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DRUGS IN ANAESTHESIOLOGY

Vipin K Dhama



4th Edition

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Contents

1. **Basics of Pharmacology** 1
2. **Skeletal Muscle Relaxant** 14
3. **Opioids** 40
4. **Volatile Anesthetics** 71
5. **Intravenous Induction Drugs** 101
6. **Inotropes** 132
7. **Anticholinergics** 153
8. **Anticholinesterases** 160
9. **Local Anesthetics** 171
10. **Nonopioid Analgesics** 206
11. **Antihypertensive Drugs** 214
12. **Antiemetics and Antacids** 228
13. **Anticoagulant and Procoagulant** 243
14. **Miscellaneous Drugs** 256
15. **Antibiotics** 275

Index *285*

4

CHAPTER

Volatile Anesthetics

INTRODUCTION

Volatile anesthetics (VAs) are different from other anesthetic drugs as they are given via respiratory route. Vapor is the gaseous state of a substance that is liquid or solid at room temperature. A special device (vaporizer) is attached to anesthetic circuit to deliver VA.

The target organ for VA is brain. To reach there, VA has to pass through anesthetic circuit, lungs, and blood. Amount of VA reaching brain affects the onset of induction of anesthesia. VA can be used as an induction agent, especially in pediatric patients. This can avoid the need of muscle relaxants during induction.

INDICATIONS FOR VOLATILE ANESTHETIC

- In operation theater (OT), for induction, maintenance of general anesthesia
- In intensive care unit (ICU), for management of bronchospasm and acute respiratory distress syndrome (ARDS), sedation in place of intravenous (IV) sedatives[1]

PHARMACOKINETICS

Volatile anesthetics pass through many tissues (alveoli, blood, and brain) and perform their clinical effects. It is important to understand the changes at each level and then read the whole pharmacokinetics of VA.

Like other drugs, pharmacokinetics deals with VA in respect to their:

- Absorption (uptake from alveoli into pulmonary capillary blood)
- Distribution
- Metabolism
- Elimination

Multiple factors are involved in determining concentration of VA at target organ (brain) as instead of going into blood directly; VA is inhaled from vaporizer to breathing circuit to lung and then into blood circulation to reach brain **(Table 1 and Flowchart 1)**.

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TABLE 1: Factors affecting volatile anesthetic (VA) concentration.

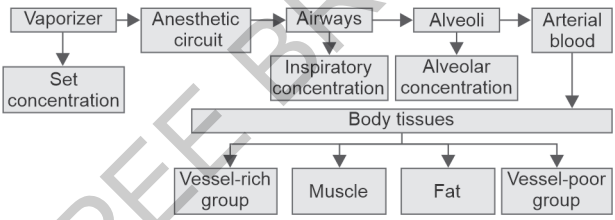
Factors affecting inspiratory concentration:
- Breathing circuit volumes
- Fresh gas flow (FGF) rate
- Absorption of the inhaled anesthetics in the rubber or plastic components of the breathing system

Factors affecting alveolar concentration:

Uptake (absorption from alveoli into blood)
- Uptake = Solubility × cardiac output × partial pressure difference (between alveolar gas and venous blood)
- Alveolar ventilation
- Concentration effect and second gas effect
- Factors affecting solubility—hematocrit and fat content of blood
- Factors affecting partial pressure difference— tissue solubility and tissue blood flow

Factors affecting arterial concentration:
- Shunts
- Dead space

Flowchart 1: Schematic path of volatile anesthetic (VA) to body tissues.



Fate of Inhaled Anesthetic

Volatile anesthetics are available in liquid form. Vaporizer converts this liquid into gas/vapor form. This vapor mixes with gases (nitrous oxide/air/oxygen) in anesthetic circuit and enters respiratory system. To understand about pharmacokinetics of VA, it will be easier if one recalls the gas laws (for ideal gases).

Partial Pressure

When a gas mixture is kept in a container, the molecules of the gas mixture exert pressure on the walls of the container. The part of the total pressure that results from any one gas in the mixture is called the partial pressure of that gas. The total pressure of the mixture is the sum of the partial pressures of the constituent gases **(Fig. 1)**. For example, total atmospheric pressure is 760 mm Hg.

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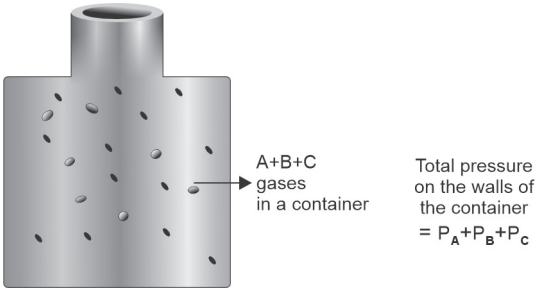

Fig. 1: Partial pressure (sum of partial pressures of individual gases).

As the amount of oxygen is 21% (out of 100 parts of air, 21 parts are of oxygen), the partial pressure of oxygen would be 21/100 × 760 = 159.6 mm Hg.

When we breathe room air, this is the partial pressure of oxygen. Similarly, we can calculate the partial pressures of other gases in atmospheric air.

If we add all the pressures of gases present in the air, the total would be 760 mm Hg.

Equilibrium

Meaning of this word is "balance". VAs are in gas form when they mix with alveoli air. From alveoli, VAs enter blood and then move to target organ (brain). When VA molecules enter into alveoli, they mix with gases present in alveoli then movement into pulmonary blood occurs. After that VA molecules reach different tissues of body via circulation through blood.

The movement of VA through different phases (alveoli air, blood, and tissue) occurs after equilibrium is achieved in different phases. Once partial pressures of VA are equal in different phases, it would mean that these phases are in a state of equilibrium.

Partition Coefficient

Partition coefficient is the ratio of the concentrations of the VA in two phases at equilibrium.

In other words, it measures relative solubilities of a VA in two phases. Blood/gas coefficient of isoflurane is 1.4 at 37°C. This means that each milliliter of blood holds 1.4 times isoflurane as alveolar gas does at this temperature.

Note that after achieving equilibrium, blood contains more isoflurane than alveolar gas. High blood gas coefficient means that agent is more soluble in blood than in gas.

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Blood/gas coefficient = Affinity of agent for blood/Affinity of agent for gas or, in simple words, Blood/gas coefficient = No. of molecules in blood/No. of molecules in alveolar air

Gases (VA) always move from high partial pressure to low partial pressure. Concentration of gases in solutions does not determine their movement. So, a solution can have higher concentration of a gas than adjacent solution, but the movement of gases will be decided by partial pressure only. Concentration can be thought of as dissolved VA (solubility) while the movement is of undissolved VA.[2]

Movement of VA across will depend on partial pressure. Concentration may be more in a compartment (alveoli/blood/brain), but it will not have any effect on movement.

Factors Affecting Inspiratory Concentration

We fill the vaporizer with VA and initiate flow of gases. Gas mixture leaving the vaporizer carries the concentration set on vaporizer, but patient's lung may receive a different concentration. It is affected by:

- Breathing circuit volumes
- Fresh gas flow (FGF) rate
- Absorption of the inhaled anesthetics in the rubber or plastic components of the breathing system. [High FGF, low-circuit volume, and low-circuit absorption reduce difference between concentration set on vaporizer and inspired concentration (FI)].

Note that concentration set at point A is not the same as delivered at point B (patient's airways). Also note that concentration delivered at point B is called as FI **(Fig. 2)**.

Factors Affecting Alveolar Concentration

Concentration of inhaled anesthetic at point B is the FI. Concentration achieved at point C is the alveolar concentration (FA) **(Fig. 3)**. The concentration of VA that a

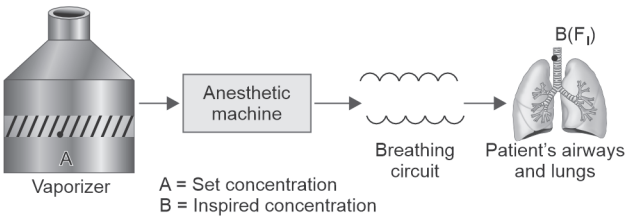

Fig. 2: Inspiratory concentration.

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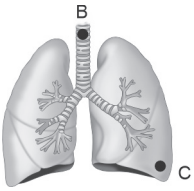

Fig. 3: Assessment point of inspired and alveolar concentration.

patient inspires need not be the same as that achieved in the alveolus. The factors governing FA of VA are:

- Uptake (absorption from alveoli into blood)
- Ventilation
- Concentration and second gas effect

Uptake: As soon as the inhaled anesthetic reaches the alveolus, it starts getting absorbed into the blood. Thus, absorption from alveoli (uptake) depends upon the solubility of the agent in the blood compared to alveolar gas, cardiac output, and partial pressure difference between alveolar gas and venous blood.

As can be seen in the formula below, there is no effect of VA concentrations in alveolar gas or blood on uptake.

Uptake = Solubility × cardiac output × partial pressure difference between alveolar gas and venous blood

Solubility

If a drug is highly soluble in blood, it will easily diffuse out of the alveolus into the pulmonary capillaries and concentration of the drug at point C (representing alveoli) will decrease **(Fig. 3)**. Thus, it will take longer time for FA to become equal to FI.

So, we can conclude that highly soluble drugs, i.e., drugs with high blood/gas coefficient takes longer time to achieve a given FA. Now delay in rise of FA means delay in achieving a definite brain tissue concentration and finally delay in induction of anesthesia. Thus, highly soluble drugs will take longer time for induction.

Solubility means VA is mixed in blood. High solubility means more VA will dissolve before partial pressure rises. This will lead to more time for transfer from blood. So, induction will be slow.

Blood: Gas partition coefficient means solubility of VA in blood in comparison to alveolar gas. This coefficient is affected by hematocrit and fat content of blood.

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Hematocrit: The higher the hematocrit, the higher will be the solubility. That is why anemic patients show a more rapid induction of anesthesia. The decreased solubility in blood in anemic patients reflects the decrease in lipid-dissolving sites normally present on erythrocytes. As a result, FA rises faster in anemic patients leading to faster induction.

Fat contents of the blood: Fat acts as a large reservoir for inhalational anesthetics. Increasing the fat content of the blood, e.g., postprandial lipidemia results in increased solubility of the drug in blood. Final impact is modest slowing of rate of induction.

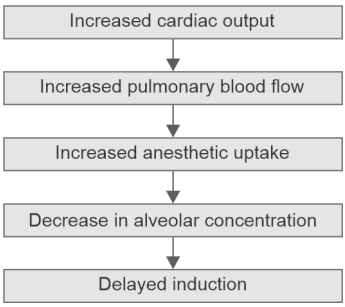
Cardiac Output

Increased cardiac output leads to increase in pulmonary blood flow per unit time. Hence, more VA will be removed from alveoli leading to decrease in VA alveolar concentration. As VA alveolar concentration determines speed of induction of anesthesia, induction will be delayed with increase in cardiac output **(Flowchart 2)**. On other hand, decrease in cardiac output will lead to increase in alveolar concentration of VA. As VA cause myocardial depression, decrease in cardiac output will further increase speed of induction. Remember that change in cardiac output affects soluble agents more than insoluble agents. In clinical practice, there is not much effect of cardiac output on VA induction in healthy patients.

Volatile anesthetics that depress cardiac output can exert a positive feedback response in this regard **(Flowchart 3)**.

In clinical practice, there is not much effect of cardiac output on VA induction. At the start of induction, concentration of agent in body tissues is nil. After dissolving in blood, agent diffuses into tissues. This transfer also depends on three factors: Tissue solubility, tissue blood

Flowchart 2: Cardiac output and volatile anesthetic (VA) induction.



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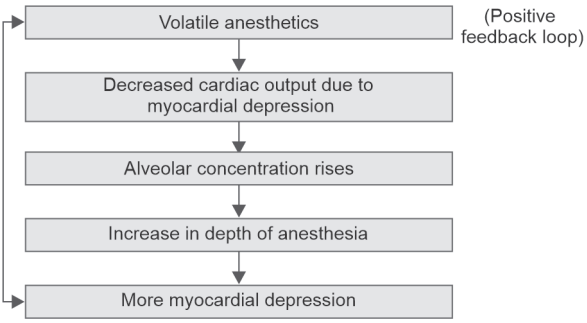
Flowchart 3: Myocardial depression and volatile anesthetic (VA) induction.



flow, and partial pressure difference between arterial blood and tissues.

Tissues are divided into four groups, depending on the blood flow:

1. Vessel-rich group (brain, heart, kidney, and endocrine organ)
2. Muscles
3. Fat
4. Vessel-poor group (bone, ligament, teeth, and cartilage)

Out of these four groups, vessel-rich group gets the largest share of cardiac output (around 75%) and that is why a large amount of VA is delivered to these tissues.

They achieve a rapid equilibrium with arterial blood (approximately 8 minutes). Uptake by vessel-rich group is minimal, so it does not influence the FA. Muscle group has lower perfusion in relation to tissue mass, so equilibrium takes place after 2–4 hours of induction depending upon tissue/blood partition coefficient. Once equilibrium with muscle is complete, only fat continues to store VA. Fat has higher affinity for VA than muscles. It takes days to fill. Absence of significant blood flow to vessel-poor group means that these tissues do not take part in uptake process.

Alveolar Ventilation

Increasing the ventilation means increasing the quantity of VA being deposited in the alveoli. The net effect is a more rapid rate of increase in FA toward the FI. Thus, there is faster induction of anesthesia. Volatile agents depress ventilation and thus sets in a negative feedback loop in this regard **(Flowchart 4)**.

Another point of concern is that increasing the minute ventilation will cause CO_2 washout and thus lead to

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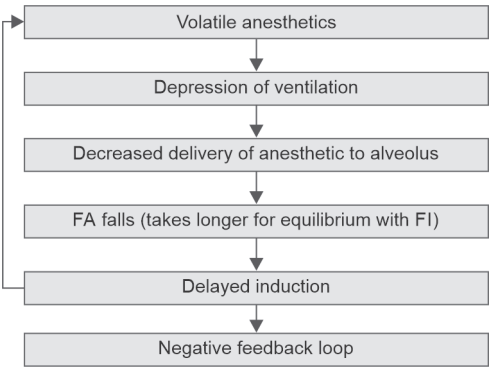
Flowchart 4: Ventilation and VA induction.



(FA: alveolar concentration; FI: inspired concentration; VA: volatile anesthetic)

hypocapnia, which, if significant, will lead to cerebral vasoconstriction and decreased delivery of anesthetic to the brain. Ventilation affects the FA of soluble agents more than insoluble agents.

Functional Residual Capacity

A larger functional residual capacity (FRC) dilutes the FI of gas resulting initially in a lower alveolar partial pressure and therefore slower onset of anesthesia.

Concentration Effect

It means that increasing the FI of an anesthetic agent increases its rate of rise of alveolar concentration (FA/FI). In other words, the greater the FI, the more rapidly FA approaches FI. It is caused by two factors:

1. Concentrating effect
2. Augmentation of tracheal inflow

This effect is more significant with nitrous oxide as it can be used in much higher concentration. Nitrous oxide is more soluble in blood than nitrogen. When a patient is given an anesthetic mixture containing N_2O, some part of nitrous oxide is absorbed in the pulmonary vasculature. As a result, total volume of gas in the alveolus diminishes and fractional concentration of VA increases.

Augmentation of tracheal inflow: Loss of alveolar total gas volume due to absorption (uptake) of nitrous oxide will cause more anesthetic mixture to be filled in from the airways into the alveolus. This will cause further rise in FA of VA.

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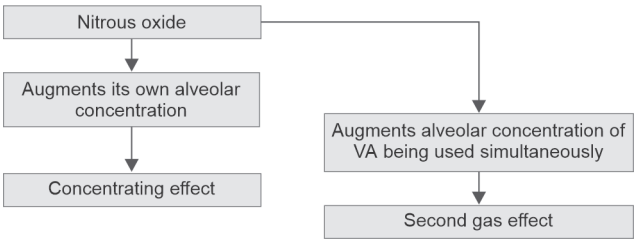
Flowchart 5: Concentration effect.



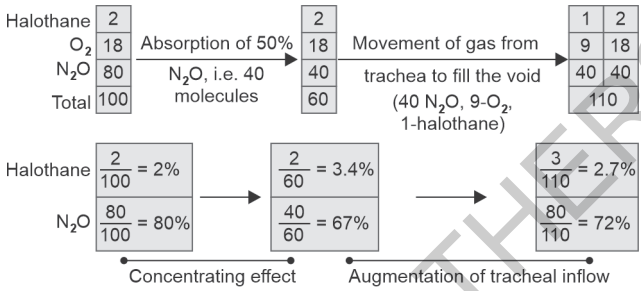


Fig. 4: Percentage changes due to concentration effect.

Second Gas Effect

Increasing the concentration of nitrous oxide augments not only its own uptake but also of VA being given along with nitrous oxide. This is called second gas effect **(Flowchart 5)**.[3]

Suppose the anesthetic gas mixture contains 2% second gas (2 molecules), 18% oxygen (18 molecules), and 80% N_2O (80 molecules).

Alveolar concentration of anesthetic mixture is...

- 2%—second gas (VA)
- 18%—O_2
- 80%—N_2O

As seen in **Figure 4**, concentration of second gas (VA) changes from 2% to 3.4% after absorption of N_2O.

This is called as second gas effect. After absorption of 40 molecules of N_2O, its concentration does not decrease by 50% but comes to 67% from 80% (concentrating effect). Due to tracheal flow, it again increases to 72%.

Factors Affecting Arterial Concentration

Shunts: A right-to-left shunt causes venous blood to mix with arterial blood without being exposed to VA in the alveoli. This dilutional effect of right-to-left shunt causes

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decrease in partial pressure of VA in arterial blood. So, we conclude that rate of induction of anesthesia is slowed with right-to-left shunt.

A left-to-right shunt has exactly the opposite effect. It causes re-exposure of the arterial blood to alveolar ventilation and VA. As a result, partial pressure of VA in the blood rises. These shunts have little clinical impact.

Dead space: Increase in dead space increases the difference between alveolar partial pressure of VA and the partial pressure of VA in the arterial blood. [Dead space is the area which is ventilated but not perfused].

Rate of induction is not affected provided minute ventilation remains the same. The rate of induction is more rapid in infants and children than adults. This may be due to a larger ratio of alveolar ventilation to FRC, increased cardiac output, and greater delivery of cardiac output to vessel-rich tissues.

Factors Affecting Recovery from Inhalational Anesthesia

Solubility and duration of anesthesia: Prolonged duration of anesthesia will hinder recovery from anesthesia due to soluble agents (e.g., isoflurane and halothane).

Duration of anesthesia will have little effect on recovery if less soluble agents are used (e.g., sevoflurane and desflurane). The reason being soluble agents show higher uptake by the body, thus filing the fat and muscle reservoirs. The more the amount of drug stored in the reservoir, the longer the duration required to empty them. Thus, prolonged anesthesia will prolong recovery from soluble agents such as isoflurane unlike desflurane.

Obese patients have a larger fat compartment. So, prolonged emergence time will occur due to the high absorption and slow release of anesthetic agents from fat tissue. Emergence time from VA with lower fat tissue solubility (e.g., sevoflurane, desflurane, and xenon) will not be much affected.

Removal of anesthetic agent from the body is via exhalation, metabolism, and transcutaneous loss. Therefore, metabolism also contributes to removal of anesthetic agent from the body along with alveolar ventilation and hastens recovery. However, metabolism plays an important role with halothane and methoxyflurane only. Both these drugs undergo extensive metabolism in liver. This is in contrast

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to rate of induction of anesthesia, which is not influenced by metabolism even for drugs such as halothane and methoxyflurane.

The basic difference between induction and recovery is that we can increase the speed of induction by increasing the FI of inhaled anesthetic and thus overcome the effect of solubility, but rate of recovery cannot be increased as FI cannot become less than zero. Once the drug is inside the body, it will take its own time to come out.

Another point is that, at the beginning of induction, all the tissues have same VA partial pressure, i.e., zero; while during the recovery, partial pressures are variable in different tissues. At the beginning of recovery, vessel-rich group has partial pressure in equilibrium with alveolar partial pressure. Anesthetic partial pressure of inhaled anesthetic in muscles equilibrates with alveolar partial pressure only after 2–4 hours of anesthesia. Fat continues to take up anesthetic unless the alveolar partial pressure falls below partial pressure in fat. Thus, muscle and fat act as reservoirs of anesthetic agent. The more the anesthetic stored in them, the more the time will be required for elimination. In clinical practice, differences in recovery time observed between the VA in postoperative ward are not much different.[4]

Mechanism of Action of Inhalational Agents

All the currently used VAs are halogenated hydrocarbons. Many theories have been proposed to explain the mechanism of action of inhalational agents, but exact site of action is still not clear **(Table 2)**.

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TABLE 2: Receptor volatile anesthetic interaction.

Target	*Binding site*	*Effect*
Gamma-aminobutyric acid A (GABA-A) receptor	A specific binding site at the GABA-A receptor is suggested	Receptor response ↑ Inhibitory postsynaptic potential (IPSP)↑
Nicotinic acetylcholine receptor	Several binding sites	Receptor response ↓ Action potential ↓
TREK-1	Disruption of the cell membrane and release of lipid raft fragments	Lipid raft fragments → Activation of phospholipase D2 → Activation of TREK-1

Unitary hypothesis: According to this theory, all VAs have a common mechanism of action, which is probably by interaction with a specific molecular structure in central nervous system (CNS). The property that correlates most with anesthetic potency is lipid solubility.

Therefore, binding site of inhaled anesthetic should be lipophilic/hydrophobic. This theory is supported by Meyer and Overton rule, which states that product of anesthetizing partial pressure and lipid solubility as measured by oil gas partition coefficient is constant for all VAs. If a VA has high lipid solubility, then less partial pressure will be required to produce a specific clinical effect.[5]

It has been proposed that anesthesia occurs when specific number of VA molecules attach themselves to a specific hydrophobic site in brain. Note that number of molecules is important, not the type of molecule. So, it does not matter whether isoflurane or sevoflurane is used. Only the number of molecules is important for clinical effect [increase in minimum alveolar concentration (MAC) will lead to increase in clinical effect].

$$\text{VA} + \text{receptor site} \rightarrow \text{clinical effect}$$

As the number of receptor sites occupied by VA increases, intensity of effect also increases.

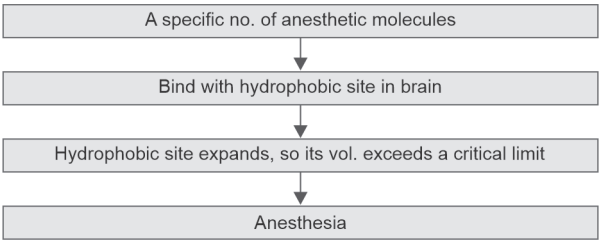
Exception to this theory: Enflurane and isoflurane are isomers with same lipid solubility but different potencies.

Certain lipid-soluble compounds are convulsants rather than being anesthetic agents.

Many volatile lipid-soluble polyhalogenated agents lack anesthetic property.

Volume expansion hypothesis: Absorption of VA molecules could expand the volume of a hydrophobic area in cell membrane and distort the ion channels required for neural transmission. As anesthesia can be reversed by compressing the expanded hydrophobic regions in experimental studies, volume expansion theory gains some credibility **(Flowchart 6)**.

Flowchart 6: Volume expansion theory.



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Decreasing the temperature should increase the anesthetic requirement, while exactly the reverse is true. MAC reduces in hypothermia.

Minimum Alveolar Concentration

The alveolar concentration of VA that prevents movement in 50% patients in response to a standardized stimulus is MAC of that agent. Standardized stimulus in human is initial surgical skin incision. MAC concept describes a concentration versus response relationship. MAC remains the standard index for comparison of VA potency.[6]

Minimum alveolar concentration intubation: MAC of anesthetic agent that allows intubation without movement or coughing by the patient.

Minimum alveolar concentration bar: MAC of anesthetic that inhibits increase in concentration of catecholamine levels in venous blood in response to skin incision.

Minimum alveolar concentration intubation > MAC skin incision > MAC laryngoscopy.

Minimum alveolar concentration is not a reliable indicator of hypnosis or unconsciousness. Modern perioperative management incorporates various types of drugs, complicating the interpretation of MAC. The use of neuromuscular blockade to produce paralysis renders the concentration of inhaled anesthetic required to suppress movement uninformative.

Advantages of Minimum Alveolar Concentration Concept

Unlike blood concentration measurement of IV drugs, brain concentration cant be measured for VA. After equilibrium, alveolar level will correspond to level in brain, so MAC can be used to assess brain levels of VA.

Potencies of different VAs can be compared. We can know the clinical effects of different VAs and compare the amount of VA needed for a particular clinical effect. MAC produces a standard for experimental studies.

Minimum alveolar concentration is roughly additive, e.g., 0.5 MAC halothane + 0.5 MAC enflurane = Same CNS depression as 1.0 MAC isoflurane. However, effect on other organs is different, e.g., 0.5 MAC halothane causes more myocardial depression than 0.5 MAC isoflurane.

Roughly, 1.3 MAC of any anesthetic (e.g., for isoflurane 1.3 × 1.2 = 1.56%) prevents movement in 95% of patients (approximation of ED_{95} of other drugs).

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TABLE 3: Factors affecting MAC.

Effect on MAC	*Factors*
Decrease	Hypo-/hyperthermia, acute alcohol intoxication, hypoxia (PaO_2 < 40 mm Hg), hyponatremia, anesthetic drugs (opioids/BZDs/barbiturates/ketamine/LA except cocaine), and old age
Increase	Chronic alcoholism, MAC is maximum at 6 months of age then decreases
No change	Gender of the patient, blood pressure, hyper-/hypocarbia, potassium concentration, and duration of anesthesia

(BZDs: benzodiazepine; LA: local anesthetic; MAC: minimum alveolar concentration; PaO_2: partial pressure of arterial oxygen)

TABLE 4: Minimum alveolar concentration (MAC) values.

	0–1 month	*1–12 months*	*Adult*
Halothane	0.87	1.2	0.75
Isoflurane	1.6	1.8	1.2
Sevoflurane	3.3	2.6	2.0
Desflurane	9.1	8.7	6.0

Factors Affecting Minimum Alveolar Concentration

Minimum alveolar concentration in pregnancy decreases progressively (at term by up to 40%). It returns to normal within a week after delivery. Changes in progesterone and endogenous opiate levels during pregnancy have been implicated. MAC is reduced by 4% per decade of age over 40 years. Maximum MAC is at 1–6 months of age **(Tables 3 and 4)**.[7]

PHARMACODYNAMICS (TABLES 5 TO 8)

Compound A-induced Nephrotoxicity

Sevoflurane is degraded by CO_2 absorbent (soda lime/baralyme) into compound A ($CH_2FOC(CF_3)=CF_2$) and compound B. Out of these, compound A is the major degradation product and is nephrotoxic. Factors increasing production of compound A are low flow, fresh absorbent, high absorbent temperature, use of baralyme, and high sevoflurane concentration. FGF < 2 L/min should be avoided. Isoflurane can be preferred in patients with elevated renal risk.[8]

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TABLE 5: Cardiovascular system effects.

	Halothane	*Isoflurane*	*Desflurane*	*Sevoflurane*
Myocardial contractility Normal heart	All agents depress contractility in normal ventricular myocardium in the following order: Halothane = Enflurane > Isoflurane = Desflurane = Sevoflurane This is due to inhibition of Ca^{2+} movement by affecting both L and T type Ca channels. Other mechanisms include inhibition of Na-Ca exchange and reduction of intracellular calcium concentration Decrease mean arterial pressure in a dose-dependent manner as concentrations of VA increases, primarily due to decreased SVR, except for halothane, which lowers mean arterial pressure by directly affecting the myocardium and reducing cardiac output			
Myocardial contractility Diseased heart	VA produces beneficial decrease in preload and afterload in patients with heart failure and coronary artery disease. Thus, during moderate LV dysfunction, despite reduction in contractility, there is simultaneous improvements in LV loading condition and relative maintenance of cardiac output. There is no effect on early ventricle filling during diastole In severe dysfunction, all VAs impair ventricular function			
SVR	Calculated SVR does not change significantly due to vasodilation in some beds and vasoconstriction in other beds. Has least effect on SVR	All these three drugs cause fall in SVR due to potent vasodilator action Cutaneous and skeletal blood flow is increased		

Contd...

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- Emphasis has been on clinically relevant features of drugs.

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