECE)

The T piece (Fig. 66.11) first described by Philip Ayre in 1937, was developed in response to the need for apparatus with low resistance and minimal deadspace for paediatric anaesthesia.

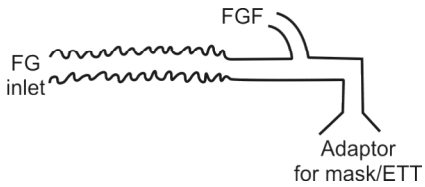

Fig. 66.11: T piece systems

Description of the Equipment

It is a simple piece of metal with three limbs and a port at the end of each limb. One port is for the fresh gas, second port attaches to the endotracheal tube or mask connector. The third limb is the

expiratory limb. A length of tubing may be attached to form a reservoir for the gases. A one cm diameter tubing attached to the expiratory limb has a volume of 2cc per inch of the tubing.

Modifications

Innumerable modifications of the T piece have been made.

- Bain modification: Here fresh gas flow is incorporated into the expiratory limb.
- Jackson Rees modification: Here a reservoir bag is incorporated into the expiratory limb.

Harrisons Classification of the Modifications of T Piece

Type I: No expiratory limb

Type II: Expiratory limb volume greater than patients tidal volume.

Type III: Expiratory limb volume less than tidal volume.

Techniques of Use

May be used with either spontaneous or controlled ventilation. Manually controlled ventilation is possible with alternatively occluding the expiratory limb with thumb. This makes it difficult to gauge the depth of respiration.

Functional Analysis (Fig. 66.12)

When the patient exhales, exhaled gases pass down the expiratory limb and mix with fresh gases entering via the fresh gas inlet. During the expiratory pause the fresh gas flow continuously sweep the exhaled gases away from the T piece and down the expiratory limb.

During inspiration, the gases initially inhaled come from the fresh gas reserves plus the fresh gas which inflows constantly through the fresh gas port. So rebreathing is minimal.

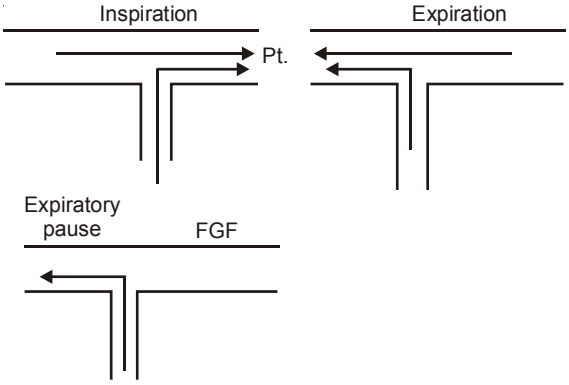

Fig. 66.12: T piece systems functional analysis

Hazards of the T Piece System

Excessive pressure caused by obstruction of the expiratory limb. Prolonged occlusion can cause over inflation and barotrauma.

Obstruction to the inflow of fresh gas. Dangerous air dilution can occur during spontaneous breathing.

JACKSON REES MODIFICATION OF T PIECE (MAPLESON F) (FIG. 66.13)

This is the most commonly used form of the T piece system.

Description of the Equipment

A reservior bag with an opening in it is connected to expiratory limb of the T piece. The hole in the bag is most commonly in the tail and some times in the side of the bag.

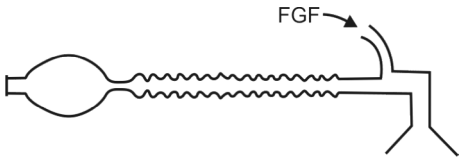

Fig. 66.13: Jackson-Rees (Mapleson F)

Techniques of Use

The system can be used with spontaneous, controlled or assisted respiration. For spontaneous respiration the valve is left fully open. For assisted or controlled respiration the relief mechanism is sufficiently occluded to allow the bag to remain distended. Alternatively the hole in the bag can be occluded by the users finger during inspiration.

Functional Analysis

The system functions very much like a Mapleson E system. The addition of a bag to the expiratory limb will not affect the function of the system provided the bag is separated from the patient by a tube with an internal volume which exceeds the patients tidal volume.

Fresh gas flow requirement: 2-3 times the minute volume.

Advantages

- Simple to assemble and use
- Easily disinfected or sterilized
- Light weight. No excessive drag on the face mask accidental extubation.
- No moving parts. So the equipment is not prone to malfunction
- Less resistance circuits.
- The equipment is inexpensive
- The system can be conveniently positioned.

Disadvantages

- System requires relatively high gas flows.
- Correct gas flow is difficult to determine.
- Dangerous rebreathing if the fresh gas flow is low.
- Mapleson E and F systems are difficult to scavenge.

Out of these circuits, those used in paediatric anaesthetic practice are

- Ayre's T piece.
- Jackson Ree's Modification of T piece.
- Bain's circuit.

The characteristics of an ideal anaesthetic circuit are:

- Low resistance
- Least dead space
- No rebreathing
- Light weight
- Simple to use

Some anaesthetic circuit are known as co axial circuits because both the fresh gas tube and tube carrying exhaled gas run one inside the other. They are:

- Bain circuit.
- Lack circuit and
- Humphrey circuit.

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67 Positive End Expiratory Pressure

Positive end-expiratory pressure exists whenever the airway pressure is greater than the ambient pressure just prior to the next inspiration.

This can be used both during controlled and spontaneous ventilation. When used for spontaneous ventilation, it is termed as CPAP.

Positive end expiratory pressure (PEEP) is produced mainly by two types of devices:

- Gravity-dependent devices
- Non-gravity dependent devices.

GRAVITY DEPENDENT DEVICES

Under Water Column

Expiratory tubing of the circuit pass through a column of water. 'Gases bubble through the column of water. We can adjust PEEP by adjusting the level of water above the port of gas entry.

Water Weighted Diaphragm

Here the expiratory gases will have to push off a diaphragm (above which is water) in order to be vented out, thus producing a PEEP.

NON-GRAVITY DEPENDENT DEVICES

Venturi PEEP Valve

Here, instead of using gravitational pressures high pressure jet gas applied to one side of a one way valve through a venturi tube, the other side of the valve being connected to the patient's expiratory port.

Spring Loaded Disk Valve

An adjustable spring (like in the expiratory valves) provides tension (force) against a disk which rests on the expiratory port.

Pressurised Exhalation Balloon Valve

Inflation of the baloon (which opposes the patient's exhalatory pressure) by any of the three devices. (Venturi jet/adjustable pressure reducing valve/adjustable needle valve).

Magnetic PEEP Valve

Valve is a metal component resting on the exp. gas flow pathway. A variable position magnet (by means of screw) can be moved closer to the valve to increase the attractive force, only on overcoming which the exp. gas can be vented out.

Fixed or Adjustable Orifice

Here PEEP is produced by increasing the resistance to a continuous gas flow by adjusting the orifice of the outflow pathway.

MEASUREMENT

PEEP is measured by connecting a 'T' to the pressure gauge and the plateau pressure during expiration is observed. This measurement should always be made at the Y-connector (Patient-ventilator interphase) (Fig. 67.1).

INDICATIONS

The commonly suggested indications for PEEP are:

- Inability to maintain an acceptable PaO_2 (60 mmHg or 8 KPa) in spite of an inspired O_2 concentration of 50% (i.e., FiO_2 of 0.5).
- Degree of pulmonary shunt (QS/QT ratio) greater than 10 to 15%.
- Conditions such as pulmonary aspiration where terminal airway closure would be expected. Thus, the goal of PEEP is to increase lung volume and avoid alveolar collapse during expiration.

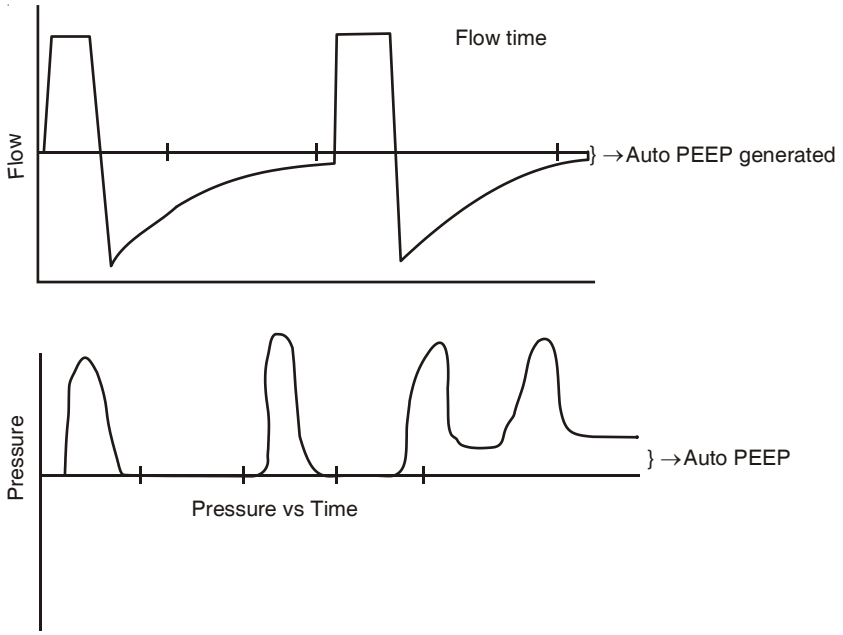

Fig. 67.1: Auto PEEP in ventilator graphics

EFFECTS

The effects which PEEP produce can broadly be divided into two:

Pulmonary

- *Increased FRC:* PEEP increases FRC by two mechanisms.
  - *Increasing alveolar volumes:* Especially upto a PEEP value of 10 cm of H_2O. This, by increasing the surface area of exposure of alveolar gas and capillary blood secondary to elongation of juxta alveolar vessels, effects better gas exchange.
  - *Alveolar recruitment:* This is beneficial because of an increase in FRC secondary to inflation of previously collapsed alveoli. Especially seen beyond a PEEP value of 10 cm H_2O. Thus, peep keeps the alveoli 'splinted open'.
- *Redistribution of lung extra avascular water:* By facilitating movement of water from the less compliant interstitial space between the alveolar epithelium and capillary endothelium where gas exchange occurs, to the more, compliant interstitial spaces around the peribronchial and hilar areas; PEEP improves O_2 diffusion across the alveoli capillary membrane and may play a major role in improving pulmonary mechanisms and oxygenation in severe noncardiogenic pulmonary oedema.
- *Intrapulmonary shunting:* PEEP therapy results in distention of poorly ventilated alveoli. Hence shunt is decreased and more responsive hypoxaemia results.
- *Alterations in dead space ventilation:* Overdistention of alveoli, compression of surrounding capillaries, diminished perfusion and increased dead space ventilation can result.

Cardiovascular and Haemodynamic Effects

- Decreased cardiac output: Occurs due to:
  - Reduction in venous return because of increased intrathoracic pressure.
  - Right ventricular dysfunction because of reduced right ventricular emptying against an increased pulmonary vascular resistance due to PEEP. This reduced pulmonary blood flow reduces LV input and thus reduces cardiac output.

    Also reduced RV ejection leads to increased RV end diastolic volume and a leftward shift of interventricular septum, reducing the LV volume. This also decreases cardiac output.
  - Due to the increased pulmonary pressure above atmosphere produced by PEEP, the lungs exert a smothering or tamponade like effect on heart preventing it from diastolic filling properly, thus reducing the cardiac output.
- *Effects on haemodynamic measurements:* An increase in intrapleural and mediastinal pressure is associated with PEEP. Good correlation exists between pulmonary artery occlusion pressure (PAOP), which is a measure of pulmonary, venous pressure) and LA pressure upto a PEEP of 10 cm. H_2O, whereas with higher levels of PEEP this measurement may be fallacious.

Optimal PEEP

Optimal PEEP coincides with the greatest reduction in QS/QT (intrapulmonary shunt) ratio in the absence of "detrimental effect" on cardiac output. Now redefined as that which provides maximum O_2 delivery and the lowest VD/VT.

Auto PEEP/Inadvertent PEEP/Intrinsic PEEP

This PEEP occurs as a result of inadequate emptying of lung at the end of expiration usually associated with airflow obstruction. This produces 3 major problems:

- Barotraumas
- Hypotension
- Increased work of breathing

How to reduce auto PEEP?

- Reduce Ve (minute ventilation) using lower tidal volumes. Reduce respiratory rate to give enough expiratory time.
- Treat airway obstruction.
- Raise inspiratory flow (square wave).
- Accept reasonable limits of hypercapnoea.

Monitoring

Common monitors used while employing PEEP are:

- Arterial O_2 tension (PaO_2)
- Cardiac output (QT)
- Arteriovenous O_2 content difference ($A\text{-}VO_2$)
- O_2 delivery (cardiac output × arterial O_2 content × 10 expressed as ml/mt)
- Intrapulmonary shunting (QS/QT)—Requires FiO_2 arterial and pulmonary artery blood samples.
- Dead space ventilation (VD)—Can be calculated by VD/VT ratio or monitored by noting changes in the difference between the arterial and the end tidal CO_2.
- Lung compliance—obtained by calculating the static compliance or rather effective static compliance (ESC).

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68 Intermittent Mandatory Ventilation

DEFINITION

This is a method of "augmented" ventilation in which the patient breathes spontaneously at his own rate and the ventilator delivers mechanical breaths of predetermined volume at predetermined mandatory time intervals.

Abolition of spontaneous breathing requires suppression of the ventilatory drive by sedation, muscle paralysis or hyperventilation. Thus IMV is superior to CMV in a patient who has some spontaneous respiration, but this being inadequate also requires mechanical ventilation. In IPPV, the patient does not get fresh gas even if he breathes spontaneously in between. While in IMV, he does receive fresh gas if he breathes spontaneously in between ventilator delivered breaths (Fig. 68.1).

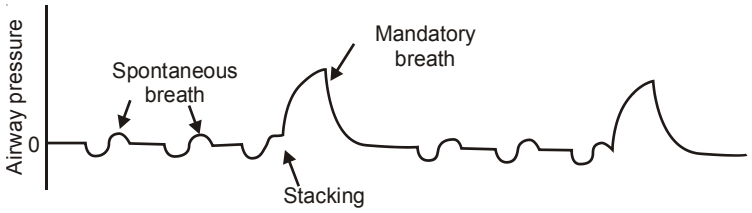

Fig. 68.1: Intermittent mandatory breath

APPARATUS

The breathing system and the composition of the inspired gas (humidity, FiO_2 and temperature) are the same for both spontaneous and mechanical breaths, only the force generating is different. In most IMV systems, a one way valve permits spontaneous ventilation from a reservoir (Fig. 68.2). The ventilator also draws the gas from the same reservoir, the one way valve connecting the patient to the reservoir being closed temporarily by the pressure generated by the mechanical breath.

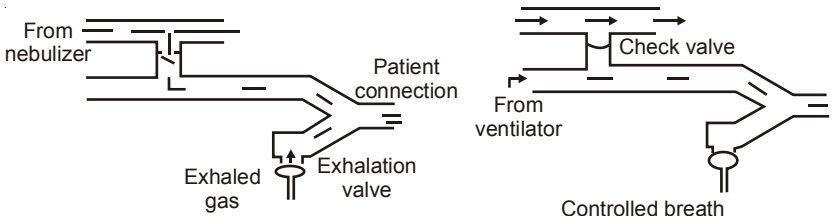

Fig. 68.2: Spontaneous ventilation

Since the patient also breathes spontaneously through the same breathing system, all additional mechanical dead space should be removed, before instituting IMV. Otherwise rebreathing may occur during spontaneous breathing. When spontaneous and mechanical breaths coincide, the machine usually 'overrides' the patient's effort and patient seem to quickly learn to 'ride along'. If there is pronounced spontaneous ventilation, IMV can lead to minute ventilation and respiratory alkalosis.

SYNCHRONIZED INTERMITTENT MANDATORY VENTILATION (FIG. 68.3)

Synchronized intermittent mandatory ventilation (SIMV) is IMV with minimal difference. After the mandatory period lapses, the ventilator waits for the next spontaneous breath so that the additional volume supplied by the mechanical breath is delivered "in phase" with the patient's spontaneous breath. The ventilator senses the negative pressure during spontaneous ventilation which can be pre-set, and delivers the set tidal volume. IMV without synchronization may allow the mandatory breath to be delivered at the end of a spontaneous inspiration. This may result in an increase in tidal volume and an increased risk of pulmonary barotrauma. This problem is avoided in SIMV. In case the patient does not take a breath despite an appropriate waiting period the ventilator goes ahead and delivers the mechanical breath at the set rate "stacking" of breaths is thus avoided.

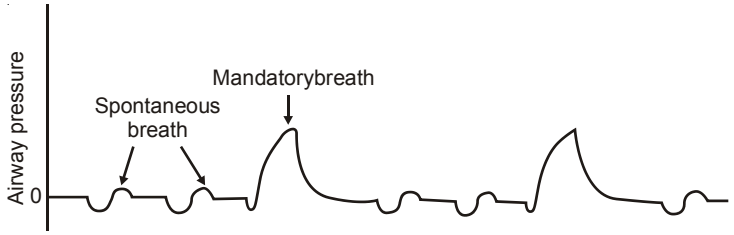

Fig. 68.3: Synchronized intermittent mandatory ventilation

SIMV is the mechanical version of a manoveure, anaesthesiologists have used for decades, while the anaesthestised patient breath spontaneously, assisting every fourth or fifth breath by manually squeezing the reservoir bag of the anaesthetic circuit. SIMV is also known as Intermittent demand ventilation or Intermittent assisted ventilation.

CLINICAL APPLICATIONS OF IMV

Intermittent mandatory ventilation was originally introduced into practice to wean difficult patients from mechanical ventilation. IMV is nevertheless a safe satisfactory and convenient mode of weaning patients.

A patient is considered fit for weaning when he is conscious responsive and haemodynamically stable and when the pathology which necessitated mechanical ventilation has been reversed adequately. When patient is judged to be ready for starting the weaning process, IMV is started at a

rate equal to half the respiratory rate for his age. The patient is closely monitored for the signs of respiratory distress and IMV rate is reduced by one breath at appropriate intervals, (which may very from 4 to 24 hrs) till the rate is two breaths per minute at which stage IMV can be discontinued. Any deterioration in the patient's condition may necessitate going back several steps and even resorting to full mechanical ventilation.

In patients who have had prolonged ventilation, weaning should proceed at a much slower rate; in such patients, the IMV rate is reduced by one breath per minute every 12 to 24 hours only. ABG must also be estimated frequently and one must always be prepared to go back to a higher IMV rate and slow the weaning process if the patient is not yet ready.

Though IMV was originally introduced as a method for weaning, IMV has now become an independent mode of ventilation in its own right in patients with some spontaneous breathing. In combination with CDP, has now become the corner stone of therapy for patients with respiratory failure. This combination allows goal directed treatment of the two distinctly different forms of respiratory failure i.e. ventilatory failure and failure of oxygenation.

The clinical conditions in which IMV is very often used include:

- ARDS - low IMV rates in combination with CDP
- Ventilatory failure due to central depression and to neuromuscular disease or in immediate postoperative period.
- Respiratory failure in chronic obstructive airway disease.
- Flail chest.

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69 Pressure Support Ventilation

Pressure support ventilation (PSV) is essentially a method of assisting the inspiratory effort of a spontaneously breathing patient.

To understand pressure support ventilation, we must have a brief understanding of the work of breathing. Work of breathing (WOB) is the work that must be performed to cause the lungs to expand during inspiration. Expiration is a passive process. Work = Force × distance, in physics. In applications to the lung physiology.

$$\text{WOB} = \text{Pressure} \times \text{Volume}$$

There are two components which pressure needs to overcome before lung volume can increase.

- Elastic recoil encountered in stretching lung and rib cage.
- Nonelastic (viscous resistance) which comprises airway resistance and tissue resistance.
  - When pulmonary compliance decreases, the work performed to overcome elastic resistance increases.
  - When airway resistance increases, the work to overcome the resistance and hence the work of breathing also increases.

Minute volume is Tidal volume × Respiratory rate. Each MV has an optimum respiratory rate at which the WOB is minimal.

Pressure support ventilation is essentially a method of assisting the inspiratory effort of a spontaneously breathing patient. Hence, the patient's respiratory drive must be intact.

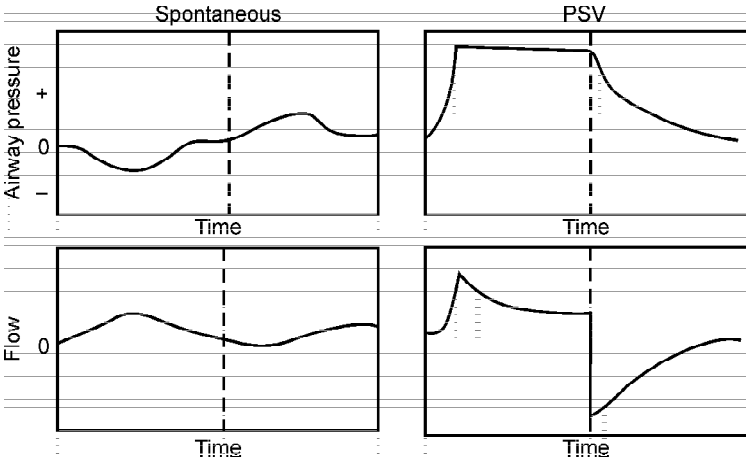

Fig. 69.1: The airway pressure and flow waveforms during spontaneous breathing and P&V.

Mechanism

The patient has to 'trigger' the ventilator to receive a pressure supported breath. Once triggered, the ventilator supplies the patient with a clinician selected constant level of positive pressure during most of the inspiratory phase.

Pressure support is terminated towards the end of spontaneous inspiration. The cycling from inspiration to expiration is related to flow. When the flow decreases to 25% of the peak flow then inspiration stops and expiration occurs.

In interest of patient safety, two over-riding mechanisms are usually incorporated.

- The first is pressure cycling, flow being terminated when airway pressure exceeds the level of PSV by predetermined amount.

  This back-up supports the patients lungs from barotrauma should cough or strain.
- The other is time cycling backup which prevents a prolonged inspiration in event of significant leak in the breathing circuit of ventilator.

In pressure support ventilation (PSV), when the patient's spontaneous inspiration opens the demand valve, the ventilator provides a variable flow of gas that rapidly increases, until the inspiratory circuit pressure reaches a preselected level above baseline. A microprocessor controlled servo mechanism adjust the flow to keep this circuit pressure constant. As patient's airway pressure approaches the preselected circuit pressure, flow into the system diminishes and inspiration ends when a preset low system flow is reached (Fig. 69.1).

The tidal volume delivered with PSV is dependent upon: (1) preselected pressure level, (2) patients inspiratory effort, (3) pulmonary mechanism.

This volume variability makes it difficult to compare with the standard techniques based on existing data.

When pressure support level is raised sufficiently (20 to 30 cm H_2O), most of the ventilatory work is done by the ventilator, and mode is functionally similar to AMV/Assist mode. When set at 3 to 6 cm H_2O and used is conjunction with SIMV, the system functions similarly to continuous flow IMV system, with respect to spontaneous work of breathing.

Thus, PSV helps

- Initially to overcome the resistance offered by the artificial airway and breathing circuit.
- When the PSV is set appropriately high (PMAX), it greatly assists the inflation of the lung during inspiration and attenuates the elastic workload.

PSV MAX

It is pressure support that deliveries a tidal volume of 10 to 12 ml/kg, where WOB is completely taken over by the ventilator, except for the minimum effort of triggering on ventilator.

Uses of PSV

- PSV is used to reduce work of breathing by the patient and reduces respiratory fatigue.
- It is used in weaning or gradual discontinuance of ventilatory support.
- It is used for conditioning the respiratory muscles for endurance and strength.

Once PSV is established, the level of pressure support should be gradually reduced at a speed tolerated by the patient.

If airway reflexes are intact, the patient can be extubated at a pressure support of 5-7 cm H_2O which is the level required to overcome the resistance of artificial airway and ventilatory circuit.

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70 Weaning Modes of Ventilation

DEFINITION

Withdrawal of mechanical ventilation by gradual process is called Weaning. A patient who has been ventilated for only a few hours (either because of inadequate recovery from anesthesia or prophylactically following a major procedure) may be rapidly removed from the ventilator as soon as his recovery is adequate. Ideally the term weaning can be used only in those minority of patients who can not be discontinued from the ventilator support, without gradual manoeuvres taking more than 12 hours.

CRITERIA FOR WEANING

- The underlying indication for ventilatory support is significantly reversed.
- Measurement of cardiopulmonary reserve are judged adequate for spontaneous ventilation.
- General clinical examination and laboratory measurements presents no contraindication to maintain adequate spontaneous ventilation and cardiopulmonary homeostasis.

CLINICAL CRITERIA

- Fully awake and alert patient.
- Lack of residual hypnotic/opioid /relaxant effects.
- Free from fatigue and pain.
- Satisfactory cardiovascular status—
  - Free from tachycardia, hypotension, hypertension
  - Absence of dysrhythmia
  - Optimal haemoglobin
- Satisfactory respiratory reserve
  - Vital capacity 10 to 15 ml/kg
  - $FEV_1 > 10$ ml/kg
  - Tidal volume > 15 ml/kg
  - Peak inspiratory pressure $\geq$ 20 cm H_2O
  - PaO_2 (at fiO_2 of 0.4) 8 KPa
  - Dead space/tidal vol. < 0.6

WEANING METHODS

T Piece Weaning

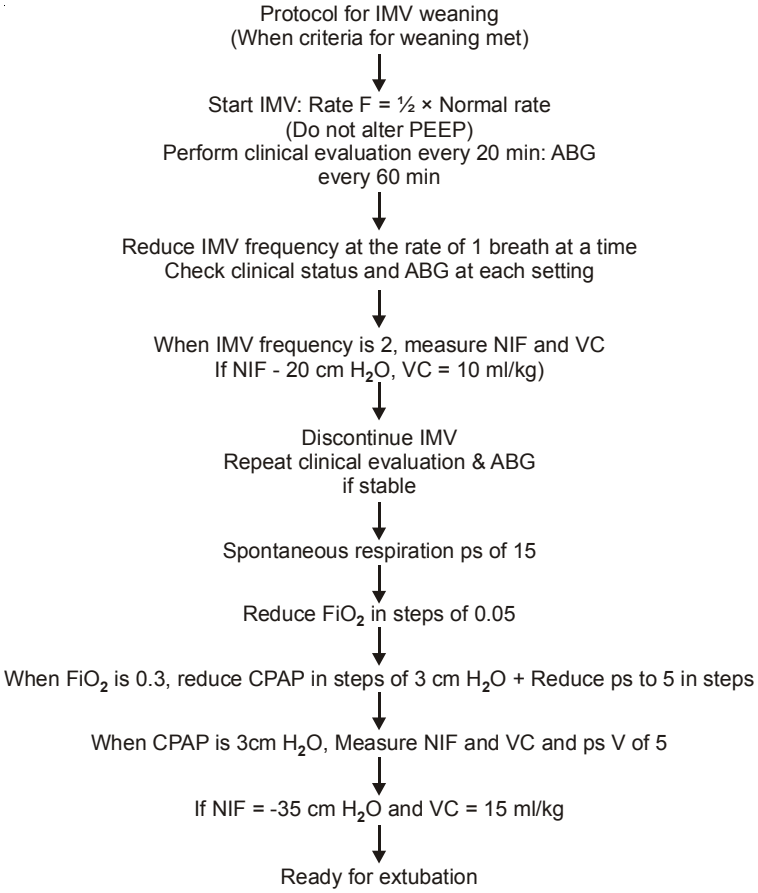
Prior to the use of IMV as a weaning tool, patients were weaned from the mechanical ventilator with the use of "T tube" trials. Even now used in patients who have been ventilated for less than 48-72 hours are weaned with T tubes. In some situations T piece weaning may succeed when IMV weaning has failed.

IMV Weaning (Flow chart 70.1)

A patient who has been on respiratory support for several days is probably best weaned with the help or IMV or SIMV.

IMV was originally introduced into clinical practice to wean difficult patients from mechanical ventilation. IMV is nevertheless a safe satisfactory and convenient mode for weaning patients.

Flow chart 70.1: Protocol for IMV weaving



IMV is an augmented ventilation in which the patient breathes spontaneously at his own rate and the ventilator delivers mechanical breaths of predetermined tidal volume at predetermined mandatory time intervals.

Insynchronized mandatory ventilation (IMV) there is synchronization of the mechanical breath on the spontaneous next breath. In IMV and SIMV the minute volume is the sum of the volume delivered by the mandatory mechanical breaths during that minute and the patients spontaneous minute volume.

In any of the above steps, if the patient deteriorates, go back to the previous settings. Be prepared to go back by several steps if required.

IMV/SIMV-Pressure Support Ventilation

This has the advantage of allowing the patient gradually to assume more work of breathing. It is recommended that SIMV (Fig. 70.1) without pressure support not to be used at ventilator rates of less than 4/minutes because the demand may unnecessarily increase the work of breathing.

PSV is a form of mechanical ventilatory support in which the patient's spontaneous effort is assisted by the ventilator, upto a clinician selected level of P_{aw} (airway pressure). It acts mainly by reducing the work of breathing.

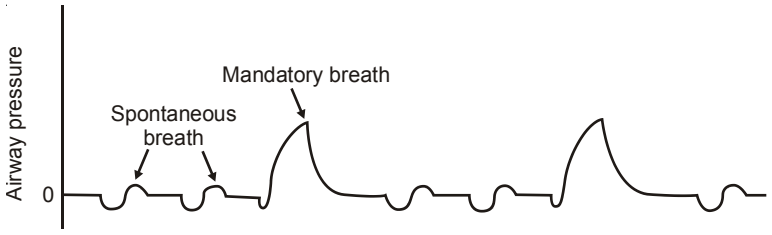


Fig. 70.1: Synchronised intermittent mandatory ventilation (SIMV)

It is ideal to start with a PSV max and progressively reduce the level of PSV down to 5 cm H_2O. If the patient maintains a normal rate and normal ABG, the individual is ready for extubation.

CPAP and Weaning

There are circumstances in which the use of 5 to 10 cm of CPAP aids the ability to maintain spontaneous ventilation. Benefit includes: (1) adequate maintenance of FRC, (2) improved lung compliance, and, a subjective sensation of better lung inflation.

As a general rule, patients already receiving PEEP therapy should receive 5 to 7.5 cm H_2O of CPAP during weaning, and trachea should be extubated only depending on the set CPAP level.

Airway Pressure Release Ventilation (Fig. 70.2)

As in the diagram, the patient breathes with a CPAP of 10 cm H_2O. Suddenly at point (A), a valve opens in the circuit and P_{aw} falls to point B, i.e. a lower level of CPAP. It remains therefore 1 second and again CPAP is reapplied to the original level.

It is claimed that APRV provides good gas exchange at lower mean airway pressure and that it is easier to wean a patient from APRV. This is still in the experimental stage.

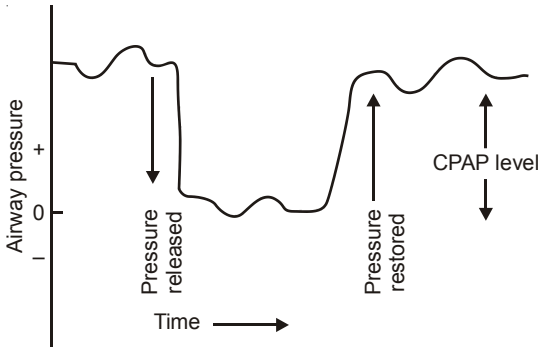


Fig. 70.2: Airway pressure release ventilation

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71 High Frequency Positive Pressure Ventilation

INTRODUCTION AND HISTORICAL PERSPECTIVE

High frequency positive pressure ventilation (HFPPV) constitutes one of the three modalities of high frequency ventilation, the others being high frequency jet ventilation (HFJV) or high frequency flow interruption (HFFI) and high frequency oscillatory ventilation (HFOV).

Jacobs and Spoerel coined the term high frequency jet ventilation (HFJV).

During early 1970s Sjostrand and associates described high frequency positive pressure ventilation (HFPPV).

DEFINITION

HFPPV is defined as a ventilation with a frequency more than 60 breaths/minute (i.e., one breath/second) and tidal volume close to anatomical dead space.

CHARACTERISTICS

The characteristics of different modalities of high frequency ventilation may be understood with the help of the Table 71.1.

Table 71.1: Characteristics of values types of HFPPV

Characteristic	*HFPPV*	*HFJV*	*HFOV*
Rate per minute	60-80	100-200	900-3,000
Flow pattern	Square wave	Square wave	Sine wave
Gas entrainment	No	Yes	No
Flow	Turbulent	Turbulent	Turbulent/Laminar
Flow generation	Pneumatic	Solenoid Pneumatic	Piston
Expiration	Passive	Passive	Active

High frequency ventilation differs from the usual modes of ventilation (conventional mechanical ventilation) in the following ways:

- Very high frequency of the order of 60 to 3000/mt
- Small tidal volumes (equal to or less than the anatomical dead space)
- Very short inspiratory time
- Low IE ratio.

Thus, this mode of ventilation results in:
- Low peak inflation pressures
- Low mean airway pressures
- Low mean intrathoracic pressures.

So this mode of ventilation provides less airway and thoracic pressures leading to less chance of pulmonary barotrauma and less interference with the venous return.

THEORIES

Theories which facilitate gas transport in HFV are:
- Direct alveolar ventilation with tidal volumes greater than 0.8 VD. This mechanism accounts for significant amount of gas transport.
- Convective streaming—High rate of ventilation with HFV enhances convective streaming which is predominantly seen in upper airways.
- Taylor's dispersion theory—This type of enhancement of molecular dispersion mainly occurs in the lower airways where the gas velocities are low.
- Pendeluft due to differences in regional time constants. This tends to produce improvement in gas distribution which can produce a favourable reduction in intrapulmonary shunt.
- Other proposed theories are cardiogenic mixing and molecular diffusion. The contribution from each mechanism, for improvement of gas exchange is still controversial.

FEATURES

Features of high frequency ventilation are:
- It produces an increase in FRC resulting in better gas exchange without the need for PEEP.
- Minimal interference with circulation as the negative intrathoracic pressure is not abolished.
- Good reflex suppression of spontaneous respiratory activity without the need for relaxants and sedatives.
- Renal and hepatic circulations are well preserved because of maintenance of adequate circulatory volume.
- Risk of aspiration is reduced as there is constant efflux of gas.
- Less chance for pulmonary barotrauma.
- Movement of the thoracic cavity and adjoining viscera is reduced which can improve the surgeon's access to the field.

USES AND INDICATIONS

- For bronchoscopy and laryngoscopy
- During surgeries like microlaryngeal and tracheal reconstruction where an immobile surgical field is needed.
- In the management of respiratory distress syndrome (both adult and infantile).
- In extracorporeal shock wave lithotripsy to provide an immobile target (stone).

- In neurosurgeries for better maintenance of ICP, as there is no increase in intrathoracic pressure.
- In oral and maxillofacial surgeries, percutaneous transtracheal jet ventilation is preferred as there may be difficulty in securing the airway.
- For ventilating patients with bronchopleural fistula which will reduce the amount of air leak through the fistula.
- In thoracic surgery, to provide an immobile nondependent lung ventilation during one lung anaesthesia.

Relative indications are:

- Aortic aneurysm surgery
- Oesophageal surgery
- Pulmonary oedema and severe asthma.
- To assist weaning from long-term ventilation.

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72 Sterilisation of Anaesthetic Equipments

INTRODUCTION

Anaesthetic equipments can act as a carrier for infection between patients and implicated as a cause for nosocomial infection. Most of patients undergoing anaesthesia and surgery are more likely to develop respiratory infection, because of their underlying disease (their resistance may be low), and the anaesthetic agent (decreases the ciliary and mucous activity) and the surgery (may interfere with their ability to cough breath). So prevention of cross contamination is important for limiting the incidence of postoperative infection.

There are so many limitations for sterilizing anaesthetic equipments. First of all many forms of sterilization may damage the equipment. The liquid and gas sterilization may leave residue in the equipment. That can subsequently cause harm to the patient. Even though some of the items are easily sterilized, others (e.g., rubber items) not easily sterilized with present technique.

Furthermore, sterilization is very costly and requires additional manpower.

Even though there are so many limitations or sterilization, routine sterilization of equipment which come in close proximity to the patient is essential after every use. Sterilization of all equipments used for anaesthesia is essential whenever used in infected patient especially with virulent organisms like TB and *Pseudomonas*.

Stages of the processes of sterilization are:

- Decontamination
- Disinfection
- Sterilization

Decontamination

This consists of the physical removal of infected matter and can be referred to as a thorough washing or scrubbing. The processes of decontamination include prerinsing, preparing the equipment, soaking, removal of soil, rinsing and drying. Some detergents are particularly useful to facilitate the process of decontamination.

Decontamination will only reduce the bacterial count, but it is equally important. Unless an article is decontaminated the disinfection or sterilization may not do well.

Decontamination devices are now available that include automatic washing machine and ultrasonic washes.

Disinfection

Disinfection implies the removal or killing of most or all infective organisms except spores. This is regarded as enough and suitable for many purposes. Disinfection can be done by physical or chemical methods.

- *Pasteurization:* This consists of heating the article to a temperature of 70° C for 20 min or 80° C for 10 min. This is most conveniently done in a water bath or in a low pressure autoclave. It is not so efficient as boiling. This is adequate for perishable article, where absolute sterility is not necessarily required.
- *Boiling*: The processes should be timed, from the point when the water return to the boiling point after the introduction of last item to be treated. This is satisfactory for any article made entirely of metal and also for those made up of rubber.
- *Chemical disinfection*
  - *Formaldehyde vapour:* Produced from paraformaldehyde tablets or neutralization of formalin solution.
  - *Alcohol* can use in the form of 70%. Ethyl alcohol or 70% isopropyl alcohol.
  - *Chlorhexidine (Hibitane)*
    i. Nondetergent chemical disinfectant
    ii. 0.05% solution in water can be used, but article should be soaked for 1 hr.
    iii. 0.5% solution in 70% alcohol can be used for quicker sterilization.
  - *Cidex (Gluteraldehyde)*
    i. It is to be mixed with an activator before use.
    ii. Very high bacteriological range and is active against various spores.
    iii. The solution may be kept in use for up to 14 days.
    iv. Expensive
    v. Popular for disinfection of endoscopic instruments.
  - Mists or fogs of sterilising agent can be used for equipment such as ventilators. The solution used for this are:
    i. 70% alcohol or stericol
    ii. Mixture of dimethyl phenol in alcoholic/aminodetergent base and 18%, phenol or H_2O_2
  - Fumigation cabinet are also used as another system for sterilizing anaesthetic equipments and ventilators.

Sterilisation

Sterilisation infers the killing of all organisms including spores and viruses. Sterilization can be done by physical or chemical methods.

AUTOCLAVING

Most effective method of sterilization. The articles to be treated are placed in a chamber with a close fitting door and steam is applied to the chamber. The time pressure and temperature recommendation are as follows:

Time	*Pressure*	*Temperature*
30 min	15 lb/m^2	122°C
10 min	20 lb/m^2	126°C
3 min	30 lb/m^2	134°C

The article suitable for sterilization by dry heating e.g. glass syringes, may be wrapped in special Craft paper and then placed in a thermostatically controlled hot air oven at 150 to 170°C of 20 to 30 minutes.

GAMMA IRRADIATION

It requires large expensive and sophisticated plant, which is appropriate for sterilization of large quantities of disposable goods.

ETHYLENE OXIDE OR PROPYLENE OXIDE GAS

For this, special sterilizer is required. This consists of a chamber resembling an autoclave in which both humidity and temperature are controlled. Ethylene oxide is very inflammable and is used as a 5-10% mixture with a gas such as Arcton-12 to prevent explosion risk. After sterilization, a period of 5-7 days must elapse before the gas is entirely eliminated, from rubber and plastic.

RECOMMENDATIONS FOR STERILISATION OF ANAESTHETIC EQUIPMENTS

Anaesthetic Machine

External surface of anaesthetic machine should be cleaned with solution containing soap or 70% alcohol. Disinfection can be done with 0.5% chlorhexidine in 70% alcohol.

Breathing attachment should be removed and the decontamination of corrugated hose, rebreathing bag expiratory valve, elbow, catheter mounds etc. can be done with hot soapy water, and rinsed, and hung upto dry. Disinfection can be done by soaking in 0.1%. chlorhexidine solution in water for 1 hour. Alternatively, these can be autoclaved, but too frequent autoclaving should be avoided.

Circle Absorbers

- Corrugated tubing and rebreathing bag can be sterilized as described above.
- Unidirectional valve may be unscrewed decontaminated and disinfection can be done with 70% alcohol or 0.5% chlorhexidine solution.
- Soda lime canister should be cleaned well and disinfection done with 70% alcohol.

Ventilators

The tubing and reservoir bag should be removed and treated on the same manner described above.

The exterior of ventilator should be swabbed and cleaned with an antiseptic solution, like 70% alcohol in chlorhexidine.

Face Masks

Immediately after use face masks should be dropped into a bucket containing soap solution. At the end of everydays work, the face masks should be taken out, thoroughly washed in a hot soapy solution, rinsed in hot water and hung up to dry. It should not be autoclaved.

Endotracheal Tube and Airway

Should be dropped into a bucket of soap solution immediately after use. They should later be cleaned out with a long narrow brush, specially made for this purpose thoroughly rinsed. Then it is either autoclaved or boiled for 3 minutes. Those made with latex rubber should be boiled carefully.

Laryngoscope Blade and Other Metal Accessaries

Preferably autoclaved especially after use in infected cases. Alternative is soaking in 0.5% chlorhexidine in 70% alcohol solution. The laryngoscope handle should be swabed with alcohol or chlorhexidine.

Special Precautions for Infected Cases

Use disposable equipments whenever possible. All reusable equipments should be sterilized in the most effective way described already.

Storage of Sterile Objects

Face masks and other parts of breathing attachments after drying can be stored in a dust free cupboard or drawer. Endotracheal tube and airway may be stored together in a sterile tray and kept covered with a cloth or polythene sheeting.

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73 Electrical Hazards in Operation Theatres

The electrical hazards in the operation theatre may happen either from (1) Normal or (2) faulty electric currents.

FROM NORMAL ELECTRIC CURRENTS

Static Electricity

Static charges may be produced by friction, movement and induction. Large accumulation of static electricity occurs in the theatre and this may cause explosions, if particular care is not taken. The trolleys when moved in the theatre can acquire dangerous static charges. This can be reduced by wheels fitted with conducting rubber tyres. Friction may be produced by woollen blankets when they are shaken and drawn over a trolley, or may be induced on equipment by the presence of certain apparatus in the theatre. Here, there is no contact between the source of current and the article on which the current is induced.

Materials like Nylon which acquire static charges should be treated with antistatic wax. Plastic aprons and woollen garments should not be worn by theatre staff, and if at all necessary, should be rendered antistatic. Conducting footwear should be worn by everyone in the theatre involved in anaesthesia, woollen blankets should not be brought to the theatre, and should be replaced by that of cotton.

The explosion risk comprises to an area of lesser than 25 cm round the anaesthetic equipment. The prime precaution is to see that all breathing tubes and masks are antistatic. All nonessential articles in the theatre which readily acquire static charges are to be removed and earthing should be provided to all equipments close to the anaesthesia circuit. This is to ensure that there is a resistance of not more than 100 mega Ohms and not less than 50,000 ohms between each person or article in the theatre and earth.

Floors of the theatre should be conducting, as that of the items resting on it. This is achieved by Terrazo or concrete flooring with a resistance of 0.1 to 10 mega ohms. Alternatively conducting rubber or plastic sheet may be spread on the floor; or temporarily a moistened sheet will do. When the antistatic properties of rubber tubings are in doubt, they can be moistened by 1% solution of a wetting agent. Rubber tubings are made antistatic by the addition of carbon to the rubber 'mix'. It owes the conductivity due to the closeness of carbon particles and therefore are susceptible to wear and tear, reducing conductivity. Therefore, all antistatic rubber should be tested once in a year. In doubtful cases the rubber material should be kept moist. The instruments used to measure conductivity is 500 volt DC insulation tester. Two leads are applied to different sites on the testing material and the handle of the instrument is rotated and deflection noted.

A high relative humidity reduces the risk of static explosion and articles are to be exposed for more than 10 minutes for moisture to collect on their surface for this purpose. When full air conditioner works, a relative humidity over 70% is quite comfortable provided that the temperature does not rise above 20 to 21° C.

Diathermy

This is a radiofrequency power oscillation with the patient in the circuit, producing intense heat at the small electrode used by the surgeon in the wound, and producing slight warmth on the earth plate.

The apparatus is used in the theatre quite frequently and can cause explosion, burns or electrocution or may interfere with the monitors and on cardiac pace makers. The sparks produced may ignite volatile gases. Spark-gap sets give 350 KHz (coagulation) and 450 KHz (cutting). Valve and transistor sets produce bursts of mega hertz oscillations at verying intervals (50 Hz–50 KHz). Poor contact of the earth plate with the skin may give rise to electrical burns. A broken plate may earth the patient to the operating table and acquire burns. A sparking earth plate may ignite ether or spirit based skin preparation. Accidental placing of the diathermy electrode on the wrong part of the patient or the surgeon or assistant may cause burn.

PREVENTION OF HAZARDS DUE TO DIATHERMY

- Use a "ground free" diathermy set.
- Use a monitored plate lead on the patients, which gives an alarm when broken and it should be tested regularly.
- Place the earth plate on a large area with good blood supply and as near as possible to the operation site.
- Place ECG and other monitoring equipments as away as possible from the diathermy set. Use bipolar diathermy on patients with cardiac pace makers, and understand the type of pace maker and to treat emergency.
- The surgeon should be encouraged to use the ground pedal himself. This should have an audible warning during current flow and as little current as possible should be used.
- Encourage the return of the active electrode to its quiver, when not in use. Avoid pushing the foot pedal under the operating table.
- Diathermy should be used with caution in the neighbourhood of plastic drapes, especially when mixtures of nitrous oxide and oxygen or other anaesthetic mixtures are used, lest they ignite the plastic drapes.
- Avoid diathermy when operating on patients with cardiac pace makers.
- Grounded electric blankets should not be used on patients on diathermy machine.
- The diathermy apparatus should be regularly serviced.
- The theatre staff should be trained on precautions during the use of diathermy.

The Other Normal Electric Currents

- Sparks from motors
- Sparks from X-ray machine
- Sparks from switches, etc.

FAULTY ELECTRIC CURRENTS

The great majority of electrical hazards in the theatre are from the mains supply. This can occur due to short circuits in electrical apparatus, faulty wires and cables and breaking of bulbs. Mains electricity can be dangerous, if the live lead in a grounded systems makes contact with a grounded person (Macroshock). To prevent this, equipments working on electricity (Mains) should be enclosed in grounded metal cases and should be checked periodically. Mains frequency of 50 Hz (usual supply) can cause ventricular fibrillation. Dry skin can give a resistance of 5 mega ohms, this can be reduced to 500 ohm. with wet electrode jelly mA (milli ampere) AC or 5 mA DC gives tingling sensation on the skin, 15 mA AC or 75 mA DC causes muscle paralysis, 70 mA AC or 300 mA DC causes ventricular fibrillation, even if the flow is for less than 20 minutes. 0.5 mA current direct across the ventricles cause ventricular fibrillation. Direct current with an open circuit to the skin causes tissue destruction with punched out open sore.

Prevention

All portable electrical apparatus should be fitted with explosion proof switches and three wire flex (color coded), the earth wire being connected to the outer casing of the apparatus and to the earth point on the wall plug. The plug should have a locking device making it impossible to remove the plug while the current is switched on make sure that all electrical equipments in the theatre is periodically tested by experienced person and that proper maintenance is given from time to time and that they are of standard.

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74 Brain Death

An organism exists only when the cell aggregation is under the control of the central nervous system, endocrine system and the immune system. Death may be defined as "the cessation of life, the ceasing to exist; defined by physicians as a total stoppage of the circulation of blood, and a cessation of the animal and vital functions consequent thereon, such as respiration, pulsation, etc. —Black's Law Dictionary.

Thus, the neurological determination of death, more commonly known by the term "Brain Death" is prerequisite to declare the person dead.

The Minnesota Criteria for Brain Death can be summarised as:

- Absence of spontaneous movement.
- Absence of spontaneous respiration over a 4 minute period.
- Absence of brain reflexes as evidenced by: fixed dilated pupils; absent gag, corneal and ciliospinal reflexes, absent doll's eye movements, absent response to caloric stimulation, and absent tonic neck reflex.
- Unchanged status eye at least twelve hours.
- Responsible pathological process deemed irreparable.

Absent spinal reflexes and EEG activity are missing from this list.
Require for the Diagnosis of Brain Death are:

THE ESTABLISHMENT OF CESSATION OF ALL BRAIN FUNCTIONS

Clinical Neurological Examination

- Absent pupillary response to light
- Absent oculocephalic reflex.
- Absent corneal reflex.
- Absent oculovestibular reflex. (Caloric Test)
- Absent gag reflex.
- No reaction to deep central pain.
- Apnoea test.

Paraclinical/Laboratory Confirmatory Tests

- EEG
- Evoked Responses
- Cerebral Angiography

- Cerebral Radionuclide Angiogram
- Transcranial Doppler
- Computed Tomography
- MRI
- Position Emission Tomography
- Bispectral Index (BIS)

DEMONSTRATION OF IRREVERSIBILITY IS ESTABLISHED BY:

- Determination of the cause of loss of brain functions.
- Exclusion of Reversible conditions
- Cessation of brain functions persists for an appropriate period of observation.

Exclusions for Brain Death are:

- Hypothermia (temp < 35°C)
- Drugs (no depressant or relaxants)
- Acid/base abnormalities
- Metabolic/Endocrine disease
- Markedly elevated $paCO_2$
- Severe hypothermia.

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