Anesthesia, Pain Management, Sedation

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*www.ASA.org* - practice guidelines developed by American Society of Anesthesiologists (ASA).

Types of anesthesia:

1. **monitored anesthesia care (MAC)** - supplements local anesthesia performed by surgeons.
2. **regional anesthesia** - patients remain awake and, if needed, can receive intravenous sedation or analgesics.

N.B. inadequate regional anesthetic may require rapid transition to heavy sedation or general anesthesia!

1. **general anesthesia** - reversible state of unconsciousness; four components of general anesthesia:
2. amnesia
3. analgesia
4. inhibition of noxious reflexes
5. skeletal muscle relaxation

Basis of anesthetic environment:

1. Drugs
2. Equipment
3. Monitors

PHARMACOLOGIC PRINCIPLES

* general anesthesia components are achieved by combination of intravenous anesthetics / analgesics, inhalational anesthetics, ± muscle relaxants.
* ***intravenous induction agents*** most commonly initiate anesthetic state;
* ***inhalational agents*** remain core of anesthesia maintenance (± supplementation with i/v opioids & muscle relaxants).

Inhalational Agents

Mechanism

- remains speculative, but some evidence suggests ***N*-methyl-D-aspartate receptor**\* as well as **GABAB**\*\* and **glycine**\*\* receptors.

* in general, action is ***nonselective*** – affect all body cells.

\*excitatory receptors of glutamate (→ Na+, Ca2+ into cell → depolarization)

\*\*inhibitory receptors (→ Cl- out of cell → hyperpolarization)

Historical inhalational anesthetics:

1. **Ether** - *slow* induction and equally delayed emergence but could produce unconsciousness, amnesia, analgesia, and lack of movement.
2. **Diethyl ether**.
3. **Chloroform** - *hepatic toxicity*, occasional fatal *cardiac arrhythmias*.

Obsolete inhalational anesthetics:

**methoxyflurane** – causes *nephrotoxicity* due to plasma [F -]↑↑↑ during methoxyflurane metabolism.

* most potent known inhalational anesthetic!!!
* still used in obstetrics (does not relax uterus when inhaled briefly).

Modern inhalational anesthetics:

all (except nitrous oxide) are *volatile, halogenated hydrocarbons*

| **Anesthetic****agent** | **MAC\***(potency) | **Oil/Gas partition coefficient**1 | **Blood/Gas partition coefficient**2(induction speed) | **Brain/Blood****coefficient**3 | **Muscle/Blood coefficient**3 | **Fat/Blood coefficient**3 |
| --- | --- | --- | --- | --- | --- | --- |
| Nitrous oxide – **safest!!!** | 104.5 | 1.4 | 0.47 | 1.1 | 1.2 | 2.3 |
| Desflurane | 6.00 | 18.7 | 0.44 | 1.3 | 2.0 | 27 |
| Sevoflurane | 2.00 | 47.2 | 0.65 | 1.7 | 3.1 | 48 |
| Enflurane | 1.68 | 96.5 | 1.85 | 1.4 | 1.7 | 36 |
| Isoflurane | 1.15 | 90.8 | 1.4 | 1.6 | 2.9 | 45 |
| Halothane | 0.74 | 224 | 2.4 | 1.9 | 3.4 | 51 |

**\*MAC (minimal alveolar concentration)** – concentration needed to eliminate movement in 50% patients challenged by standardized skin incision.

* expressed as percent of gas mixture at 1 atm.
* inversely correlates with anesthetic potency (lower MAC - more potent agent.).
* inhaliacinių anestetikų narkozės gylį lengva valdyti keičiant inhaliuojamą koncentraciją; anestetikai labai greitai pasišalina iš organizmo (vs. intraveniniai anestetikai).

Gas concentration = ***partial pressure*** × ***solubility***\*

\*solubility is expressed in terms of ***partition coefficient***:

1**Oil/gas partition coefficient** – lipid solubility (correlates with anesthetic potency).

2**Blood/gas partition coefficient** - inversely correlates with induction speed:

*less soluble agents* have more rapid induction and emergence (i.e. blood concentration more rapidly equilibrates with inspired concentration - both at anesthesia beginning when inspired concentration is high and at end when inspired concentration is zero).

* least soluble is ***desflurane*** (coefficient ≈ 0.44).

*more soluble agents* - concentration in blood rises more slowly (because greater amounts of drug must dissolve) – slower changes in anesthesia depth in response to changes in inhaled concentrations.

* most soluble are ***ether*** (coefficient ≈ 12) & *halothane* (coefficient ≈ 2.4).

3**Tissue/blood coefficients** - solubility in various tissues:

***brain, heart, liver, kidney, endocrine glands*** – highly perfused tissues – rapidly attain steady-state with gas partial pressure in blood.

***skeletal muscles*** – poorly perfused (during anesthesia), large volume – very slowly achieve steady-state.

***fat*** - poorly perfused, anesthetics are very fat-soluble - very slowly achieve steady-state.

***bone, cartilage, ligaments*** - poorly perfused, low storage capacity – slight impact on gas distribution time.

Common effects:

* didina smegenų perfūziją (ICP↑), bronchodilatuoja, relaksuoja gimdą.
* potencijuoja nedepoliarizuojančių miorelaksantų poveikį.

Adverse effects

| **Anesthetic****agent** | **Arrhythmias, sensitization to catecholamines** | **Cardiac output, BP** | **Respiratory reflexes** | **Hepatotoxicity** | **Nephrotoxicity** |
| --- | --- | --- | --- | --- | --- |
| Nitrous oxide – **safest!!!** | - | - | - | - | - |
| Desflurane | - | +/- |  |  |  |
| Sevoflurane | - | - | ↓ | - | + |
| Enflurane | + | ↓ → N | ↓ | + | +/- |
| Isoflurane | - | ↓ | ↑ | - | - |
| Halothane | ++ | ↓ | ↓ | +++ | - |

* all potent inhalational agents occasionally trigger *malignant hyperthermia*!

Individual Drugs

Nitrous Oxide (N2O, “laughing gas”)

[see p. Op100 >>](file:///D%3A%5CViktoro%5CNeuroscience%5COp.%20Operative%20Techniques%5COp100.%20General%20Principles%20of%20Operative%20Neurosurgery.doc#NITROUS_OXIDE)

Halothane

neurosurgical aspects – [see p. Op100 >>](file:///D%3A%5CViktoro%5CNeuroscience%5COp.%20Operative%20Techniques%5COp100.%20General%20Principles%20of%20Operative%20Neurosurgery.doc#HALOGENATED_AGENTS)

* prototype to which newer agents are compared.
* pleasant odor, nonirritating - facilitates mask induction (esp. important in children)!
* potent ***bronchodilator*** effects – agent of choice in asthmatics.
* MAC is only 0.74 vol% and vapor pressure of halothane is 240 mm Hg (30% of atmospheric pressure) – so halothane can be administered at concentrations greatly exceeding MAC (technique called *overpressure*) at anesthesia beginning (to increase alveolar & blood concentration more quickly).
* highest blood & tissue solubility – steady-state is achieved very slowly (slow induction, slow emergence) H: *overpressure*.
* 70% eliminated unchanged (nonmetabolized); another part is liver-metabolized to tissue-toxic hydrocarbons and bromide ion [Br-].
* disadvantages (→ almost complete replacement by newer agents):
1. **powerful cardiac depressant** (potentially beneficial in patients with ischemic heart disease but may precipitate acute decompensation in patients with severe left ventricular dysfunction).
2. **sensitizes myocardium to catecholamines** (arrhythmogenic).

N.B. kadangi halothane sukelia hipotenziją, jos gydymui negalima skirti simpatomimetikų, bet tiesioginius vazokonstriktorius (e.g. phenylephrine).

1. fulminant **halothane hepatitis** (microscopically indistinguishable from viral hepatitis) – depends on enzyme induction and liver hypoxia.

N.B. damage is metabolic (halothane is not direct hepatotoxin)!

* + rare (≈ 1 in 10,000).
	+ manifests on 2-5th day (fever, anorexia, vomiting, etc.).
	+ mortality 20-50% due to liver necrosis.
	+ hepatitis incidence is increased sevenfold if halothane is repeated within 3 months (except children < 8 years of age - halothane hepatitis has not been reported and halothane can be used repeatedly).

N.B. halothane is agent of choice for children!

* + for medicolegal prudence halothane should be avoided in cases of *potential postoperative liver problems* or if patient has taken *enzyme-inducing drugs* (phenobarbital, isoniazid).
1. **weak analgesia** – must be used with other anesthetic (e.g. N2O).

Isoflurane

neurosurgical aspects – [see p. Op100 >>](file:///D%3A%5CViktoro%5CNeuroscience%5COp.%20Operative%20Techniques%5COp100.%20General%20Principles%20of%20Operative%20Neurosurgery.doc#HALOGENATED_AGENTS)

***Most commonly used potent inhalational agent!***

* more cost-effective than newer agents (sevoflurane, desflurane).
* advantages (over halothane):
1. **less** **cardiac depression, less sensitization to catecholamines**
2. **minimal metabolism** (no organ toxicity!!!).
3. does not increase cerebral blood flow & ICP; effectively depresses cerebral metabolic activity.
* disadvantages:
1. **pungent odor** - precludes use for inhalational induction.
2. **isoflurane-induced tachycardia** (can increase myocardial oxygen consumption) - observe heart rate in patients with CAD.

Sevoflurane

neurosurgical aspects – [see p. Op100 >>](file:///D%3A%5CViktoro%5CNeuroscience%5COp.%20Operative%20Techniques%5COp100.%20General%20Principles%20of%20Operative%20Neurosurgery.doc#HALOGENATED_AGENTS)

* approved for use in 1995.
* rapid induction and emergence.
* advantages (over isoflurane):
1. faster emergence (postoperative somnolence↓ and nausea↓).
2. pleasant to inhale (suitable for inhalational induction in children).
3. minimal cardiovascular side effects.
4. effectively reduces airway resistance (58%); vs. halothane (69%) or isoflurane (75%).
* clinical differences between halothane and sevoflurane are subtle.
* disadvantage - considerable metabolic transformation → plasma [F -]↑ (comparable to enflurane but less than methoxyflurane) → ***nephrotoxicity***.

Desflurane

neurosurgical aspects – [see p. Op100 >>](file:///D%3A%5CViktoro%5CNeuroscience%5COp.%20Operative%20Techniques%5COp100.%20General%20Principles%20of%20Operative%20Neurosurgery.doc#HALOGENATED_AGENTS)

* most volatile of potent inhalational agents (lowest B/G solubility coefficient) – most rapidly taken up and eliminated!!! (net greičiau negu N2O)
* pungent odor (precludes inhalational induction).
* generally ***benign hemodynamically*** (but tachycardia & hypertension if concentration is increased too rapidly).
* negligible organ toxicity.

Enflurane

neurosurgical aspects – [see p. Op100 >>](file:///D%3A%5CViktoro%5CNeuroscience%5COp.%20Operative%20Techniques%5COp100.%20General%20Principles%20of%20Operative%20Neurosurgery.doc#HALOGENATED_AGENTS)

* isomer of isoflurane.
* no wide popularity.
* *less cardiac sensitization* to catecholamines than halothane.
* disadvantage - metabolized to fluoride (F - ) → after prolonged administration (esp. in obese patients) → mild **renal dysfunction**.

Intravenous Induction Agents

1. sodium thiopental
2. ketamine – vienintelis turintis gerą analgeziją!
3. propofol
4. etomidate\*
5. midazolam\*

\*benzodiazepines

* used for **rapid anesthesia** **induction** →:
	+ 1. inhalational agents.
		2. potent opioids (total intravenous anesthesia).
* intravenous induction is ***rapid, pleasant, safe***\* - most adults and many older children prefer intravenous induction to inhalational induction.

\*there are situations in which intravenous induction introduces hazards.

* all lower ICP (except ketamine).

Sodium Thiopental

* extremely rapidly acting (in 30-60 sec) barbiturate; duration of action is ultra-short (≈ 30 min).
* oldest intravenous induction agent.
* well tolerated by wide variety of patients.
* disadvantages:
1. **respiratory depression**.
2. patients with *reactive airway disease* may develop **bronchospasm** (H: use propofol or ketamine).
3. **vasodilation & cardiac depression** – hazardous only in *hypovolemia* or *CHF* (H: use etomidate or ketamine).
4. **no analgetic properties** (potent anesthetic & weak analgetic).
* emergence:
1. *in usual doses* - rapid emergence (redistribution from brain to peripheral tissues, particularly fat).
2. *in higher doses* - thiopental action must be terminated by hepatic metabolism (eliminates only 10% per hour).

N.B. once injected, little can be done to facilitate removal!

* other less commonly used barbiturates: thiamylal, methohexital.

Ketamine

- see [p. Rx3 >>](file:///D%3A%5CViktoro%5CNeuroscience%5CRx.%20Treatment%20Modalities%5CRx3.%20Other%20Sedatives-Anxiolytics.doc#Ketamine)

Propofol

- see [p. Rx3 >>](file:///D%3A%5CViktoro%5CNeuroscience%5CRx.%20Treatment%20Modalities%5CRx3.%20Other%20Sedatives-Anxiolytics.doc#Propofol)

Midazolam (Versed®)

- see [p. Rx3 >>](file:///D%3A%5CViktoro%5CNeuroscience%5CRx.%20Treatment%20Modalities%5CRx3.%20Other%20Sedatives-Anxiolytics.doc#Midazolam)

Etomidate

- see [p. Rx3 >>](file:///D%3A%5CViktoro%5CNeuroscience%5CRx.%20Treatment%20Modalities%5CRx3.%20Other%20Sedatives-Anxiolytics.doc#Etomidate)

Opioids

* used in most general anesthesia cases.
* advantages:
	+ **profound analgesia** (extends through early postemergence interval); often added to local anesthetic solutions in epidural / intrathecal blocks to improve analgesia.
	+ reduce MAC of potent inhalational agents.
	+ **minimal cardiac depression** (meperidine has negative inotropic effects)
	+ **blunt hypertensive-tachycardic response** to endotracheal intubation and incision.
	+ increase CSF absorption and minimally reduce cerebral metabolism, slow EEG (but will not produce isoelectric tracing)
* disadvantages:
	+ ***inconsistent hypnosis & amnesia*** (but in doses 10-20 times analgesic dose, opioids act as complete anesthetics - provide hypnosis and amnesia – may be used as sole anesthetic agents).
	+ ***ventilatory depression*** → ICP↑
	+ ***postoperative nausea***

morphine, hydromorphone, meperidine - inexpensive, intermediate-acting agents; cause histamine release → cerebrovascular vasodilation → ICP↑ (plus, meperidine neuroexcitatory metabolite nor-meperidine can cause seizures).

Synthetic opioids - do not cause histamine release!!!

fentanyl (100-150 times more potent than morphine, crosses BBB) – inexpensive, short-acting.

sufentanil, alfentanil - more potent then fentanyl, short-acting (alfentanil - shortest duration of all narcotics), but more expensive; *raise ICP* - not appropriate for neurosurgical cases!

remifentanil - particularly short-acting, but most expensive narcotic in clinical use; large doses may be neurotoxic to limbic system; used for awake craniotomy

Neuroleptanalgesia

– combination of droperidol (neuroleptic drug – sedative, antiemetic, anticonvulsant) with fentanyl.

* useful for diagnostic and minor surgical procedures.
* fixed ratio preparation exists – called **Innovar**.
* little c/v effects.
* adverse effects: respiratory depression, extrapyramidal symptoms.
* if anesthesia is necessary – add 65% N2O - **neuroleptanesthesia**

Neuromuscular Blockers

* in the 1950s, anesthesia was conducted using single potent inhalational agent that produced all components of general anesthesia (including muscle relaxation); depth of anesthesia necessary to produce profound muscle relaxation was much deeper than that necessary to provide hypnosis and amnesia.

Muscle relaxants:

1. Neuromuscular blockers - block cholinergic (acetylcholine) transmission between *motor nerve ending* and **nicotinic receptor** *on neuromuscular end-plate of skeletal muscle*;

 used in general anesthesia and ICP treatment protocols.

1. Central muscle relaxants (diazepam, baclofen) – act at **GABA receptors** in CNS;

 used to control spastic muscle tone.

1. Direct muscle relaxants (dantrolene) – block **Ca2+ release** from sarcoplasmic reticulum;

 used in malignant hyperthermia, neuroleptic malignant syndrome.

Neuromuscular blockers

* allow to deliver smaller doses of inhalational / intravenous agents – just to achieve hypnosis, amnesia, and analgesia. Safer anesthesia!
* do not affect sensorium, memory, sensations!
* ***structural analogs of* *acetylcholine***.
* block *cholinergic (acetylcholine) transmission* between **motor nerve ending** and **nicotinic receptor on neuromuscular end-plate of skeletal muscle**.
* neuromuscular blockers act as:
	+ 1. noncompetitive agonists (depolarizing type)
		2. competitive antagonists (nondepolarizing type)
* > 90% receptors must be blocked for complete relaxation.

Depolarizing agents

- agonists at cholinergic neuromuscular junction:

**phase I**: drug attaches to nicotinic receptor and (like Acch) opens Na+-channels → membrane depolarization → initial transient visible ***fasciculations***;

**phase II**: drug is not destroyed by acetylcholinesterase → continuous depolarization → gradual repolarization, but receptor remains desensitized to Acch → profound ***flaccid paralysis***.

Succinylcholine

* the only depolarizing agent still in use.
* remains popular for endotracheal intubation:
* **fastest acting** paralytic agent (especially useful if gastric regurgitation is risk);
* **shortest duration of action** paralytic agent (rapidly hydrolyzed by plasma *pseudocholinesterase*) – muscles regain 25% of their strength in just 3-8 minutes – if not successfully intubated, patient can be mask ventilated until spontaneous respiration resumes.
* administered i/v/i.
* dose in children < 10-12 yrs is higher (2 mg/kg) than in adults (1.5 mg/kg).

Side effects:

* 1. Causes muscle fasciculations → **postoperative muscle pain**.
	2. **Intraocular pressure**↑ (due to extraocular musculature fasciculations) - contraindicated in ocular trauma, glaucoma.
	3. **Intragastric pressure**↑ (due to abdominal musculature fasciculations) – hazardous if at risk for aspiration.

1, 2, 3 can be reduced by pretreatment with small (less-than-paralytic) *"precurarizing", s. “priming” dose of nondepolarizing agent*;

 probably not needed in young children.

* 1. Triggering **malignant hyperpyrexia** when combined with volatile agent (esp. halothane) in genetically susceptible patients.
	2. Severe, life-threatening **hyperkalemia** (in patients with burns, paraplegia, quadriplegia, massive trauma).

N.B. succinylcholine is contraindicated in paraplegia / quadriplegia!

* 1. **Bradycardia** (esp. in children) – due to some stimulation of muscarinic receptors.
	2. **Prolonged paralysis** in cases of abnormal plasma pseudocholinesterase (e.g. some pregnant women, hepatic or renal failure, Eaton-Lambert syndrome in bronchogenic carcinoma).

N.B. succinylcholine has no pharmacologic antagonists!

Nondepolarizing agents

- compete for receptor sites with acetylcholine (i.e. combine with nicotinic receptor and prevent acetylcholine binding) – ***prevent myocyte membrane depolarization***.

* + - block magnitude depends on:
1. **agent affinity** for receptor.
2. **acetylcholine availability**, i.e. block can be overcome by increasing acetylcholine concentration in synaptic gap (clinically achieved by cholinesterase inhibitors).

N.B. at *high doses* also block ion channels directly (↓ cholinesterase inhibitor ability to reverse blockade).

* + - skeletal muscle sensitivity varies – in order of susceptibility:

eye & face > fingers > limbs, neck, trunk > intercostal muscles > diaphragm (paralyzed lastly)

First drug was curare, which native hunters of Amazon used to paralyze game.

* + - in early 1940s tubocurarine was purified.
		- **maksimalios blokados** trukmė daugmaž visų vienoda – svyruoja nuo 1 min (rocuronium) iki 6 min (doxacurium).
		- skliausteliuose nurodytas laikas, per kiek minučių **raumenys atgauna 25% savo jėgos**:

Long-acting:

1. Metocurine (107 min)
2. Pipecuronium (95 min)
3. cis-Atracurium (90 min) – may induce slight histamine release.
4. Pancuronium (86 min) – vagolytic (heart rate↑)
5. Doxacurium (83 min)
6. Vecuronium (44 min) – does not induce histamine release!
7. Rocuronium (43 min) – the only paralytic approved for RSI; may induce histamine release
8. D-tubocurarine (38 min) – may induce histamine release, promote ganglionic blockade → bronchospasm, blood pressure↓, bradycardia.

Short-acting:

1. Mivacurium (16 min)
2. Rapacuronium (8 min) – shortest action (≈ as succinylcholine).

Pharmacokinetics

* administered only i/v.
* dozės svyruoja nuo 0.03 mg/kg (D-tubocurarine) iki 0.6 mg/kg (rocuronium).
* do not enter cells, do not cross BBB.
* metabolism:
1. **not metabolized** (action is terminated by redistribution and excretion in urine unchanged) – most drugs!
2. **deacetylation in liver + excretion in bile unchanged** – vecuronium, rocuronium.
3. **spontaneous degradation in plasma** by ester hydrolysis – atracurium, mivacurium – the only drugs that dose is not to be reduced in renal failure!

Reversal

sugammadex (Bridion) – FDA approved to reverse effects of rocuronium and vecuronium.

Dosing considerations

1. Neuromuscular blockers **prevent movement in response to noxious stimuli** (can mask signs of inadequate anesthesia / intraoperative awareness).
2. **Higher doses are required for intubation than for surgical relaxation** (if nondepolarizer is used only after intubation, smaller doses are required).
3. Other drugs may **potentiate actions** of nondepolarizing agents:
	* succinylcholine used for intubation decreases subsequent requirements for nondepolarizers.
	* ***halogenated hydrocarbon anesthetics*** variously potentiate action (e.g. desflurane potentiates vecuronium effects 20% more than does isoflurane).
	* ***aminoglycosides*** (inhibit presynaptic Acch release), ***Ca-channel blockers*** potentiate action.
4. **Individual responses vary widely**.
5. Pancuronium must not be used in patients at high risk for ***pulmonary complications***.
6. Prolonged use → **muscle atrophy**, **irreversible CNS changes** (mechanism unknown; may be related to steroid-like molecule moiety of the drug)
7. **Subtle blockade** can be difficult to detect - can be associated with postoperative problems!
* subtle residual paralysis is quantified using:
1. ***train-of-four (TOF) fade ratio***: magnitude of four muscle twitches (in response to supramaximal stimuli delivered at 0.5-second intervals to ulnar nerve) is evaluated; magnitude of fourth twitch is compared with first twitch;

TOF ratio > 0.70 (fourth twitch is at least 70% of magnitude of first twitch) - adequate return of neuromuscular function.

1. ***sustained 5-second head-lift*** (commonly used clinical index of adequate reversal) – atitinka TOF > 0.60.
* nondepolarizing relaxants are pharmacologically reversed with ***cholinesterase inhibitors*** (neostigmine or edrophonium)\* + atropine or glycopyrrolate (to counteract muscarinic effects of cholinesterase inhibitors).

\*in too high doses may cause depolarizing block!

ANESTHESIA EQUIPMENT

1. **Anesthesia machine**:
2. ***gas sources*** (oxygen, nitrous oxide, and air) - small, *attached tanks* that are pin-indexed to prevent accidental connection of incorrect tanks or *wall outlets* that provide gas from large, remote tanks.
3. ***flowmeters*** - independent adjustment of individual gases (but are designed to minimize chance of delivering hypoxic gas mixture by "fail-safe" valves that require pressurization of oxygen line before nitrous oxide can be delivered).
4. ***flow-proportioning device*** - automatically reduces nitrous oxide flow if oxygen flow is reduced below safe concentration.
5. **Anesthesia vaporizers** - agent-specific, temperature-compensated devices that divert proportion of fresh gas through system that saturates diverted gas with anesthetic vapor before it rejoins remainder of gas flow.
6. **Anesthetic circuit** - circle system with one-way valves that direct inspired and expired gas flow.
* typical circuit includes **carbon dioxide absorber** - contains either ***soda lime*** (sodium hydroxide, calcium hydroxide, potassium hydroxide) or ***Baralyme*** (barium hydroxide + calcium hydroxide) - insoluble carbonates are formed until absorbent is exhausted.
* most have built-in **mechanical ventilator** - capable of ventilating most anesthetized patients (but are not suitable for ventilating occasional patient with severe respiratory failure).
1. **Anesthetic gas scavenging system** - attached to exhaust valve of anesthetic circuit → elimination of anesthetic gases and vapors into air outside hospital.

general consensus is that exhausting gases into operating room risks potential harm to personnel and their offspring.

1. **Monitoring**

ASA established standards for basic anesthetic monitoring:

**Standard I** - ***qualified anesthesia care provider*** must be continuously present in operating room during anesthesia - patient status must be continuously monitored by practitioner.

**Standard II** - ***continuous assessment*** of ventilation, oxygenation, circulation, and temperature:

1. **Oxygen analyzer** with low concentration alarm (detects delivered oxygen concentration (FIO2).
2. **Pulse oximetry** (hemoglobin oxygen saturation) - required during all anesthetics.
3. Ventilation adequacy:
	1. continuously assured by **clinical evaluation** – physical assessment of chest expansion, auscultation of breath sounds.
	2. CO 2 content in expired gas - **end-tidal carbon dioxide (ETCO2) monitoring** (normal difference between ETCO2 and PaCO2 is 2-5 mmHg - reflects dead space ventilation).
4. Device capable of detecting disconnection of breathing system components (gives audible signal when its alarm threshold is exceeded).
5. Continuous ECG; BP and heart rate at least every 5 minutes.

BP monitoring:

* 1. noninvasive – automated oscillometric BP analyzers.
	2. invasive - most commonly cannulated radial artery.
1. Means of temperature evaluation (**esophageal temperature** is most commonly measured during general anesthesia).

#### CNS monitoring

**Bispectral array (BIS)** is modified EEG – is believed to monitor awareness during anesthesia - reports numbers from 0 to 100 (100 represents complete awareness and 0 complete suppression of brain wave activity).

PREOPERATIVE EVALUATION, PREMEDICATION

surgical risk – see 2210 p.

Airway Examination

* even if regional anesthesia is planned, potential for general anesthesia must be considered.
* goal is to identify characteristics that could hinder *mask ventilation* or *tracheal intubation*.
1. mouth opening
2. tongue size
3. neck mobility
4. thyromental distance.

Cardiovascular Disease

Five major predictors of *postoperative myocardial ischemia*: cardiac risk factors – see 2210 p.

* + 1. ECG evidence of left ventricular hypertrophy
		2. history of hypertension
		3. diabetes mellitus
		4. definite CAD
		5. use of digoxin.
			- prophylactic drugs to decrease risk for cardiac complications:
1. perioperative **β-blockers** - should not be *routinely* used in noncardiac surgery (decrease MIs, but increase strokes)
2. combined oral and transdermal clonidine
	* + - in general, *antihypertensive medications* should be continued throughout perioperative period.
			- if heart disorder cannot be corrected - use monitoring with Swann-Ganz catheterization.

Pulmonary Disease

* + - * pulmonary function testing remains controversial (cannot define threshold above which surgery is prohibitive).

Neurologic Disease

Careful documentation is required in:

1. patients with neurologic impairment
2. regional anesthetic procedures.

Renal and Hepatic Disease

* renal and hepatic dysfunction alter metabolism and disposition of many anesthetic agents.
* Child-Pugh Classification of Preoperative Risk in Liver Disease.

Endocrinology

Principles of management in **diabetic patients** undergoing surgery → see 2750 p. (*endocrine*)

Chronic (> 3-6 months) **glucocorticoid** administration → see 2210 p. (*surgery*)

Assessment of Physical Status

**ASA I** - no organic, physiologic, biochemical or psychiatric disturbance

**ASA II** - patient with mild systemic disease that results in ***no functional limitation*** (e.g. well-controlled hypertension, uncomplicated diabetes mellitus).

**ASA III** - patient with severe systemic disease that results in ***functional impairment*** (e.g. diabetes mellitus with vascular complications, prior myocardial infarction, uncontrolled hypertension).

**ASA IV** - severe systemic disease that is ***constant threat to life*** (e.g. congestive heart failure, unstable angina pectoris).

**ASA V** - moribund condition in patient who is ***not expected to survive*** with or without operation (e.g. ruptured aortic aneurysm, intracranial hemorrhage with ICP↑).

**ASA VI** - declared brain death patient whose organs are being ***harvested for transplantation***.

**E** - ***emergency operation*** is required.

Example: *ASA IE,* otherwise healthy patient for emergency appendectomy.

Preoperative medications

1. benzodiazepines (anxiolysis & amnesia)
2. H2-receptor antagonists (gastric acidity↓)
3. anticholinergics (to prevent bradycardia, respiratory hypersecretion)
4. opioids (analgesia)

GENERAL ANESTHESIA STRATEGIES

Key Considerations in General Anesthesia

1. Does condition / surgery suggest **additional monitoring** techniques beyond monitors that are used in every patient?
2. Any conditions that **contraindicate** specific drugs?
3. Is **endotracheal intubation** necessary for this procedure?
4. If endotracheal intubation is required, are there any anticipated difficulties with **oral translaryngeal intubation**?
5. Are **neuromuscular blockers** required for adequate exposure during surgery?
6. Specific **surgical requirements** (e.g. if surgical procedure will include intraoperative nerve stimulation, neuromuscular blockers would interfere with assessment).
7. Are substantial **blood loss / large fluid shifts** anticipated?

N.B. complications of anesthesia are most likely during induction and emergence!

Steps in General Anesthesia

Immediate Preparation

In holding area:

1. Ascertain **patient identity**.
2. Confirm that **informed consent** is obtained.
3. Check any **pending diagnostic tests**.
4. Note any **acute changes in health status**.
5. **Intravenous catheterization** (intravenous catheterization usually is deferred in small children until after inhalational induction).
6. **Intravenous premedication** (for apprehensive patients who have not received oral premedication): midazolam or diazepam.

In operating room:

1. Cooperative patient is asked to **breathe oxygen** from face mask.
2. **Monitoring devices** are applied: *chest stethoscope, noninvasive blood pressure, ECG, pulse oximetry*.
	* *capnography* is initiated after airway has been secured.
	* *temperature monitoring* may or may not be initiated before induction (depending on type of temperature monitor to be used).

Anesthesia Induction

* start only when surgeon is available.
* begins when drug accumulates (reaches sufficient concentrations) in CNS.
* ***patient rapidly loses consciousness*** - ceases to maintain airway, abruptly reduces / ceases spontaneous ventilation, receives drugs that depress myocardium / change vascular tone.
* induction stages:

**stage 1** (awake + analgesia)

**stage 2** ("excitement stage") – hazardous stage (delirium, combative behavior).

**stage 3** (surgical level of anesthesia) – eye movement cease, fixed pupils.

**stage 4** (medullary paralysis) – severe respiratory & vasomotor depression → death.

A. Awake Intubation

- indicated (in small fraction of patients) **when** **inducing anesthesia without first securing airway is risky**:

1. inadequate mouth opening
2. facial trauma
3. cervical spine injury, severe chronic cervical spine disease
4. lesions in upper airway.
* awake intubation may be supplemented with **sedatives, opioids, topical / local anesthesia**.
* techniques:
1. **"blind" nasal route** - endotracheal tube is inserted through nose and guided into trachea by listening to patient's breath sounds as they are transmitted through tube.
2. **fiberoptic bronchoscopy** - passing endotracheal tube through nose / mouth into pharynx, → passing bronchoscope through tube (visualizing larynx-trachea) → threading tube over bronchoscope.
3. **direct visualization**.
* induction drugs are given when airway is secured.

B. Intravenous Induction

- used commonly in **elective adult cases**.

1. patient is first **preoxygenated with 100% oxygen** - to wash out nitrogen (79% of room air) from lungs and to provide patient with oxygen reservoir (equivalent to functional residual capacity) should mask ventilation or intubation be difficult.
* avoid gastric filling by unnecessary respiratory assistance.

N.B. most patients do not need extreme mechanical ventilation (vigorous “bagging”) – pakanka to take several full-volume breaths via bag-valve-mask just prior intubation.

1. **premedicate** (at anesthesiologist choice):
* opioids or benzodiazepines.
* lidocaine blocks increase of ICP & BP (useful in brain injury) by decreasing laryngeal reflexes during intubation.
* atropine blocks muscarinic bradycardia of succinylcholine (esp. in children < 6 years).
1. rapidly acting **intravenous induction agent** (quickly renders patient unconscious & apneic; e.g. thiopental causes unconsciousness in 25 sec).
2. anesthesiologist determines ***whether patient can be manually ventilated*** (using anesthesia face mask and reservoir bag on breathing circuit).
3. if mask ventilation is satisfactory → give **neuromuscular blocker**.
4. patient is adequately relaxed → **endotracheal intubation**.
5. ***tube position*** in trachea is confirmed by **auscultation & capnography**.

Drawbacks associated with intravenous induction:

1. *spontaneous ventilation* is abolished ***without certainty*** that *manual ventilation* can be accomplished.
2. endotracheal intubation is performed while patient is *lightly anesthetized* - potentially precipitating ***hypertension, tachycardia, bronchospasm***;

adjuvant agents (lidocaine, opioids) may blunt reflex responses to intubation.

C. Rapid-Sequence Induction

- indicated in patients who are at **high risk for acid aspiration**:

1. obese patients
2. obstetric patients
3. symptomatic gastroesophageal reflux
4. bowel obstruction.
5. recently eaten

N.B. ***emergency surgery patients usually are considered to have "full stomachs"*** (uncertainty regarding recent food ingestion + pain / injury delays gastric emptying).

Concept is to **progress rapidly from awake state to anesthetized, endotracheally intubated state**:

atropine (± lidocaine\*) premedication → intravenous induction agent\*\* → immediate succinylcholine (without first assuring that manual ventilation can be accomplished) → wait ≈ 1 minute\*\*\* → intubate trachea.

\*decreases airway stimulation - prevents ICP increase.

\*\*thiopental, etomidate, midazolam, or ketamine (contraindicated in head and eye injuries - may increase ICP and IOP).

\*\*\*papildomai ventiliuoti nereikia – turi pakakti preoksigenacijos.

* all that time (from induction drug until endotracheal tube position confirmation) assistant applies firm cricoid cartilage pressure (*Sellick's maneuver*) - to prevent passive stomach regurgitation.

Risk is that anesthesiologist gives paralyzing dose of succinylcholine without proving that mask ventilation is possible!

* *contraindication* – distorted airways!

D. Inhalational Induction

* was the only option before thiopental:
* induction consisted of having patient inhale anesthetic (ether, chloroform) through face mask while anesthesiologist gradually assumed airway maintenance.
* induction was often turbulent and hazardous because ***patients spend several minutes in stage 2 ("excitement stage")*** - agitated and combative (required physical restraint), at risk for laryngospasm, vomiting-aspiration.
* modern inhalational anesthetics have ***rapid stage 2***.
* indicated in:
1. **small children** - progress through stage 2 rapidly + postinduction intravenous catheterization avoids trauma of insertion in awake, struggling children.
2. adult patients at **severe risk for bronchospasm**
3. some patients in whom airway may be difficult to secure.

E. Combined Intravenous-Inhalational Induction

- ***smooth, rapid hypnosis*** + ***deep level of inhalational anesthesia before airway instrumentation***.

1. preoxygenation
2. **intravenous** induction agent
3. potent **inhalational** agent (+ nitrous oxide) by face mask to deepen anesthetic.
4. **airway maintenance** & ventilation:
5. face mask (more difficult to master than intubation); chin tilted, neck extended; mandible displaced anteriorly; ± oral / nasal airway.

if procedure is brief, case can be completed using face mask.

1. short-acting **muscle relaxant** (succinylcholine) → endotracheal intubation.
2. laryngeal mask airway (does not protect against aspiration!) - avoids unnecessary intubations (e.g. fewer hypertensive & tachycardic episodes).

Difficult Airway Management

* the most crucial skill in anesthesia.
* difficult direct laryngoscopy occurs in 1.5-8.5%, failed intubation - in 0.13-0.3%.
* temporizing or life-saving interventions:

LMA (laryngeal mask airway)

Combitube

lighted stylet

Bullard laryngoscope.

Anesthesia Maintenance

Ensure that patient has **no pain** and **no recall** – use titratable combination of:

1. intravenous opioids and hypnotics
2. nitrous oxide + potent inhalational agent.
3. muscle relaxants.
* if *no potent inhalational agents* are used, technique becomes more dependent on amnesia from sedative-hypnotics (e.g. midazolam) and is often termed **nitrous-narcotic** **technique**.
* some prefer to use **total intravenous anesthesia** - continuous infusion of short-acting hypnotic (e.g. propofol) and short-acting opioid.
* titration of agents for anesthetic depth:
1. changes in ***BP & heart rate*** - indirect evidence of adequate anesthesia; satisfactory but can result in either inadequate anesthesia or in administration of more agent than is necessary.
2. ***BIS monitor*** – more reliable *see above*

Emergence from Anesthesia

1. **withdrawal** of potent inhalational agents + cessation of continuous infusion of intravenous agents.
* drug *redistribution from brain* (rather than drug metabolism\*) to muscles → adipose tissue underlies recovery from anesthesia.

\**drug metabolism* is usually very slow due to accumulation in adipose tissue!

* inhalational agents are most rapidly eliminated from body (no postoperative respiratory depression).
1. **extubation** occurs when:
2. patient ***follows commands*** and ***demonstrates sufficient strength*** to breathe spontaneously and protect airway.
3. patient is still ***deeply anesthetized***, i.e. *deep extubation* (only if patient is not at risk for aspiration) - emergence occurs with natural airway; indicated in patients who are at risk for bronchospasm with trachea stimulation during emergence.
4. **monitors removed, patient is transported** to PACU / ICU.
* *risk for hypoxemia* during transport (continue oxygen or pulse oximetry).
* if received invasive monitors intraoperatively, monitoring is often continued *en route* to PACU / ICU.

Intraoperative Fluid And Blood Management

* isotonic saline.
* surgical stress is associated with hyperglycemic response - glucose is not required intraoperatively.
* simplest formula for replacement of fluid losses (in addition to replacement of blood loss):

4 ml × kg -1 × h -1 - for procedures with **minimal** trauma

6 ml × kg -1 × h -1 - for those involving **moderate** trauma.

8 ml × kg -1 × h -1 - for those involving **extreme** trauma.

* ***perioperative interstitial fluid expansion*** → ***mobilization & return to ECV and plasma volume*** on postoperative day 3 (if cardiovascular system and kidneys cannot effectively function, hypervolemia and pulmonary edema may occur).
* *moderate normovolemic anemia* may be preferable to transfusion of homologous blood in most surgical patients (i.e. transfusion trigger should be at lower than traditional 10 g per 100 ml).
* in 1996, Task Force on Blood Component Therapy of ASA concluded, that transfusion should not be based on single hemoglobin trigger but rather on ***individual patient's risk for inadequate tissue oxygenation***.

POSTANESTHESIA CARE

ASA established standards of postanesthesia care:

1. All patients (general, regional, or monitored anesthesia) receive appropriate postanesthesia care as dictated by responsible anesthesiologist.
2. All patients will be accompanied to PACU by anesthesia provider who is aware of patient's condition.
3. In PACU, patient's condition will be reassessed and report given to care provider assuming responsibility for care.
4. Patient's condition is evaluated continually in PACU.
5. Physician is responsible for discharge from PACU.

Most patients stay in PACU for 30-60 minutes; criteria for discharge from PACU:

1. awake and oriented with stable vital signs.
2. breathing without difficulty, able to protect airway, and oxygenating appropriately.
3. pain, shivering, nausea & vomiting adequately controlled.
4. no evidence of surgical complications (such as postoperative bleeding).
5. resolution of block (in patients receiving regional anesthesia).

#### 1. Postoperative Agitation & Delirium

* 1. pain & anxiety
	2. serious physiologic disturbances (hypoxemia, hypercarbia, acidosis, hypotension, hypoglycemia)
	3. surgical complications
	4. adverse drug reactions.

N.B. ensure that serious underlying conditions are not cause of agitation before empirically treating with pain medications, sedatives, or physical restraints.

#### 2. Respiratory Complications

- most frequent major complications in PACU.

1. **Airway obstruction**:

1. most commonly - ***obstruction of oropharynx by tongue*** caused by residual effects of general anesthetics, pain medications, muscle relaxants.

H: oxygen , head-tilt and jaw-thrust maneuver ± oral / nasopharyngeal airway.

1. ***laryngospasm***

H: continuous positive airway pressure (if ineffective → 10-20 mg succinylcholine).

1. ***glottic edema, postextubation croup*** (in children)

H: humidified oxygen, systemic steroids, racemic epinephrine by nebulization.

1. blood, vomitus or debris in airway
2. vocal cord paralysis
3. external compression by hematoma, dressing, cervical collar.

2. **Hypoxemia**:

1. ***hypoventilation*** (e.g. residual effects of anesthetic agents and muscle relaxants, reluctance to inspire deeply after abdominal or thoracic surgery)
2. most commonly – ***atelectasis*** H: incentive spirometry, vigorous encouragement to inspire deeply and cough.

#### 3. Postoperative Nausea & Vomiting

- one of the most annoying problems; prophylaxis & treatment:

1. **propofol** for anesthesia induction.
2. **droperidol** - effective in subsedative doses.
3. **ondansetron** (and related drugs).
4. **metoclopramide**.

N.B. intravenous coadministration of ondansetron and metoclopramide can produce bradyarrhythmias!

#### 4. Hypothermia

1. risk for ***increased oxygen consumption*** postoperatively (particular problem in CAD - shivering could trigger myocardial ischemia).
2. hypothermia could increase rate of ***surgical infections***.

#### 5. Circulatory Complications

* 1. **Hypotension** – many causes. H: fluids, inotropic agents, Trendelenburg position, oxygen.
	2. **Hypertension** (due to pain, anxiety, hypoxemia and hypercarbia) H: correct underlying cause.

Pain Management

Nociception pathway:

1. **Peripheral nociceptors**.

*NSAIDs* block production of prostaglandins, which, in turn, sensitize nociceptors.

1. **Pain fibers** (unmyelinated Type C and thinly myelinated Type A-δ fibers)

*transcutaneous electrical nerve stimulation* blocks pain pathway by stimulating large myelinated sensory fibers in area.

*local anesthetics* directly inhibit neural transmission.

1. **Modulatory interneurons** in dorsal horn of spinal cord.

*opioids* stimulate inhibitory pathways to modulatory interneurons (intrathecal or epidural infusion bypasses blood-brain barrier)

1. **Spinothalamic tracts** to rostral areas: **thalamus, limbic system, cortex**.

Preoperative Planning & Education

* pain control is as much achieved by **preoperative psychological preparation** (education and allaying of fears) as it is by heavy doses of postoperative analgesics.
* ignorance is often basis for unnecessary anxiety.
* many patients have inappropriate fear that narcotic medications will lead to *postoperative narcotic addiction* - such fears must be eliminated with proper education.

Surgical Techniques

* **videotelescopic / minimal access** surgical approaches result in less pain than more traditional access procedures.
* infusing supplemental **local anesthetic** in wound at procedure time.

Preprocedure Analgesia & Anxiolysis

***Hypersensitization*** occurs when neurons mediating nociception markedly increase their frequency of discharge → ↑degree of perceived pain.

***Central sensitization*** - spinal dorsal horn neurons exhibit "wind-up" phenomenon through activation of *N*-methyl-D-aspartic acid receptors – this is avoided by **preemptive analgesia** (analgesia administered before generation of nociceptive messages).

* favored medication is fentanyl 25-50 μg intravenously, with 3-4 minute lags between doses, and titration up to 100 μg total.
* *naloxone, oxygen* and *suction* must be available in case of oversedation.

Anesthetic Methods and Strategies

##### Multimodal Analgesia incorporates local / regional anesthesia in addition to general anesthesia:

1. **Wound infiltration** with *local anesthetic*
2. **Nerve block** with *local anesthetic* (e.g. interpleural, intercostal, paravertebral blocks are useful in supplementing postoperative pain control for abdominal and chest incisions).
3. **Spinal or epidural blockade** using:
4. *local anesthetics*.
5. *opioids*:
	* dose required is extremely small compared to dose required systemically to provide similar levels of analgesia).
	* regionally specific - spares sensory and motor function (patient can ambulate freely, no risk of urinary retention, skin sores, or other sensory loss-related problems).
	* ***subarachnoid catheters*** are generally avoided (risk of meningitis) – use single dose of morphine (longer duration).
	* optimal opioid for ***epidural catheter*** analgesia is fentanyl (most lipid soluble of opioids).
	* combined local anesthetic and opioid by epidural route may provide even greater analgesia than either group of drugs alone.
6. *clonidine* - drug whose role has not yet been defined for spinal and epidural anesthesia.

Postoperative Medications

1. **intermittent intravenous bolus** of narcotics - appropriate for initial rapid control ± maintenance.

N.B. intramuscular administration of narcotics is to be avoided - more painful, slower effect, more difficult to monitor for adverse reactions.

* doses: 2-10 mg morphine, 50-100 μg fentanyl, 25-50 mg meperidine.
* some patients are accustomed to narcotic analgesics - require increased amounts of medication - administer increasing doses as long as there is clear indication for it and there are no signs of oversedation.
1. **constant infusion** of narcotics - standard of care; concept of PCA (patient-controlled analgesia) - patient controls small fixed boluses at preset intervals.
* surgeon decides on narcotic amount at each dose, and maximal number of doses per hour(*lockout period*) - to avoid oversedation.
1. **oral analgesics** (dosing every 3-4 hours)
	1. narcotic (oxycodone, hydromorphone, meperidine, codeine)
	2. nonnarcotic (acetaminophen, aspirin).

##### Opioid Analgesics

Narcotic analgesics remain mainstay for controlling postoperative pain.

* produce analgesia through direct action on CNS.
* all provide ***excellent analgesia***, and all have similar side effects, although to varying degrees.
* morphine is most commonly used.

##### Nonopioid Analgesics

* ability to relieve pain and decrease opioid consumption.
* contraindicated after operations such as cataract surgery in which bleeding would be catastrophic.
* one form of parenteral NSAID: ketorolac tromethamine.

##### Tramadol - acts by both *catecholamine reuptake inhibition* and *agonist at μ1 receptors*.

##### potency ≈ oral meperidine or codeine.

CONSCIOUS SEDATION

- altered level of consciousness (patient is able to maintain airway and is able to respond to verbal commands or physical stimulation appropriately).

* patient must be monitored by trained health care professional.
* parameters that must be monitored every 5 minutes: BP, pulse, respiratory rate, level of consciousness, oxygen saturation (the single most sensitive and affected parameter!).

Medications for conscious sedation:

- rapid onset of action and duration of action is relatively short.

1. Fentanyl (reversed by naloxone)
2. Midazolam (reversed by flumazenil) - profound amnestic effect!; no analgesic properties.
3. Propofol
4. Ketamine
5. Droperidol

For *all* medications used for conscious sedation in *all* patients, key is **titration** - never give whole dose as bolus infusion (could lead to oversedation in large percentage of patients)!!!

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