Anestesia Generale

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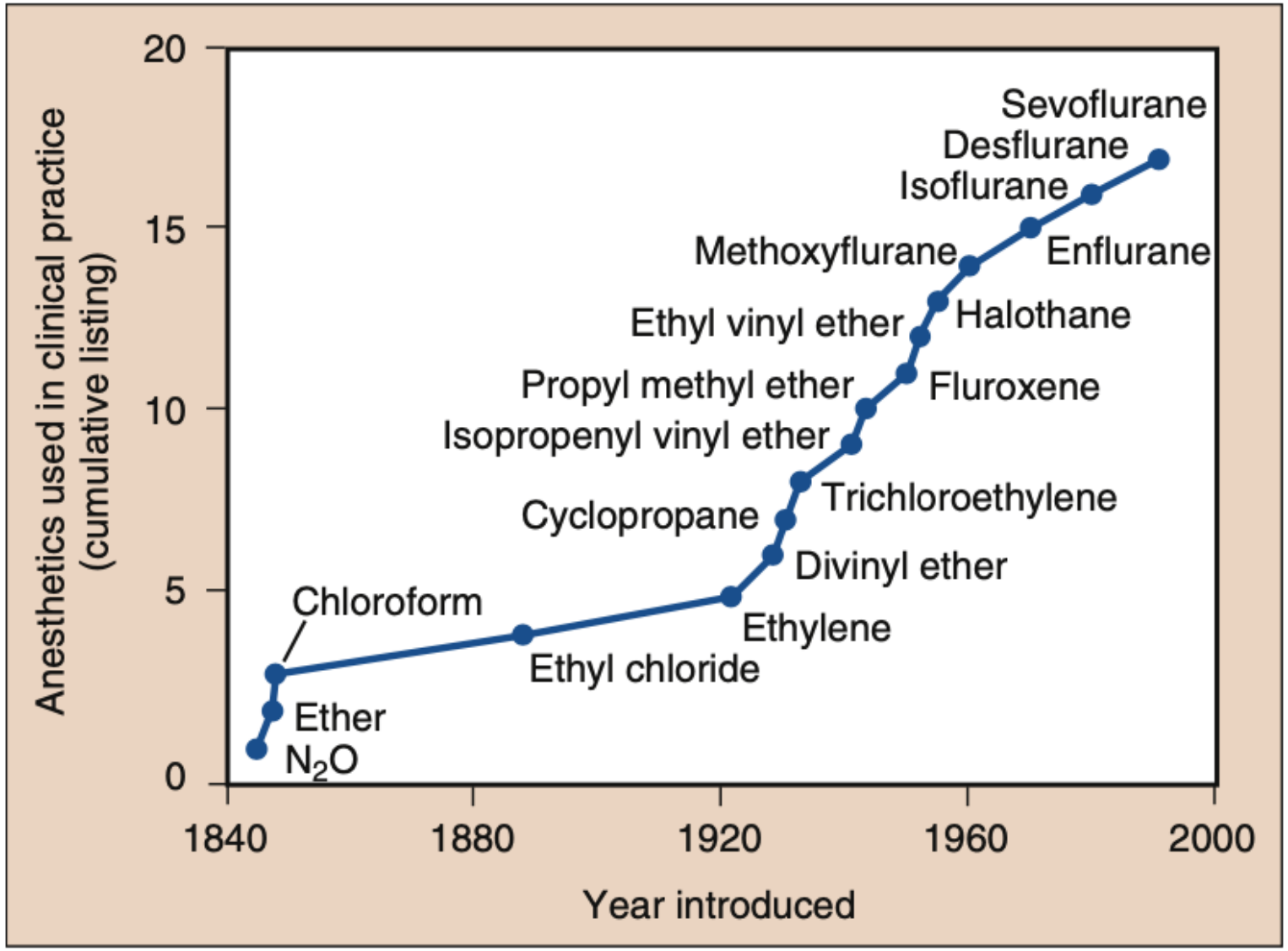
Anestesia Generale

L’anestesia generale è uno stato di soppressione della coscienza farmacologicamente indotto e reversibile, durante il quale il paziente non risponde agli stimoli, compresi quelli dolorosi.

L’anestesia generale ha come obiettivi:

1. Perdita coscienza (“ipnosi”)
2. Amnesia
3. Analgesia
4. Immobilità
5. Areflessia

Anestetici volatili



Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

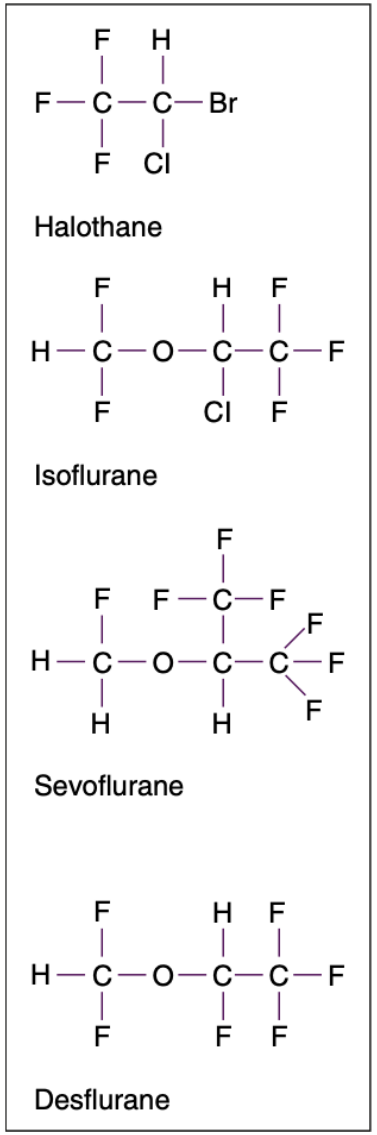
TABLE 25-1 OVERVIEW OF CANDIDATE SITES OF ANESTHETIC ACTION

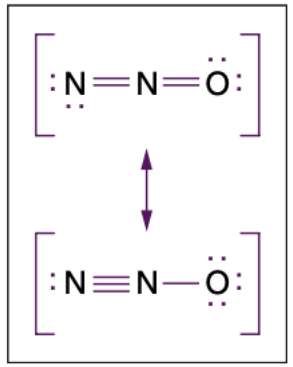
	Site	Effect	Targets
Proteins	Amphiphilic binding sites	Conformational flexibility, ligand binding	Ion channels, receptors, signaling proteins
Action potential	Nervous system	Small reduction in amplitude	Na^+ channels
	Cardiovascular system	Reduced amplitude, duration	Ca^{2+} channels, K^+ channels
Synaptic transmission			
Inhibitory	Presynaptic terminal	Enhanced transmitter release	?
	Postsynaptic receptors	Enhanced transmitter effects	Glycine, $GABA_A$ receptors
Excitatory	Presynaptic terminal	Reduced transmitter release	Na^+ channels, K_{2P} channels
	Postsynaptic receptors	Reduced transmitter effects	NMDA receptors, nicotinic acetylcholine receptors
Neuronal networks	Neuronal circuit	Altered long-term potentiation (LTP)/ long-term depression (LTD)	Synaptic plasticity
	Neuronal integration	Altered rhythmicity, coherence	HCN channels, K_{2P} channels, extrasynaptic $GABA_A$ receptors, etc.
Central nervous system	Neocortex, hippocampus, amygdala	Sedation, amnesia	Slow 1-Slow 4, δ-, α-, θ-, γ-rhythms, cross-frequency coupling
	Diencephalon (thalamus), brainstem (reticular formation)	Unconsciousness	γ-band transfer entropy?, cross-frequency coupling? cortical integration capability?
	Spinal cord	Immobility	Thalamic deafferentation? Nocifensive reflex
Cardiovascular system	Myocardium	Negative inotropy	Excitation-contraction coupling
	Conduction system	Dysrhythmias	Action potential
	Vasculature	Vasodilation	Direct and indirect vasoregulation

HCN, Hyperpolarization-activated cyclic nucleotide; *NMDA, N*-methyl-D-aspartate.

Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Cohen NH, Young WL. Miller's Anesthesia E-Book. Elsevier Health Sciences; 2014.

Anestetici volatili





La pressione totale esercitata da una miscela ideale di gas ideali è uguale alla somma delle pressioni parziali che sarebbero esercitate dai gas se fossero presenti da soli in un eguale volume.

Pressione di vapore: massima pressione parziale di un volatile, aumenta con la temperatura

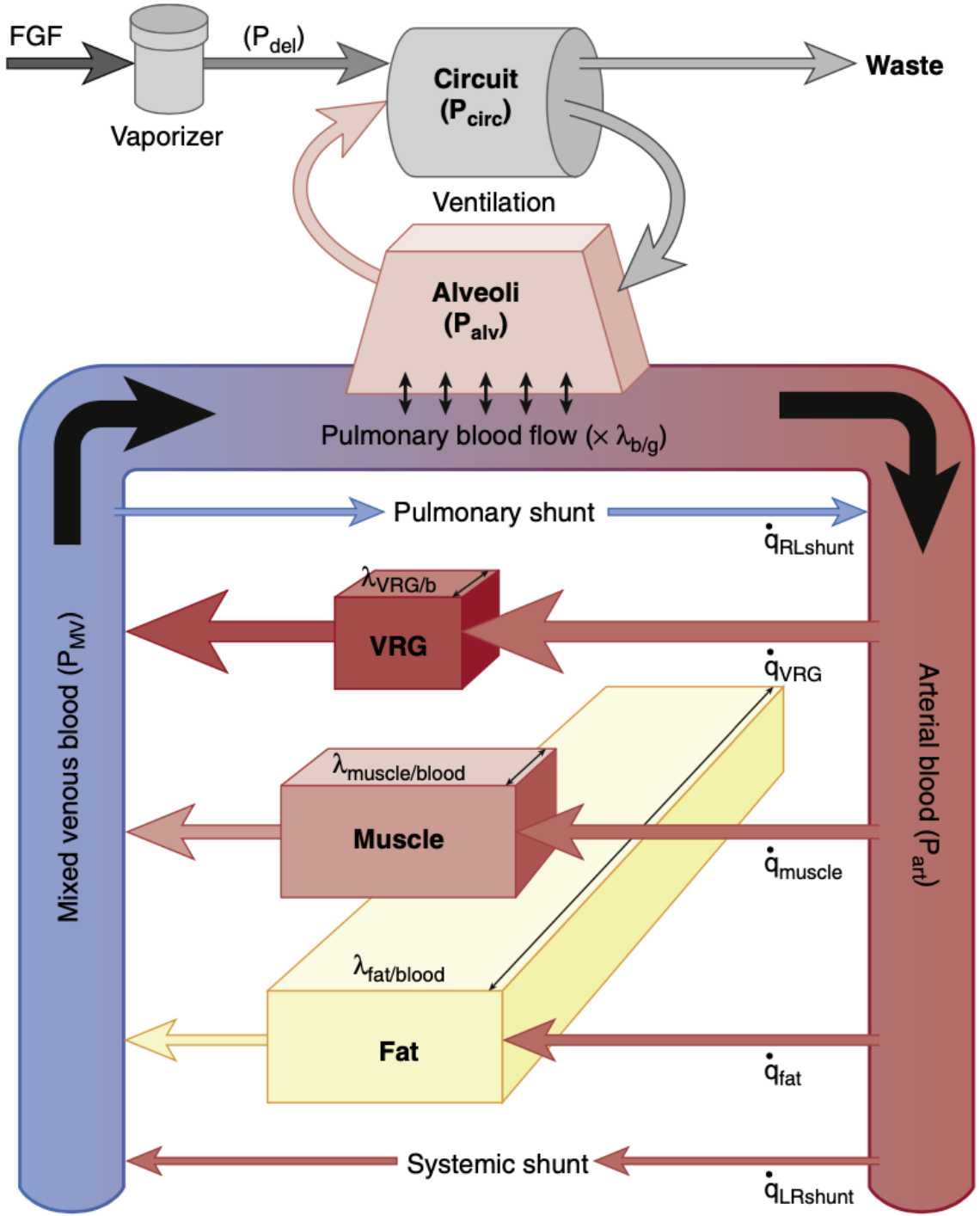
Anestetici volatili: pressione di vapore<1 e temperatura di ebollizione >20°C

Anestetici gassosi: pressione di vapore>1 e temperatura di ebollizione >20°C

Idrofobia e solubilità nei tessuti: valutati con coefficiente di ripartizione acqua/olio

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.
Gropper MA, Eriksson LI, M.D LAF, et al. Miller's Anesthesia. Elsevier; 2019.

$$dVA_{del}/dt = P_{del} \times FGF.$$



Gropper MA, Eriksson LI, M.D LAF, et al. Miller's Anesthesia. Elsevier; 2019.

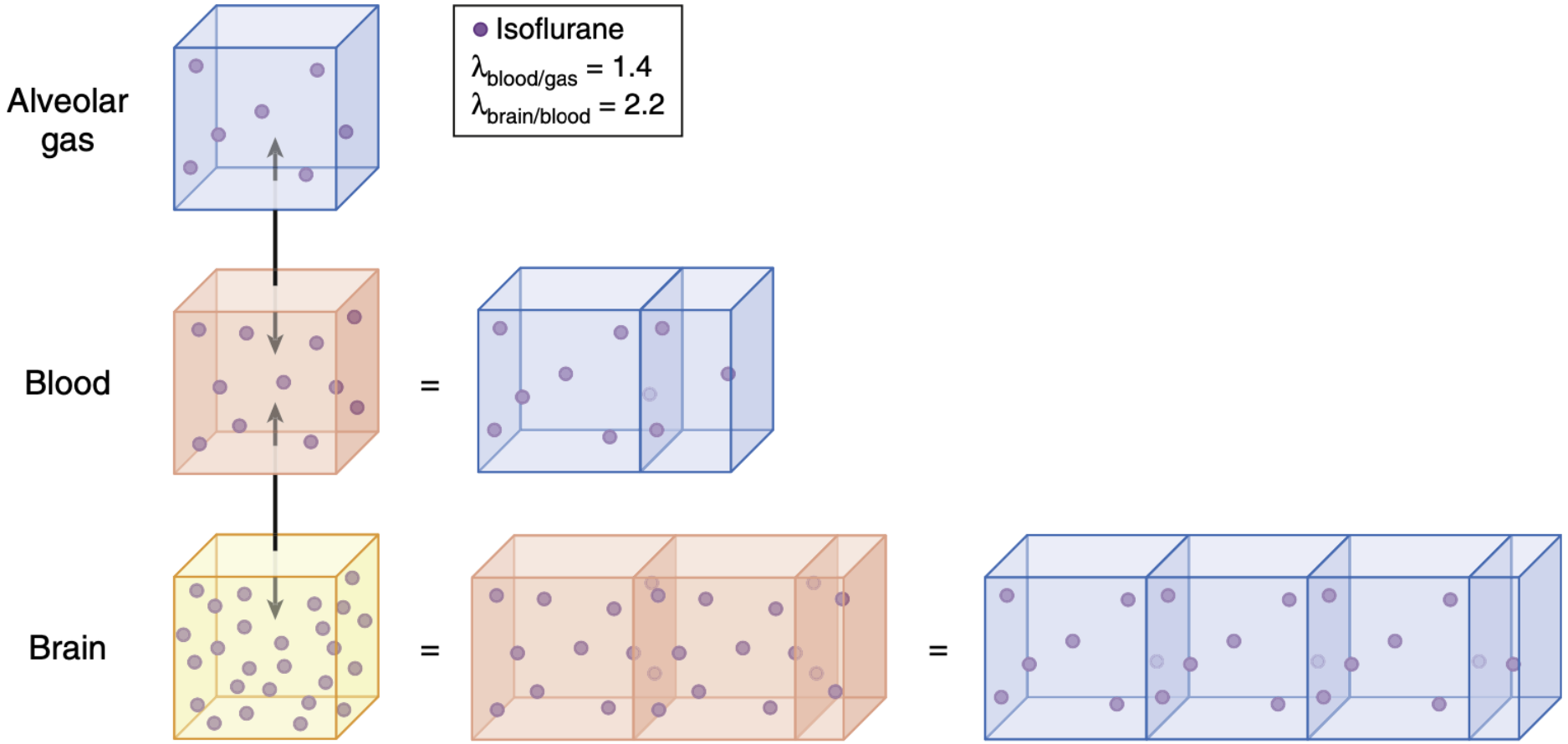


Fig. 20.1 Partitioning of anesthetic gases between different biophases. Left, Depicts the partitioning of isoflurane between gas phase *(blue)*, blood *(red)* and brain *(yellow)*. The blood:gas partition coefficient ($\lambda_{b/g}$) for isoflurane is 1.4 and the brain:blood partition coefficient ($\lambda_{CNS/blood}$) is 2.2 (see Table 20.2). At equilibrium, defined as equal isoflurane partial pressure in all compartments, a volume of blood contains 1.4-fold the quantity of isoflurane as the same volume of alveolar gas, whereas a volume of brain tissue contains 2.2-fold the quantity of isoflurane as the same volume of blood. Right, We also depict partition coefficients as effective (equivalent) volumes of another biophase. For example, 1 volume of blood contains the same amount of isoflurane as 1.4 volumes of alveolar gas, whereas 1 volume of brain contains the same amount of isoflurane as 2.2 volumes of blood or 3.1 volumes of gas.

Gropper MA, Eriksson LI, M.D LAF, et al. Miller's Anesthesia. Elsevier; 2019.

MAC

Minima concentrazione alveolare di un anestetico inalatorio a cui corrisponde l'immobilità a uno stimolo standard, i.e. incisione della cute, nel 50% dei soggetti

Factors Increasing MAC

Drugs

- Amphetamine (acute use)
- Cocaine
- Ephedrine
- Ethanol (chronic use)

Age

- Highest at age 6 months

Electrolytes

- Hypernatremia
- Hyperthermia

Red Hair

Factors Decreasing MAC

Drugs

- Propofol
- Etomidate
- Barbiturates
- Benzodiazepines
- Ketamine
- α_2-Agonists (clonidine, dexmedetomidine)
- Ethanol (acute use)
- Local anesthetics
- Opioids
- Amphetamines (chronic use)
- Lithium
- Verapamil

Age

- Elderly patients

Electrolyte Disturbance

- Hyponatremia

Other Factors

- Anemia (hemoglobin < 5 g/dL)
- Hypercarbia
- Hypothermia
- Hypoxia
- Pregnancy

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

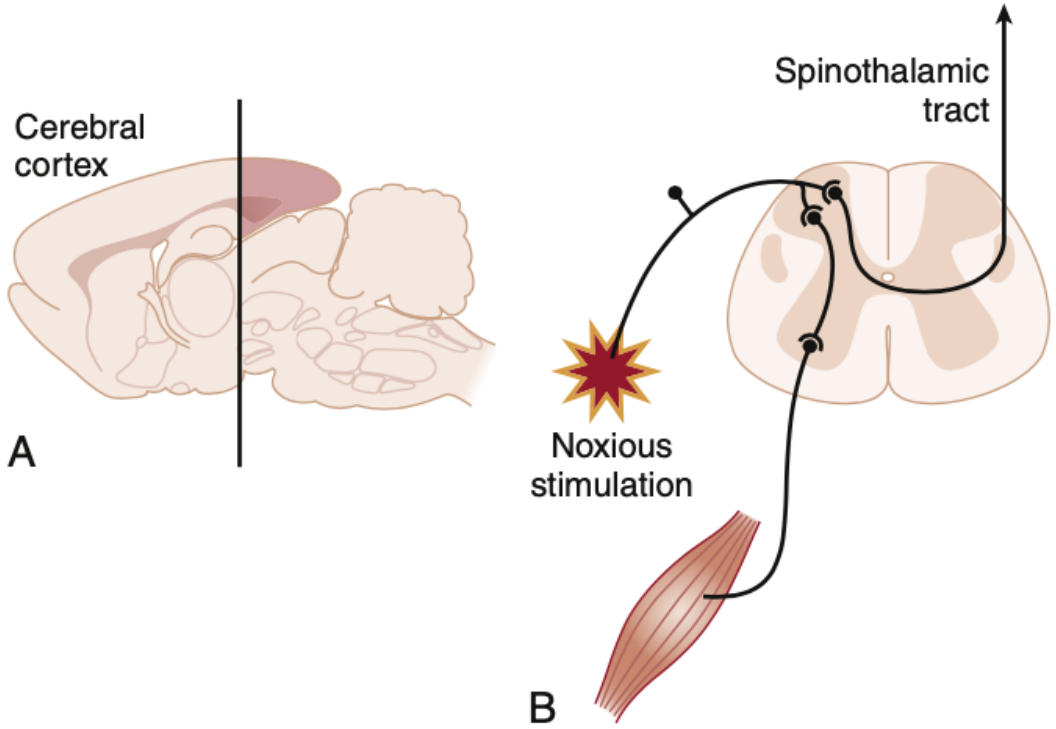


Figure 25-4. Inhaled anesthetics produce immobility at the spinal level. **A,** Decerebration by removal of the forebrain rostral to the *black line* does not alter the MAC of isoflurane in rats, indicating that volatile anesthetic immobilization does not depend on the cerebral cortex.[17,18] **B,** Anesthetics suppress the nocifensive withdrawal reflex response to noxious stimulation transmitted to the dorsal horn by sensory nerves at the spinal level. Current efforts are focused on identifying the molecular, cellular, and anatomic substrates for this effect.

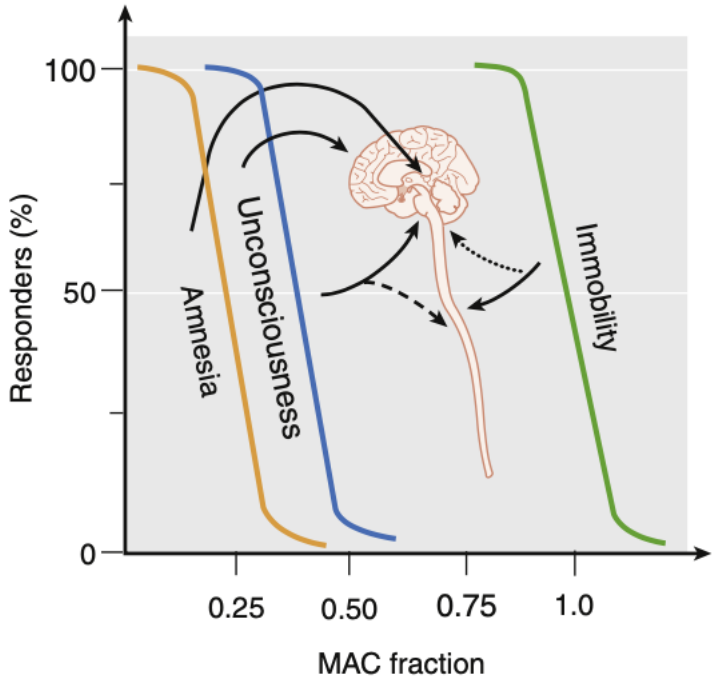


Figure 25-3. Multiple behavioral end points and sites of action underlie inhaled anesthetic action. Amnesia, the most sensitive anesthetic end point, probably involves the hippocampus, amygdala, mediotemporal lobe, and other cortical structures. Unconsciousness likely involves the cerebral cortex, thalamus, and reticular formation. Sedation and hypnosis (loss of responsiveness) are part of the consciousness-unconsciousness continuum and are not shown. Immobility occurs by anesthetic action in the spinal cord, although supraspinal effects *(dotted arrow)* are likely important for some anesthetics. Anesthetic action in the spinal cord blunts ascending impulses arising from noxious stimulation and might indirectly contribute to anesthetic-induced unconsciousness and amnesia *(dashed arrow)*. Cardiovascular responses occur at even greater MAC fractions (not shown). *(Courtesy Joseph Antognini, University of California, Davis.)*

Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Cohen NH, Young WL. Miller's Anesthesia E-Book. Elsevier Health Sciences; 2014.

Characteristic	Isoflurane	Enflurane	Halothane	Desflurane	Sevoflurane	Nitrous Oxide
Partition coefficient						
Blood-gas	1.46	1.9	2.54	0.45	0.65	0.46
Brain-blood	1.6	1.5	1.9	1.3	1.7	1.1
Muscle-blood	2.9	1.7	3.4	2.0	3.1	1.2
Fat-blood	45	36	51	27	48	2.3
MAC (age 30-55 years) % of 1 atmosphere	1.15	1.63	0.76	6.0	1.85	104
Vapor pressure at 20° C (mm Hg)	240	172	244	669	160	
Molecular weight (g)	184.5	184.5	197.4	168	200	44
Stable in hydrated CO_2 absorbent	Yes	Yes	No[a]	Yes	No[a]	Yes
Stable in dehydrated CO_2 absorbent	No		No[ab]	No[b]	No[abc]	Yes
Percent metabolized	0-0.2		15-40	0-0.2	5-8	

[a]Compound A.
[b]Carbon monoxide.
[c]Severe exothermic reactions reported.
MAC, Minimum alveolar concentration.

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

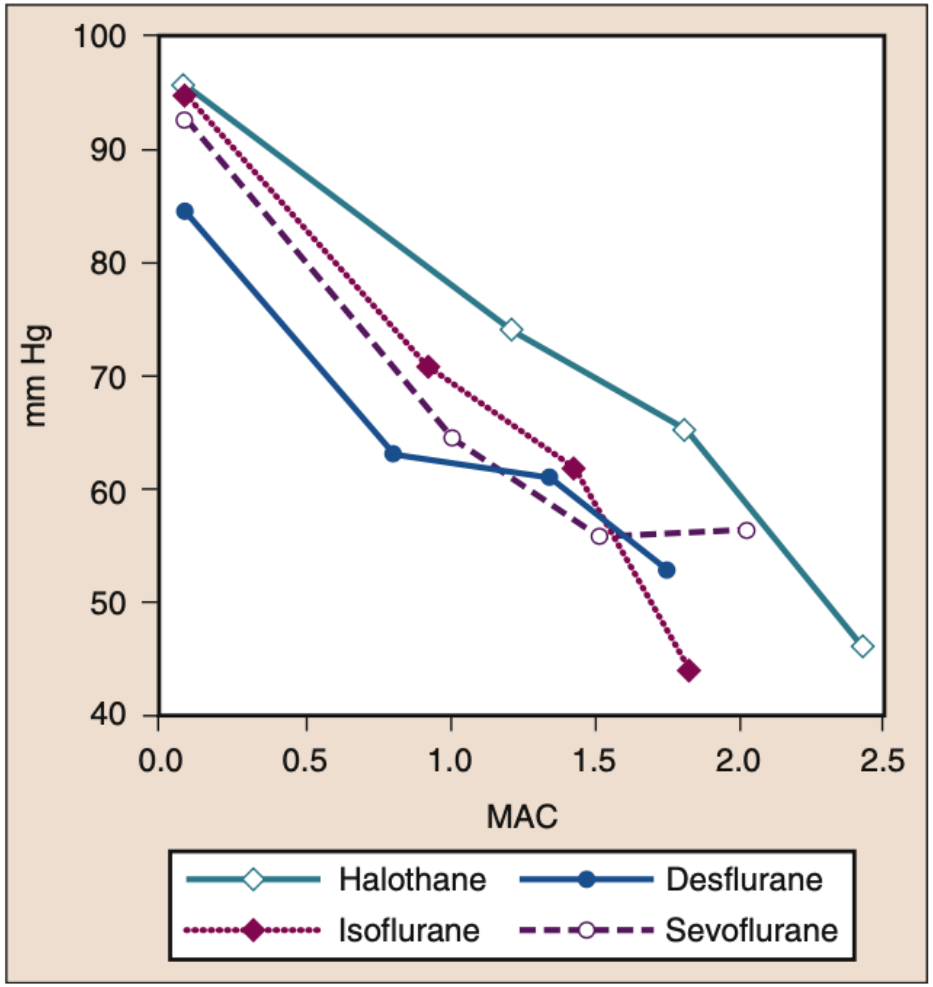


Fig. 7.8 The effects of increasing concentrations (MAC) of halothane, isoflurane, desflurane, and sevoflurane on mean arterial pressure (mm Hg) when administered to healthy volunteers. *MAC*, Minimum alveolar concentration. (From Cahalan MK. *Hemodynamic Effects of Inhaled Anesthetics. Review Courses*. Cleveland: International Anesthesia Research Society; 1996:14-18, used with permission.)

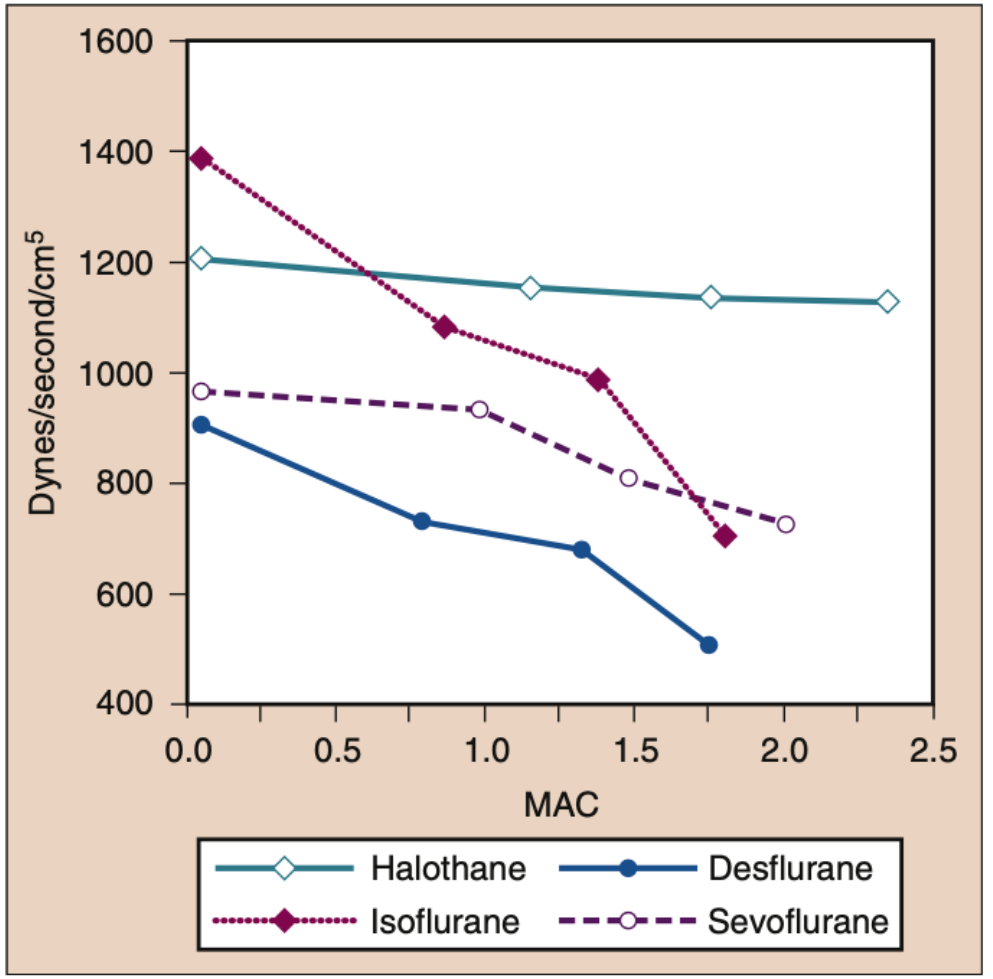


Fig. 7.9 The effects of increasing concentrations (MAC) of halothane, isoflurane, desflurane, and sevoflurane on systemic vascular resistance (dynes/sec/cm^5) when administered to healthy volunteers. *MAC*, Minimum alveolar concentration. (From Cahalan MK. *Hemodynamic Effects of Inhaled Anesthetics. Review Courses*. Cleveland: International Anesthesia Research Society; 1996:14-18, used with permission.)

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

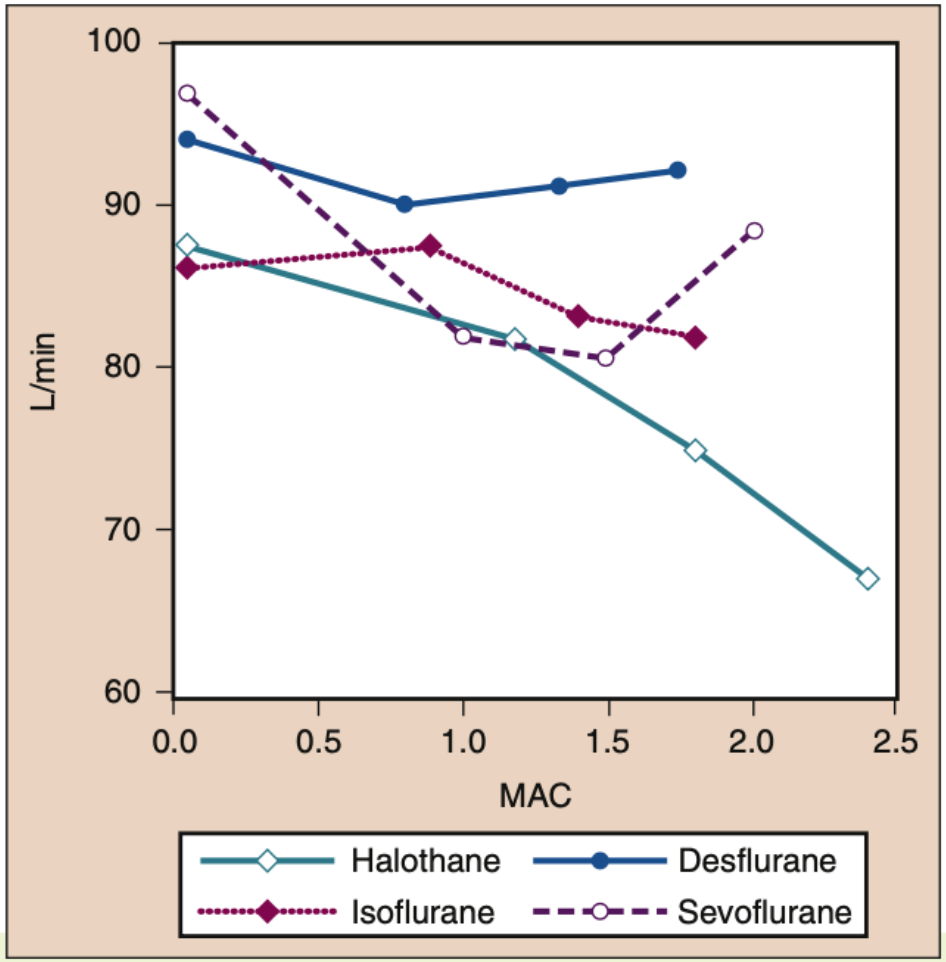


Fig. 7.10 The effects of increasing concentrations (MAC) of halothane, isoflurane, desflurane, and sevoflurane on cardiac index (L/min) when administered to healthy volunteers. *MAC*, Minimum alveolar concentration. (From Cahalan MK. *Hemodynamic Effects of Inhaled Anesthetics. Review Courses*. Cleveland: International Anesthesia Research Society; 1996:14-18, used with permission.)

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

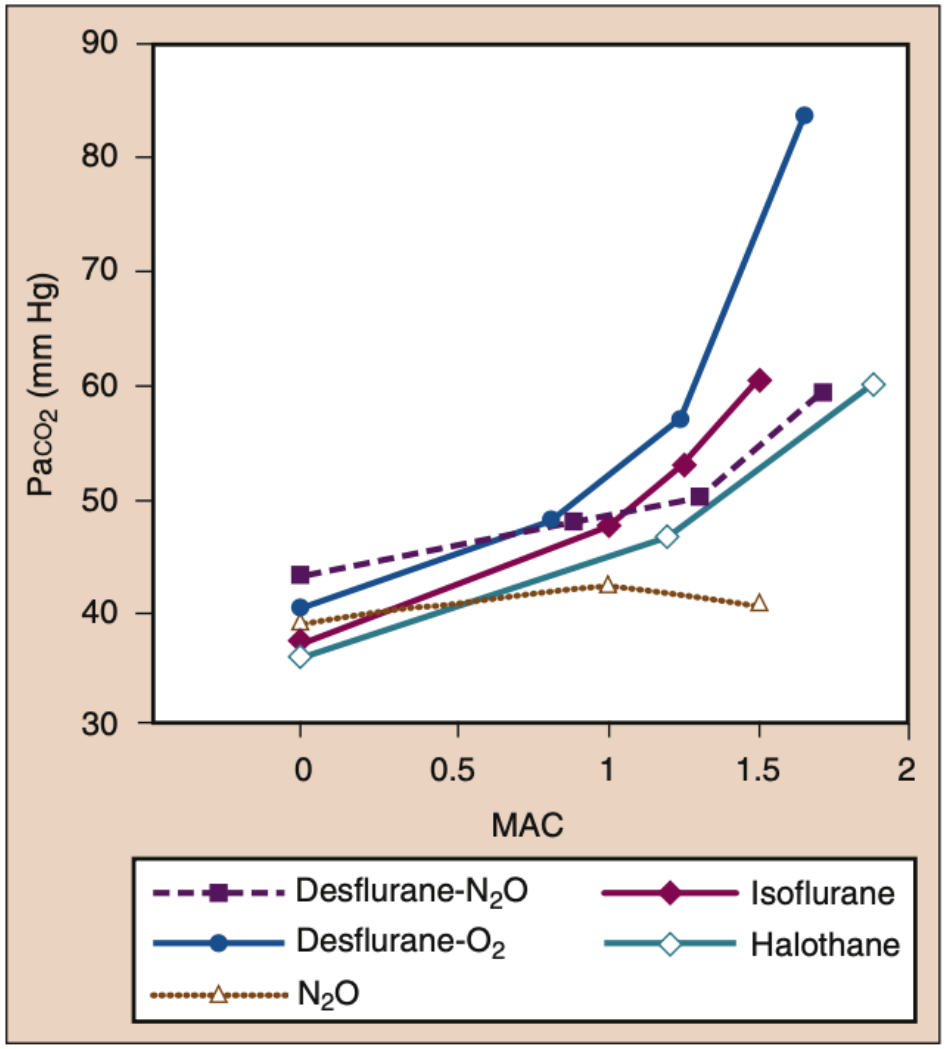


Fig. 7.14 Inhaled anesthetics produce drug-specific and dose-dependent increases in $Paco_2$. *MAC*, Minimum alveolar concentration. (From Eger EI II. *Desflurane (Suprane): A Compendium and Reference*. Nutley, NJ: Anaquest; 1993:1-119, used with permission.)

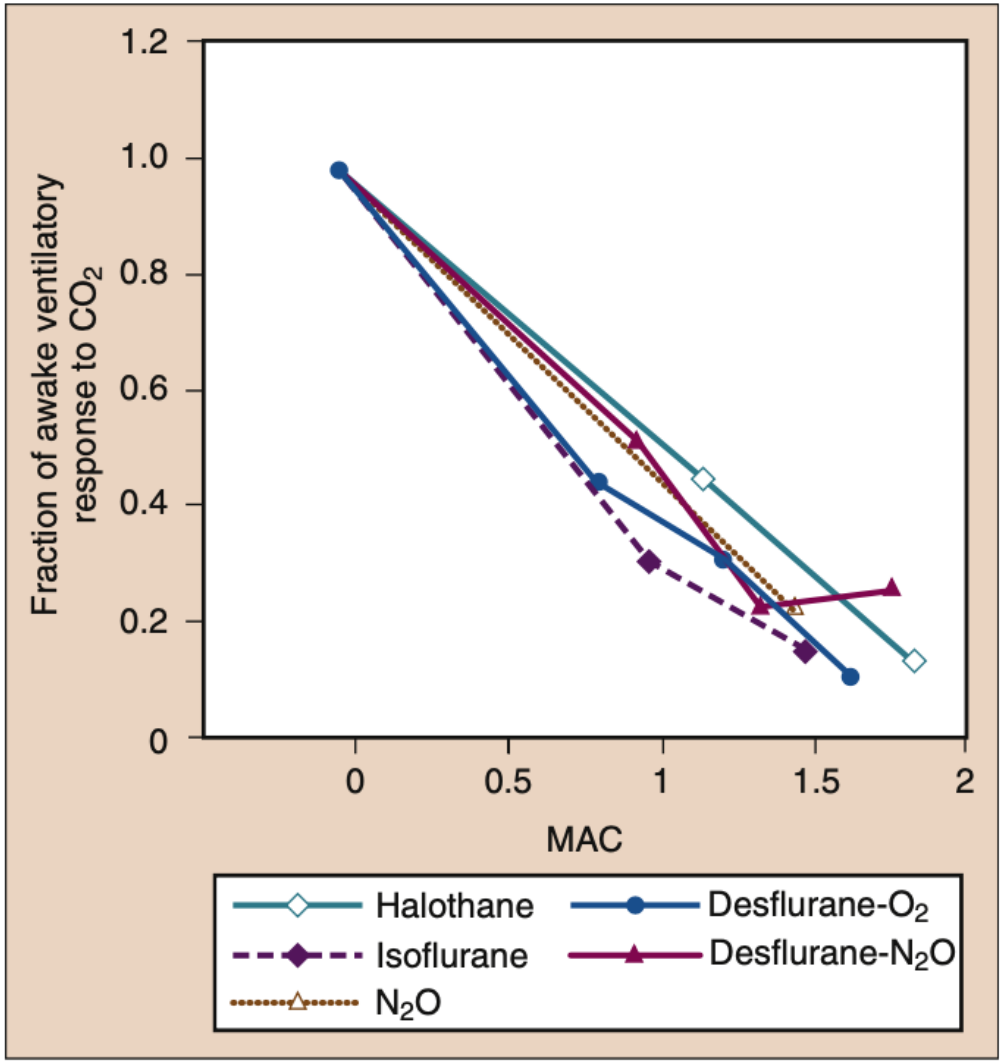


Fig. 7.15 All inhaled anesthetics produce similar dose-dependent decreases in the ventilatory responses to carbon dioxide. *MAC*, Minimum alveolar concentration. (From Eger EI II. *Desflurane (Suprane): A Compendium and Reference*. Nutley, NJ: Anaquest; 1993:1-119, used with permission.)

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

TABLE 20.3 Metabolism of Halogenated Volatile Anesthetics

Anesthetic	Halothane	Methoxyflurane	Enflurane	Isoflurane	Desflurane	Sevoflurane
Extent of tissue metabolism (%)	25	70	2.5	0.2	0.02	5
Oxidizing enzymes	CYP2E1 CYP2A6	CYP2E1 CYP1A2, 2C9/10, 2D6	CYP2E1	CYP2E1	CYP2E1	CYP2E1
Oxidative metabolites	F_3C-COOH HBr, HCl	H_3C-O-CF_2-COOH HCl_2C-COOH HOOC-COOH HF, HCl	HF_2C-O-CF_2-COOH HCl, HF	HF_2C-O-CO-CF_3 F_3C-COOH CF_2HOH HCl	HF_2C-O-CO-CF_3 F_3C-COOH CF_2HOH HF	HO-$CH(CF_3)_2$ HF
Trifuoroacetylated hepatocellular proteins	+++++	n/a	++	+	+	none
Reducing enzymes	CYP2A6 CYP3A4	n/a	n/a	n/a	n/a	
Reductive metabolites	F^-, Br^- F_2C=CHCl F_3C-CH_2Cl					
Tissue toxicities	Hepatic	Renal Hepatic	Renal Hepatic	Hepatic	Hepatic	Hepatic
Fulminant hepatitis incidence	1:20,000	Reported, incidence unknown	1:300,000	rare	rare	Few case reports
References	72-76	77-80	81-85	84,86-88	89-92	78,93-96

The plus signs indicate relative degree of protein modification. n/a, the specific enzymes are not identified in these cases. Kharasch ED. Adverse drug reactions with halogenated anesthetics. *Clin Pharmacol Ther*. 2008;84:158–162.

Gropper MA, Eriksson LI, M.D LAF, et al. Miller's Anesthesia. Elsevier; 2019.

https://www.gehealthcare.com/products/anesthesia-delivery/aisys-cs2

1 psi=

70.3 cmH_2O=

68.9 mbar=

51.7 mmHg

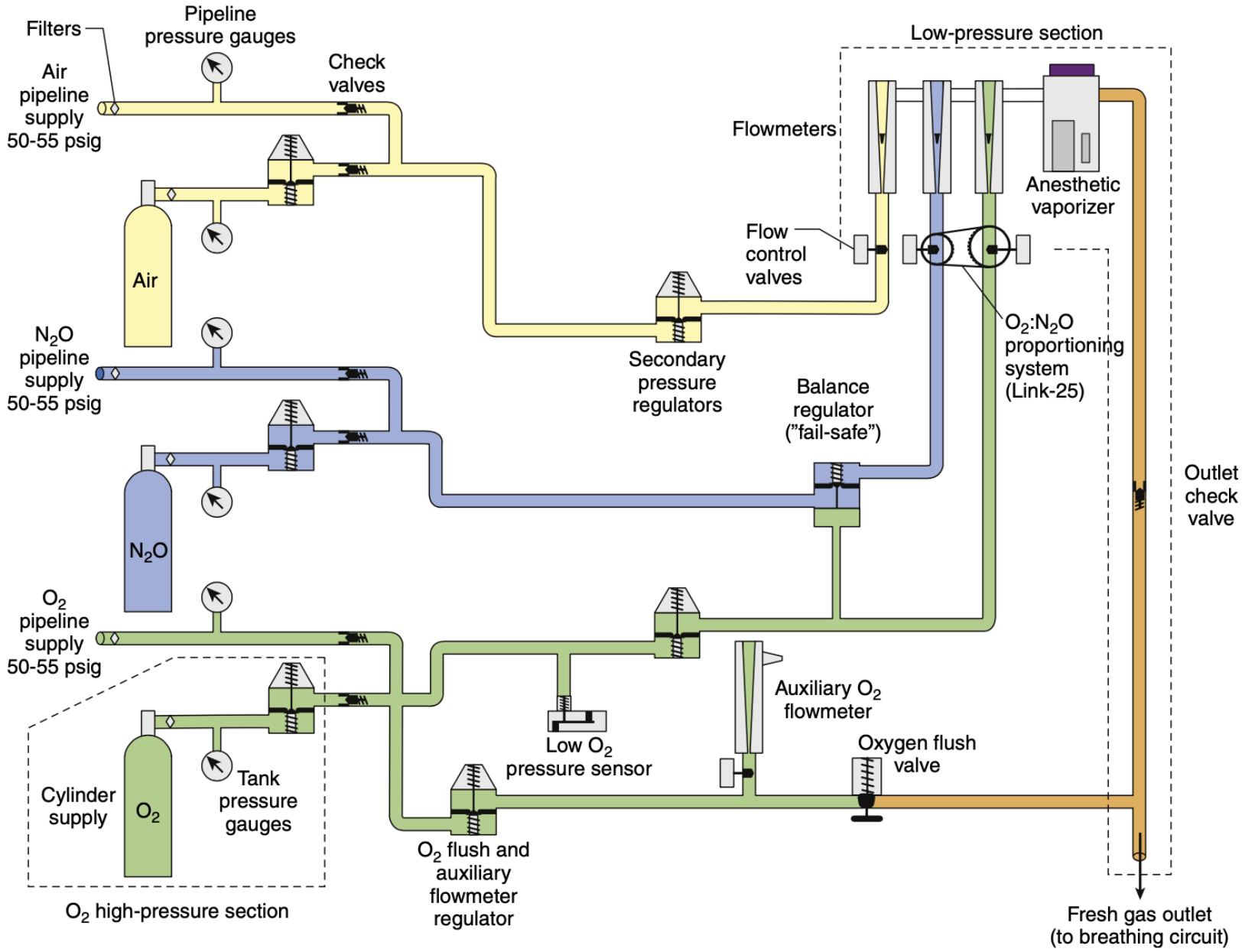


Fig. 22.1 The GE Healthcare Aespire anesthesia workstation gas supply system. The high-pressure system extends from the gas cylinders to the high-pressure regulators (*dashed lines* around O_2 high-pressure section only). The intermediate-pressure section extends from the high-pressure regulators to the flow control valves and also includes the tubing and components originating from the pipeline inlets. The low-pressure section *(dashed lines)* extends from the flow control valves to the breathing circuit. See text for additional details. (From Datex-Ohmeda. *S/5 Aespire Anesthesia Machine: Technical Reference Manual.* Madison, WI: Datex-Ohmeda; 2004.)

Gropper MA, Eriksson LI, M.D LAF, et al. Miller's Anesthesia. Elsevier; 2019.

Domande o interventi?

Anestetici endovenosi

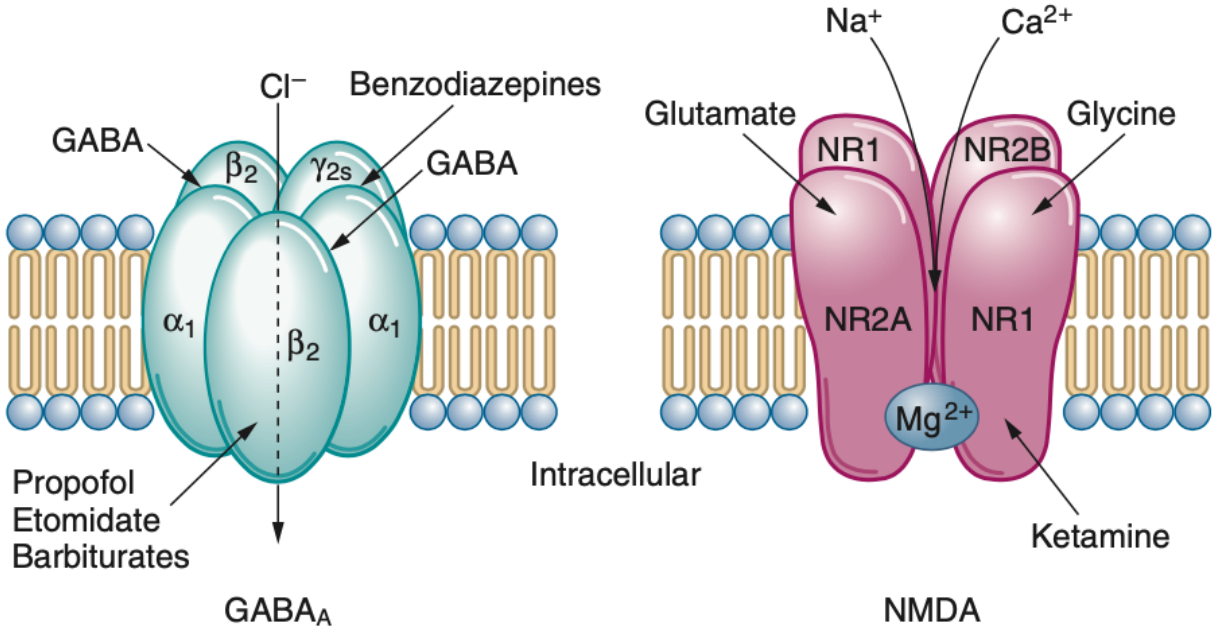
Table 8.1 Pharmacokinetic Data[a] for Intravenous Anesthetics

Drug	Induction Dose (mg/kg IV)	Duration of Action (min)	Vd_{ss} (L/kg)	$T_{½}\alpha$ (min)	Protein Binding (%)	Clearance (mL/kg/min)	$T_{½}\beta$ (h)
Propofol	1-2.5	3-8	2-10	2-4	97	20-30	4-23
Thiopental	3-5	5-10	2.5	2-4	83	3.4	11
Methohexital	1-1.5	4-7	2.2	5-6	73	11	4
Midazolam	0.1-0.3	15-20	1.1-1.7	7-15	94	6.4-11	1.7-2.6
Diazepam	0.3-0.6	15-30	0.7-1.7	10-15	98	0.2-0.5	20-50
Lorazepam	0.03-0.1	60-120	0.8-1.3	3-10	98	0.8-1.8	11-22
Ketamine	1-2	5-10	3.1	11-16	12	12-17	2-4
Etomidate	0.2-0.3	3-8	2.5-4.5	2-4	77	18-25	2.9-5.3
Dexmedetomidine	N/A	N/A	2-3	6	94	10-30	2-3

[a]Data are for average adult patients. The duration of action reflects the duration after an average single IV dose.
IV, Intravenous; *N/A*, not applicable; $T_{½}\alpha$, distribution half-time; $T_{½}\beta$, elimination half-time; Vd_{ss}, volume of distribution at steady state.

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

Anestetici endovenosi



• **Fig. 10.1** Key targets of intravenous anesthetics. $GABA_A$ receptors are critical targets for benzodiazepines, barbiturates, etomidate, and propofol. Although it is possible for the drugs and ligands to interact with this protein in multiple areas, it is generally agreed that the endogenous ligand GABA binds to the receptor in a pocket between the α and β subunits. Many of the intravenous anesthetics have their main influence on the activity of this protein in the transmembrane portion of the β subunit, whereas the benzodiazepines modulate the protein through interactions with transmembrane amino acids between the α and γ subunits near the intracellular side. NMDA receptors are activated by the agonist glutamate and co-agonist glycine only when voltage changes displace Mg^{2+} from the ion channel pore. Ketamine also acts primarily by a pore-blocking mechanism. *Ca^+,* Calcium ion; *Cl^-,* chloride ion; *GABA,* gamma-aminobutyric acid type A; *Mg^{2+},* magnesium ion; *NMDA,* N-methyl-D-aspartate.

Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018.

Propofol

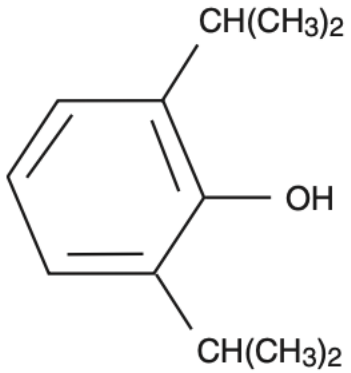
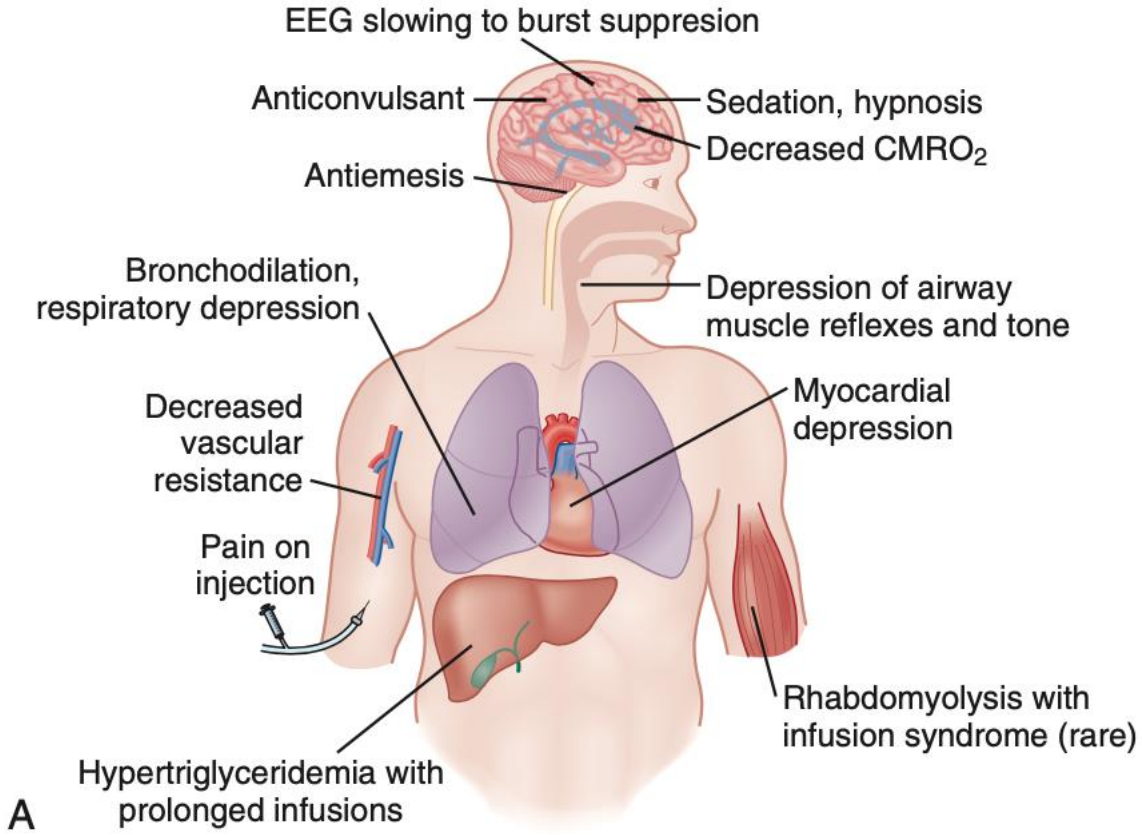


Fig. 23.1 Structure of propofol, an alkylphenol derivative.



Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018.
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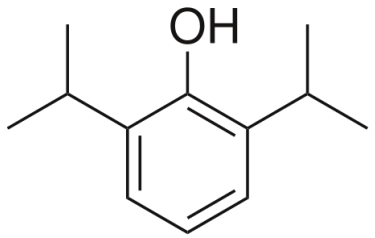
Propofol



- Alchilfenolo (2,6,-diisopropilfenolo)
- Emulsione lipidica (soia, uovo, Intralipid)
- Agonista $GABA_A$ (subunità β_2, β_3),↓ correnti Na^+ NMDA, antagonismo recettori glicina, azione su HCN Hyperpolarization-activated cyclic nucleotide-gated (Na^+, K^+)
- Metabolismo epatico

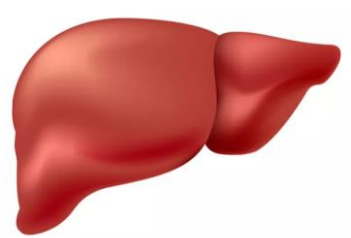
  Ossidazione (1,4-diisopropyl quinol)

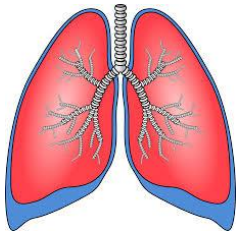
  Glucuronidazione (propofol-1-glucuronide, quinol-1- glucuronide, quinol-4-glucuronide)

  Eliminazione dei metaboliti inattivi per via renale
- <1% eliminato immodificato nelle urine, 2% immodificato nelle feci
- Metabolismo extraepatico:

  rene (30% clearance)

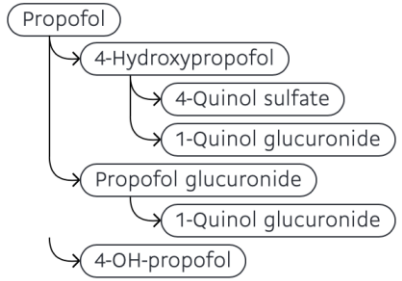
  polmone (uptake 20-30% first pass)





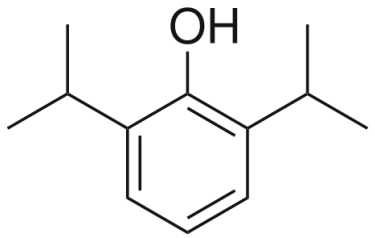
Hepatically metabolized mainly by glucuronidation at the C1-hydroxyl. Hydroxylation of the benzene ring to 4-hydroxypropofol may also occur via CYP2B6 and 2C9 with subsequent conjugation to sulfuric and/or glucuronic acid. Hydroxypropofol has approximately 1/3 of hypnotic activity of propofol.

Hover over products below to view reaction partners



Irwin MG, Wong GTC, Lam SW. Taking on TIVA: Debunking Myths and Dispelling Misunderstandings. Cambridge University Press; 2019.
Gropper MA, Eriksson LI, M.D LAF, et al. Miller's Anesthesia. Elsevier; 2019.
https://go.drugbank.com/drugs/DB00818

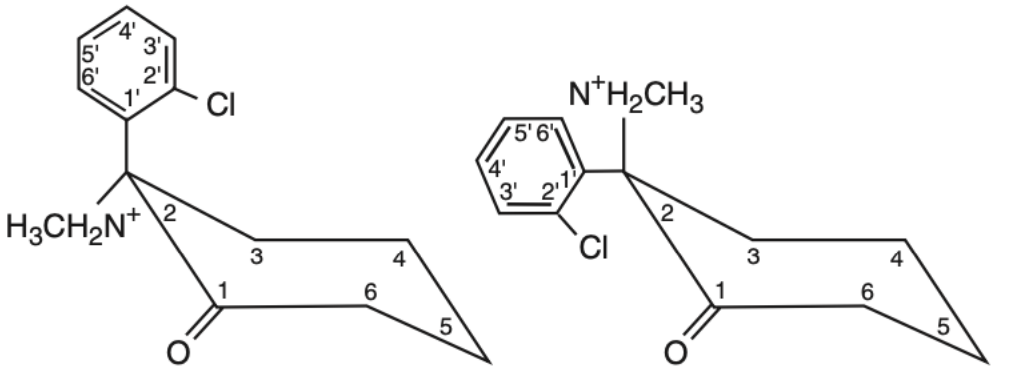
Propofol



Rapid Induction of anaesthesia	4 – 6 µg/ml	Beware of hypotension; may need higher concentrations in young unpremedicated patients
Slower induction of anaesthesia	1 - 3 µg/ml	Repeated 0.5– 1.0 µg/ml incremental increases in the target concentration
Maintenance of anaesthesia	3 – 6 µg/ml	May need to be higher if surgery very stimulating
Maintenance of anaesthesia with concurrent opioid use	2.5 – 4 µg/ml	Dose reduction due to synergistic interaction of drugs, dose reduction of propofol requirements by approx 50%
Concentration on waking	1 - 2 µg/ml	Can be variable, use Ce on induction as a guide only

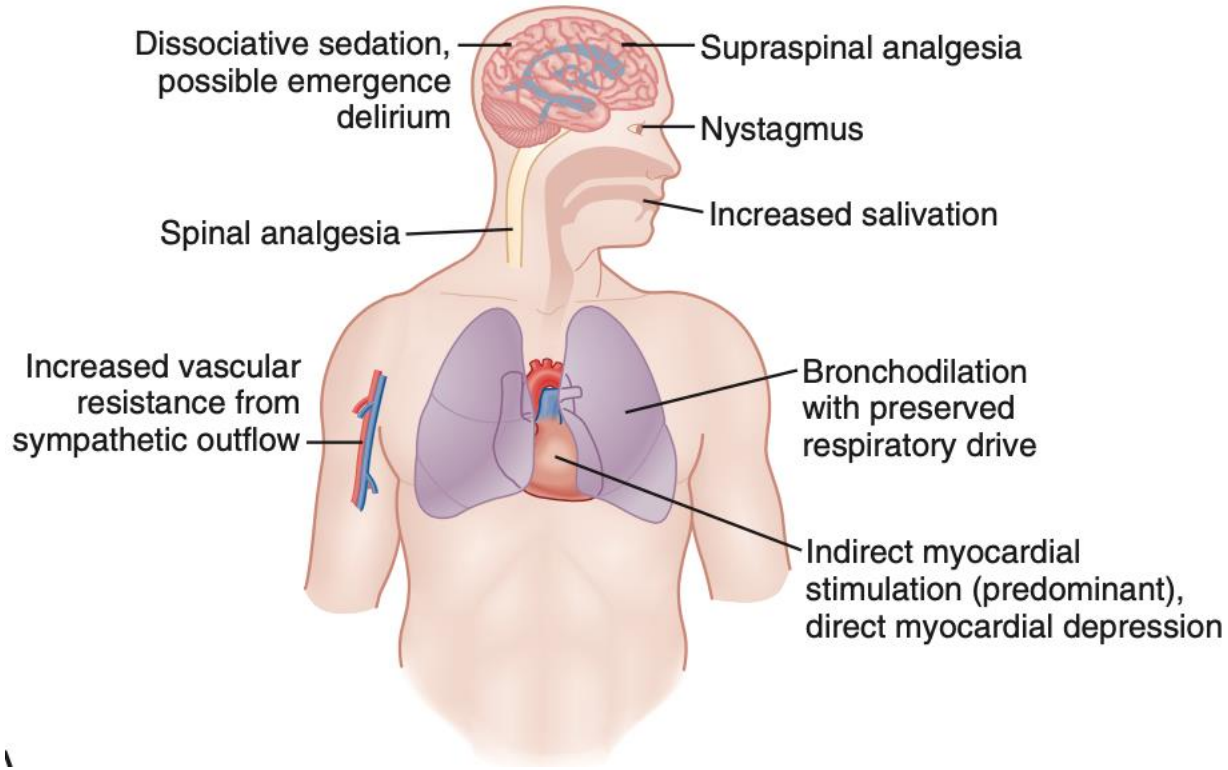
https://clinicaltoolkit.scot.nhs.uk/sjh-theatres-and-anaesthetics/local-guidelines-sops-contacts/tiva-guide/

Ketamina



S_1(+) Ketamine hydrochloride R_1(–) Ketamine hydrochloride

Fig. 23.14 Stereoisomers of ketamine as it is formulated.



Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018.
Gropper MA, Eriksson LI, M.D LAF, et al. Miller's Anesthesia. Elsevier; 2019.

BOX 23.3 Uses and Doses of Ketamine

Induction of general anesthesia*	0.5-2 mg/kg IV 4-6 mg/kg IM
Maintenance of general anesthesia	0.5-1 mg/kg IV with N_2O 50% in O_2 15-45 µg/kg/min IV with N_2O 50%-70% in O_2 30-90 µg/kg/min IV without N_2O
Sedation and analgesia	0.2-0.8 mg/kg IV over 2-3 min 2-4 mg/kg IM
Preemptive or preventive analgesia	0.15-0.25 mg/kg IV

Gropper MA, Eriksson LI, M.D LAF, et al. Miller's Anesthesia. Elsevier; 2019.

Benzodiazepine

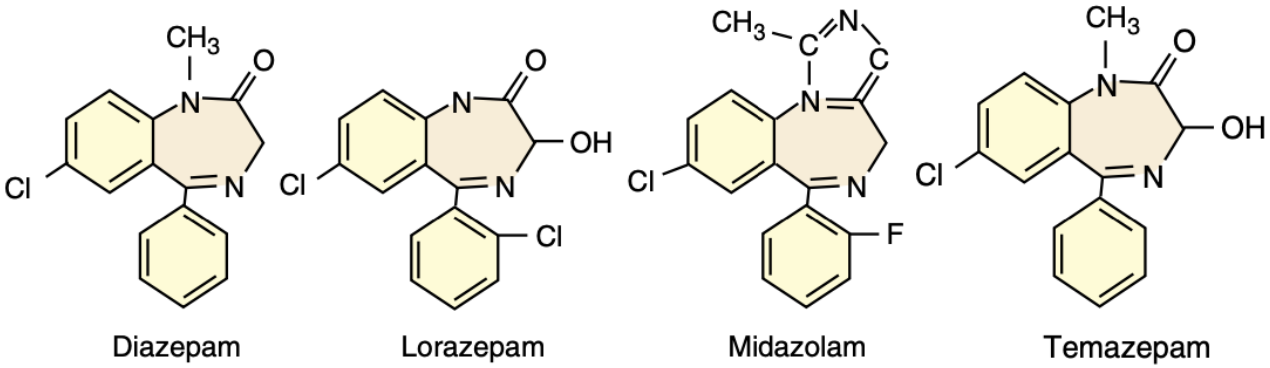
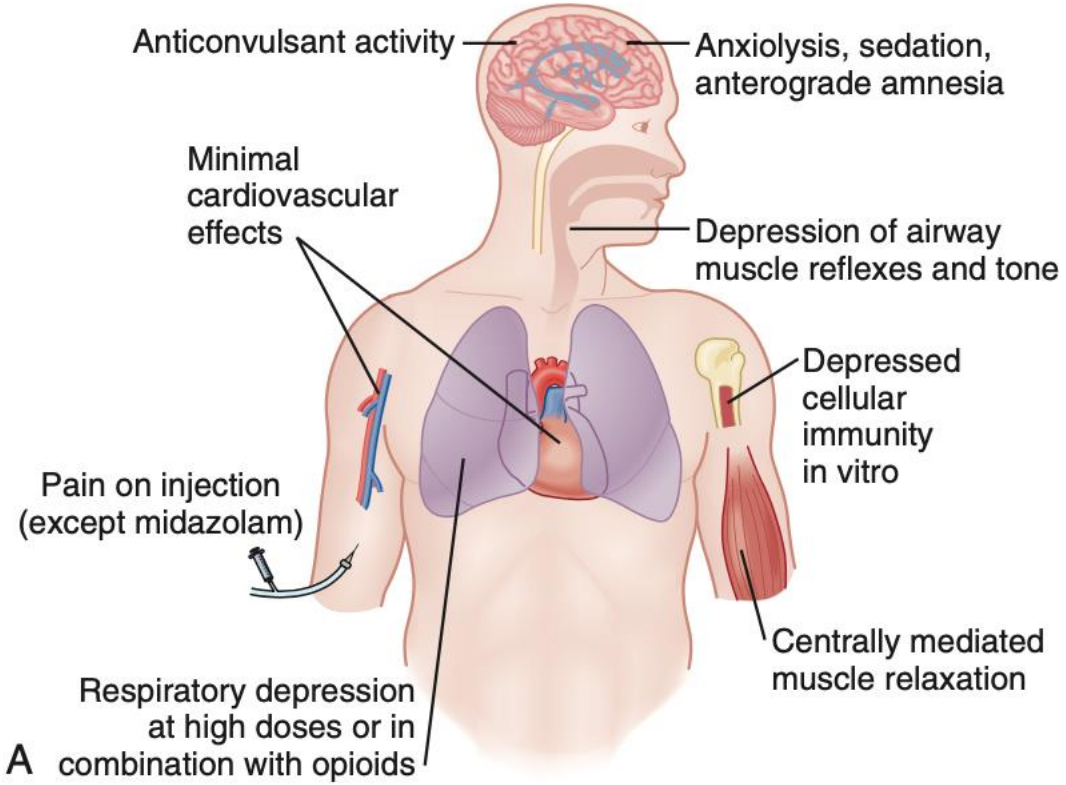


Fig. 23.8 The structures of six benzodiazepines.



Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018.
Gropper MA, Eriksson LI, M.D LAF, et al. Miller's Anesthesia. Elsevier; 2019.

TABLE 23.7 Uses and Doses of Intravenous Benzodiazepines

	Midazolam	Diazepam	Lorazepam
Induction	0.05-0.15 mg/kg	0.3-0.5 mg/kg	0.1 mg/kg
Maintenance	0.05 mg/kg prn	0.1 mg/kg prn	0.02 mg/kg prn
	1 μg/kg/min		
Sedation*	0.5-1 mg repeated	2 mg repeated	0.25 mg repeated
	0.07 mg/kg IM		

Gropper MA, Eriksson LI, M.D LAF, et al. Miller's Anesthesia. Elsevier; 2019.

Reversal delle Benzodiazepine con Flumazenil

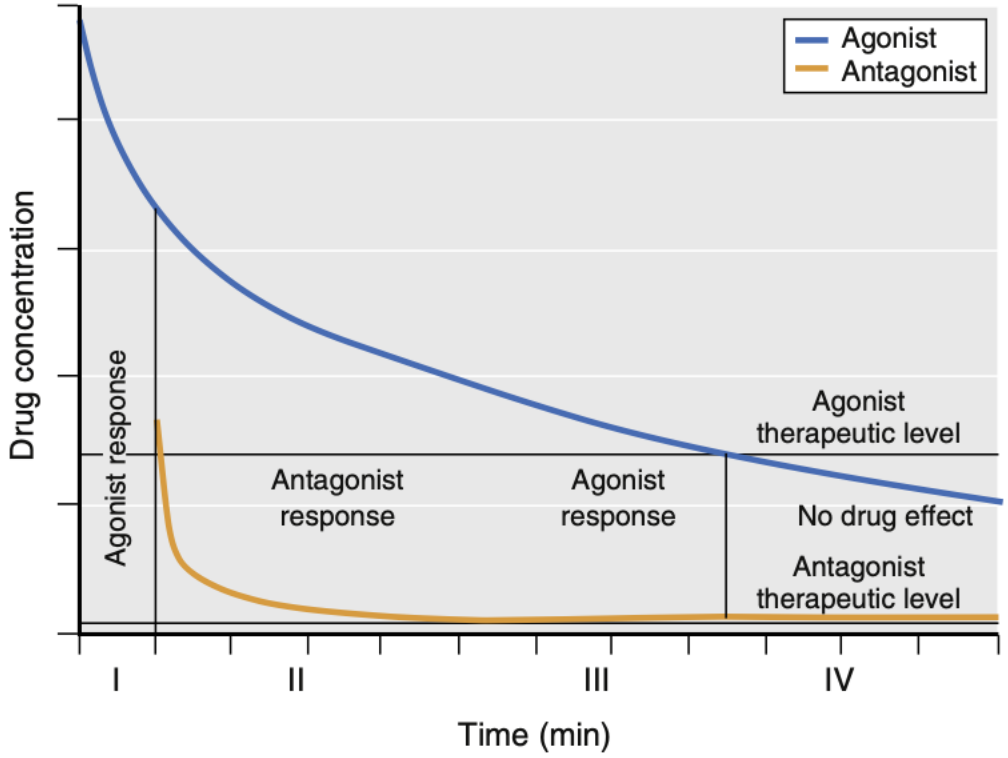


Fig. 23.13 Schematic representation of the interaction of a short-acting antagonist with a longer-acting agonist resulting in resedation. The upper curve shows disappearance of agonist from blood, and the lower curve shows disappearance of antagonist from plasma. Four conditions are represented: I, agonist response; II, antagonist response (the antagonist reverses the agonist effect); III, agonist response (resedation or resumption of agonist response with disappearance of short-lasting antagonist); and IV, no drug effect, with disappearance of agonist and antagonist (both drugs are below the therapeutic level).

BOX 23.2 Uses and Doses of Flumazenil

Reversal of benzodiazepines	0.2 mg repeated up to 3 mg
Diagnosis in coma	0.5 mg repeated up to 1 mg

Gropper MA, Eriksson LI, M.D LAF, et al. Miller's Anesthesia. Elsevier; 2019.

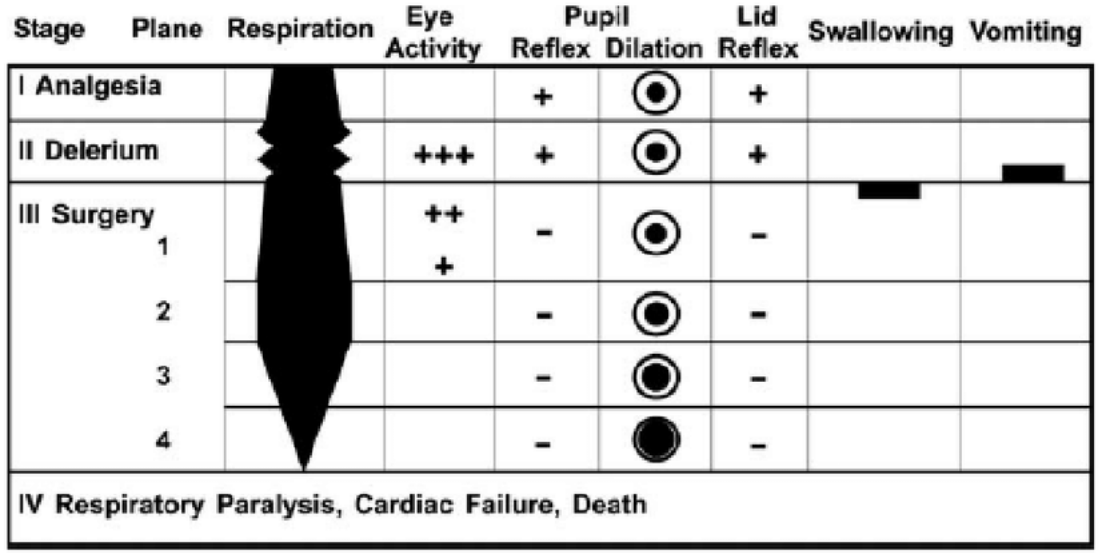
Dose/Effect	Propofol	Thiopental	Midazolam	Ketamine	Etomidate	Dexmedetomidine
Dose for induction of anesthesia (mg/kg IV)	1.5-2.5	3-5	0.1-0.3	1-2	0.2-0.3	
Systemic blood pressure	Decreased	Decreased	Unchanged to decreased	Increased *	Unchanged to decreased	Decreased †
Heart rate	Unchanged to decreased	Increased	Unchanged	Increased	Unchanged to increased	Decreased
Systemic vascular resistance	Decreased	Decreased	Unchanged to decreased	Increased	Unchanged to decreased	Decreased †
Ventilation	Decreased	Decreased	Unchanged	Unchanged	Unchanged to decreased	Unchanged to decreased
Respiratory rate	Decreased	Decreased	Unchanged to decreased	Unchanged	Unchanged to decreased	Unchanged
Response to carbon dioxide	Decreased	Decreased	Decreased	Unchanged	Decreased	Unchanged
Cerebral blood flow	Decreased	Decreased	Decreased	Increased to unchanged	Decreased	Decreased
Cerebral metabolic requirements for oxygen	Decreased	Decreased	Decreased	Increased to unchanged	Decreased	Unchanged
Intracranial pressure	Decreased	Decreased	Unchanged	Increased to unchanged	Decreased	Unchanged
Anticonvulsant	Yes	Yes	Yes	Yes?	No	No
Anxiolysis	No	No	Yes	No	No	Yes?
Analgesia	No	No	No	Yes	No	Yes?
Emergence delirium	No?	No	No	Yes	No	May reduce
Nausea and vomiting	Decreased	Unchanged	Decreased	Unchanged	Increased	Unchanged
Adrenocortical suppression	No	No	Yes?	No	Yes	No
Pain on injection	Yes	No	No	No	No	No

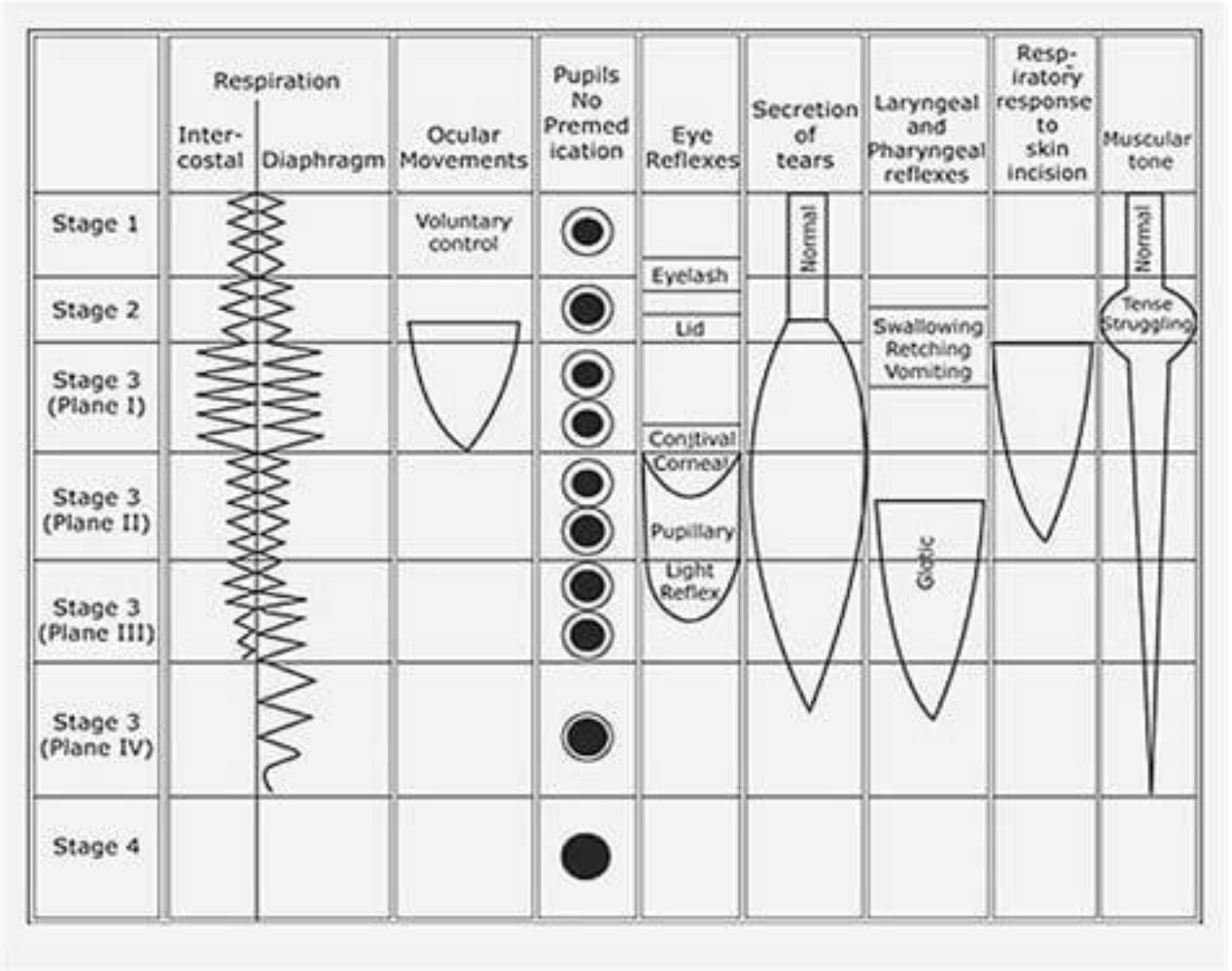
*May cause direct myocardial depression and hypotension in critically ill or catecholamine-depleted patients.
†Bolus injection may increase systemic vascular resistance and blood pressure. *IV*, Intravenous.

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

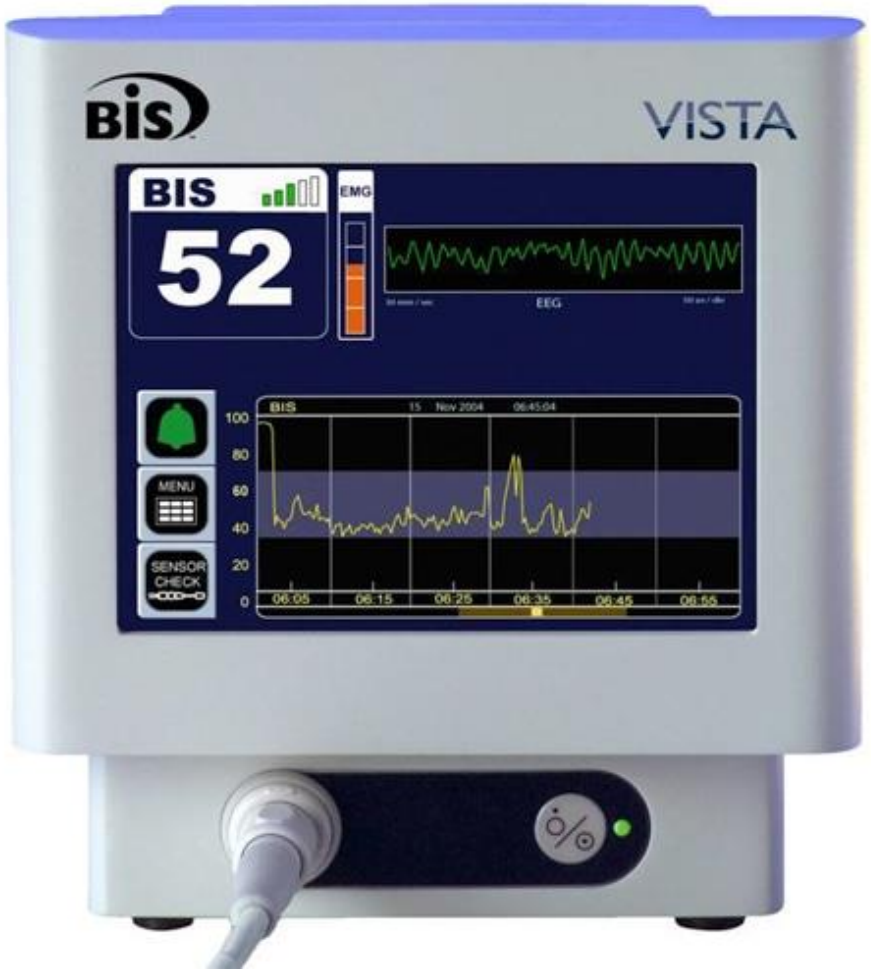
Domande o interventi?

Profondità dell'anestesia





https://www.researchgate.net/figure/Guedels-four-stages-of-anesthesia-and-their-corresponding-physiological-symptoms-taken_fig2_233835574
https://litfl.com/wp-content/uploads/2019/06/Guedel-classification-1937.jpg


BIS
VISTA
BIS
52
EMG
EEG
MENU
SENSOR CHECK
BIS
15 Nov 2004
06:45:04
100
80
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06:05
06:15
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06:35
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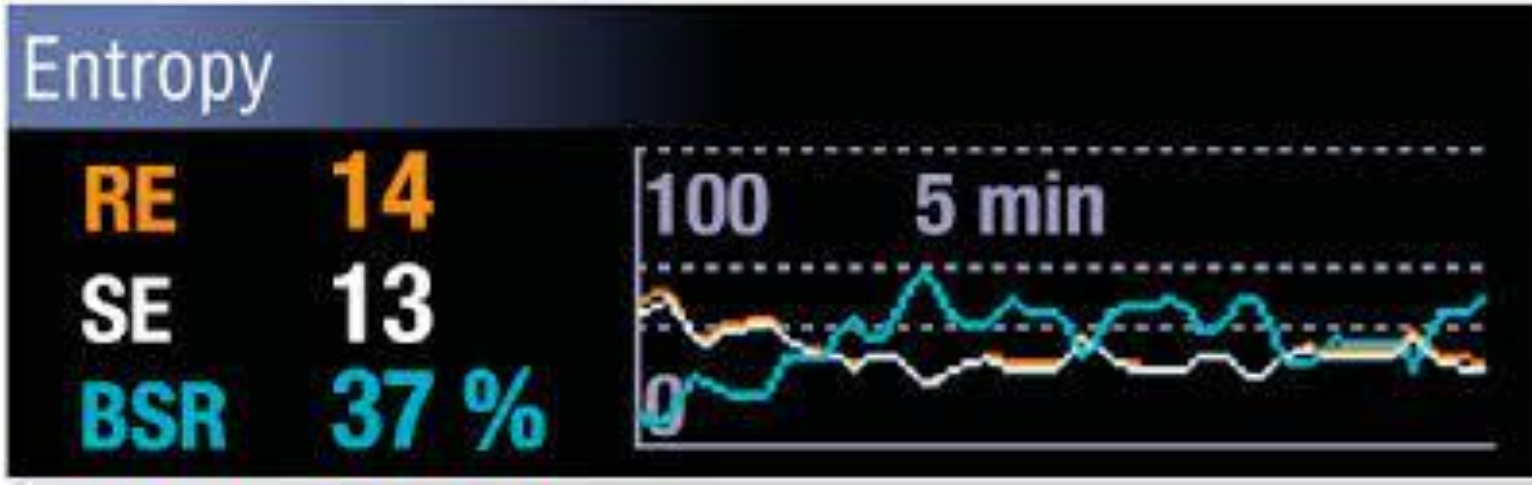
06:55


Entropy
RE 14
SE 13
BSR 37 %
100
5 min
0

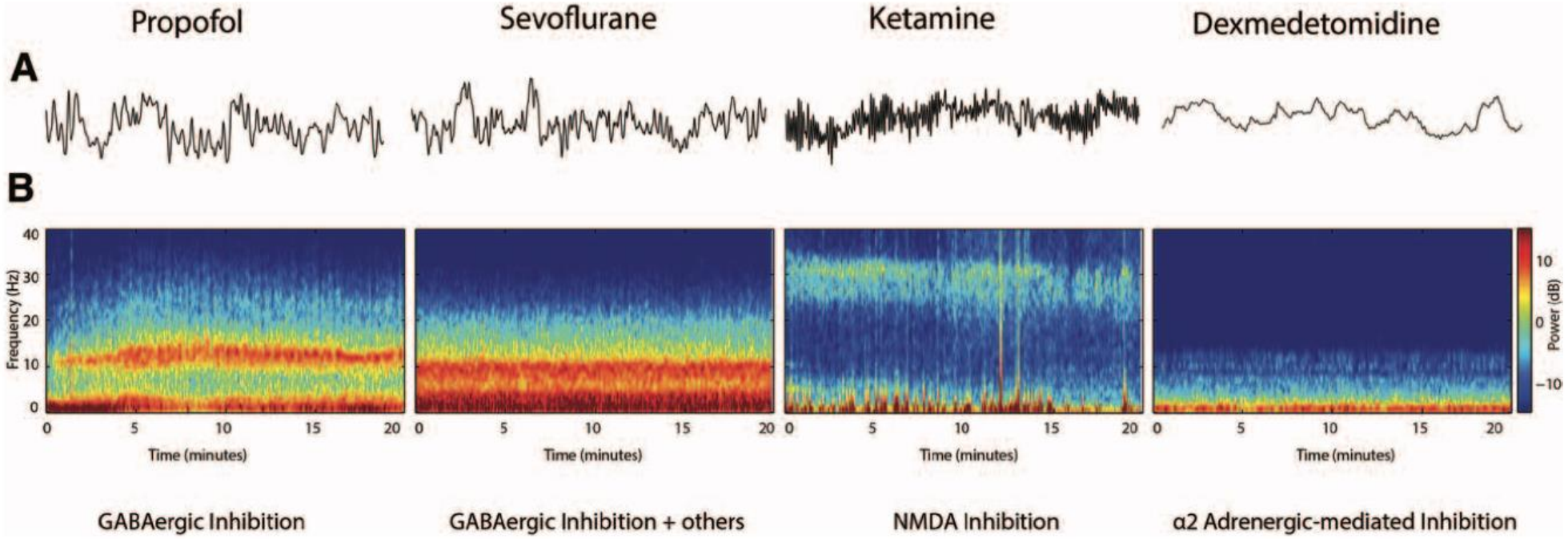


Fig. 14. Different anesthetics (propofol, sevoflurane, ketamine, and dexmedetomidine), different electroencephalogram signatures, and different molecular and neural circuit mechanisms. (*A*) Anesthetic-specific differences in the electroencephalogram are difficult to discern in unprocessed electroencephalogram waveforms. (*B*) In the spectrogram, it is clear that different anesthetics produce different electroencephalogram signatures. The dynamics the electroencephalogram signatures can be related to the molecular targets and the neural circuits at which the anesthetics act to create altered states of arousal. Propofol and sevoflurane enhance γ-aminobutyric acid (GABA)ergic inhibition, sevoflurane binds at GABA receptors and other molecular targets, ketamine blocks *N*-methyl-D-aspartate (NMDA) glutamate receptors, and dexmedetomidine is a presynaptic alpha adrenergic agonist. *A* and *B* were adapted, with permission, from Purdon and Brown, *Clinical Electroencephalography for the Anesthesiologist* (2014), from the Partners Healthcare Office of Continuing Professional Development.[69] Adaptations are themselves works protected by copyright. In order to publish this adaptation, authorization has been obtained both from the owner of the copyright of the original work and from the owner of copyright of the translation or adaptation.

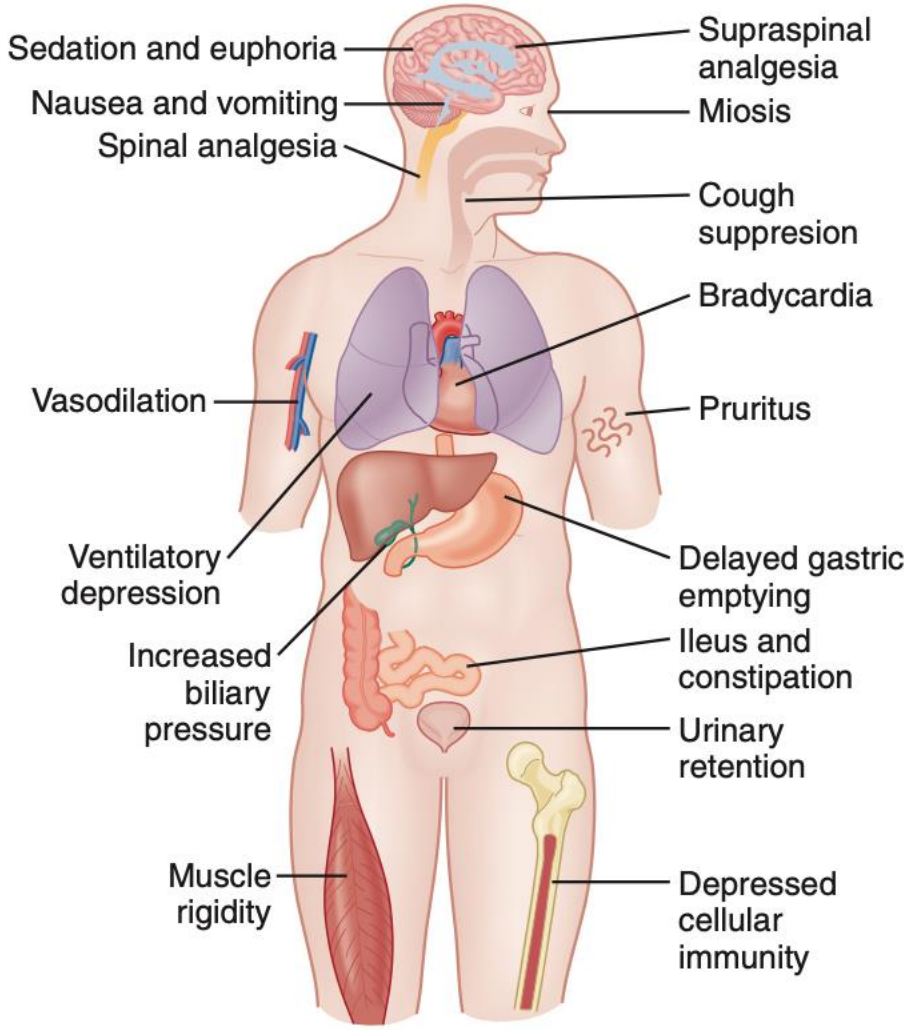
Purdon PL, Sampson A, Pavone KJ, Brown EN. Clinical Electroencephalography for Anesthesiologists Part I: Background and Basic Signatures. Anesthesiology. 2015;123(4):937-960. doi:10.1097/ALN.0000000000000841

Domande o interventi?

Oppioidi

TABLE 24.2 Pharmacologic Actions of Opioids and Opioid Receptors in Animal Models

	ACTIONS OF		
	Receptor	**Agonists**	**Antagonists**
ANALGESIA			
Supraspinal	μ, δ, κ	Analgesic	No effect
Spinal	μ, δ, κ	Analgesic	No effect
Respiratory function	μ	Decrease	No effect
Gastrointestinal tract	μ, κ	Decrease transit	No effect
Psychotomimesis	κ	Increase	No effect
Feeding	μ, δ, κ	Increase feeding	Decrease feeding
Sedation	μ, κ	Increase	No effect
Diuresis	κ	Increase	
HORMONE SECRETION			
Prolactin	μ	Increase release	Decrease release
Growth hormone	μ and/or δ	Increase release	Decrease release
NEUROTRANSMITTER RELEASE			
Acetylcholine	μ	Inhibit	
Dopamine	δ	Inhibit	



• **Fig. 17.8** Opioid pharmacodynamics. A summary chart of selected effects of the fentanyl congeners (see text for details).

Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018.
Gropper MA, Eriksson LI, M.D LAF, et al. Miller's Anesthesia. Elsevier; 2019.

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Gastrointestinal tract	μ, κ	Decrease transit	No effect
Psychotomimesis	κ	Increase	No effect
Feeding	μ, δ, κ	Increase feeding	Decrease feeding
Sedation	μ, κ	Increase	No effect
Diuresis	κ	Increase	
HORMONE SECRETION			
Prolactin	μ	Increase release	Decrease release
Growth hormone	μ and/or δ	Increase release	Decrease release
NEUROTRANSMITTER RELEASE			
Acetylcholine	μ	Inhibit	
Dopamine	δ	Inhibit	

TABLE 20–2 ■ OPIOID AGONISTS

OPIOID LIGANDS	RECEPTOR TYPES		
	μ	δ	κ
Etorphine	+++	+++	+++
Fentanyl	+++		
Hydromorphone	+++		+
Levorphanol	+++		
Methadone	+++		
Morphine[a]	+++		+
Sufentanil	+++	+	+
DAMGO[a] ([D-Ala^2,MePhe^4,Gly(ol)^5] enkephalin)	+++		
Bremazocine[c]	+	+	+++
Buprenorphine	P		– –
Butorphanol[c]	P		+++
Nalbuphine	– –		++
DPDPE[b] ([D-Pen2,5]-Enkephalin])	+++		
U50,488[c]		++	

+, agonist; –, antagonist; P, partial agonist. In potency: + < ++ < +++
[a]Protoypical μ-preferring. [b]Prototypical δ-preferring. [c]Prototypical κ-preferring.
Source: Modified with permission from Raynor K et al. Pharmacological characterization of the cloned kappa-, delta-, and mu-opioid receptors. *Mol Pharmacol*, **1994**;*45*:330–334.

Hilal-Dandan R, Knollmann B, Brunton L. Goodman and Gilman's The Pharmacological Basis of Therapeutics, 13th Edition. McGraw-Hill Education; 2017.
Gropper MA, Eriksson LI, M.D LAF, et al. Miller's Anesthesia. Elsevier; 2019.

TABLE 24.9 Approximate Opioid Loading (Bolus) Doses, Maintenance Infusion Rates, and Additional Maintenance Doses for Total Intravenous Anesthesia

	Loading Dose (μg/kg)	Maintenance Infusion Rate	Additional Boluses
Alfentanil	25-100	0.5-2 μg/kg/min	5-10 μg/kg
Sufentanil	0.25-2	0.5-1.5 μg/kg/h	2.5-10 μg
Fentanyl	4-20	2-10 μg/kg/h	25-100 μg
Remifentanil	1-2	0.1-1.0 μg/kg/min	0.1-1.0 μg/kg

From Bailey PL, Egan TD, Stanley TH. Intravenous opioid anesthetics. In: Miller RD, ed. *Anesthesia*. 8th ed. Philadelphia: Saunders; 2015. An imprint of Elsevier Inc., p. 897.

Gropper MA, Eriksson LI, M.D LAF, et al. Miller's Anesthesia. Elsevier; 2019.

TABLE 20–4 ■ DOSING DATA FOR CLINICALLY EMPLOYED OPIOID ANALGESICS

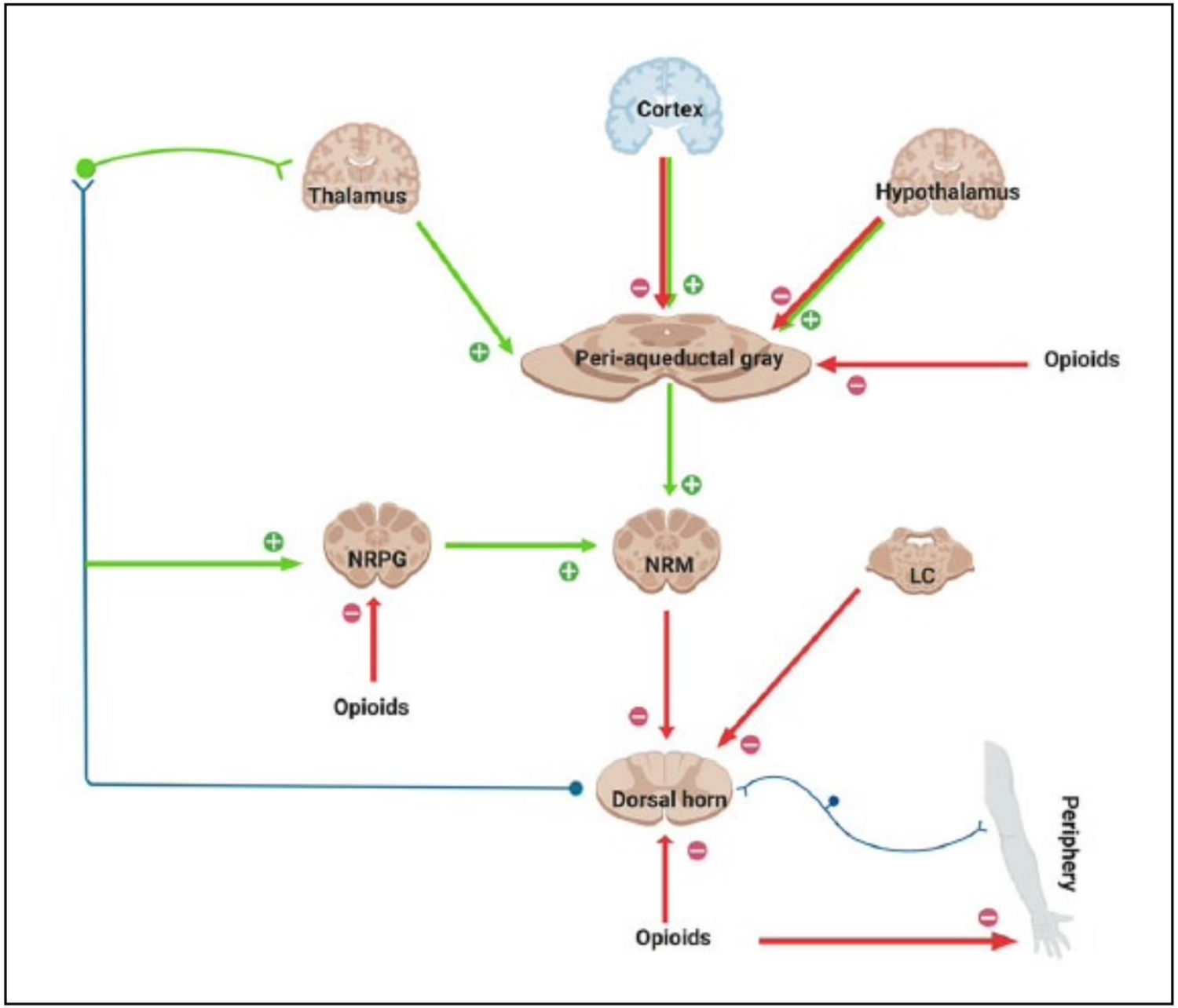
DRUG	APPROXIMATE EQUIANALGESIC ORAL DOSE	APPROXIMATE EQUIANALGESIC PARENTERAL DOSE	RECOMMENDED STARTING DOSE (Adults > 50 kg)		RECOMMENDED STARTING DOSE (Children and Adults < 50 kg)	
			ORAL	PARENTERAL	ORAL	PARENTERAL
Opioid Agonists						
Morphine	30 mg/3–4 h	10 mg/3–4 h	15 mg/3–4 h	5 mg/3–4 h	0.3 mg/kg/3–4 h	0.1 mg/kg/3–4 h
Codeine	130 mg/3–4 h	75 mg/3–4 h	30 mg/3–4 h	30 mg/2 h (IM/SC)	0.5 mg/kg/3–4 h	Not recommended
Hydromophone	6 mg/3–4 h	1.5 mg/3–4 h	2 mg/3–4 h	0.5 mg/3–4 h	0.03 mg/kg/3–4 h	0.005 mg/kg/3–4 h
Hydrocodone (typically with acetaminophen)	30 mg/3–4 h	Not available	5 mg/3–4 h	Not available	0.1 mg/kg/3–4 h	Not available
Levorphanol	4 mg/6–8 h	2 mg/6–8 h	4 mg/6–8 h	2 mg/6–8 h	0.04 mg/kg/6–8 h	0.02 mg/kg/6–8 h
Meperidine	300 mg/2–3 h	100 mg/3 h	Not recommended	50 mg/3 h	Not recommended	0.75 mg/kg/2–3 h
Methadone	10 mg/6–8 h	10 mg/6–8 h	5 mg/12 h	Not recommended	0.1 mg/kg/12 h	Not recommended
Oxycodone	20 mg/3–4 h	Not available	5 mg/3–4 h	Not available	0.1 mg/kg/3–4 h	Not available
Oxymorphone	10 mg/3–4 h	1 mg/3–4 h	5 mg/3–4 h	1 mg/3–4 h	0.1 mg/kg/3–4 h	Not recommended
Tramadol	100 mg	100 mg	50–100 mg/6 h	50–100 mg/6 h	Not recommended	Not recommended
Fentanyl	Transdermal 72-h patch (25 μg/h) = morphine 50 mg/24 h					
Opioid Agonist-Antagonists or Partial Agonists						
Buprenorphine	Not available	0.3–0.4 mg/6–8 h	Not available	0.4 mg/6–8 h	Not available	0.004 mg/kg/6–8 h
Butorphanol	Not available	2 mg/3–4 h	Not available	2 mg/3–4 h	Not available	Not recommended
Nalbuphine	Not available	10 mg/3–4 h	Not available	10 mg/3–4 h	Not available	0.1 mg/kg/3–4 h

Hilal-Dandan R, Knollmann B, Brunton L. Goodman and Gilman's The Pharmacological Basis of Therapeutics, 13th Edition. McGraw-Hill Education; 2017.

Stimolo vie discendenti

- Grigio periacqueduttale PAG e *nucleus reticularis paragigantocellularis* (NRPG)
- Attivazione dei neuroni discendenti inibitori
- ↑Attività nucleo del rafe magno (NRM)
- ↑ Output serotoninergico ed encefalinergico sulla sostanza gelatinosa del corno dorsale

Effetto diretto inibitorio sui neuroni nocicettivi della sostanza gelatinosa del midollo spinale



James A, Williams J. Basic Opioid Pharmacology — An Update. British Journal of Pain. 2020;14(2):115-121. doi:10.1177/2049463720911986

Reversal degli Oppioidi con Naloxone

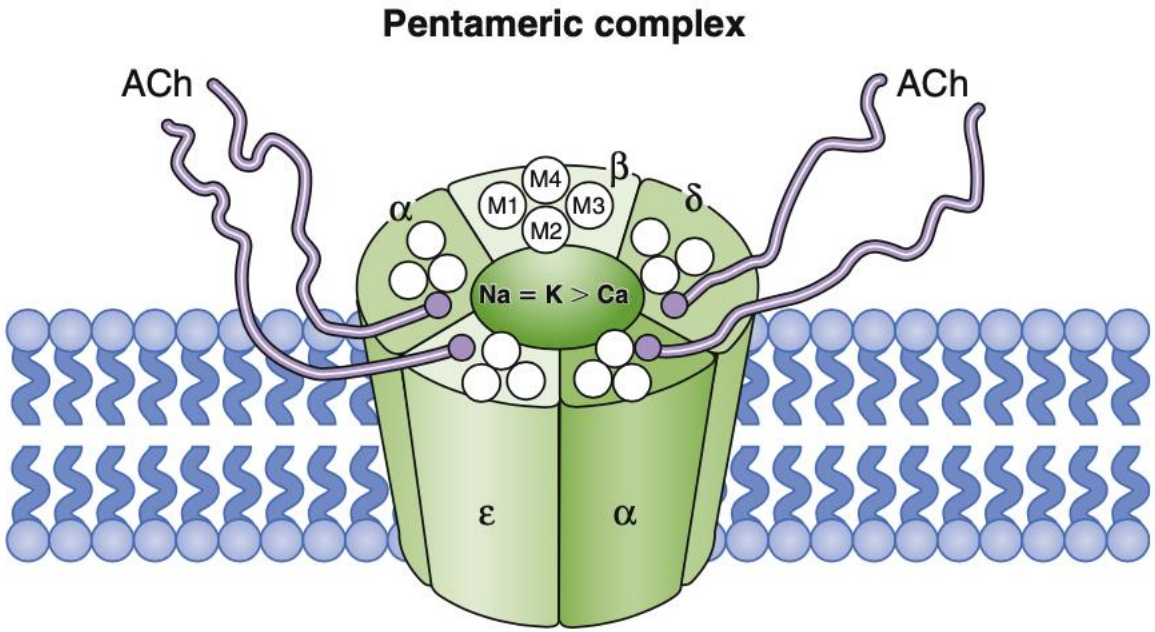
Antagonists		
Naloxone	• Antagonist at MOR/DOR/KOR • Rapid onset, moderately short acting • Rapidly reverses central and peripheral opiate effects • Used in treating opioid overdose • Autoinjector available for emergency administration	• $t_{1/2}$ ~ 64 min • Renarcotization may occur with long-lasting agonists as naloxone is metabolized • May induce moderate hyperalgesia • Known as NARCAN; used by emergency medical technicians to revive comatose opioid abusers

Although naloxone is very effective in reversing the ventilatory depression associated with opioids, it has numerous untoward effects, including acute withdrawal syndrome, nausea, vomiting, tachycardia, hypertension, seizures, and pulmonary edema, among others.[51] Recognizing that naloxone's duration of action is shorter than that of most of the μ-agonists is a key point in determining the dosing schedule; repeated doses may be necessary to sustain its effects.

Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018.
Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

Domande o interventi?

Bloccanti neuromuscolari



Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Cohen NH, Young WL. Miller's Anesthesia E-Book. Elsevier Health Sciences; 2014.

TABLE 34-2 CLASSIFICATION OF NONDEPOLARIZING NEUROMUSCULAR BLOCKERS ACCORDING TO DURATION OF ACTION (TIME TO T1 = 25% OF CONTROL) AFTER TWICE THE DOSE CAUSING ON AVERAGE 95% SUPPRESSION OF NEUROMUSCULAR RESPONSE*

	Clinical Duration			
	Long-acting (>50 min)	Intermediate-acting (20-50 min)	Short-acting (10-20 min)	Ultrashort-acting (<10 min)
Steroidal compounds	Pancuronium	Vecuronium Rocuronium		
Benzylisoquinolinium compounds	*d*-Tubocurarine	Atracurium Cisatracurium	Mivacurium	
Asymmetric mixed-onium fumarates		CW 002		Gantacurium

T1, First twitch of train-of-four.
*Most nondepolarizing neuromuscular blockers are bisquaternary ammonium compounds. *d*-Tubocurarine, vecuronium, and rocuronium are monoquaternary compounds.

Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Cohen NH, Young WL. Miller's Anesthesia E-Book. Elsevier Health Sciences; 2014.

TABLE 34-3 DOSE-RESPONSE RELATIONSHIPS OF NONDEPOLARIZING NEUROMUSCULAR BLOCKING DRUGS IN HUMAN SUBJECTS*

	ED_{50} (mg/kg)	ED_{90} (mg/kg)	ED_{95} (mg/kg)	References
Long-acting				
Pancuronium	0.036 (0.022-0.042)	0.056 (0.044-0.070)	0.067 (0.059-0.080)	98, 103
d-Tubocurarine	0.23 (0.16-0.26)	0.41 (0.27-0.45)	0.48 (0.34-0.56)	103
Intermediate-acting				
Rocuronium	0.147 (0.069-0.220)	0.268 (0.200-0.419)	0.305 (0.257-0.521)	98, 104-106
Vecuronium	0.027 (0.015-0.031)	0.042 (0.023-0.055)	0.043 (0.037-0.059)	103
Atracurium	0.12 (0.08-0.15)	0.18 (0.19-0.24)	0.21 (0.13-0.28)	103
Cisatracurium	0.026 (0.015-0.031)	—	0.04 (0.032-0.05)	107-109, 371
Short-acting				
Mivacurium	0.039 (0.027-0.052)	—	0.067 (0.045-0.081)	9, 110-112
Ultrashort-acting				
Gantacurium	0.09	—	0.19	100

Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Cohen NH, Young WL. Miller's Anesthesia E-Book. Elsevier Health Sciences; 2014.

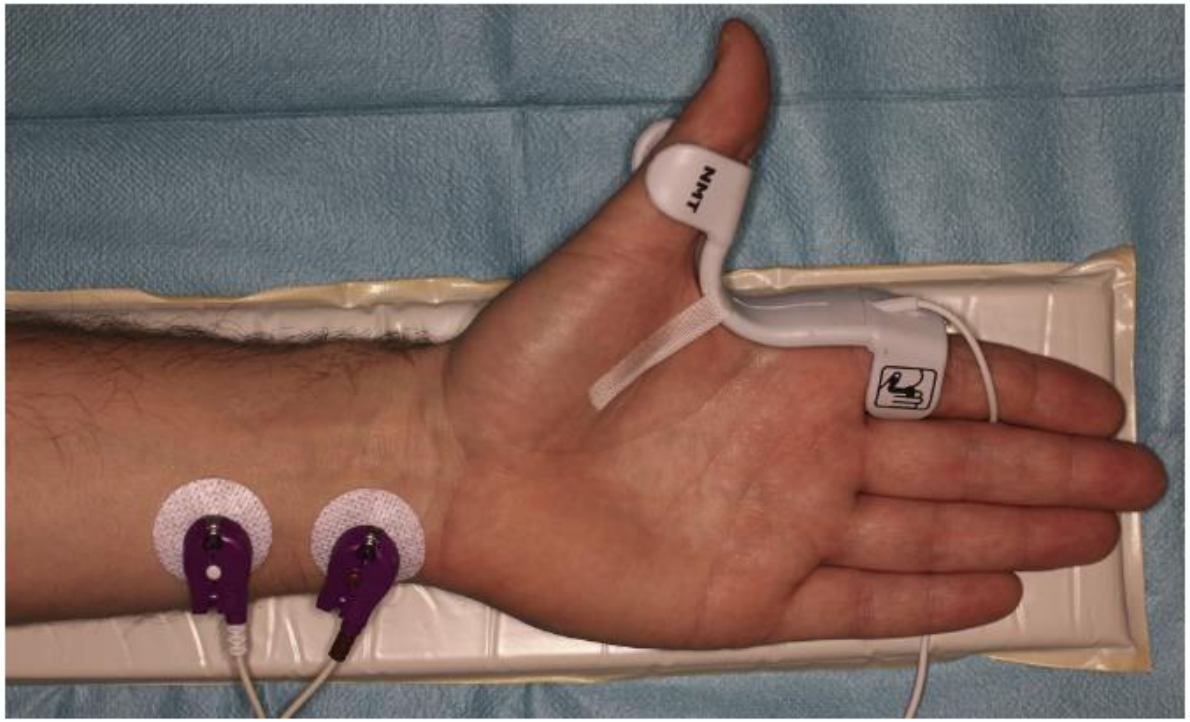


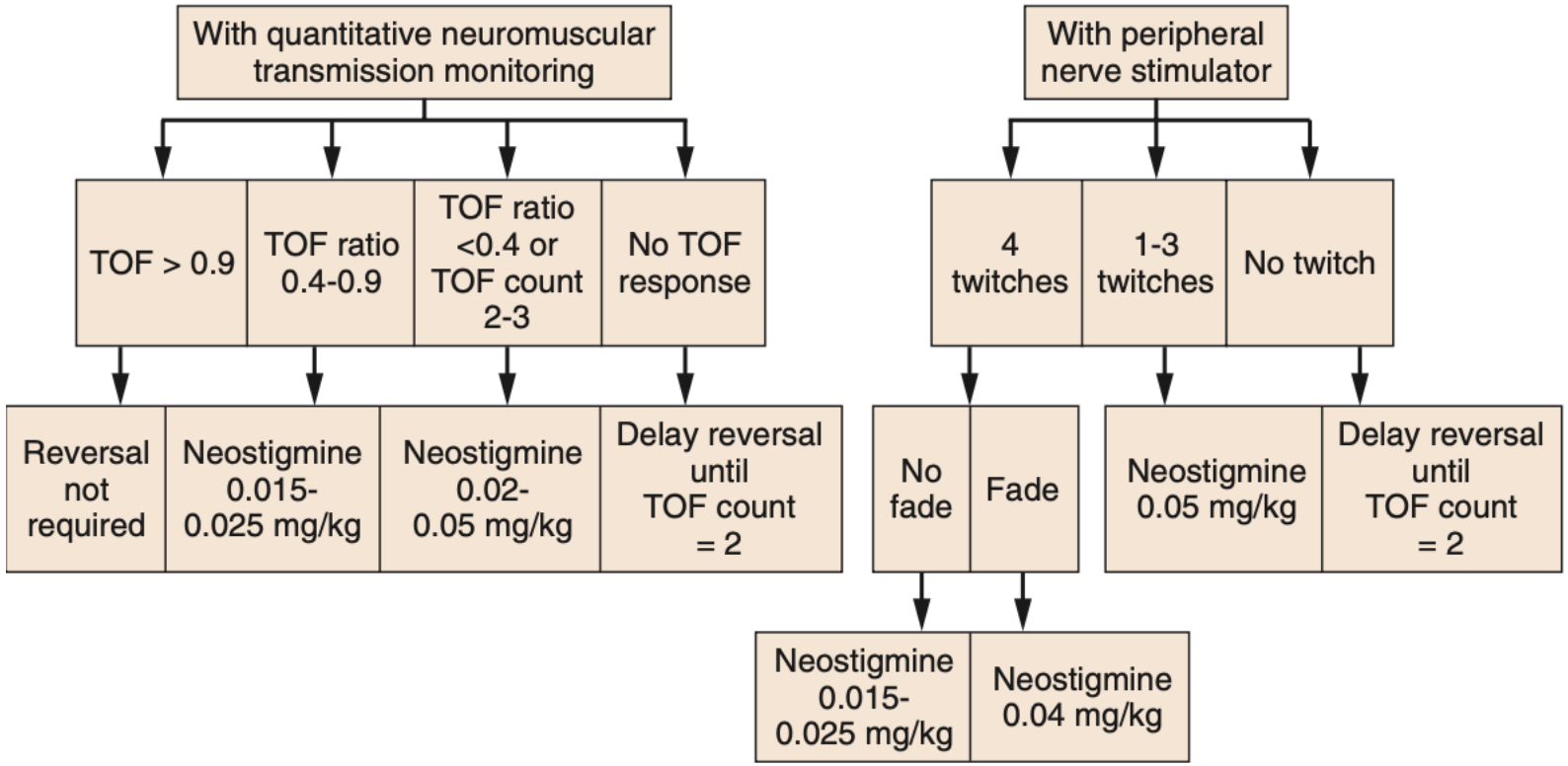
Figure 53-17. The setup of kinemyography (NMT MechanoSensor, Datex-Ohmeda, Helsinki, Finland). The response to nerve stimulation is measured by the bending of a small piezoelectric sensor positioned between the index finger and the thumb.

TABLE 53-1 CLINICAL SIGNS AND SYMPTOMS OF RESIDUAL PARALYSIS IN AWAKE VOLUNTEERS AFTER MIVACURIUM-INDUCED NEUROMUSCULAR BLOCK

Train-of-Four Ratio	Signs and Symptoms
0.70-0.75	Diplopia and visual disturbances Decreased handgrip strength Inability to maintain apposition of the incisor teeth "Tongue depressor test" negative Inability to sit up without assistance Severe facial weakness Speaking a major effort Overall weakness and tiredness
0.85-0.90	Diplopia and visual disturbances Generalized fatigue

Miller RD, Eriksson LI, Fleisher LA, Wiener-Kronish JP, Cohen NH, Young WL. Miller's Anesthesia E-Book. Elsevier Health Sciences; 2014.

Reversal dei Bloccanti neuromuscolari

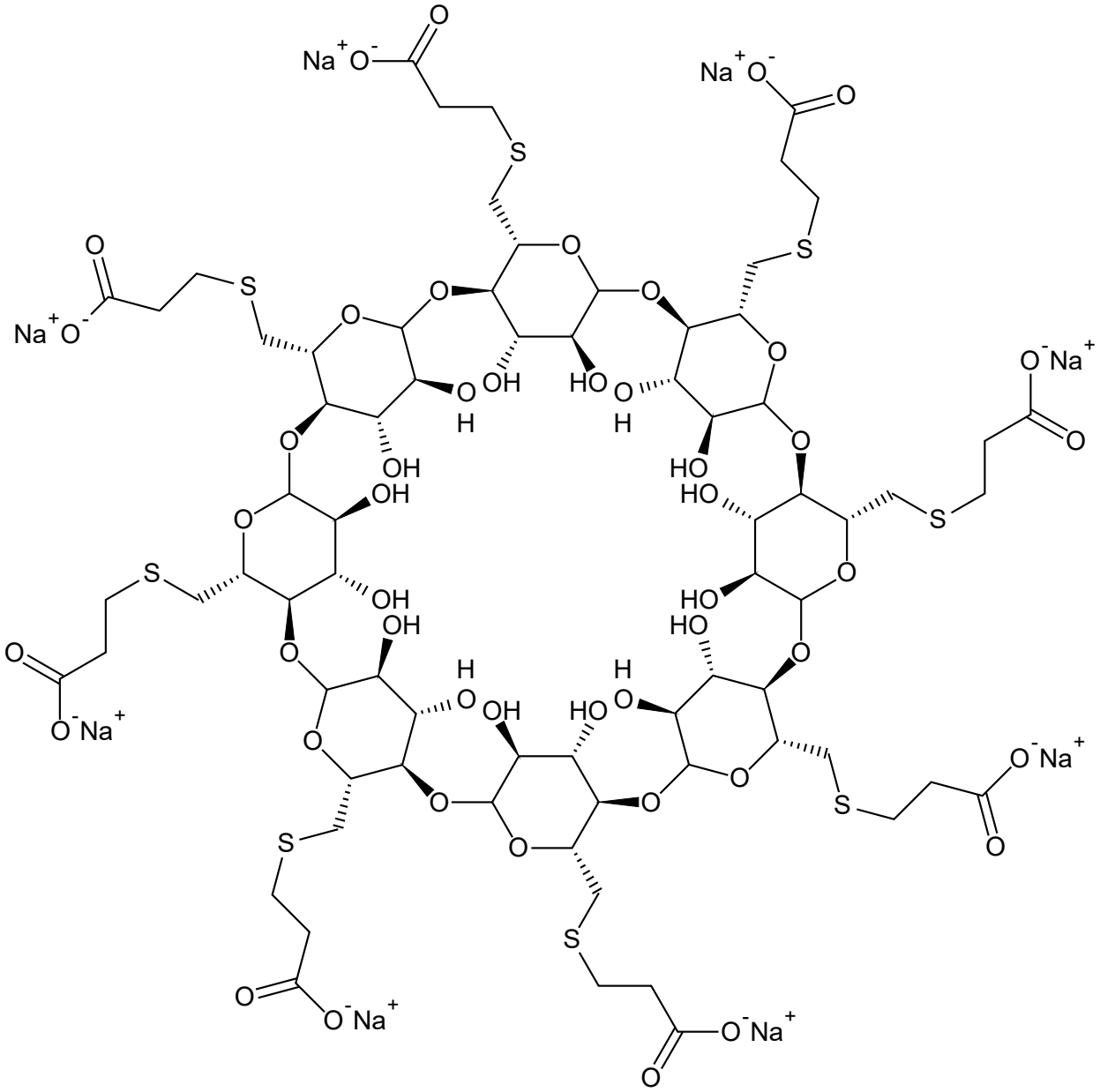


• **Fig. 22.15** Neostigmine dosing. The dose of neostigmine depends on both how depth of neuromuscular block is monitored and the degree of recovery. As little as 0.015 to 0.025 mg kg^{-1} of neostigmine is required at a train-of-four (TOF) count of 4 with no fade, whereas 0.04 to 0.05 mg kg^{-1} is needed at a TOF count of 2 or 3. If only a single twitch or none at all can be evoked, neostigmine will not reverse neuromuscular block and antagonism is best delayed until a TOF count of 2 is achieved. (From Kopman AF, Eikermann M. Antagonism of non-depolarising neuromuscular block: current practice. *Anaesthesia*. 2009;64(Suppl 1):22–30).

Hemmings HC, Egan TD. Pharmacology and Physiology for Anesthesia E-Book: Foundations and Clinical Application. Elsevier Health Sciences; 2018.

Level of neuromuscular block	Dose of sugammadex
Light block: Reappearance of fourth twitch (T4) in response to TOF stimulation	1 mg/kg*
Moderate block: Reappearance of second twitch (T2) in response to TOF stimulation	2 mg/kg†
Deep block: 1–2 PTCs and no twitch responses to TOF stimulation	4 mg/kg†

TOF: train-of-four, PTC: post-tetanic counts. *Dose obtained from the reference [35], †doses obtained from package insert recommendations.



https://www.researchgate.net/publication/332807438_The_potential_risks_of_sugammadex

Domande o interventi?

Monitoraggio

3.1. Funzione cardiocircolatoria

Razionale: Assicurare un'adeguata funzione cardiocircolatoria durante ogni tipo di anestesia.

3.1.1 ECG e frequenza cardiaca. In tutti i pazienti sottoposti alle varie tecniche di anestesia debbono essere monitorizzati in continuo il tracciato elettrocardiografico e la frequenza cardiaca con allarmi di massima e di minima.

3.1.2 Pressione arteriosa. In tutti i pazienti, la pressione arteriosa (sistolica e diastolica) deve essere misurata con tecnica non invasiva ad intervalli di 5 minuti, o ad intervalli maggiormente ravvicinati a giudizio dell'anestesista responsabile. In caso di misurazione continua dei valori pressori, l'intervallo sopra menzionato sancisce la periodicità di registrazione del dato sulla cartella anestesiologica.

3.1.3 In base alle caratteristiche del paziente e della procedura in atto, l'anestesista potrà, a sua discrezione, supplementare il monitoraggio di minima con tecniche invasive o non invasive (ecocardiografia), permettendo la determinazione di importanti parametri quali la pressione arteriosa cruenta, la pressione venosa centrale, la gittata cardiaca o parametri derivati della funzione miocardica.

3.1.4 Il rilievo dei parametri descritti non può essere inteso come sostitutivo dell'osservazione clinica di cui al punto 2.5.

3.2.1. Ossigenazione

3.2.1.1 Concentrazione inspirata di ossigeno. Durante l'anestesia generale la concentrazione di ossigeno erogata al paziente attraverso il circuito respiratorio deve essere determinata mediante un analizzatore di ossigeno dotato di allarme acustico di concentrazione minima.

3.2.1.2. *Pulsossimetro*. Durante tutte le metodiche di anestesia generale, loco-regionale o sedazione è necessario l'impiego continuo di un pulsossimetro. Il pulsossimetro deve disporre di un allarme acustico di minima ed emettere un adeguato segnale ad ogni battito cardiaco.

3.2.1.3. Il rilievo dei parametri descritti non può essere inteso come sostitutivo dell'osservazione clinica di cui al punto 2.5.

3.2.1.1. Capnometria. Durante l'anestesia generale e la sedazione profonda, la ventilazione del paziente deve essere controllata in continuo mediante capnometria. Segni clinici quali le escursioni respiratorie, la frequenza respiratoria e l'auscultazione del torace possono integrare il monitoraggio strumentale.
Nel paziente intubato o ventilato con presidio sovraglottico, il corretto posizionamento delle interfacce di ventilazione deve essere controllato sia mediante capnometria, sia mediante auscultazione del torace subito dopo il loro posizionamento.

Nel paziente in cui non siano stati applicati presidi di ventilazione o di controllo delle vie aeree, lo standard del monitoraggio capnometrico trova razionale nel fatto che, sebbene la CO_2 determinata in respiro spontaneo con sonda nasale non corrisponda alla CO_2 di fine espirazione, il trend di tale parametro rappresenta un indice indiretto certo della ventilazione.

Qualora la sedazione sia di durata ridotta e l'accesso alle vie aree del paziente sia agevole, la capnometria, benché consigliata, può essere rimessa alla decisione motivata e documentata dell'anestesista.

3.2.1.2. Volumi e pressioni di ventilazione. Quando venga impiegata la ventilazione meccanica[15], gli altri parametri misurati ai fini della sorveglianza respiratoria sono: volume corrente inspirato ed espirato, volume/minuto, pressioni di insufflazione, pressione di fine espirazione.
Un dispositivo in grado di segnalare la deconnessione del paziente dal sistema di ventilazione mediante un allarme acustico prioritario (pressione di insufflazione, volume espirato, capnometria) ed una valvola limitatrice della pressione massima di insufflazione[15] debbono essere sempre operanti.

3.2.1.3. Il rilievo dei parametri descritti non può essere inteso come sostitutivo dell'osservazione clinica di cui al punto 2.5.

3.2.2 Anestetici inalatori.

3.2.2.1 Quando l'anestesia generale è condotta con anestetici inalatori, la concentrazione inspirata e di fine espirazione deve essere misurata, al fine di assicurare adeguate concentrazioni anestetiche nella miscela respiratoria.

3. 3 Temperatura corporea.

Razionale: Assicurare l'omeostasi termica durante tutta la durata dell'anestesia.

3.3.1 Durante ogni tecnica di anestesia o sedazione, deve essere assicurata un'adeguata temperatura corporea del paziente. A tale scopo debbono essere sempre disponibili dispositivi per la misurazione e dispositivi di controllo attivo della temperatura corporea.

3.3.2 I sistemi di misura e di controllo attivo debbono essere sistematicamente utilizzati nei soggetti particolarmente vulnerabili al rischio di ipotermia non intenzionale, come ad esempio il neonato, il grande anziano o il paziente sottoposto a procedure di lunga durata e con ampia esposizione tissutale. Le stesse misure di controllo debbono essere sistematicamente applicate nei soggetti a rischio di ipertermia maligna e nei casi in cui la realizzazione della procedura richieda modifiche intenzionali della temperatura corporea.

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3.3.3 La temperatura che maggiormente si avvicina alla temperatura centrale è quella determinata a livello esofageo, naso-faringeo o timpanico. Con le relative limitazioni, possono essere misurate la temperatura rettale o vescicale[18].

3.4 Funzione neuromuscolare

Razionale: Ottimizzare la miorisoluzione durante l'anestesia ed un efficiente recupero dell'attività motoria.

3.4.1 *Monitoraggio neuromuscolare*. Quando vengano impiegati farmaci bloccanti della trasmissione neuromuscolare, deve essere disponibile uno stimolatore periferico per il monitoraggio della trasmissione neuromuscolare. Per stabilire la ripresa della normale attività, deve essere utilizzata la misurazione del Train Of Four (TOF).

3.5 Funzione cerebrale

Razionale: Determinare il livello di ipnosi e prevenire l'insorgenza di awareness intraoperatoria.

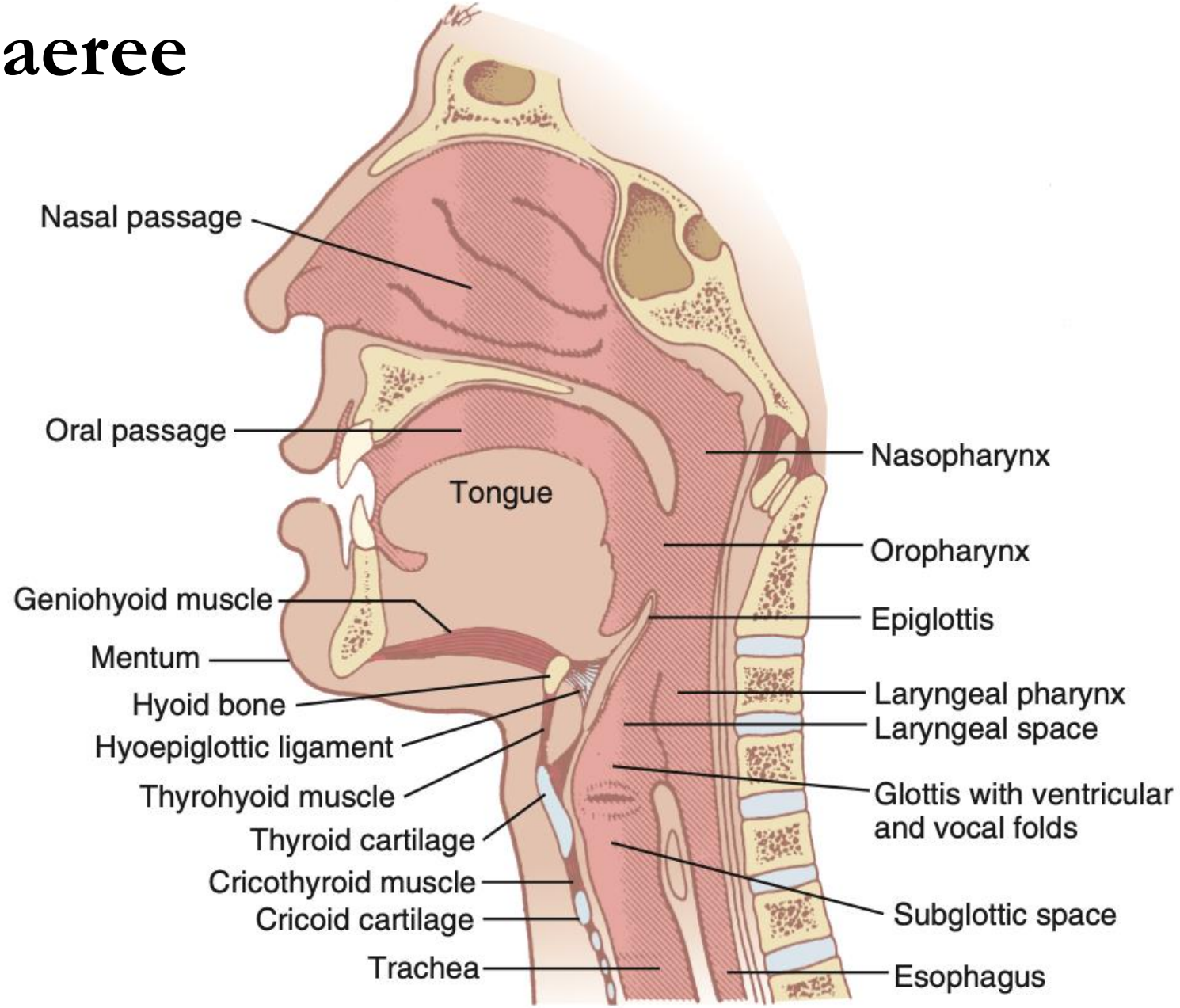
3.5.1 Specifici sistemi di monitoraggio cerebrale basati sull'elaborazione dell'EEG o sui potenziali evocati sono stati introdotti nella pratica clinica. Tuttavia il loro impiego di routine non può essere considerato parte integrale del monitoraggio standard: infatti, la letteratura sulla possibilità di prevenire l'awareness intraoperatoria mediante i sistemi di monitoraggio cerebrale è controversa[19-24].

3.5.2 Per particolari interventi e specifici pazienti indicati dalla letteratura come a maggior rischio di awareness intraoperatoria[22] o per particolari tecniche di anestesia e sedazione, la decisione di impiegare detto monitoraggio è lasciata all'anestesista.

Domande o interventi?

Gestione delle vie aeree

Figure 15-1 Normal anatomy of the airway and surrounding structures is demonstrated in a lateral view of the head and neck in the neutral position. Notice the right-angle geometry of the muscular connections from the mentum to the cricoid cartilage: mentum, geniohyoid muscle, hyoid bone, thyrohyoid muscle, thyroid cartilage, cricothyroid muscle, and cricoid cartilage. This line can be straightened by extending the head at the neck and anteriorly displacing the jaw, pulling the epiglottis and tongue away from the posterior wall of the airway.



Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.

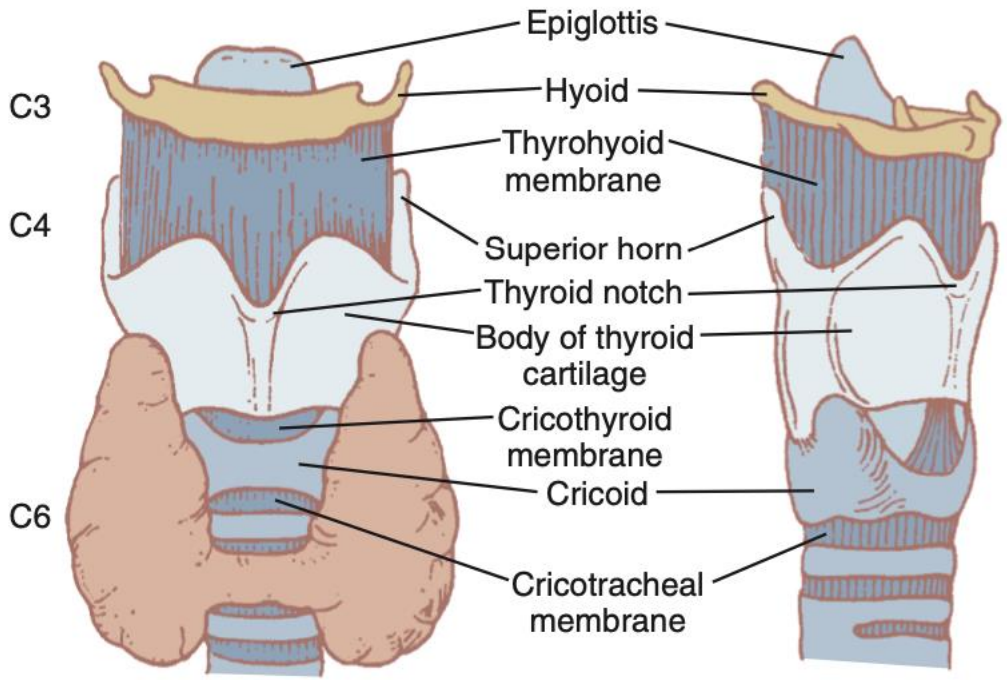
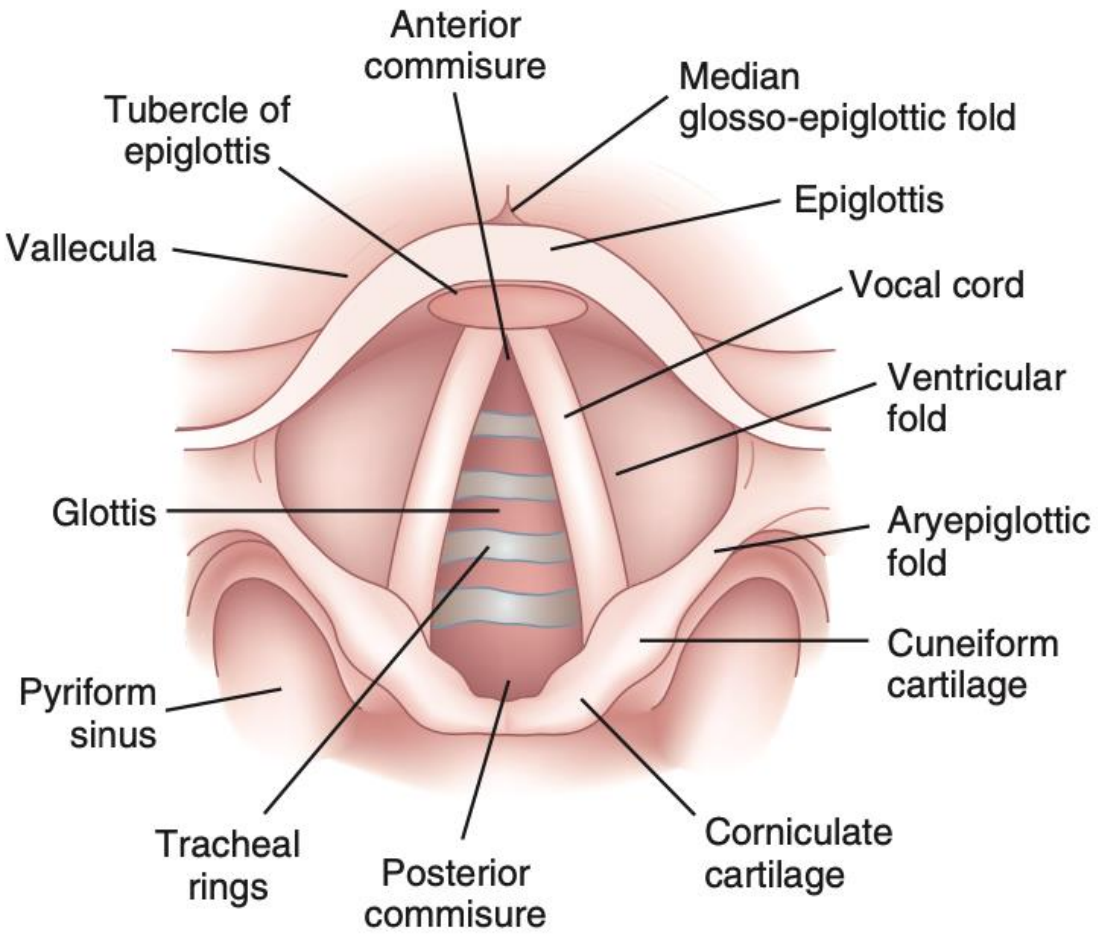


Figure 1-7 External frontal *(left)* and anterolateral *(right)* views of the larynx. Notice the location of cricothyroid membrane and thyroid gland in relation to thyroid and cricoid cartilage in the frontal view. The horn of the thyroid cartilage is also known as the cornu. In the anterolateral view, the shape of the cricoid cartilage and its relation to thyroid cartilage are shown. (Modified from Ellis H, Feldman S: *Anatomy for anaesthetists*, ed 6, Oxford, 1993, Blackwell Scientific.)



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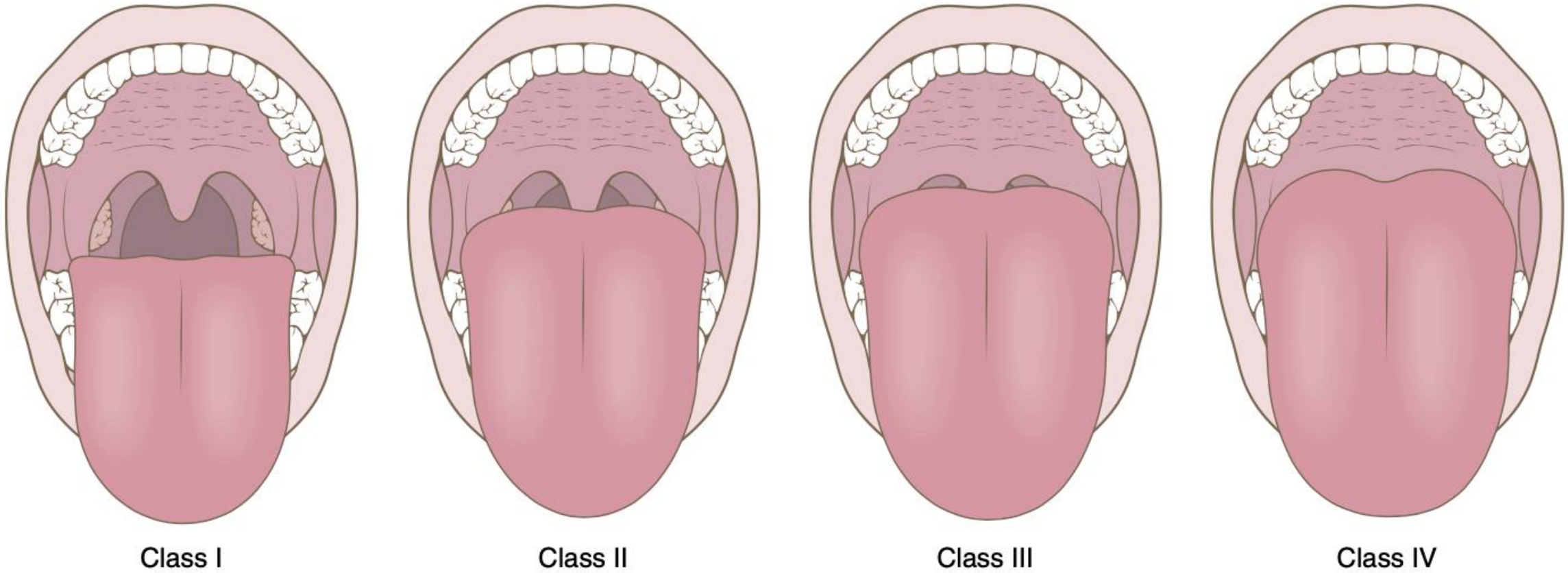


Fig. 16.6 Mallampati classification. (From Samsoon GLT, Young JRB. Difficult tracheal intubation: a retrospective study. *Anaesthesia.* 1987;42:487-490, used with permission.)

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

Table 16.4 Components of the Preoperative Airway Physical Examination

Airway Examination Component	Nonreassuring Findings
Length of upper incisors	Relatively long
Relationship of the maxillary and mandibular incisors during normal jaw closure	Prominent overbite (maxillary incisors anterior to the mandibular incisors)
Relationship of the maxillary and mandibular incisors during voluntary protrusion of the mandible	Patient cannot bring the mandibular incisors anterior to (in front of) the maxillary incisors
Interincisor distance	Less than 3 cm
Visibility of the uvula	Not visible when the tongue is protruded with the patient in a sitting position (Mallampati class higher than II)
Shape of the palate	Highly arched or very narrow
Compliance of the mandibular space	Stiff, indurated, occupied by a mass, or nonresilient
Thyromental distance	Less than three fingerbreadths
Length of the neck	Short
Thickness of the neck	Thick
Range of motion of the head and neck	Patient cannot touch the tip of the chin to the chest or cannot extend the neck

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

TABLE 13-1

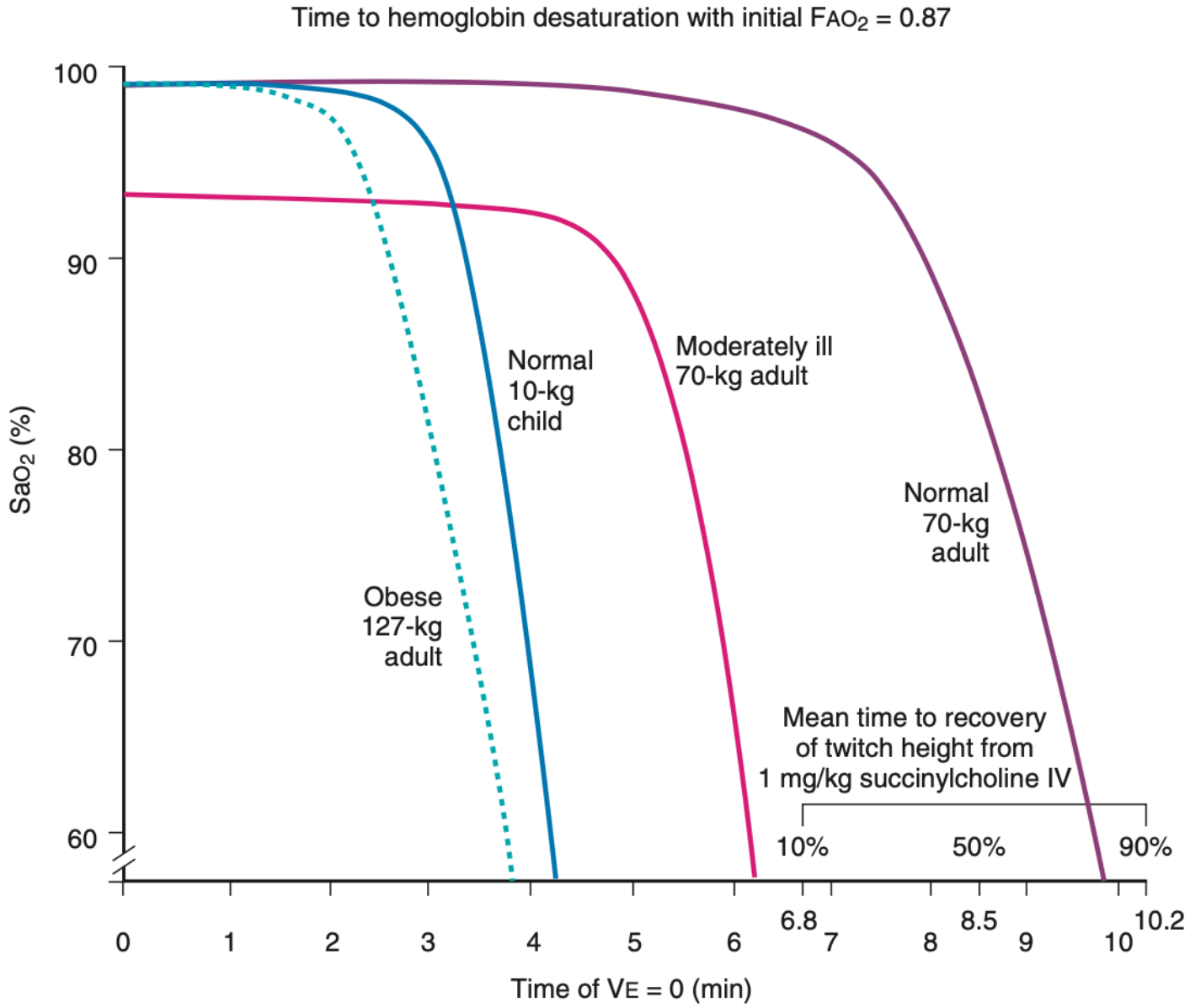
Body O_2 Stores (in mL) during Room Air and 100% O_2 Breathing

Storage Site	Room Air	100% O_2
In the lungs (FRC)	450	3000
In the blood	850	950
Dissolved in tissue fluids	50	100
In combination with myoglobin	200?	200
Total	1550	4250

FRC, Functional residual capacity; *O_2,* oxygen.

From Nunn JF, editor: *Nunn's applied respiratory physiology,* ed 4, Oxford, 1993, Butterworth-Heinemann, p 288.

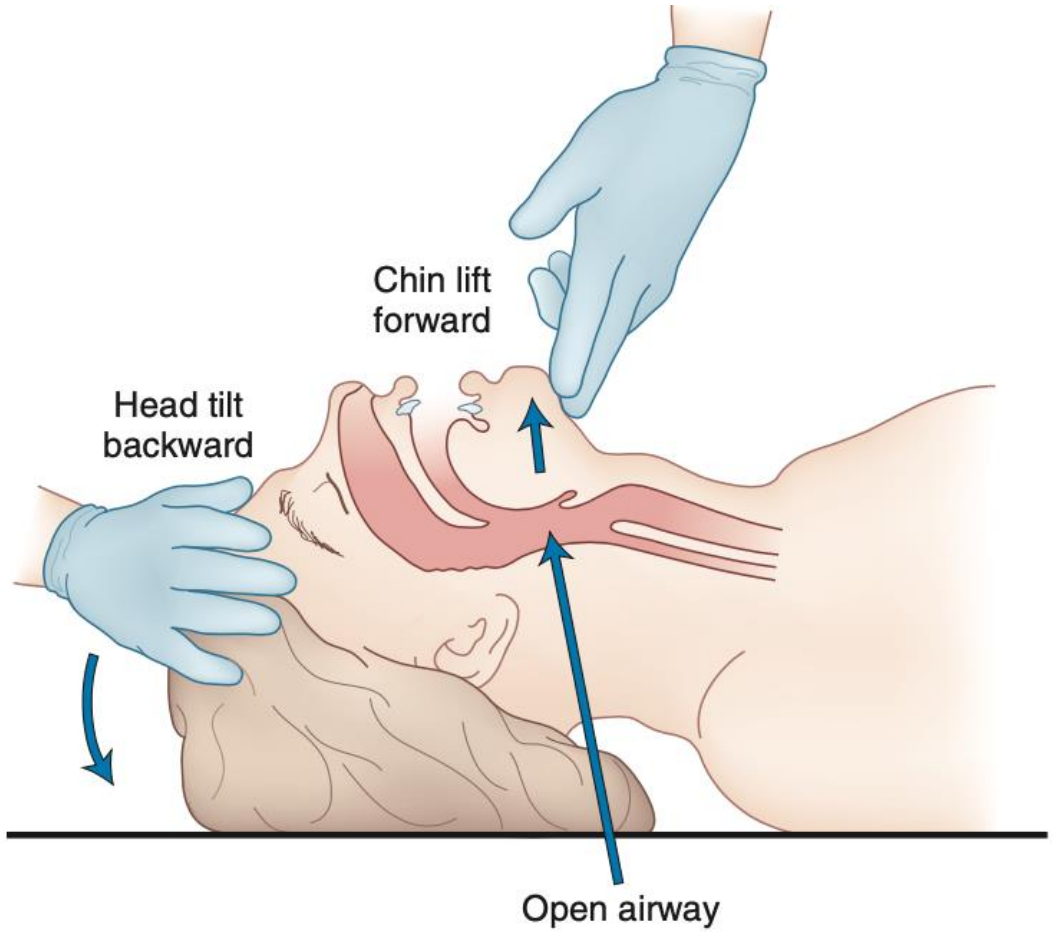
Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.



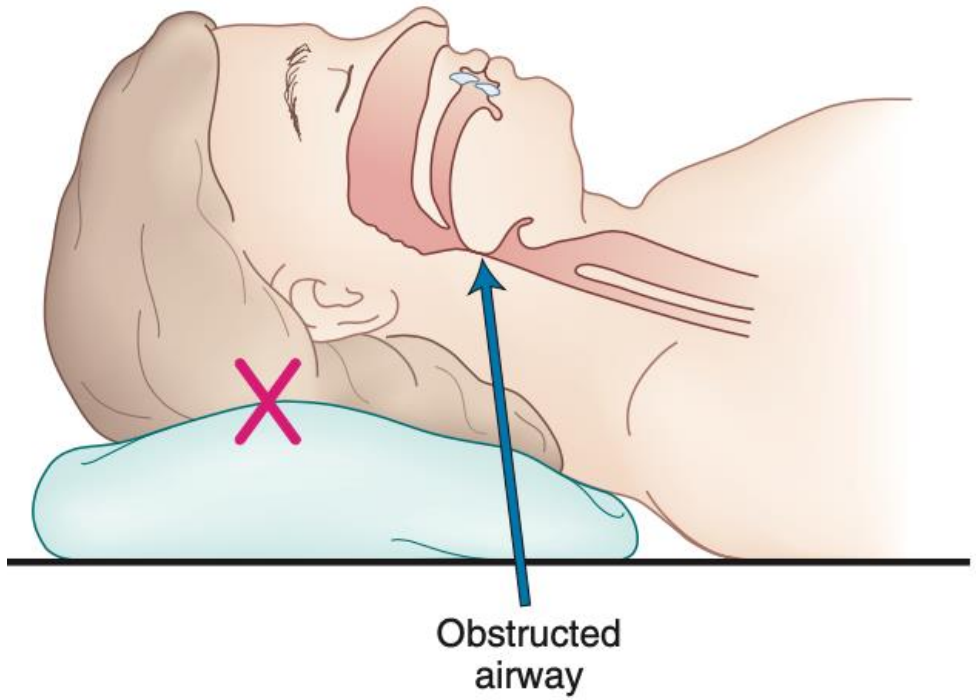
Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.

Figure 13-5 Arterial oxyhemoglobin saturation (Sao_2) versus time of apnea in an obese adult, a 10-kg child (low functional residual capacity (FRC) and high oxygen consumption ($\dot{V}o_2$)), and in a moderately ill adult, compared with a healthy adult. F_{AO_2}, Fractional alveolar oxygen concentration; *VE*, expired volume. (From Benumof JL, Dagg R, Benumof R: Critical hemoglobin desaturation will occur before return to unparalyzed state from 1 mg/kg succinylcholine. *Anesthesiology* 87:979–982, 1997.)

Domande o interventi?



B **Head tilt and chin lift to obtain extended position**



Tongue in apposition to posterior pharyngeal wall

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.

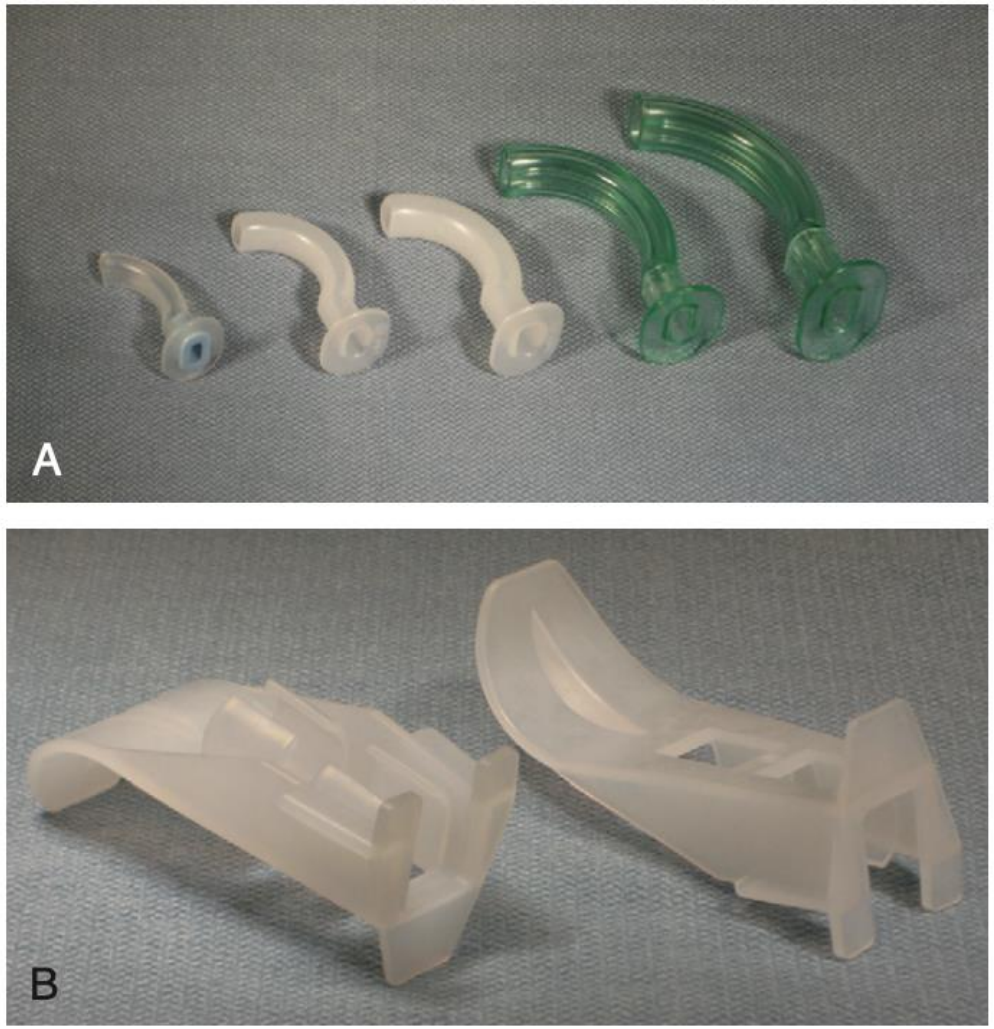
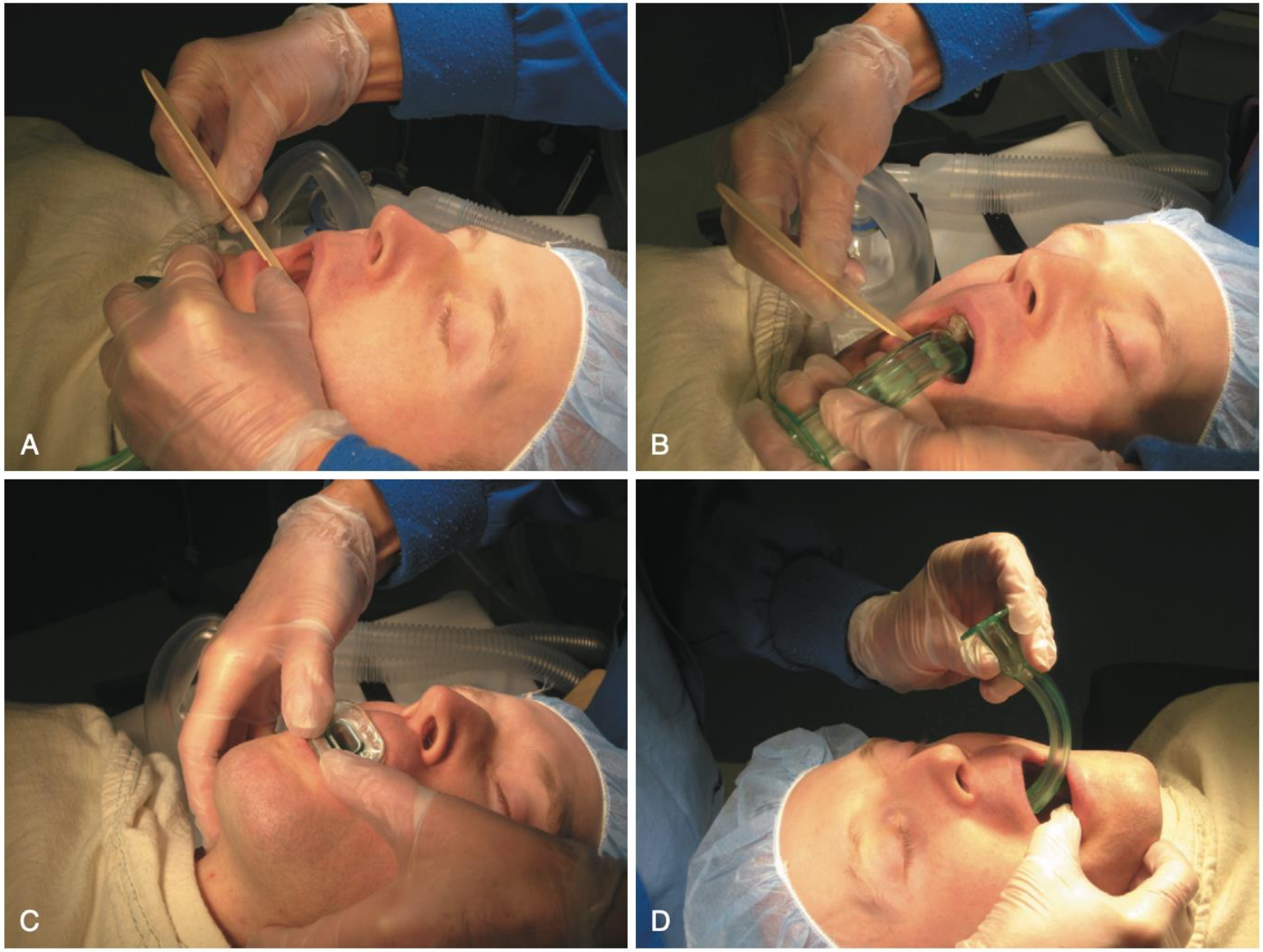


Figure 15-7 Oropharyngeal airways. **A,** Guedel Airways in sizes from neonatal to large adult. **B,** The Ovassapian Airway has a large anterior flange to control the tongue. The airway is open posteriorly (including no posterior flange) so that an endotracheal tube can be inserted with a flexible fiberoptic scope and the assembly later separated.

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.



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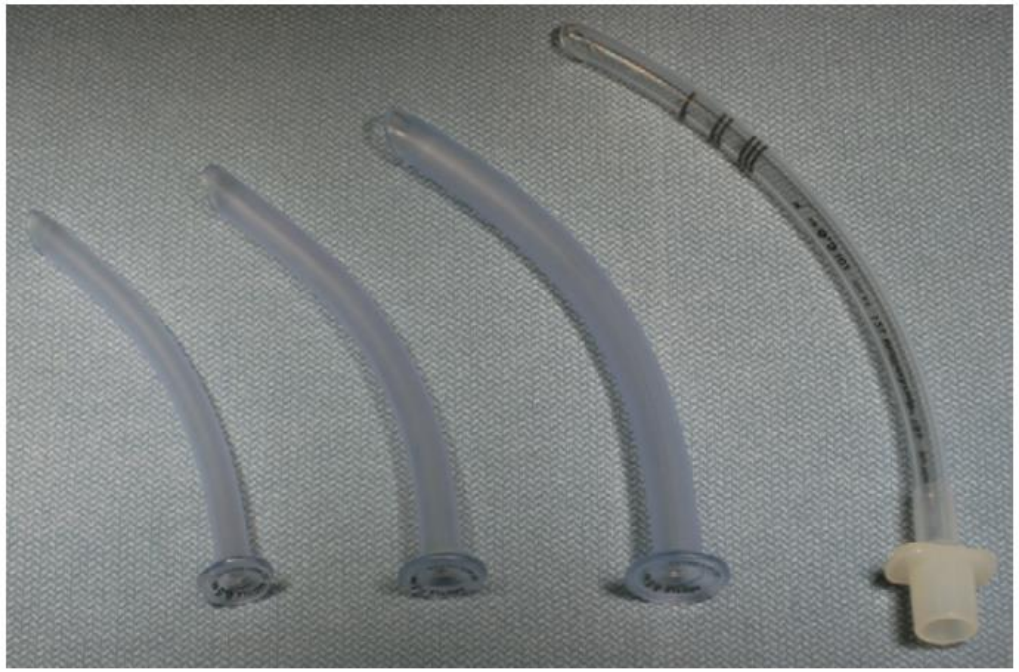


Figure 15-9 Nasopharyngeal airways. A flange prevents the outside end from passing beyond the nares, controlling the depth of insertion. Alternatively, an endotracheal tube may be cut down to provide a longer airway, with its 15-mm adapter reinserted in the cut end.

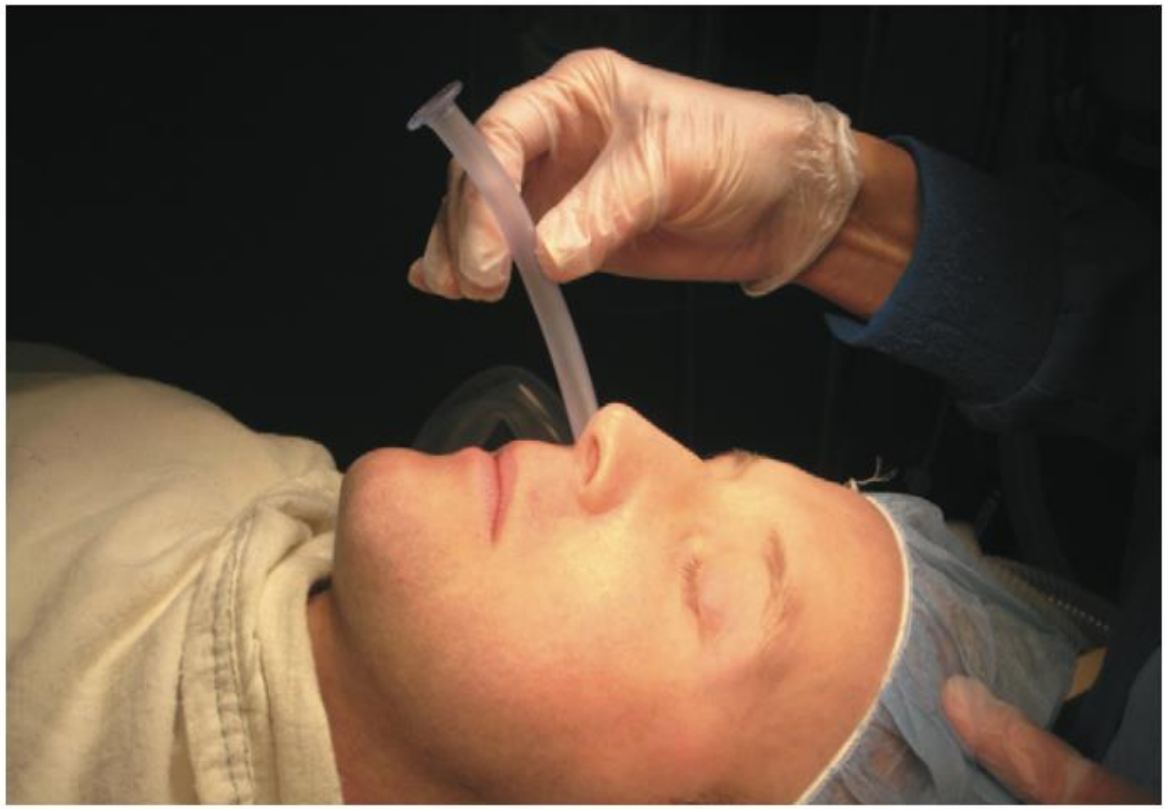


Figure 15-10 Insertion of a nasopharyngeal airway. The airway is oriented with its concave side toward the hard palate and inserted straight posteriorly. Gripping the airway near the top allows the tube to bend if there is resistance to passage. If it is gripped too close to the naris, the clinician can generate sufficient force to shear off a turbinate.

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.

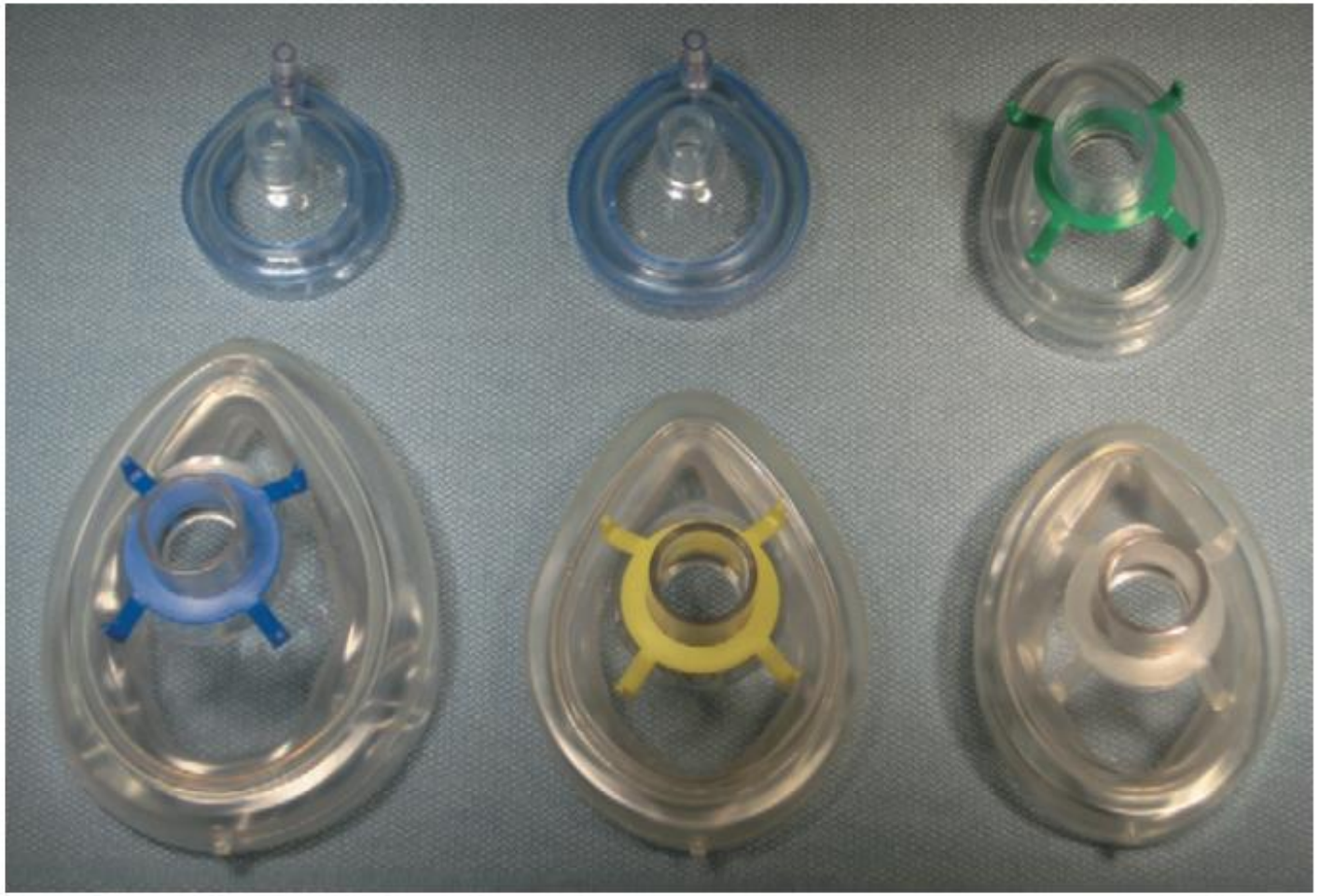


Figure 15-11 Assorted sizes of disposable, transparent face masks. The smallest masks have a 15-mm male adapter, and the larger sizes have a 22-mm female adapter to allow them to be connected to a standard breathing circuit or resuscitator bag.

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.

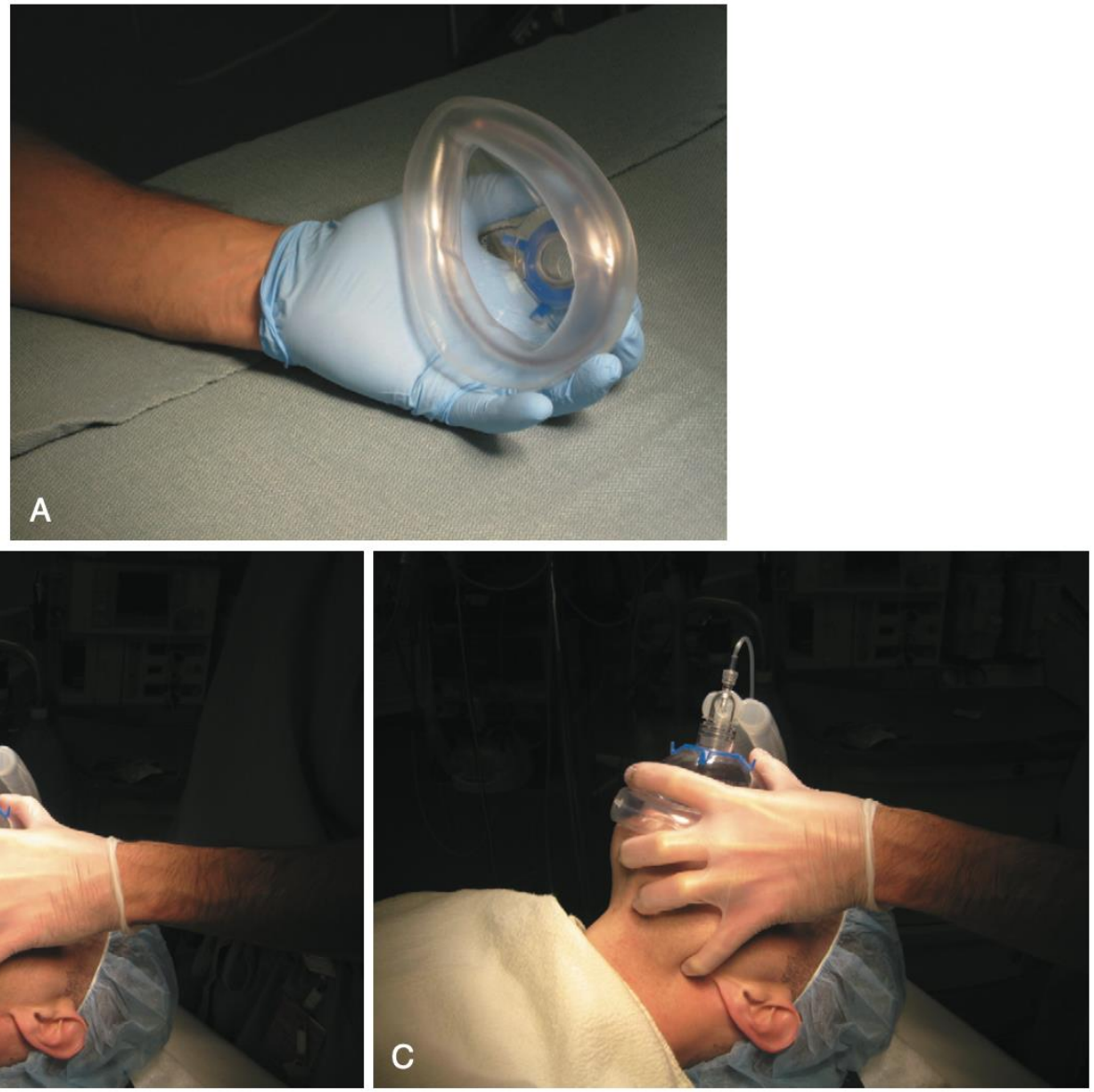


Figure 15-13 Suggested techniques for holding and supporting a face mask. **A,** In the proper hand grip of the face mask, the thumb and index finger encircle the collar while the hypothenar eminence extends below the left side of the mask. **B,** In the side view of the standard one-handed application of the face mask, the thumb and first finger (or first two) encircle the collar of the mask while the remaining fingers pull the mandible up into the mask while gently extending the head. **C,** During the one-handed mask grip with concurrent jaw thrust, notice how the little finger is located at the angle of the jaw, pulling backward and upward to maintain the jaw thrust (subluxation). Because of the increased span of the hand, only the first finger is on the mask while the middle and ring fingers pull the mandible up into the mask and extend the head.

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.

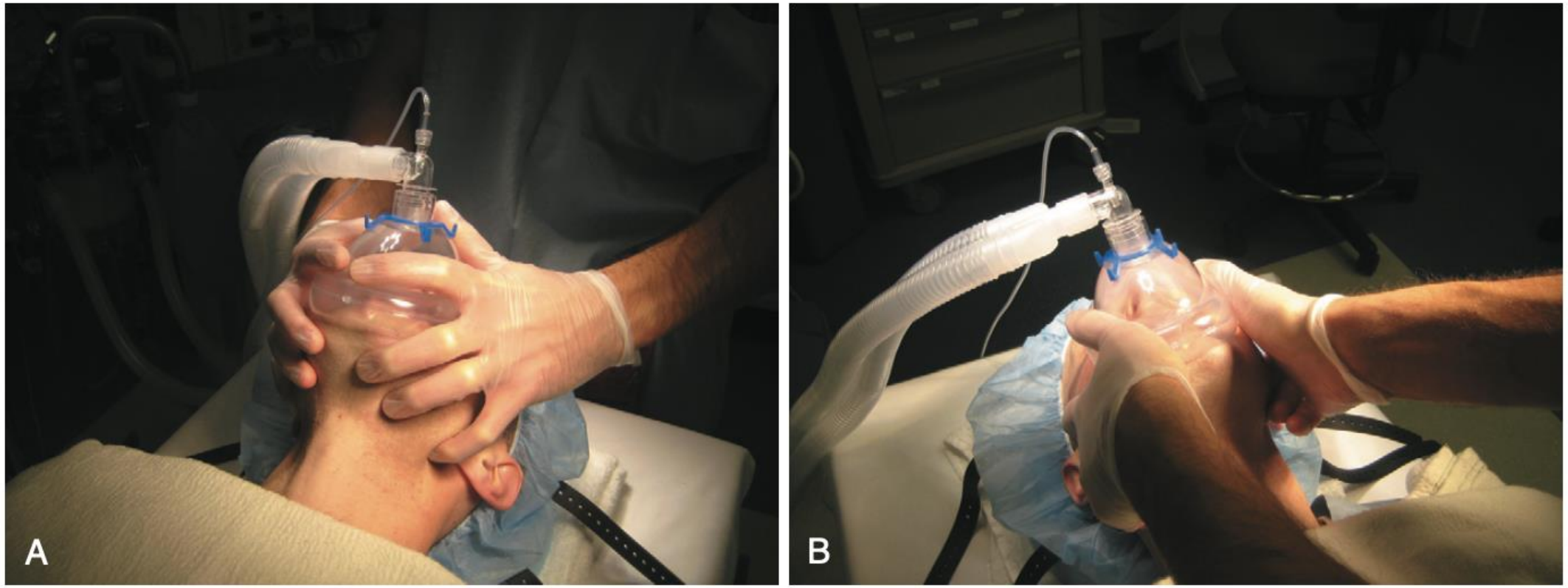


Figure 15-15 Two-hand control of a face mask. In both scenarios, a second provider must ventilate the patient. **A,** In the view of two-hand control of a face mask from above the patient, notice how the lower fingers on both hands apply a jaw thrust while the thumbs seal the mask to the face. **B,** In the view of two-hand control of a face mask from the side of the patient, the person ventilating the patient has improved access to the head as the airway is maintained from the patient's side. This arrangement is beneficial if the ventilating provider is preparing to perform laryngoscopy.

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.

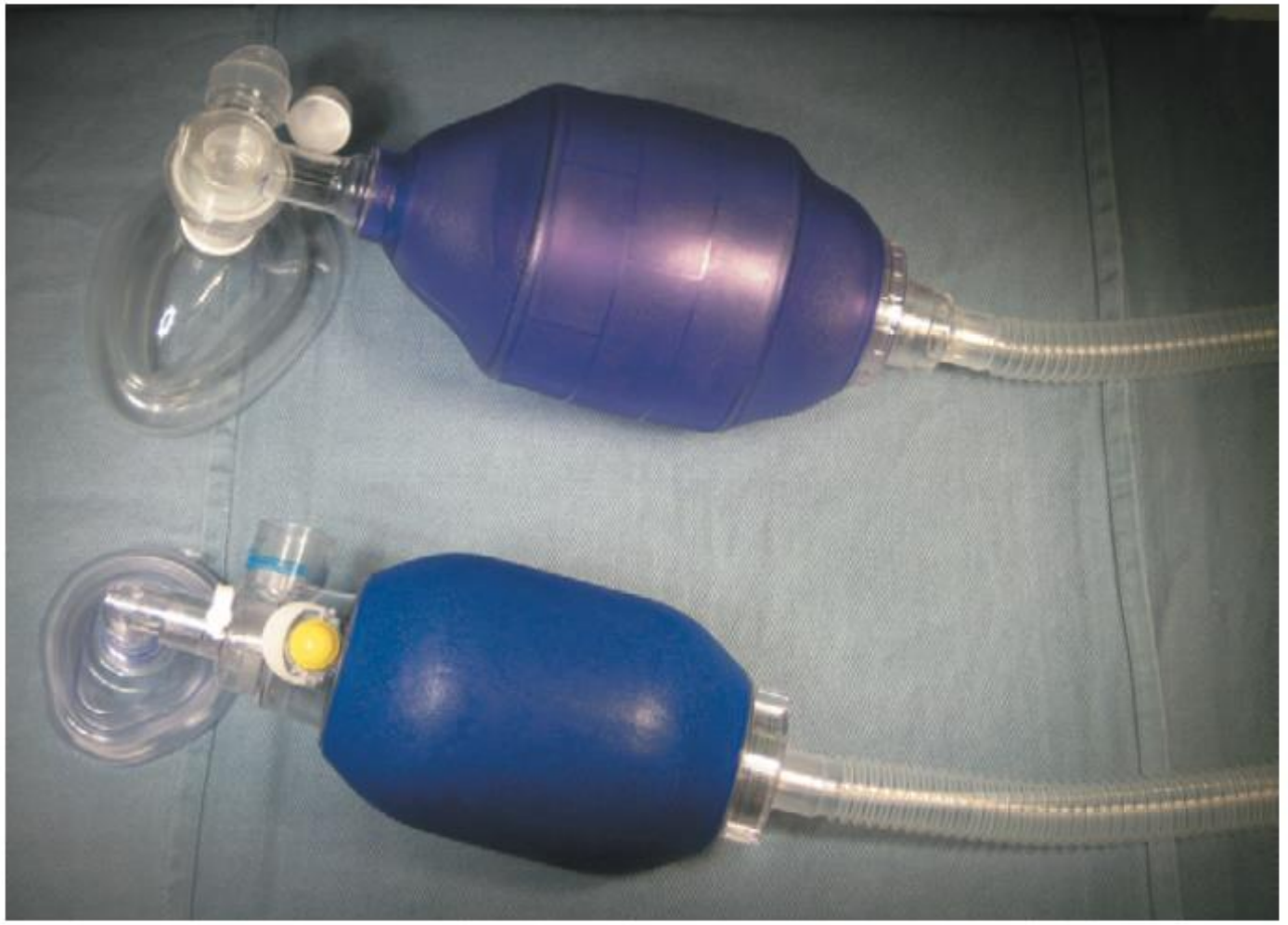
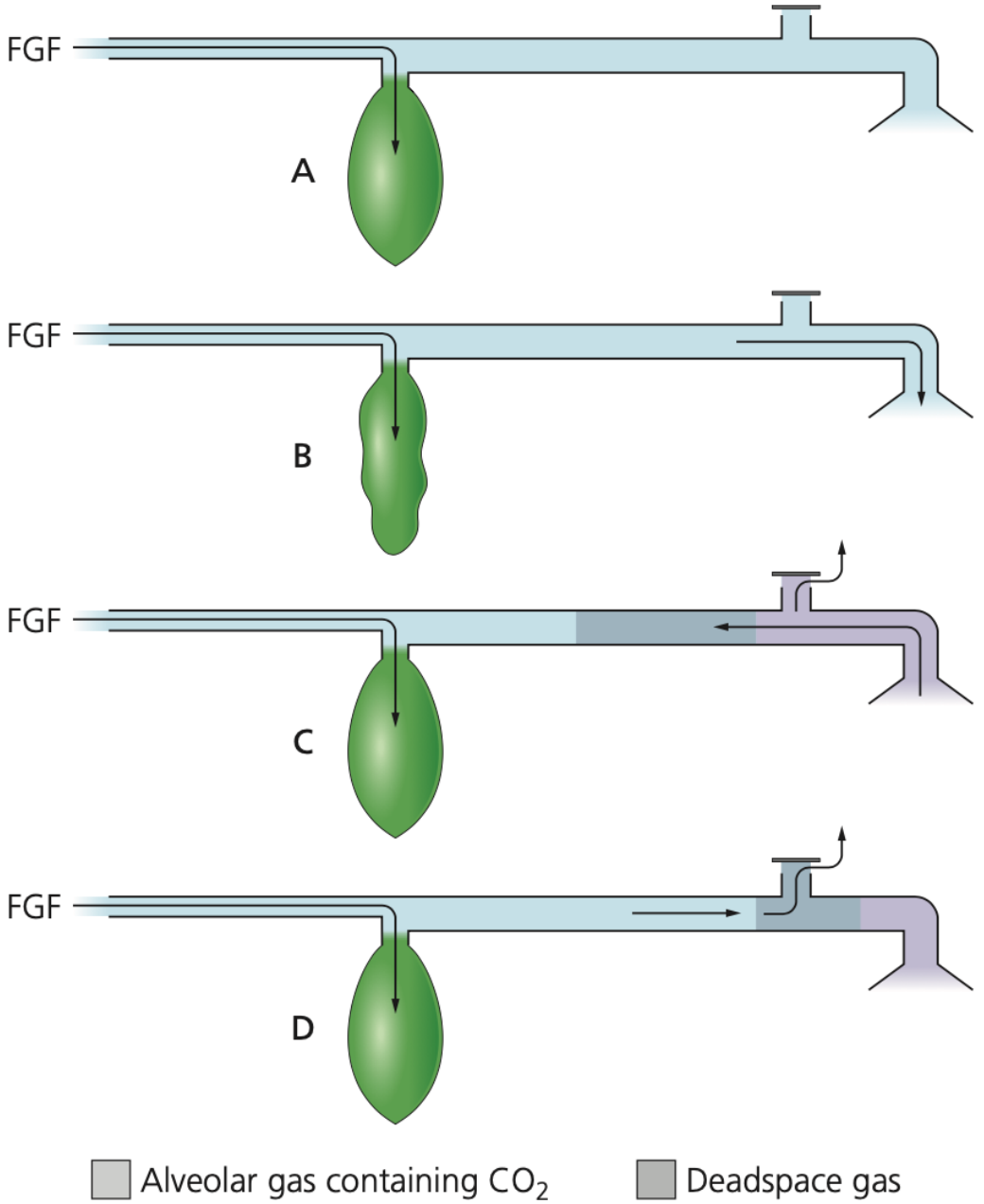


Figure 15-14 Adult and pediatric sizes of air-mask-bag unit (AMBU). The AMBU is a portable, self-inflating, easy-to-use system for the delivery of positive-pressure ventilation. It can be used with a face mask, laryngeal mask airway, or endotracheal tube.

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.

Fig. 4.8 Intersurgical adult Mapleson C system.



Al-Shaikh B, Stacey SG. Essentials of Equipment in Anaesthesia, Critical Care, and Peri-Operative Medicine E-Book. Elsevier Health Sciences; 2017.

Domande o interventi?

BOX 16-1 Benefits of Endotracheal Intubation

1. A patent airway by oral, nasal or tracheal routes
2. Controlled ventilation with up to 100% oxygen
3. Ventilation with high airway pressure
4. Airway protection from aspiration
5. Removal of secretions
6. Lung isolation
7. Administration of medication including anesthetic gases

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.

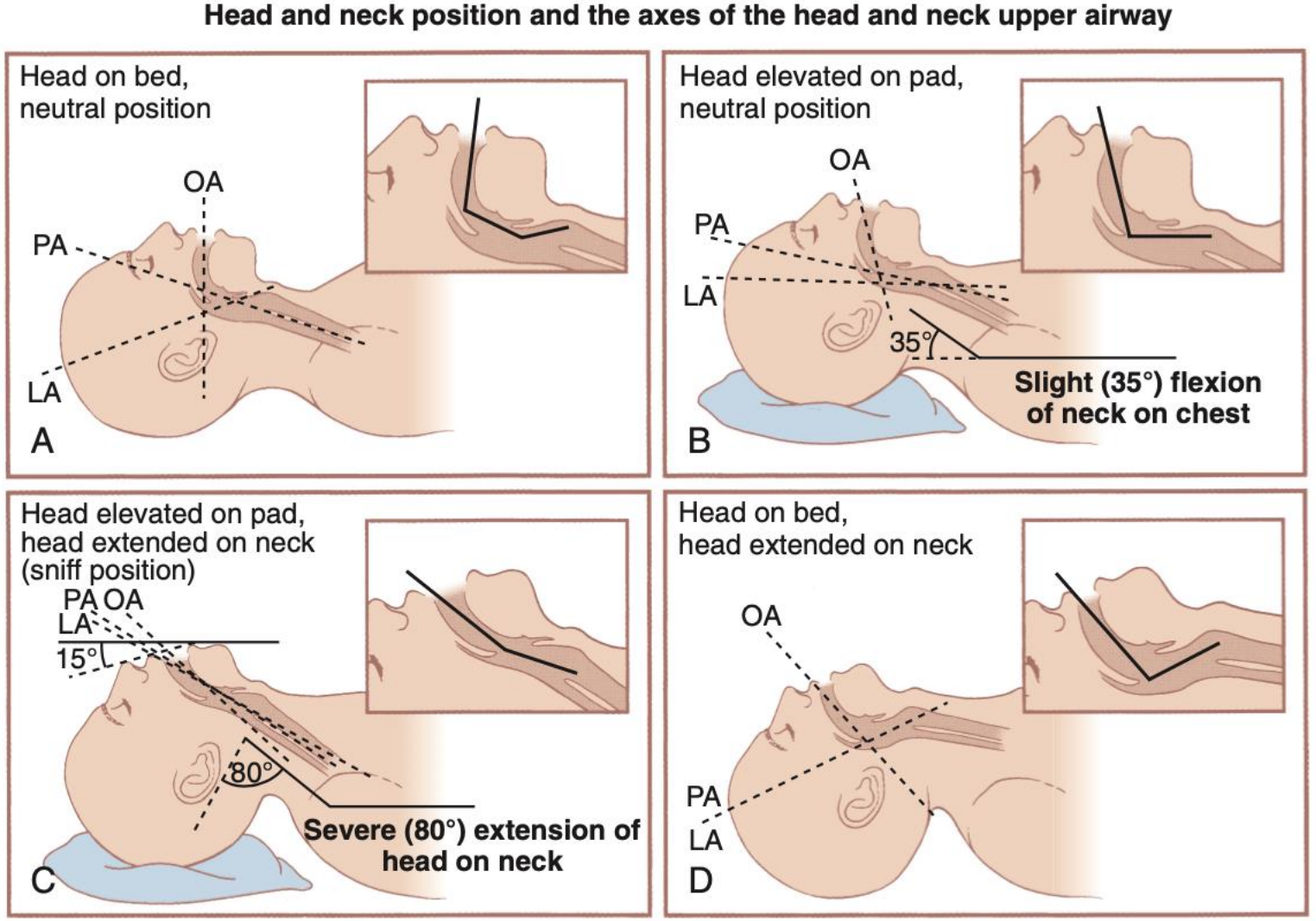


Figure 17-1 Schematic diagrams show the alignment of the oral axis *(OA)*, pharyngeal axis *(PA)*, and laryngeal axis *(LA)* in four different head positions. Each head position is accompanied by an *inset* that magnifies the upper airway (oral cavity, pharynx, and larynx) and superimposes *(bent bold line)* the continuity of these three axes within the upper airway. **A,** The head is in the neutral position with a marked degree of nonalignment of the LA, PA, and OA. **B,** The head is resting on a large pad that flexes the neck on the chest and aligns the LA with the PA. **C,** The head is resting on a pad (which flexes the neck on the chest). Concomitant extension of the head on the neck brings all three axes into alignment (sniffing position). **D,** Extension of the head on the neck without concomitant elevation of the head on a pad, which results in nonalignment of the PA and LA with the OA. (From Benumof JL, editor: *Airway management: principles and practice*, St. Louis, 1996, Mosby, p 263.)

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.

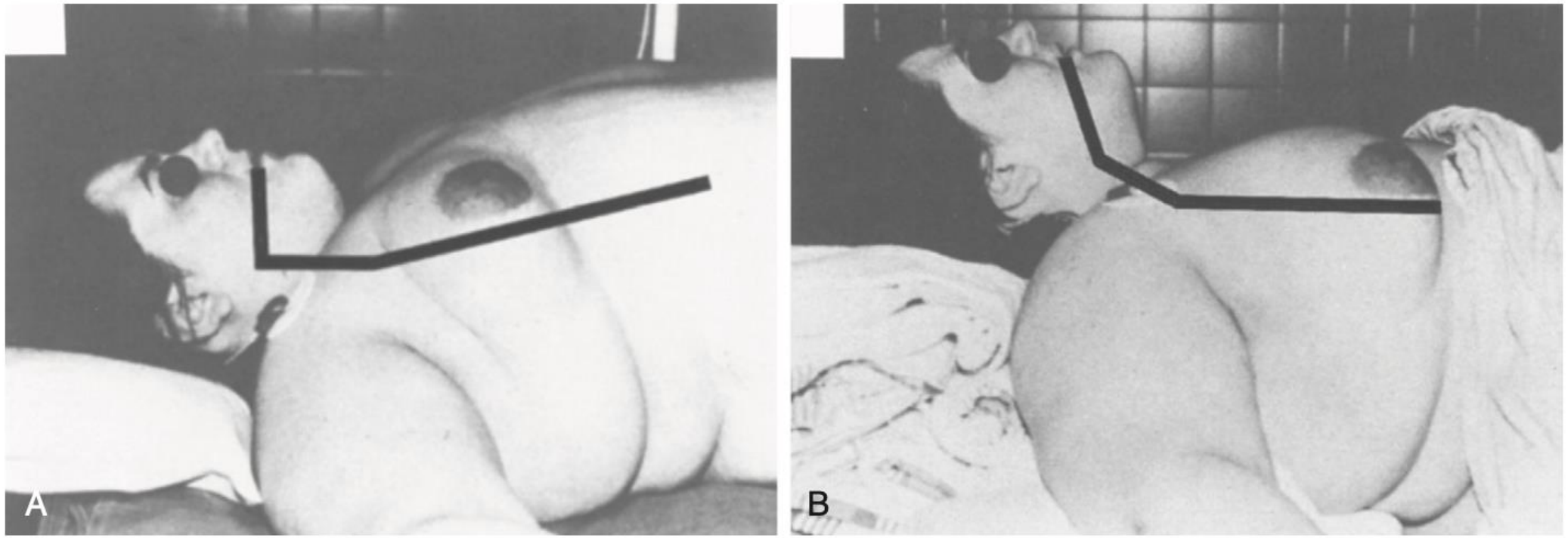
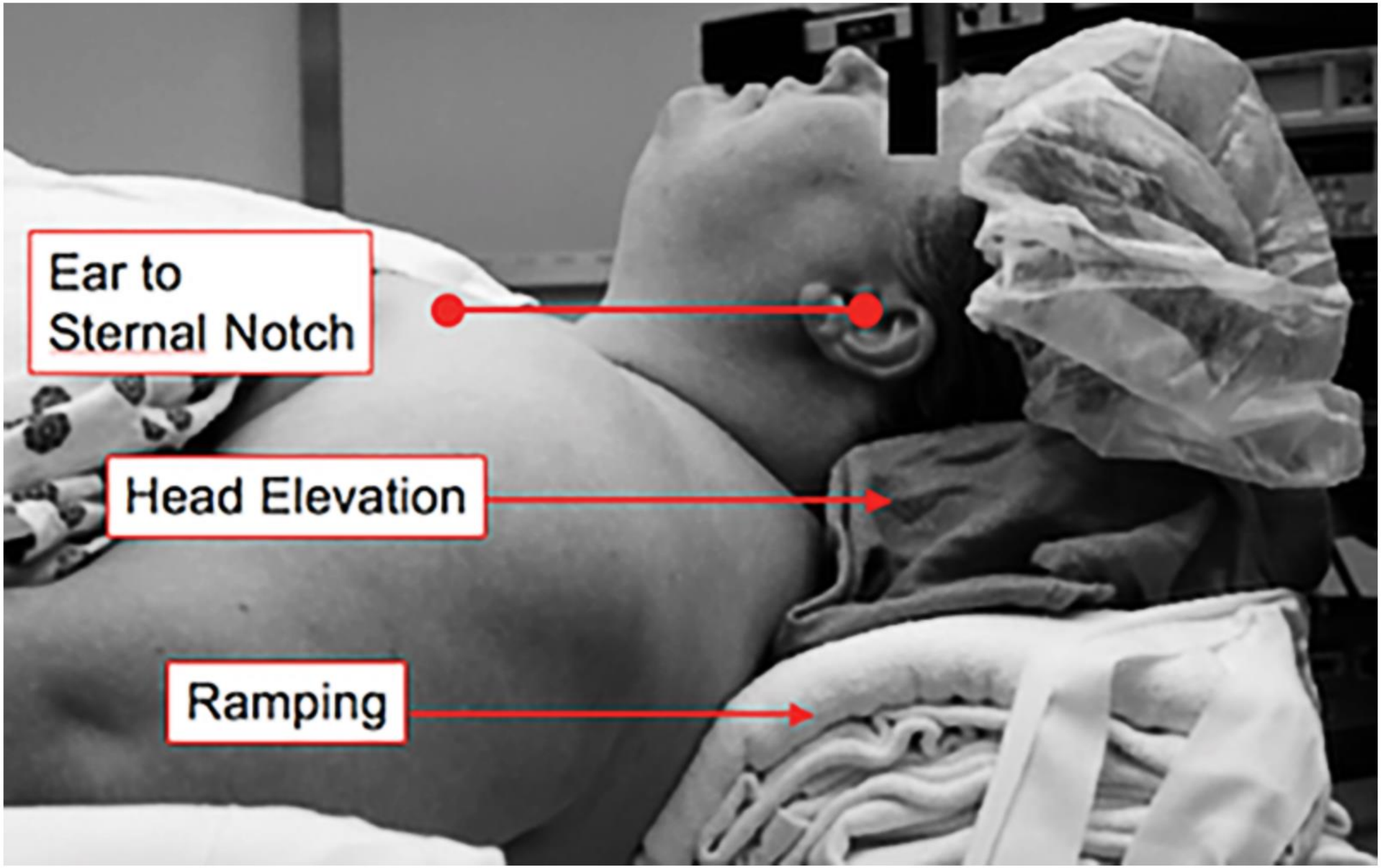


Figure 17-2 **A,** In some obese patients, placing the head on a pillow does not result in the sniffing position; in the obese patient shown and as illustrated by the overlying *bold black line*, the oral and laryngeal axes are perpendicular to one another, the neck is not flexed on the chest, and the head is not extended on the neck at the atlanto-occipital joint. **B,** In the same patient, placing support (e.g., blankets, towels) under the scapula, shoulders, nape of the neck, and head results in a much better sniffing position; the oral, pharyngeal, and laryngeal axes form only a slightly bent curve, the neck is flexed on the chest, and the head is extended on the neck at the atlanto-occipital joint. (From Benumof JL, editor: *Airway management: Principles and practice*, St. Louis, 1996, Mosby, p 264.)

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.



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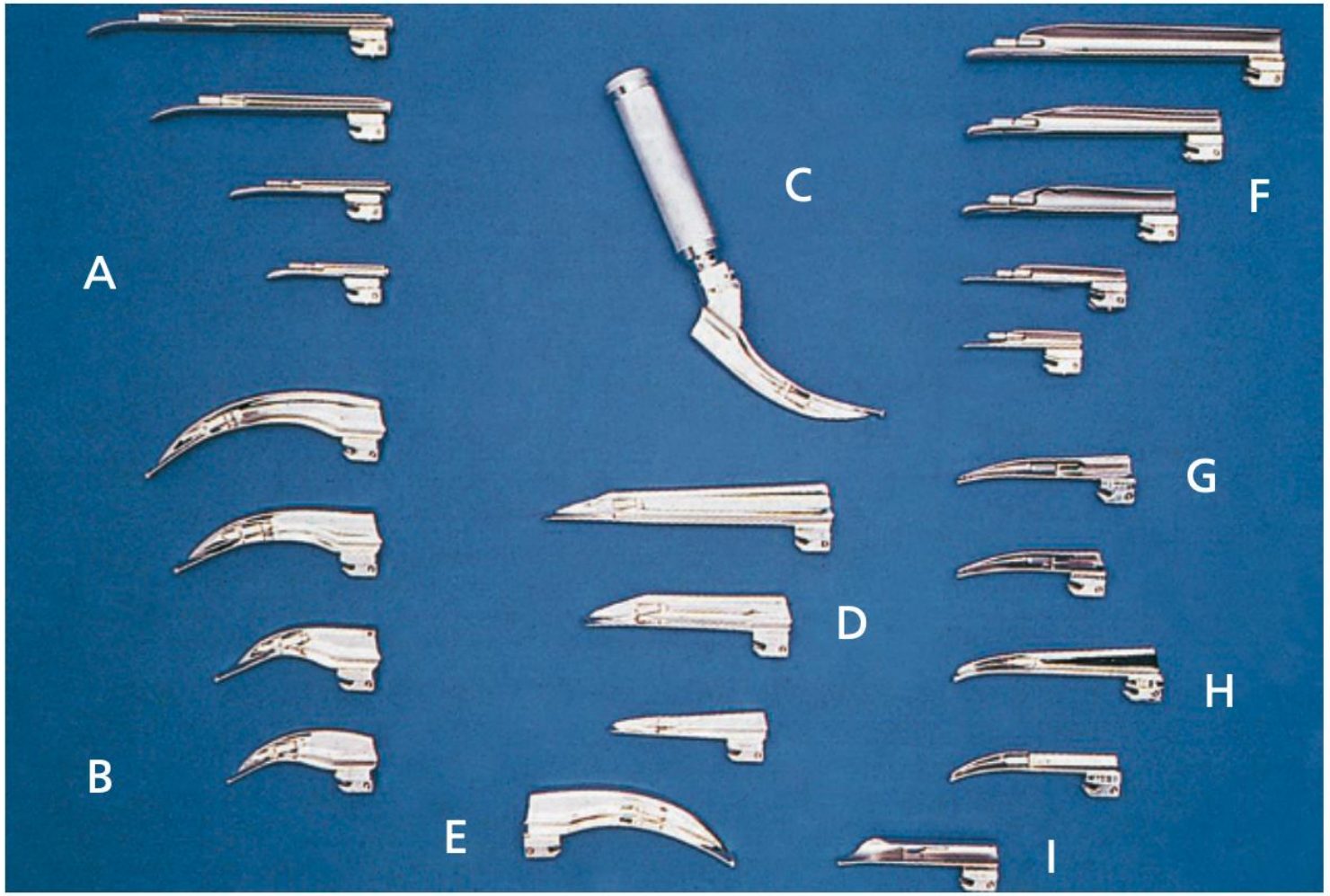
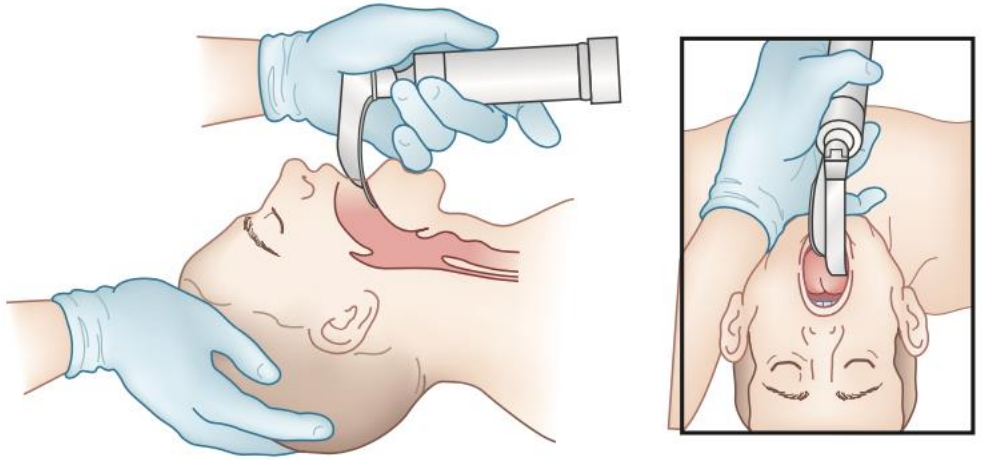


Fig. 7.2 A wide range of laryngoscope blades. (A) Miller blades (large, adult, infant, premature); (B) Macintosh blades (large, adult, child, baby); (C) Macintosh polio blade; (D) Soper blades (adult, child, baby); (E) left-handed Macintosh blade; (F) Wisconsin blades (large, adult, child, baby, neonate); (G) Robertshaw blades (infant, neonatal); (H) Seward blades (child, baby); (I) Oxford infant blade.

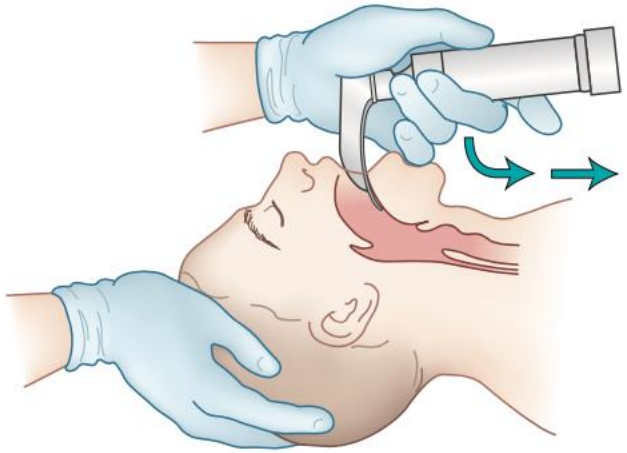
Fig. 7.6 Demonstrating the McCoy laryngoscope's hinged blade tip.

Al-Shaikh B, Stacey SG. Essentials of Equipment in Anaesthesia, Critical Care, and Peri-Operative Medicine E-Book. Elsevier Health Sciences; 2017.

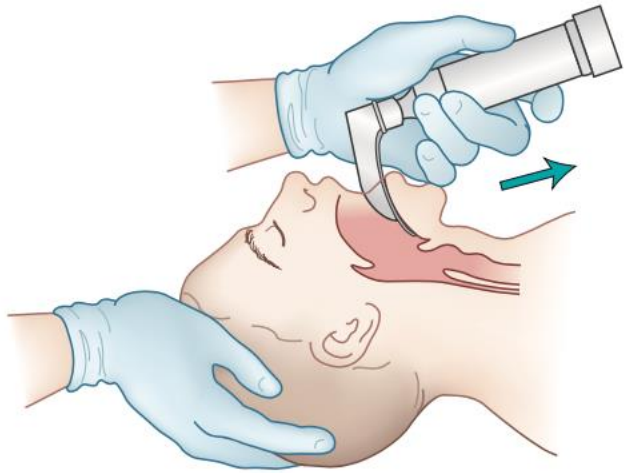
Conventional Laryngoscopy with a Curved Blade



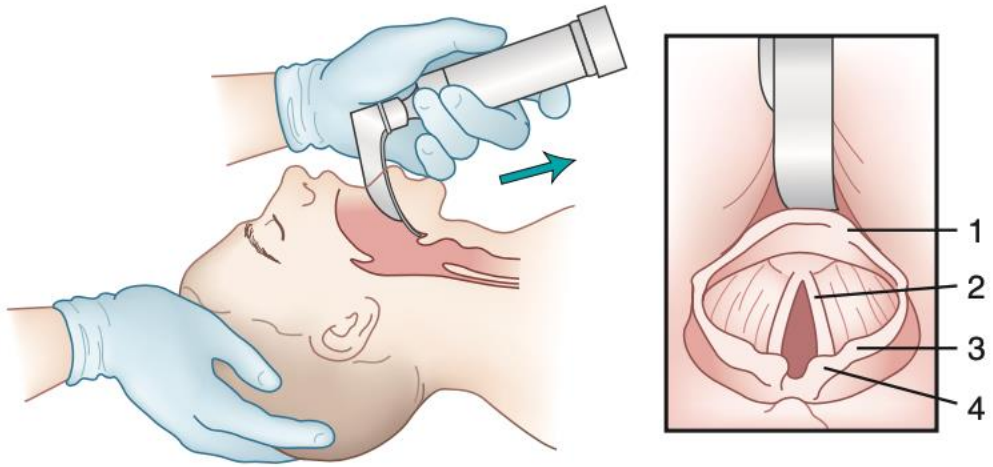
A Insert the laryngoscope blade into the right side of the mouth



B Advance the laryngoscope blade toward the midline of the base of the tongue by rotating wrist

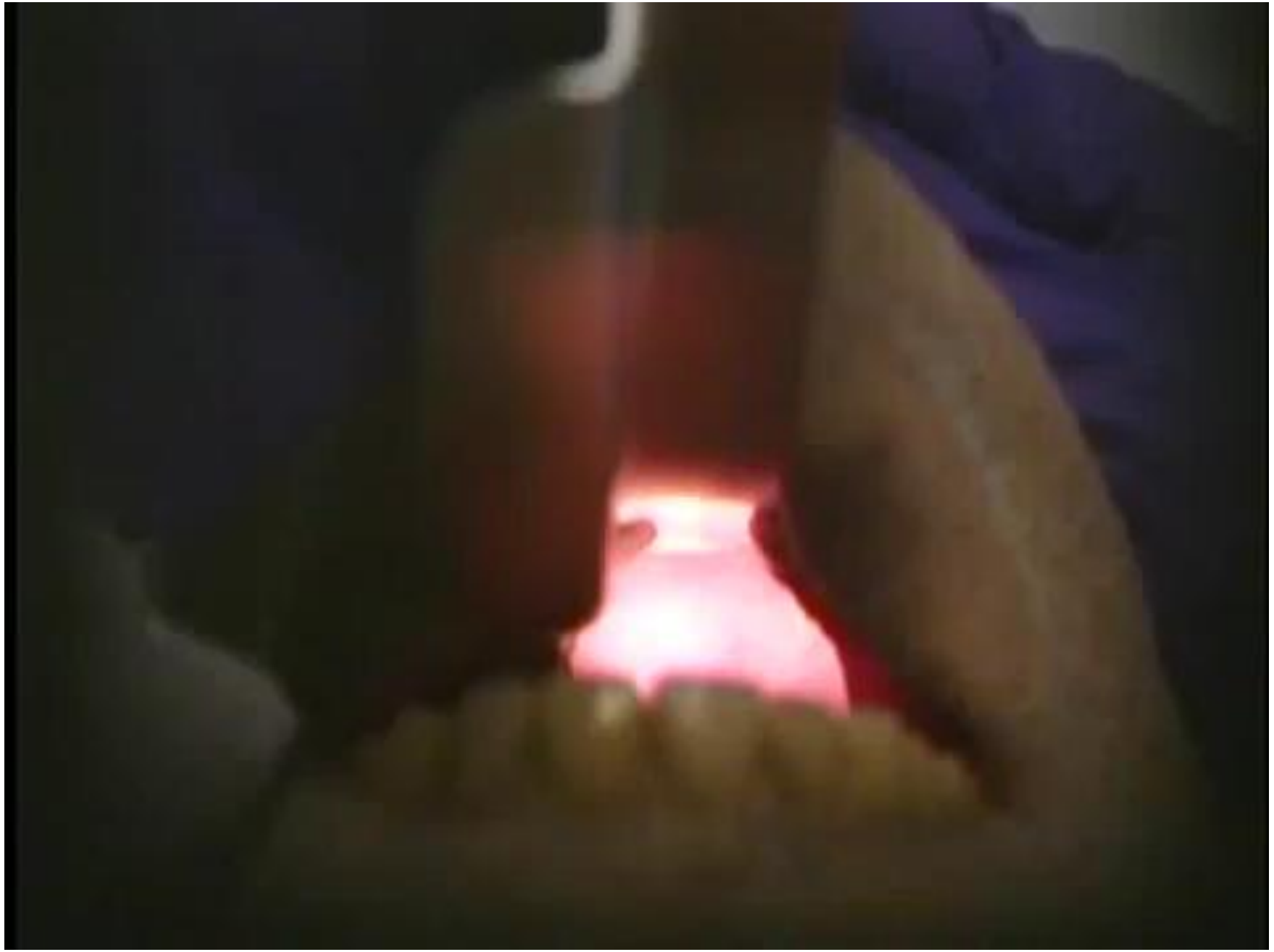


C Approach the base of the tongue and lift the blade forward at a 45° angle



D Engage the vallecula and continue to lift the blade forward at a 45° angle

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.



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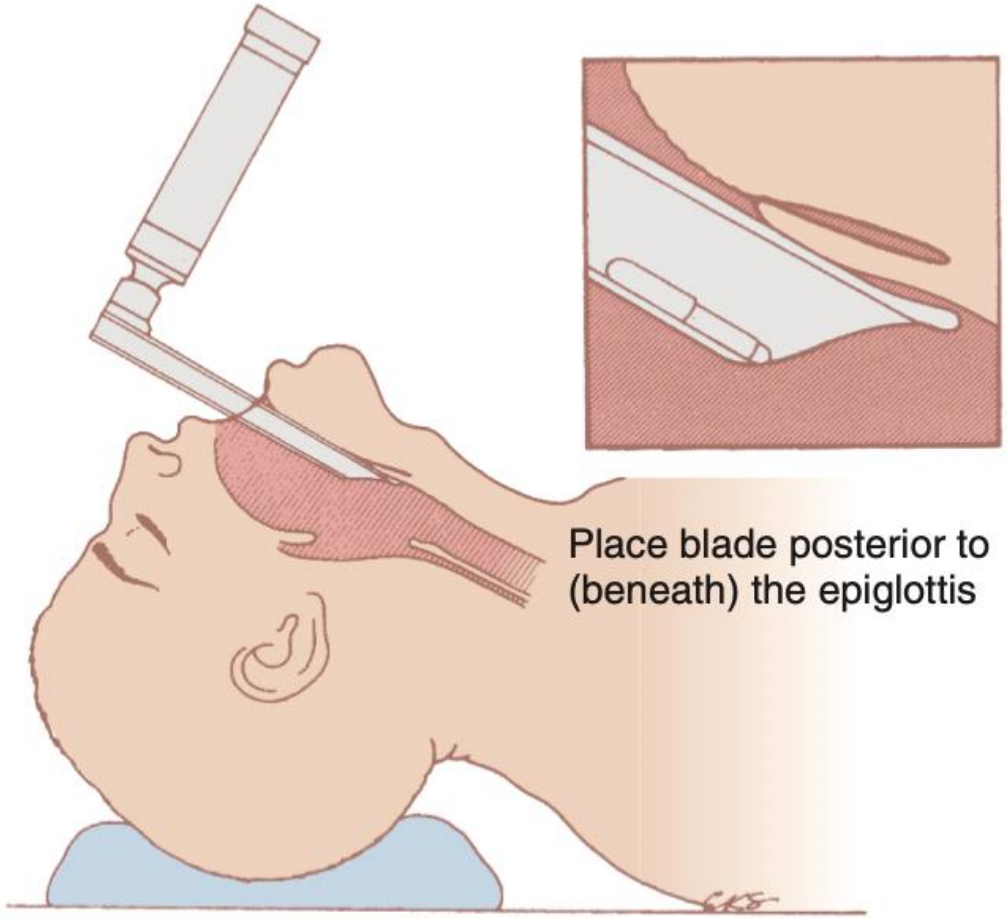
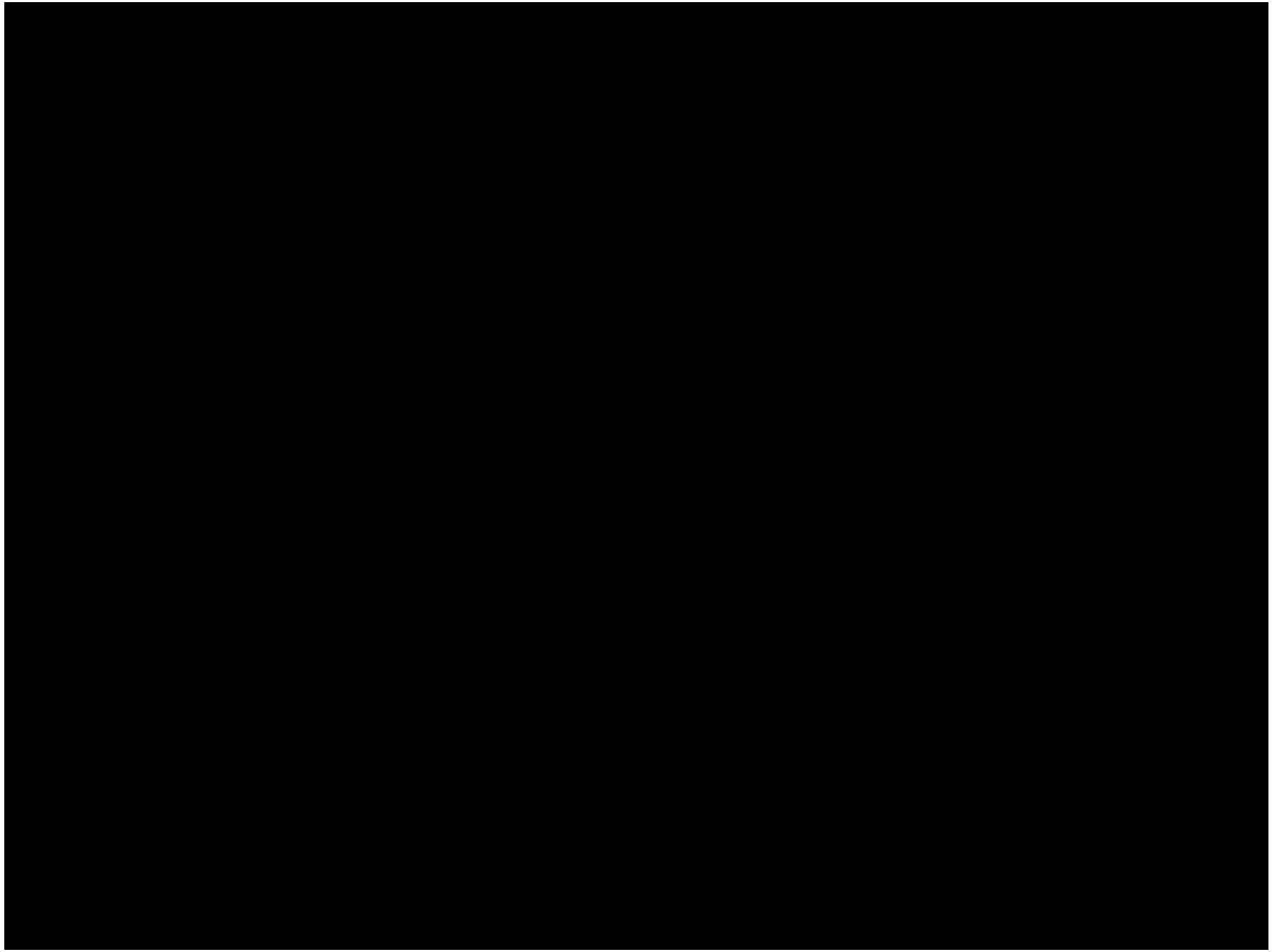


Figure 17-6 Conventional laryngoscopy with a straight blade. A straight laryngoscope blade (Miller blade) should be passed underneath the laryngeal surface of the epiglottis. The handle of the laryngoscope then should be elevated at a 45-degree angle, similar to the lifting that takes place with the use of a curved laryngoscope blade. (From Benumof JL, editor: *Airway management: Principles and practice,* St. Louis, 1996, Mosby, p 268.)

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.



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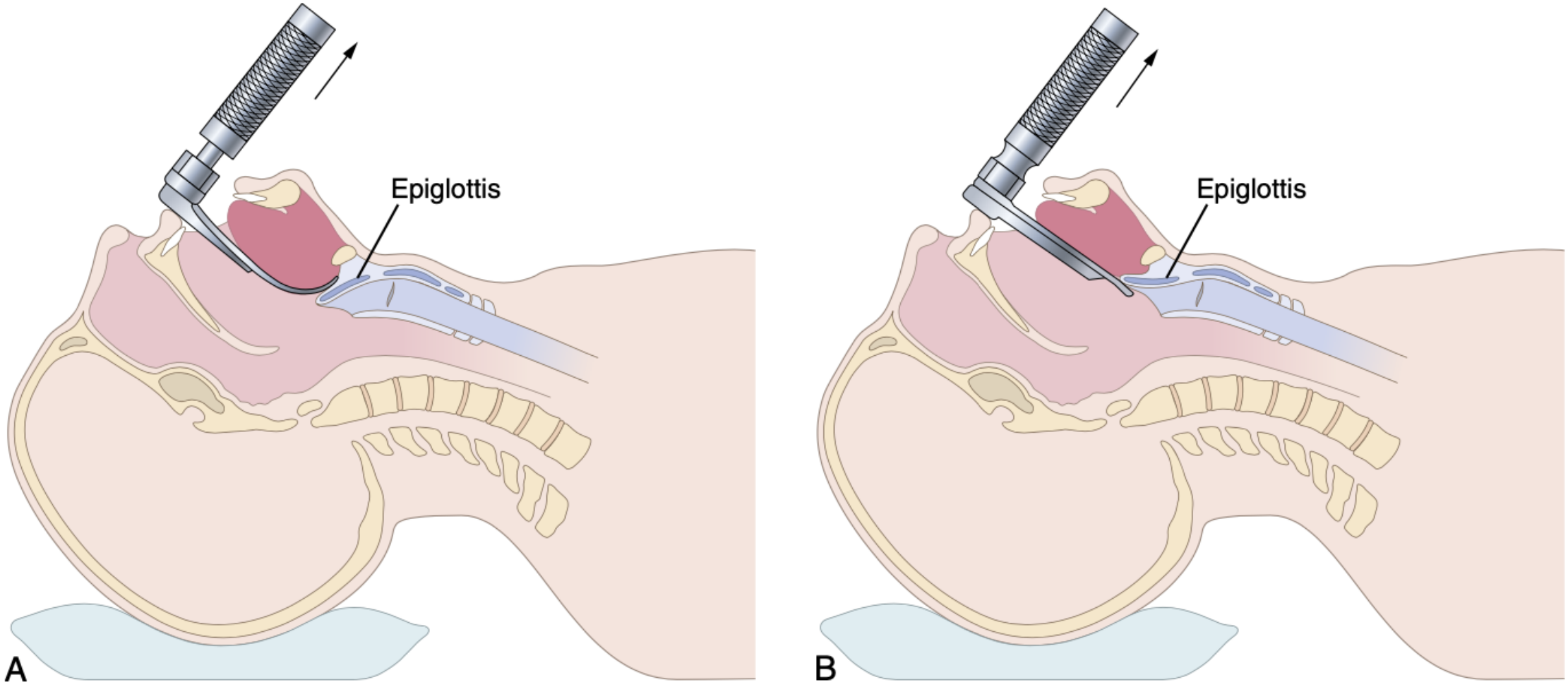


Fig. 16.13 Schematic diagram depicting the proper position of the laryngoscope blade for exposure of the glottic opening. (A) The distal end of the curved blade is advanced into the space between the base of the tongue and the pharyngeal surface of the epiglottis. (B) The distal end of the straight blade is advanced beneath the laryngeal surface of the epiglottis. Regardless of blade design, forward and upward movement exerted along the axis of the laryngoscope handle, as denoted by the arrows, serves to elevate the epiglottis and expose the glottic opening.

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

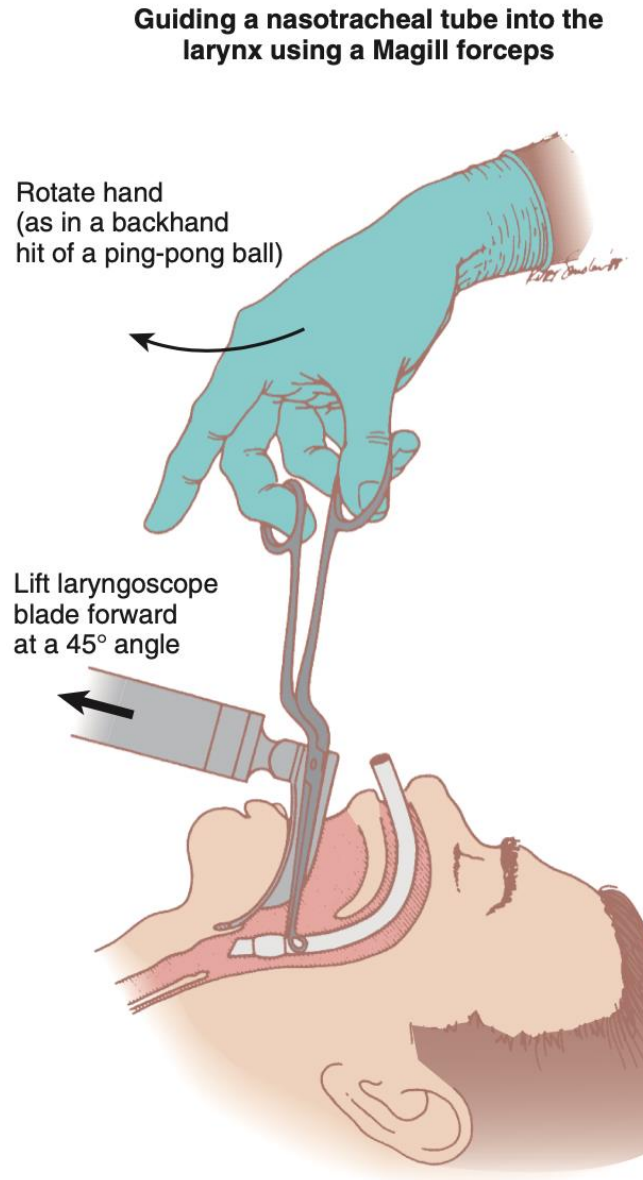
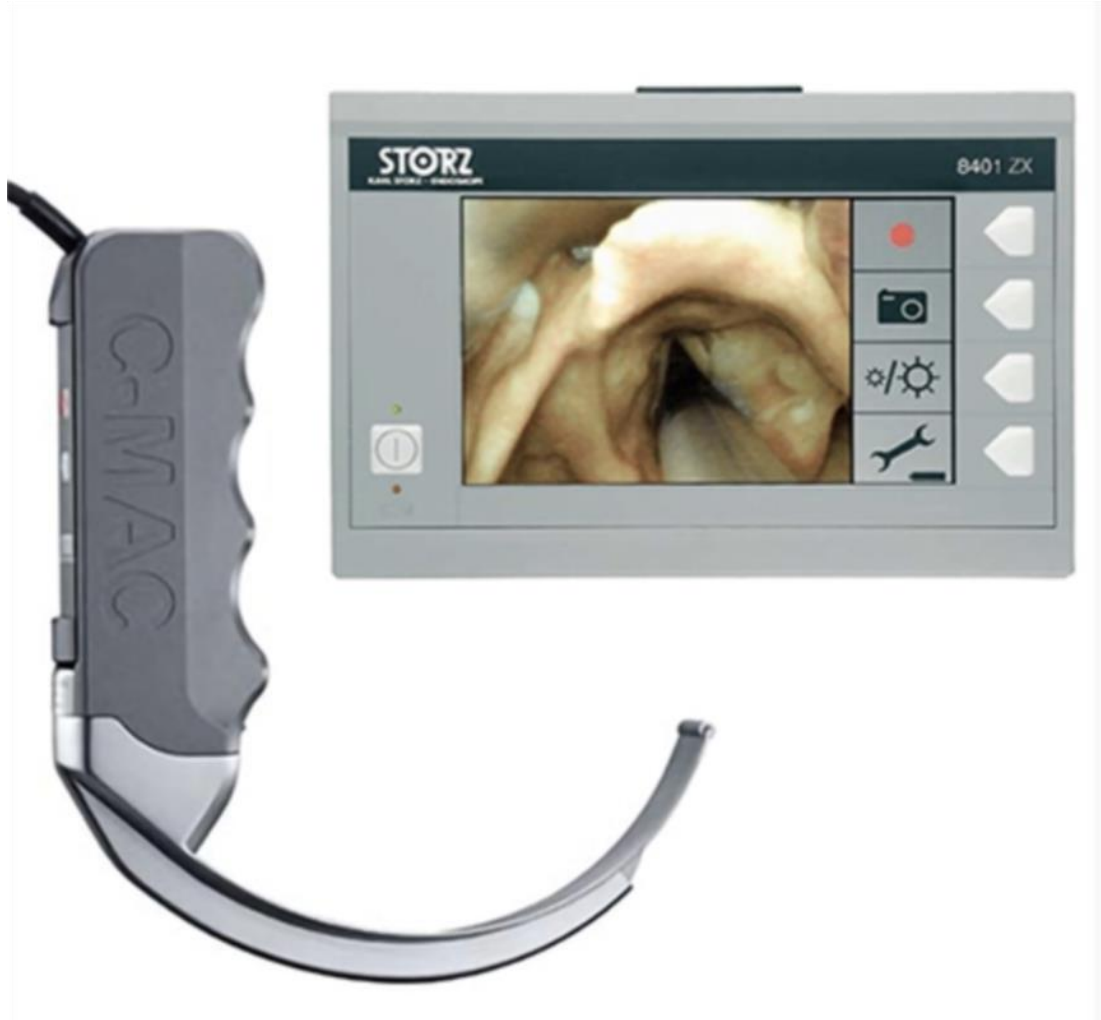


Figure 17-12 A nasotracheal tube can be guided under direct vision (laryngoscopic control) through the laryngeal aperture with a Magill forceps by rotating the hand as when using a backhand motion to hit a ping pong ball. The Magill forceps should grab the nasotracheal tube proximal to the cuff of the ETT. (From Benumof JL, editor: *Airway management: Principles and practice,* St. Louis, 1996, Mosby, p 275.)

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.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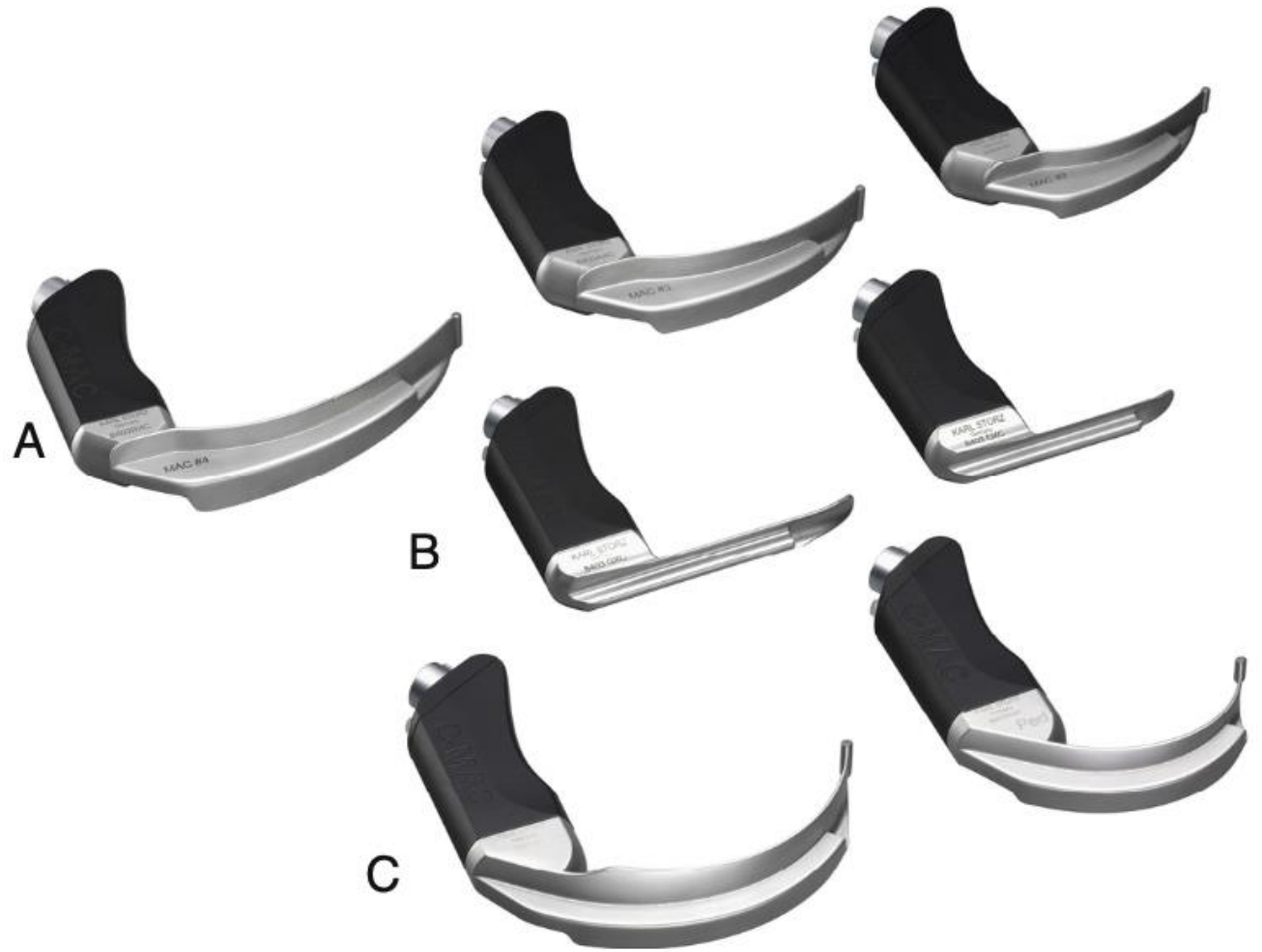


Fig. 16.16 Comparison of the different C-MAC blade types. (A) Macintosh style blade, (B) Miller style blade, and (C) D-blade. (Images courtesy of KARL STORZ Endoscopy, El Segundo, CA.)

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

Box 16.2 Complications of Endotracheal Intubation

During Direct Laryngoscopy and Endotracheal Intubation

Dental and oral soft tissue trauma
Systemic hypertension and tachycardia
Cardiac dysrhythmias
Myocardial ischemia
Inhalation (aspiration) of gastric contents

While the Endotracheal Tube Is in Place

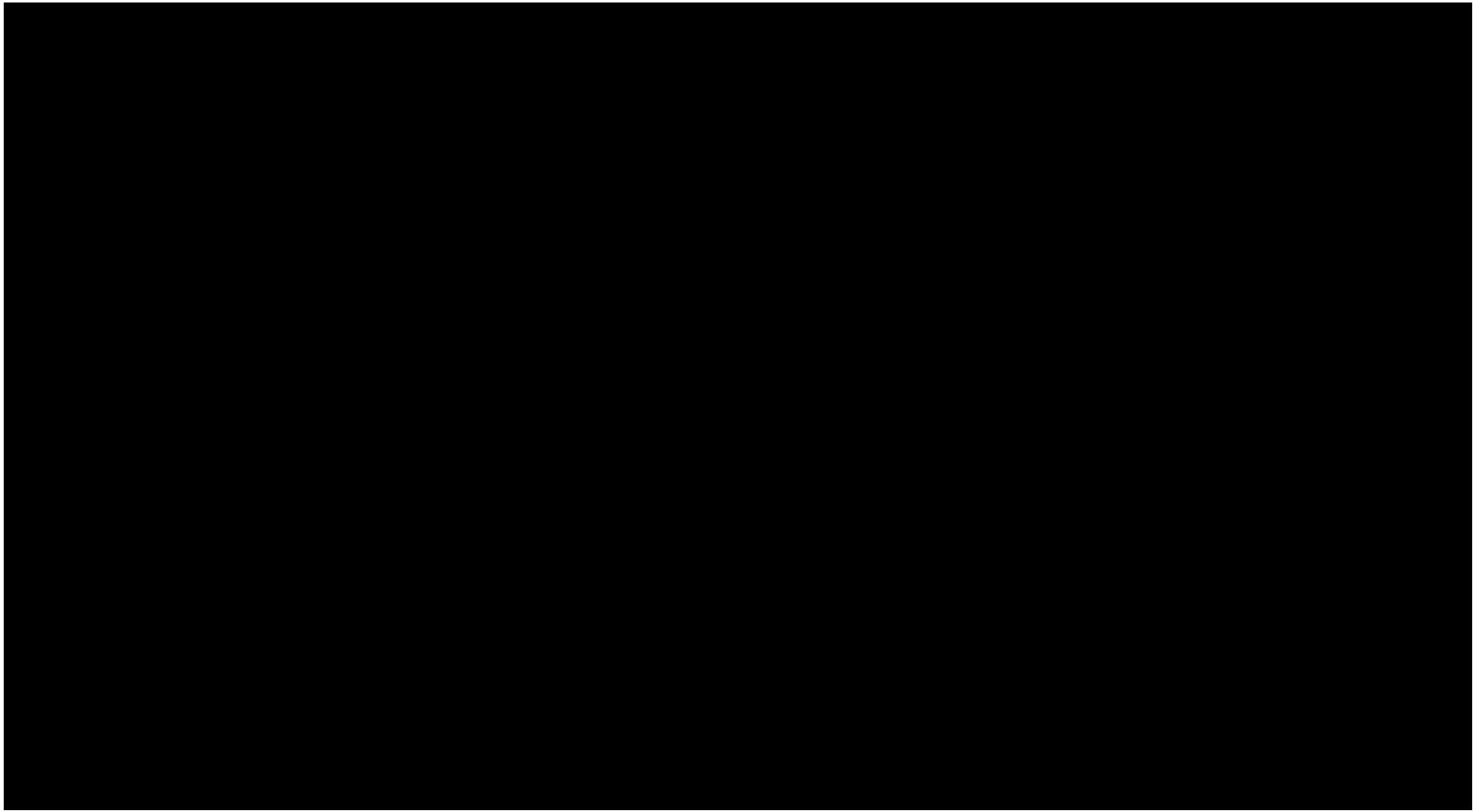
Endotracheal tube obstruction
Endobronchial intubation
Esophageal intubation
Endotracheal tube cuff leak
Pulmonary barotrauma
Nasogastric distention
Accidental disconnection from the anesthesia breathing circuit
Tracheal mucosa ischemia
Accidental extubation

Complications After Endotracheal Extubation

Laryngospasm
Inhalation (aspiration) of gastric contents
Pharyngitis (sore throat)
Laryngitis
Laryngeal or subglottic edema
Laryngeal ulceration with or without granuloma formation
Tracheitis
Tracheal stenosis
Vocal cord paralysis
Arytenoid cartilage dislocation

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

https://airwaymanagement.cookmedical.com/intubation/



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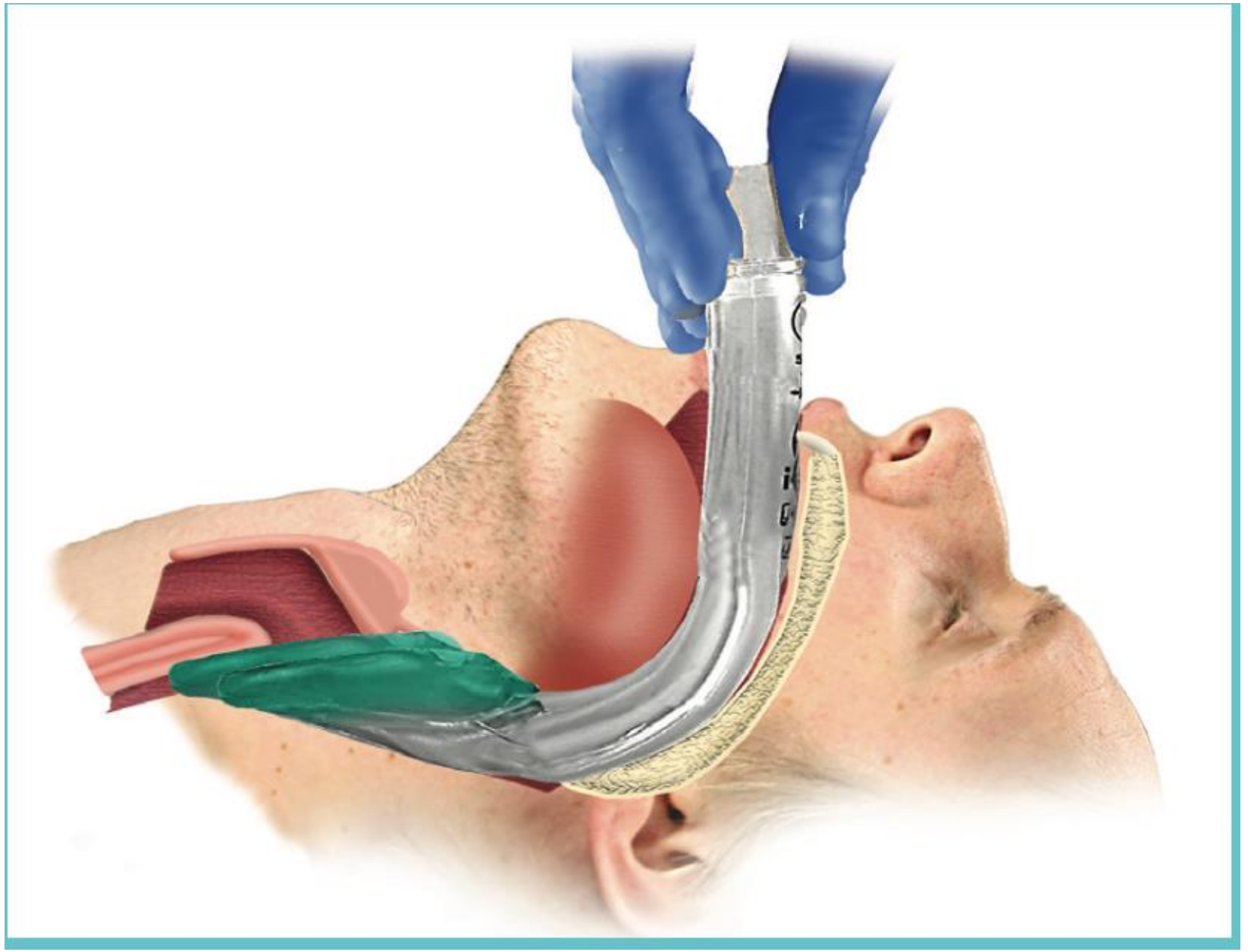
FOB

https://www.youtube.com/watch?v=QC6pQNs5AnQ



Preparations for use

Adult patient

1

Open the **i-gel** package, and on a flat surface take out the protective cradle containing the device.

2

Remove the **i-gel** and transfer it to the palm of the same hand that is holding the protective cradle, supporting the device between the thumb and index finger.

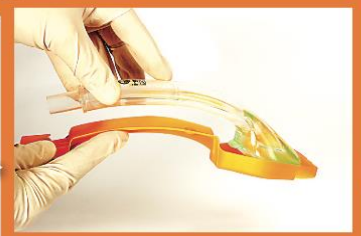
3

Place a small bolus of a water-based lubricant, such as K-Y Jelly, onto the middle of the smooth surface of the protective cradle in preparation for lubrication.

4

Grasp the **i-gel** with the opposite (free) hand along the integral bite block and lubricate the back, sides and front of the cuff with a thin layer of lubricant.

5



Place the **i-gel** back into the protective cradle in preparation for insertion.

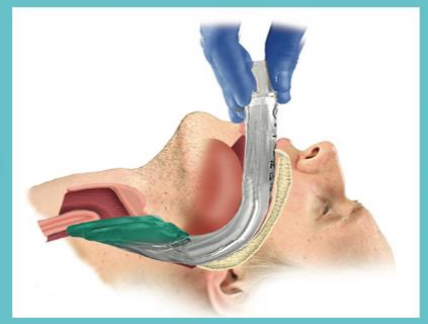
Step 6

Insertion technique

6

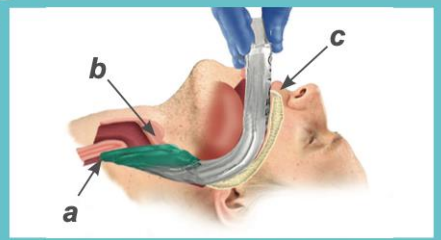
Remove the i-gel from the protective cradle. Grasp the lubricated i-gel firmly along the integral bite block. Position the device so that the **i-gel** cuff outlet is facing towards the chin of the patient. The patient should be in the 'sniffing the morning air' position with head extended and neck flexed. The chin should be gently pressed down before proceeding. Introduce the leading soft tip into the mouth of the patient in a direction towards the hard palate.

7



Glide the device downwards and backwards along the hard palate with a continuous but gentle push until a **definitive resistance** is felt .

8



The tip of the airway should be located into the upper oesophageal opening (a) and the cuff should be located against the laryngeal framework (b). The incisors should be resting on the integral bite-block (c).

9

i-gel should be taped down from 'maxilla to maxilla'.

10

If there is early resistance during insertion a 'jaw thrust' (above) or 'Insertion with Deep Rotation' (right) is recommended.

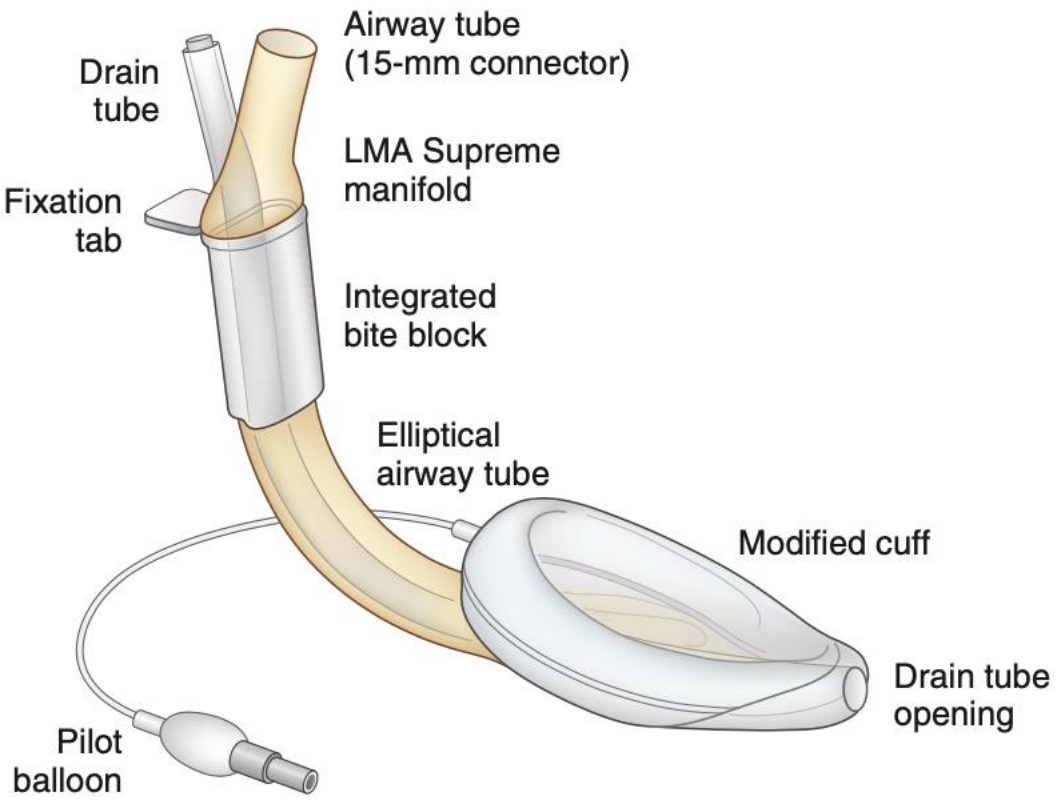


Figure 22-24 The LMA Supreme has a manifold with an integral bite block, an anatomically shaped airway tube enclosing a drain tube, a modified cuff through which the drain tube passes, and a cuff inflation line with pilot balloon.

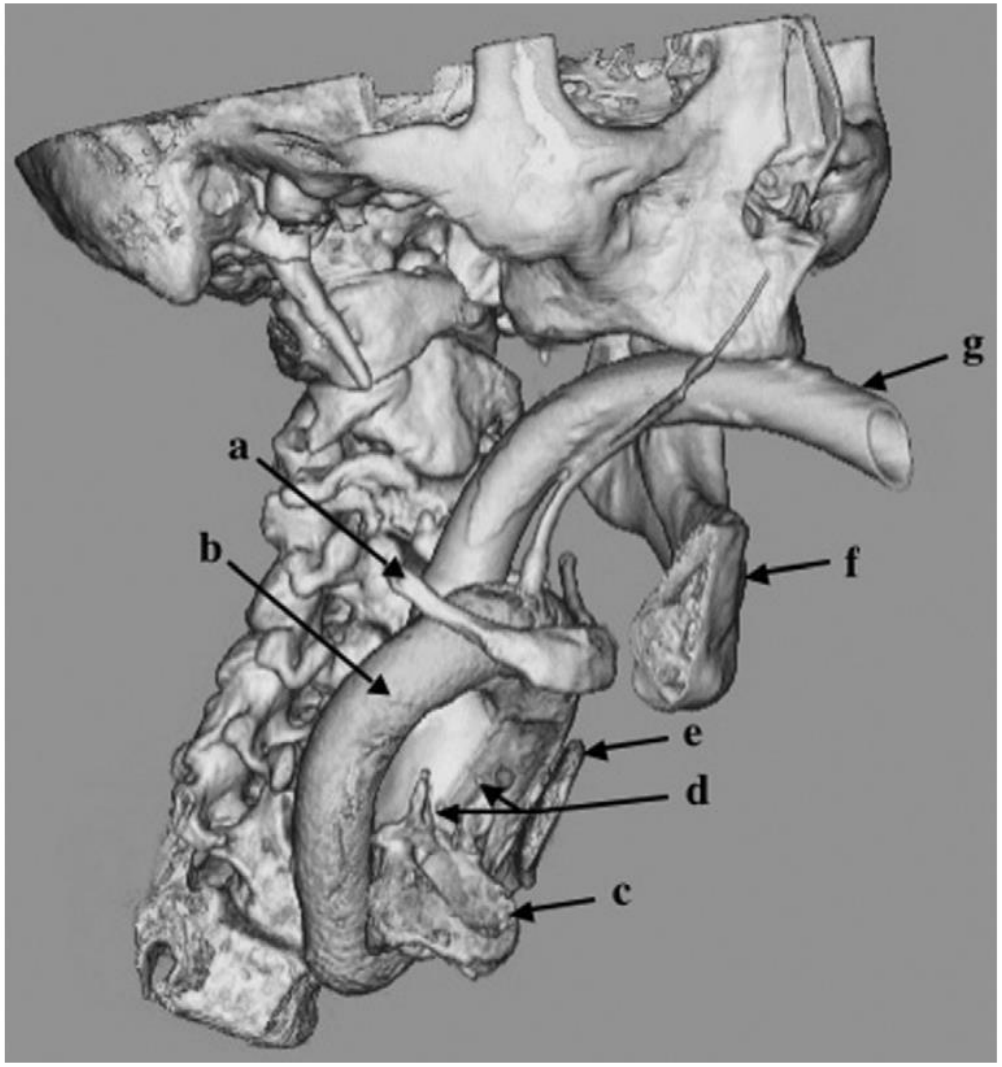
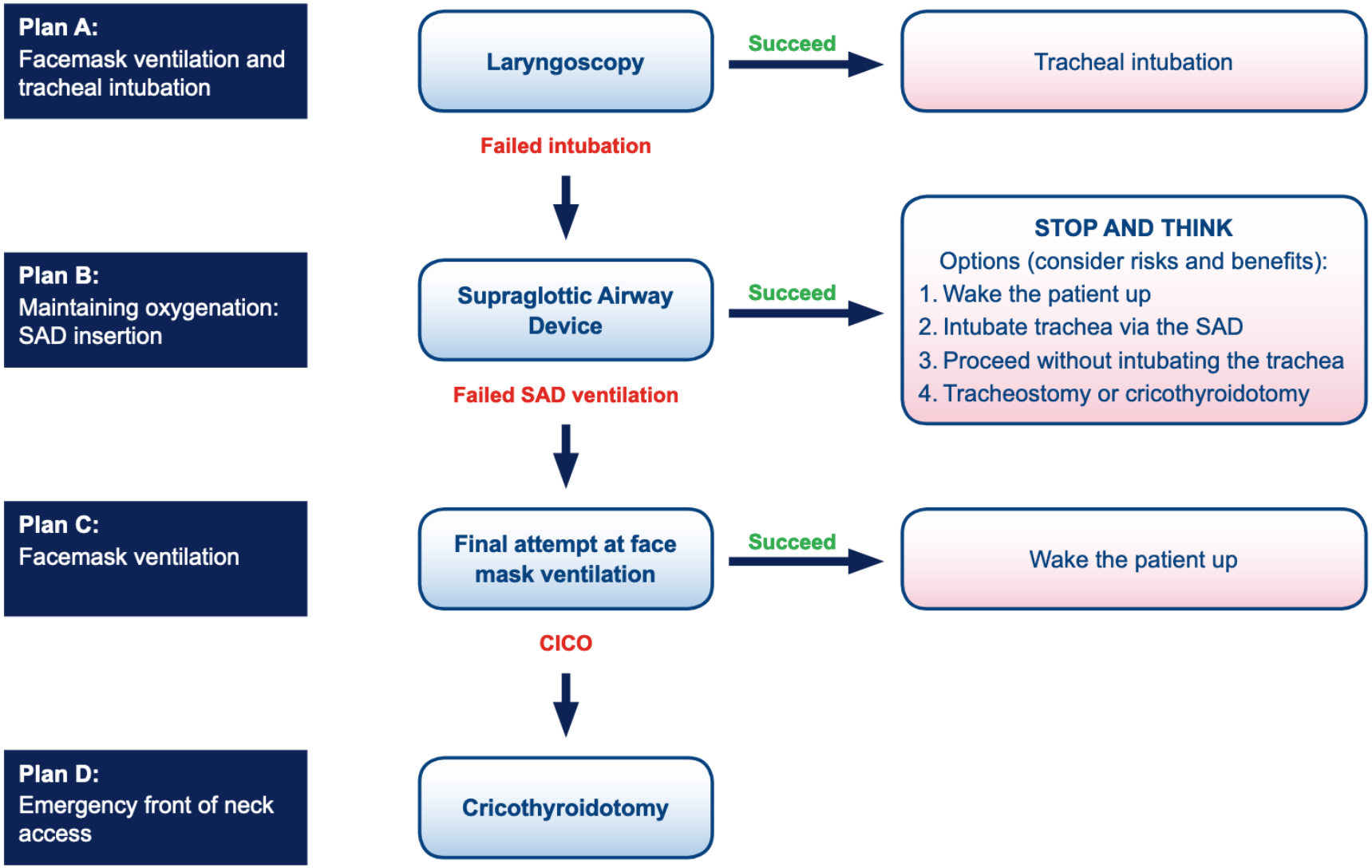


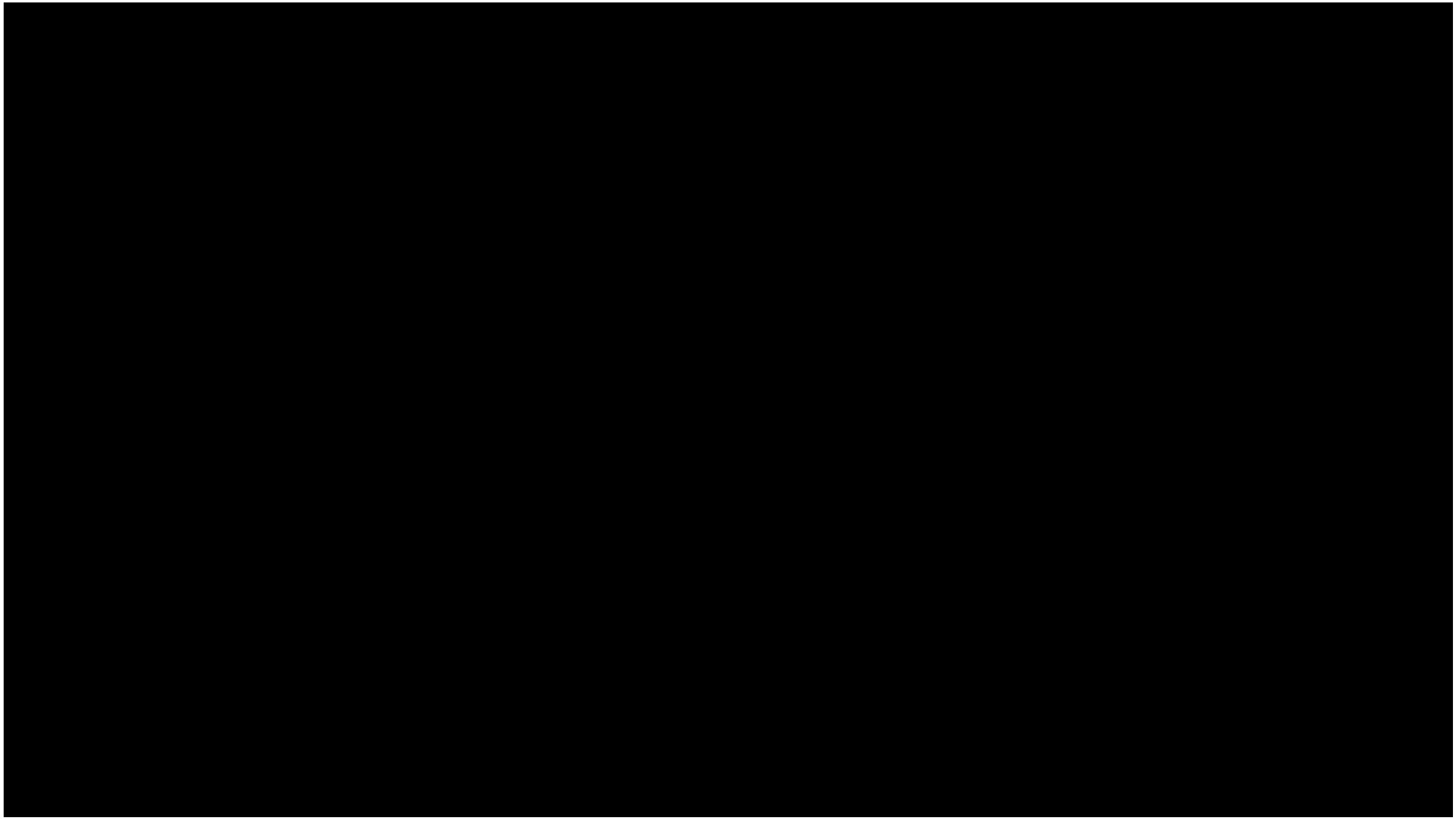
Figure 22-1 Three-dimensional radiologic reconstruction of the human airway with the laryngeal mask airway (LMA) in situ: hyoid bone *(a)*; LMA's cuff *(b)*; cricoid ring *(c)*; arytenoid cartilages *(d)*; thyroid cartilage *(e)*, which is digitally partially removed to demonstrate the position of the LMA; mandible *(f)*, which is digitally partially removed to demonstrate the position of the LMA; and the LMA's shaft *(g)*. The LMA's cuff forms a seal with the periglottic tissues and provides a continuous connection between the natural airway and the device.

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.

DAS Difficult intubation guidelines – overview



https://das.uk.com/files/das2015intubation_guidelines.pdf



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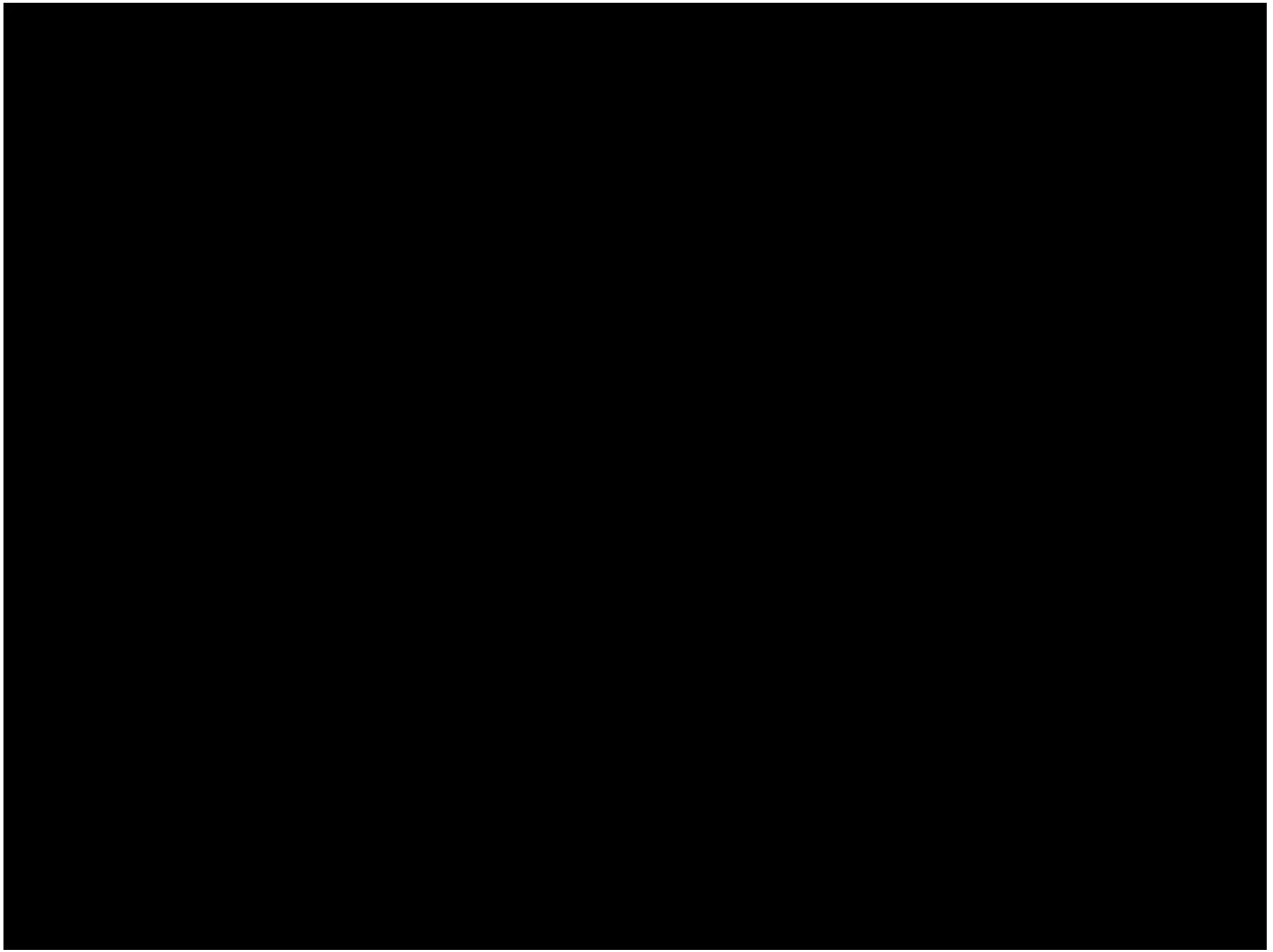

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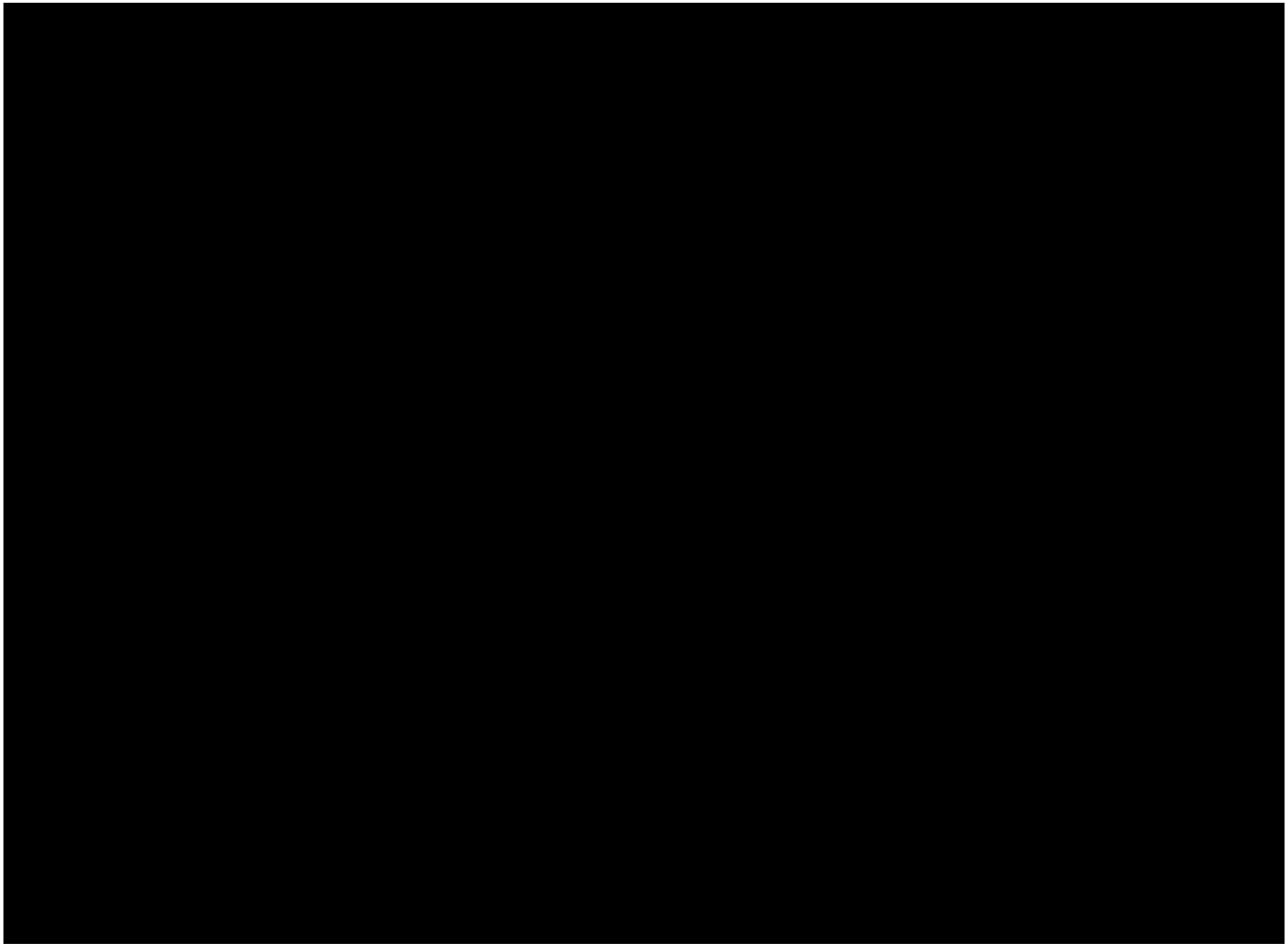

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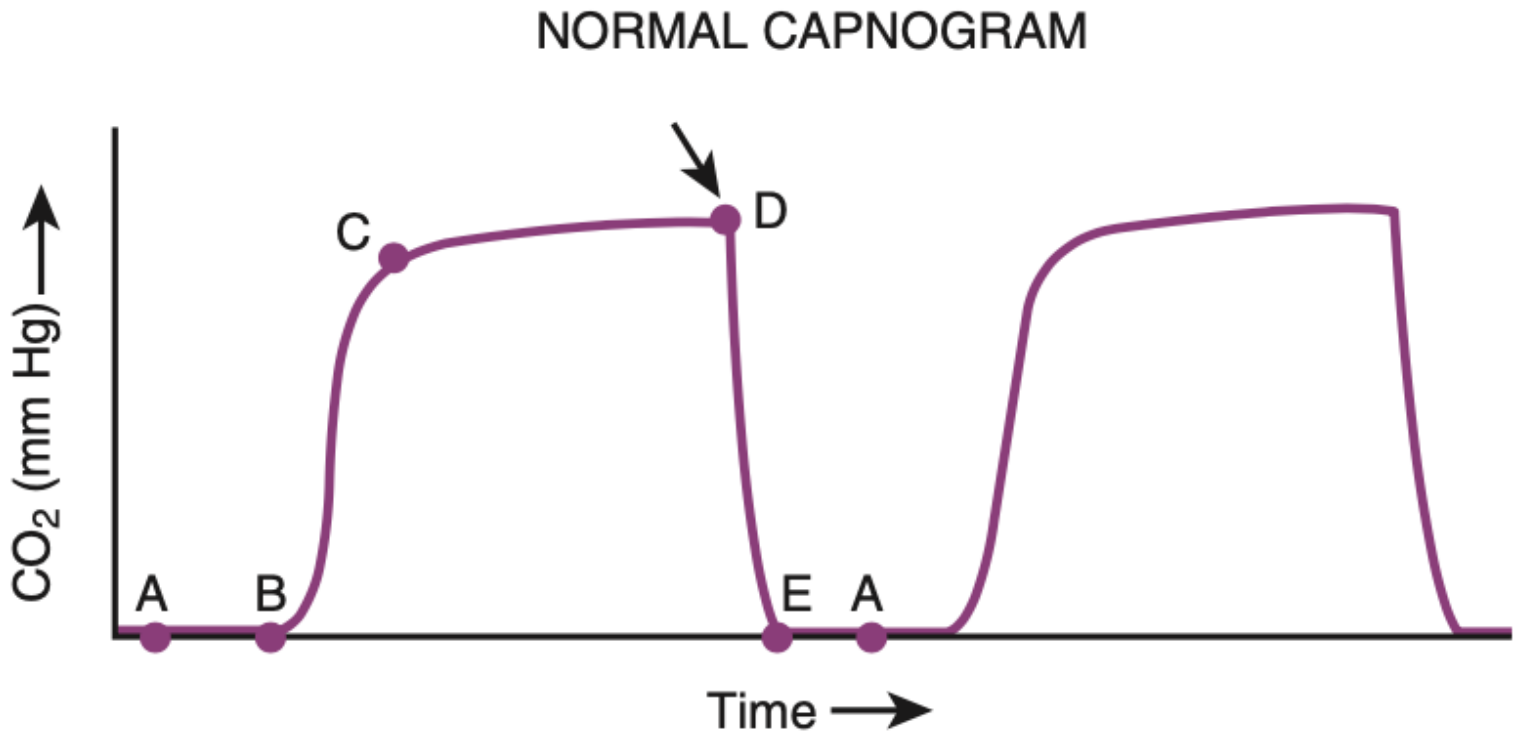
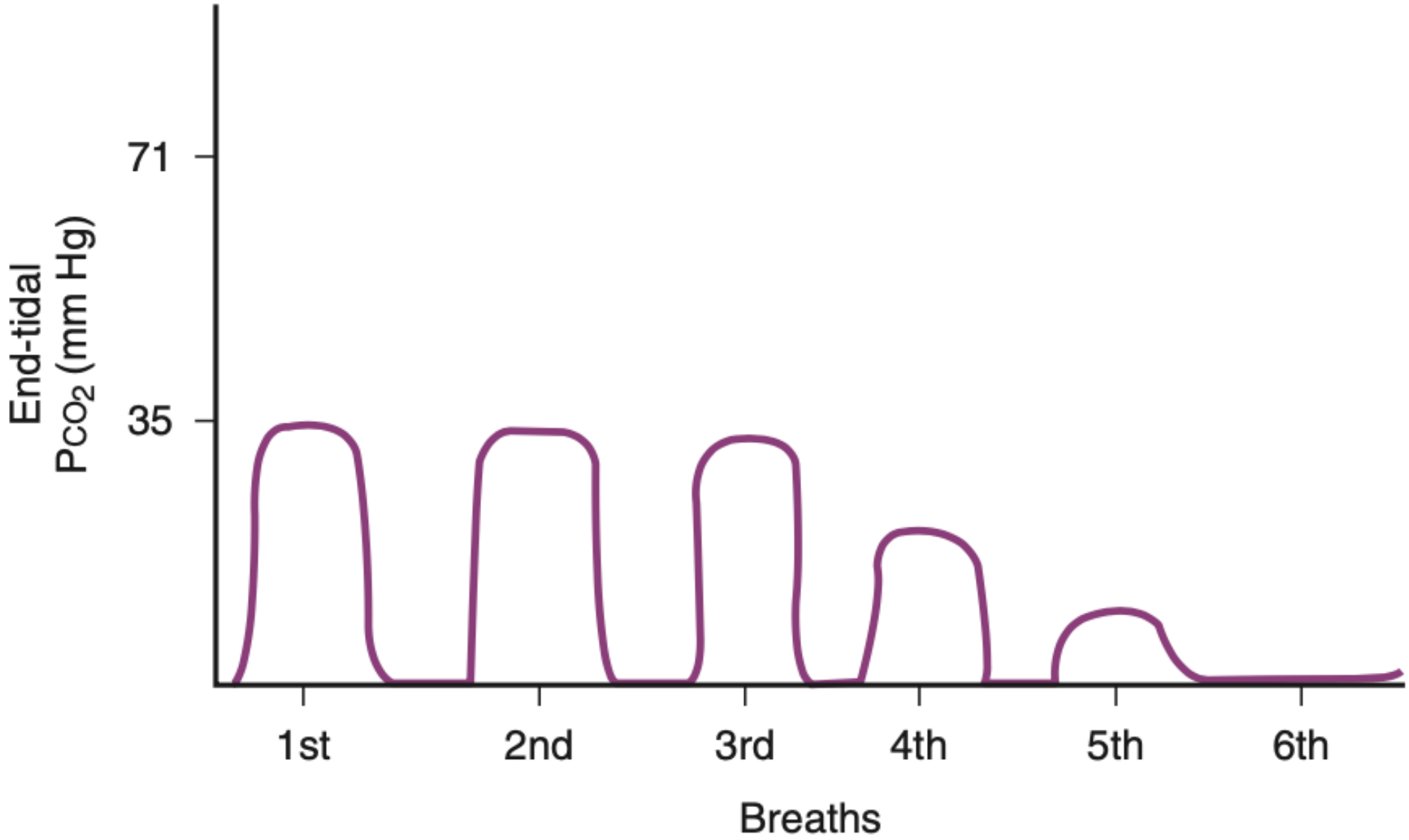


Figure 32-7 The CO_2 waveform. *A,* Expiratory pause begins. *A–B,* Clearance of anatomic dead space. *B–C,* Dead space air mixed with alveolar air. *C–D,* Alveolar plateau. *D,* End-tidal partial pressure of CO_2 registered by capnograph *(arrow)* and beginning of inspiratory phase. *D–E,* Clearance of dead space air. *E–A,* Inspiratory gas devoid of CO_2. (Modified from May WS, Heavner JE, McWorther D, Racz G: *Capnography in the operating room: An introductory directory,* New York, 1985, Raven Press, p 1.)

Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.



Hagberg CA. Benumof and Hagberg's Airway Management E-Book. Elsevier Health Sciences; 2012.

Domande o interventi?

Posizionamento

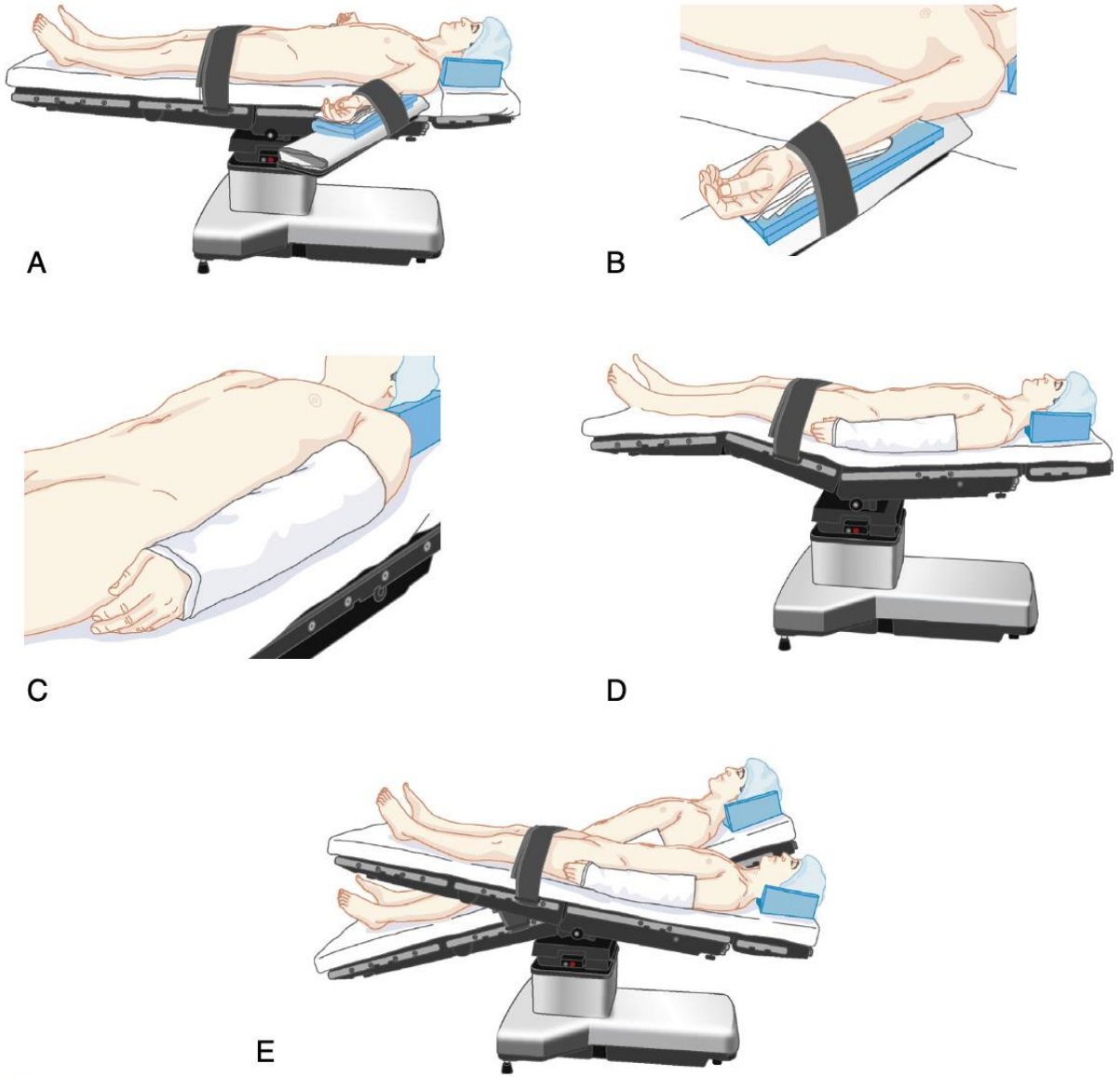


Fig. 19.1 (A) Supine positioning. Note the asymmetry of the base of the table, placing the patient's center of gravity over the base if positioned in the usual direction. (B) Arm position on the arm board. Abduction of the arm should be limited to less than 90 degrees whenever possible. The arm is supinated, and the elbow is padded. (C) Arm tucked at patient's side. Arm in neutral position with palm to hip. The elbow is padded, and one needs to ensure that the arm is supported. (D) Lawn-chair position. Flexion of the hips and knees decreases tension on the back. (E) Trendelenburg position (head tilted down) and reverse Trendelenburg position (head tilted up). Shoulder braces should be avoided to prevent brachial plexus compression injuries.

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

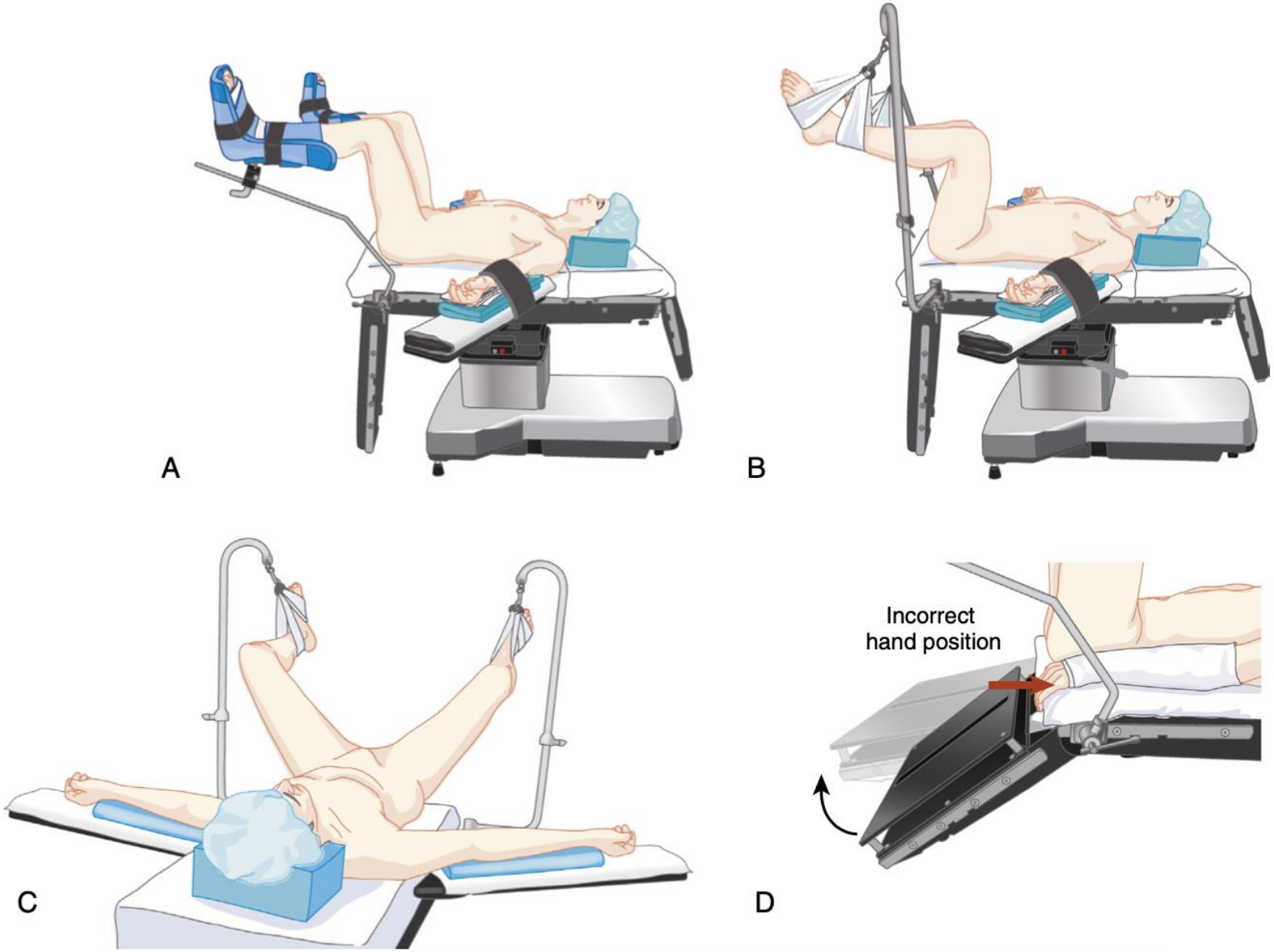


Fig. 19.2 (A) Lithotomy position. Hips are flexed 80 to 100 degrees with the lower leg parallel to the body. Arms are on armrests away from the hinge point of the foot section. (B) Lithotomy position with "candy cane" supports. (C) Lithotomy position with correct position of "candy cane" stirrups away from lateral fibular head. (D) Improper position of arms in lithotomy position with fingers at risk for compression when the lower section of the bed is raised.

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

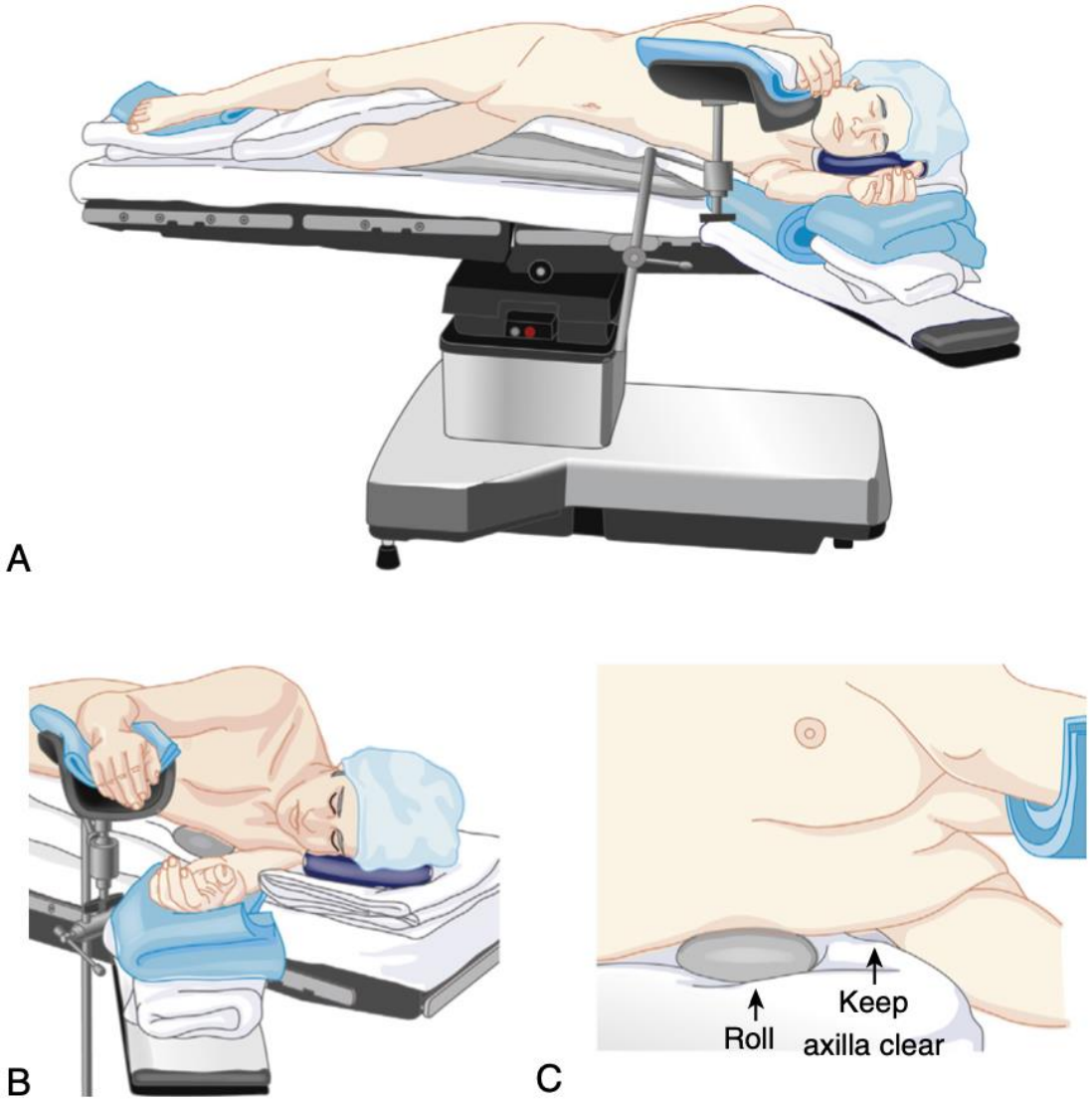
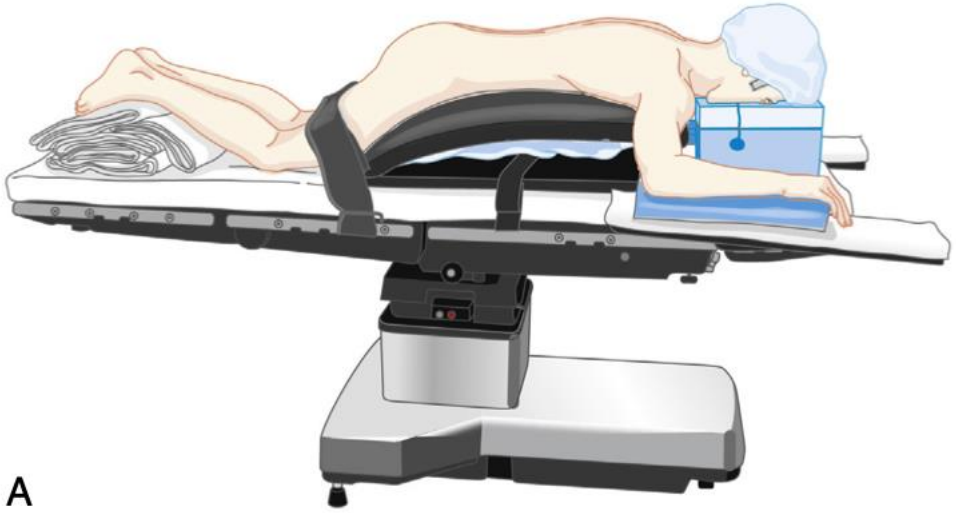
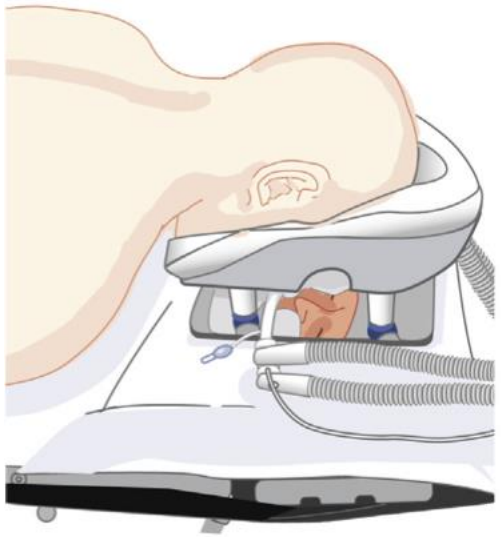


Fig. 19.3 (A) Lateral decubitus position. Note flexion of the lower leg, padding between the legs, and proper support of both arms. (B) Lateral decubitus position showing placement of arms and head. Note additional padding under headrest to ensure alignment of head with spine. Headrest should be kept away from the dependent eye. (C) Use of axillary roll in lateral decubitus position. The roll, in this case a bag of intravenous fluid, is placed well away from the axilla to prevent compression of the axillary artery and brachial plexus.

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.



A



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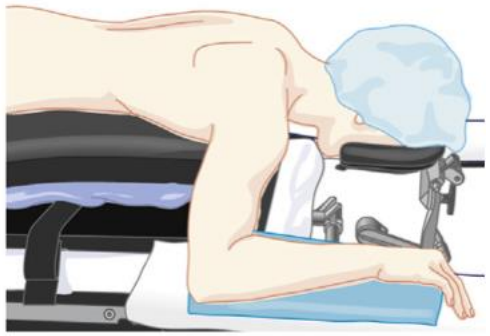

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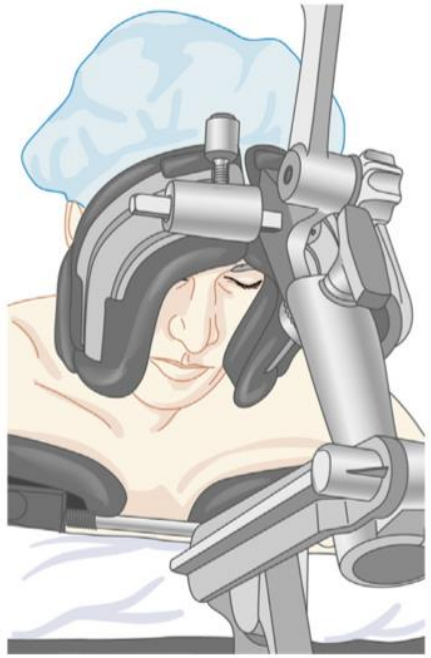

D

Fig. 19.4 (A) Prone position with Wilson frame. Arms are abducted less than 90 degrees whenever possible. Pressure points are padded, and chest and abdomen are supported away from the bed to minimize abdominal pressure and preserve pulmonary compliance. Foam head pillow has cutouts for eyes and nose and a slot to permit the endotracheal tube to exit. Eyes must be checked frequently. (B) Mirror system for prone position. Bony structures of the head and face are supported, and monitoring of eyes and airway is facilitated with a plastic mirror. (C) Prone position with horseshoe adapter. Head height is adjusted to position neck in a neutral position. (D) Prone position, face seen from below. Horseshoe adapter permits superior access to airway and visualization of eyes. Width may be adjusted to ensure proper support by facial bones.

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

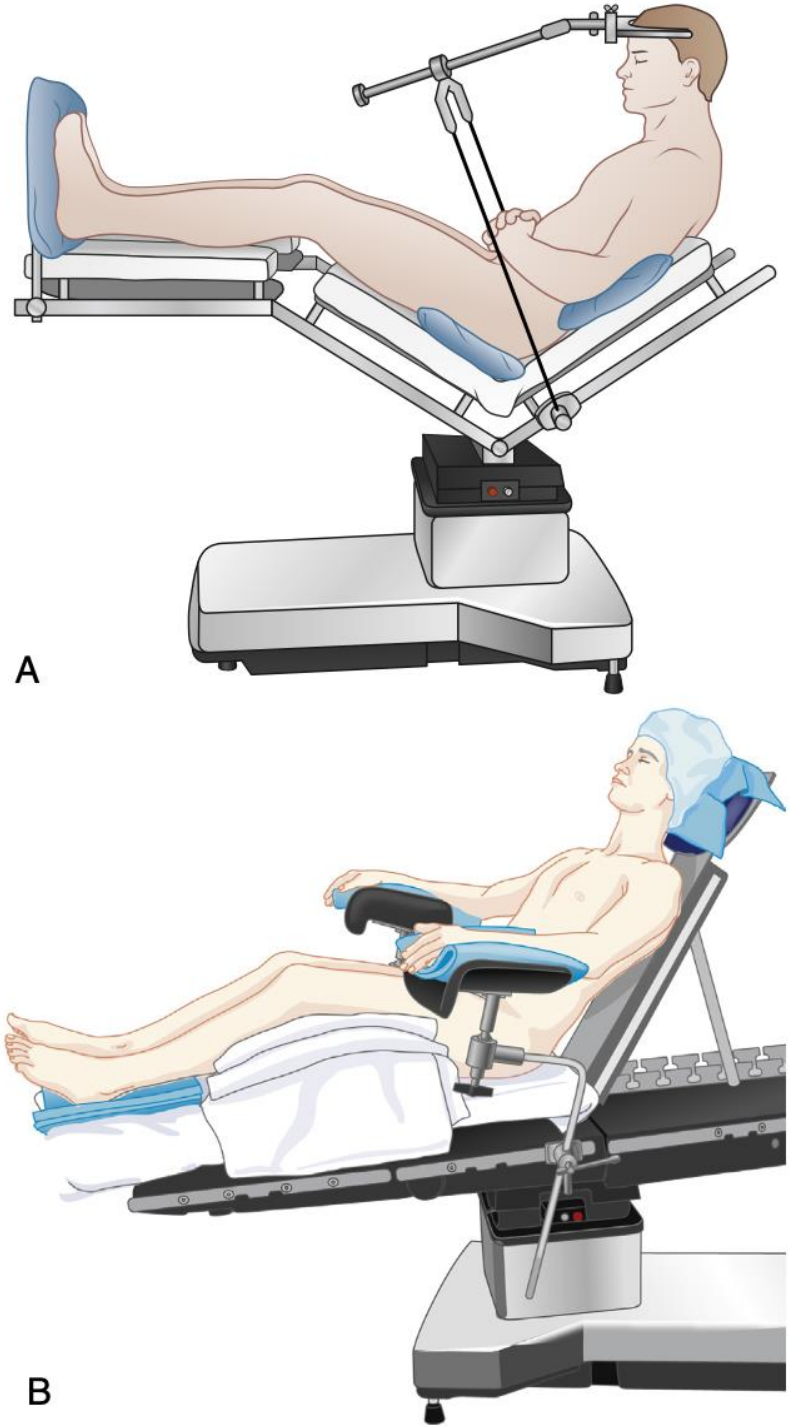

Fig. 19.5 (A) Sitting position with Mayfield head pins. The patient is typically semirecumbent rather than sitting as the legs are kept as high as possible to promote venous return. Arms must be supported to prevent shoulder traction. Note that the head holder support is preferably attached to the back section rather than the thigh section of the table so that the patient's back may be adjusted or lowered emergently without first detaching the head holder. (B) Sitting position adapted for shoulder surgery. Note the absence of pressure over the ulnar area of the elbow.

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

Domande o interventi?

Problemi al risveglio e in PACU

Box 39.1 Physiologic Disorders Manifested in the Postanesthesia Care Unit

Upper airway obstruction
Arterial hypoxemia
Hypoventilation
Hypotension
Hypertension
Cardiac dysrhythmias
Oliguria
Bleeding
Decreased body temperature
Delirium (emergence agitation)
Delayed awakening
Nausea and vomiting
Pain

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

PORC

Box 39.2 Causes of Prolonged Neuromuscular Blockade

Factors Contributing to Prolonged Nondepolarizing Neuromuscular Blockade

- Drugs
  - Inhaled anesthetic drugs
  - Local anesthetics (lidocaine)
  - Cardiac antidysrhythmics (procainamide)
  - Antibiotics (polymyxins, aminoglycosides, lincosamines [clindamycin], metronidazole [Flagyl], tetracyclines)
  - Corticosteroids
  - Calcium channel blockers
  - Dantrolene
  - Furosemide
- Metabolic and physiologic states
  - Hypermagnesemia
  - Hypocalcemia
  - Hypothermia
  - Respiratory acidosis
  - Hepatic/renal failure
  - Myasthenia syndromes

Factors Contributing to Prolonged Depolarizing Neuromuscular Blockade

- Excessive dose of succinylcholine
- Reduced plasma cholinesterase activity
  - Decreased levels
  - Extremes of age (newborn, old age)
  - Disease states (hepatic disease, uremia, malnutrition, plasmapheresis)
  - Hormonal changes
  - Pregnancy
  - Contraceptives
  - Glucocorticoids
- Inhibited activity
  - Irreversible (echothiophate)
  - Reversible (edrophonium, neostigmine, pyridostigmine)
- Genetic variant (atypical plasma cholinesterase)

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

PONV

Box 39.11 Factors Associated With Increased Incidence of Postoperative Nausea and Vomiting (PONV)

History of PONV or motion sickness
Female gender
Age less than 50 years
Postoperative opioids
Nonsmoking status
Type of surgery—eye muscle surgery, middle ear surgery, cholecystectomy, gynecologic surgery—laparoscopic approach
Duration of surgery
Anesthetic drugs—opioids, nitrous oxide, volatile anesthetics
Gastric distention—swallowed blood

Box 39.12 Commonly Used Antiemetics, With Adult Doses

Anticholinergics
Scopolamine: transdermal patch, 1.5 cm^2
Apply to a hairless area behind the ear before surgery; remove 24 hours postoperatively

Antihistamines
Hydroxyzine: 12.5-25 mg IM

Phenothiazines
Promethazine: 12.5-25 mg IV/IM
Prochlorperazine: 2.5-10 mg IV/IM

Butyrophenones
Droperidol: 0.625-1.25 mg IV
See black box warning regarding torsades de pointes: monitor the ECG for prolongation of the QT interval for 2-3 hours after administration—preoperative 12-lead ECG recommended

Nk-1 Receptor Antagonists
Aprepitant: 40 mg PO prior to induction of anesthesia

Prokinetic
Metoclopramide: 10-20 mg IV
Minimal antiemetic properties, avoid in patients with any possibility of gastrointestinal obstruction

Serotonin Receptor Antagonists
Ondansetron: 4 mg IV 30 minutes before conclusion of surgery
Granisetron: 0.35-3 mg IV near the conclusion of surgery
Tropisetron: 2 mg IV near the conclusion of surgery
Palonsetron: 0.075 mg IV with induction of anesthesia
Dolasetron: 12.5 mg IV 15-30 minutes before conclusion of surgery (no longer marketed in the United States due to risk of QTc prolongation and torsades de pointes)

Corticosteroids
Dexamethasone: 4-8 mg IV with induction of anesthesia
Methylprednisolone: 40 mg IV with induction of anesthesia

Other Antiemetics
Propofol: subhypnotic doses such as 20 μg/kg/min IV infusion intraoperatively

ECG, Electrocardiogram; *IM*, intramuscularly; *IV*, intravenously; *PO*, by mouth.

Pardo M, Miller RD. Basics of Anesthesia. Elsevier, 2018.

Dimissione dalla PACU

Table 39.1 Criteria for Determination of Discharge Score for Release From the Postanesthesia Care Unit

Variable Evaluated	Score
Activity	
Able to move four extremities on command	2
Able to move two extremities on command	1
Able to move no extremities on command	0
Breathing	
Able to breathe deeply and cough freely	2
Dyspnea	1
Apnea	0
Circulation (systemic blood pressure)	
Within 20% of the preanesthetic level	2
20% to 49% of the preanesthetic level	1
≥50% of the preanesthetic level	0
Consciousness	
Fully awake	2
Arousable	1
Not responding	0
Oxygen Saturation (pulse oximetry)	
>92% while breathing room air	2
Needs supplemental oxygen to maintain saturation >90%	1
<90% even with supplemental oxygen	0

Adapted from Aldrete JA. The post anaesthesia recovery score revisited. *J Clin Anesth*. 1995;7:89-91.

Pardo M, Miller RD. Basics of Anesthesia. Elsevier; 2018.

Domande o interventi?