Reading Material for

Anesthesia Technician

(Part-B)

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PREFACE

This is a two years post matric teaching program of Anesthesia Technician for the students of Allied Health Sciences. The purpose of this reading material is to provide basic education to the paramedics about anesthesia. This reading material attempts to cover almost all the basic theoretical knowledge required by students about anesthesia so that they can perform their work better in collaboration with anesthesiologists in operation theaters.

This reading material aims at using basic language skills to make it easier for better understanding of the subject. The contributors have put up the best efforts to make it concise and provide all the important concepts including the practical aspects.

Hopefully, this reading material provides the best of the knowledge in favour of students. .

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Section I

PRINCIPLES OF ANESTHESIA

Chapter 1

GENERAL ANESTHETICS OVERVIEW

1.1 INTRODUCTION

Anesthesia is state of complete loss of sensation, feeling, awareness and movement so that other medical and surgical procedures could be carried out. It could be:

Regional anesthesia

General anesthesia

GENERAL ANESTHESIA:

The American Society of AZAnesthesiologists (ASA) defines general anesthesia as a drug-induced loss of consciousness during which patients are not arousable, even by painful stimulation. The ability to independently maintain ventilatory function is often impaired.

It has 3 stages

1. Induction
2. Maintenance
3. Emergence

General anaesthesia may be produced by many drugs which depress the CNS, including sedatives, tranquillizers and hypnotic agents. However, for some drugs, the doses required to produce surgical anaesthesia are so large that cardiovascular and respiratory depression commonly occur, and recovery is delayed for hours or even days. Only a few drugs are suitable for use routinely to produce anaesthesia after intra venous (i.v.) injection.

1.2 INTRAVENOUS ANAESTHETIC AGENTS

Intravenous anaesthetic agents are used commonly to induce anaesthesia, as induction is usually smoother and more rapid than that associated with most of the inhalational agents. Intravenous anaesthetics may also be used for maintenance, either

alone or in combination with nitrous oxide; they may be administered as repeated bolus doses or by continuous i.v. infusion. Other uses include sedation during regional anaesthesia, sedation in the intensive care unit (ICU) and treatment of status epilepticus.

Some common IV anesthetics are given below with some description

1.2.1 PROPOFOL

This phenol derivative was identified as a potentially useful intravenous anaesthetic agent in 1980, and became available commercially in 1986. It has achieved great popularity because of its favourable recovery characteristics and its antiemetic effect.

Physical Properties and Presentation

Propofol is extremely lipid-soluble, but almost insoluble in water. The drug was formulated initially in Cremophor EL. However, several other drugs formulated in this solubilizing agent were associated with release of histamine and an unacceptably high incidence of anaphylactoid reactions, and similar reactions occurred with this formulation of propofol. Consequently, the drug was reformulated in a white, aqueous emulsion containing soyabean oil and purified egg phosphatide. Ampoules of the drug

contain 200mg of propofol in 20mL (10mgmL–1), and 50mL bottles containing 1% (10mgmL–1) or 2% (20mgmL–1) solution, and 100mL bottles containing 1% solution, are available for infusion. In addition, 50mL prefilled syringes of 1 and 2% solution are available and are designed for use in target-controlled infusion techniques. Recently, a 0.5% solution has been made available (5mgmL−1 in 20mL). This produces less pain on injection, and is intended primarily for use in children.

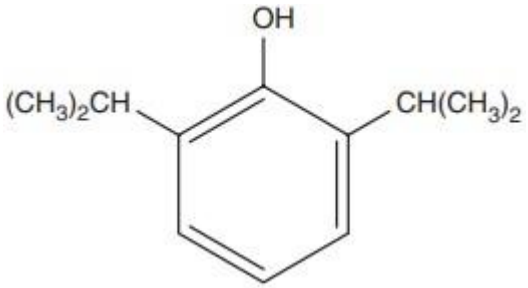


Fig. Chemical structure of propofol

Pharmacokinetics

In common with other i.v. anaesthetic drugs, propofol is distributed rapidly, and blood concentrations decline exponentially. Clearance of the drug from plasma is greater than would be expected if the drug was metabolized only in the liver, and it is believed that extrahepatic sites of metabolism exist. The kidneys excrete the metabolites of propofol (mainly glucuronides); only 0.3% of the administered dose of propofol is excreted unchanged. The terminal elimination half-life of propofol is 3–4.8h, although its effective half-life is much shorter (30–60min). The distribution and clearance of propofol are altered by concomitant administration of fentanyl. Elimination of propofol remains relatively constant even after infusions lasting for several days.

Adverse Effects

- Cardiovascular depression.
- Respiratory depression.
- Excitatory phenomena.
- Pain on injection.
- Allergic reactions.

Indications

- Induction of anesthesia.

- Sedation during surgery. · Total i.v. Anesthesia · Sedation in ICU.

Absolute Contraindications

- Airway obstruction
- known hypersensitivity to the drug

1.2.2 THIOPENTAL SODIUM

Physical Properties and Presentation

Thiopental sodium, the sulphur analogue of pentobarbital, is a yellowish powder with a bitter taste and a faint smell of garlic. It is stored in nitrogen to prevent chemical reaction with atmospheric carbon dioxide, and mixed with 6% anhydrous sodium carbonate to increase its solubility in water. It is available in single-dose ampoules of 500mg and is dissolved in distilled water to produce 2.5% (25mgml–1) solution with a pH of 10.8; this solution is slightly hypotonic. Freshly prepared solution may be kept for 24h.

The oil/water partition coefficient of thiopental is 4.7, and the pKa 7.6.

Pharmacokinetics

Blood concentrations of thiopental increase rapidly after i.v. administration. Between 75 and 85% of the drug is bound to protein, mostly albumin; thus, more free drug is available if plasma protein concentrations are reduced by malnutrition or disease. Protein binding is affected by pH and is decreased by alkalemia; thus the concentration of free drug is increased during hyperventilation. Some drugs, e.g. phenylbutazone,

occupy the same binding sites, and protein binding of thiopental may be reduced in their presence.

Thiopental diffuses readily into the CNS because of its lipid solubility and predominantly un-ionized state (61%) at body pH. Consciousness returns when the brain concentration decreases to a threshold value, depending on the individual patient, the dose of drug and its rate of administration, but at this time nearly all of the injected dose is still present in the body.

Metabolism of thiopental occurs predominantly in the liver, and the metabolites are excreted by the kidneys; a small proportion is excreted unchanged in the urine. The terminal elimination half-life is approximately 11.5 h. Metabolism is a zero-order process; 10–15% of the remaining drug is metabolized each hour. Thus, up to 30% of the original dose may remain in the body at 24 h. Consequently, a 'hangover' effect is common; in addition, further doses of thiopental administered within 1–2 days may result in cumulation.

Adverse Effects

- Hypotension.
- Respiratory depression.
- Tissue necrosis.
- Intra-arterial injection.
- Laryngeal spasm.
- Bronchospasm.
- Allergic reactions.
- Thrombophlebitis.

Indications

- Induction of anesthesia
- Maintenance of anesthesia – thiopental is suitable only for short procedures because cumulation occurs with repeated doses
- Treatment of status epilepticus · Reduction of intracranial pressure.

Absolute Contraindications

- Airway obstruction – intravenous anesthesia should not be used if there is anticipated difficulty in maintaining an adequate airway e.g. epiglottitis, oral or pharyngeal tumors.

- Porphyria – barbiturates may precipitate lower motor neuron paralysis or severe cardiovascular collapse in patients with porphyria.
- Previous hypersensitivity reaction to a barbiturate.

1.3 INHALATIONAL ANAESTHETIC AGENTS

Volatile and gaseous anesthetic agents are used widely for maintenance of anaesthesia and, under some circumstances, for induction of anaesthesia. In many situations, it is appropriate to use a mixture of 66% N2O in oxygen and a small concentration of a volatile agent to maintain anaesthesia, although for reasons discussed below there are occasions when an anaesthetist might wish actively to avoid the use of nitrous oxide.

1.3.1 AGENTS IN COMMON CLINICAL USE

In Western countries, it is customary to use one of the four modern volatile anaesthetic agents – isoflurane, desflurane, sevoflurane or halothane – vaporized in a mixture of nitrous oxide in oxygen or air and oxygen. The use of halothane has declined because of medicolegal pressure relating to the very rare occurrence of hepatotoxicity. The use of sevoflurane has increased rapidly, particularly in paediatric anaesthesia because of its superior quality as an inhalational induction agent. Desflurane produces rapid recovery from anaesthesia, but it is very irritant to the airway and is therefore not used as an inhalational induction agent.

The following account of these agents, with a comparison of their pharmacological properties, may tend to exaggerate the differences between them. However, an equally satisfactory anaesthetic may be administered in the majority of patients with any of the four agents.

ISOFLURANE

Isoflurane (1-chloro-2,2,2-trifluoroethyl difluoromethyl ether) is an isomer of enflurane and was synthesized in 1965. Clinical studies were undertaken in 1970, but because of early laboratory reports of carcinogenesis (which were not confirmed subsequently) it was not approved by the Food and Drug Administration in the United States until 1980.

Physical Properties

Isoflurane is a colourless, volatile liquid with a slightly pungent odour. It is stable and does not react with metal or other substances. It does not require preservatives. Isoflurane is non-flammable in clinical concentrations. The MAC of isoflurane is 1.15% in oxygen and 0.56% in 70% nitrous oxide.

Advantages

In summary, the advantages of isoflurane are:

- rapid recovery
- minimal biotransformation with little risk of hepatic or renal toxicity· very low risk of arrhythmias · muscle relaxation.

Disadvantage

Its disadvantage is:

· a pungent odour which makes inhalational induction relatively unpleasant, particularly in children.

SEVOFLURANE

Sevoflurane (fluoromethyl-2,2,2-trifluoro-1-ethyl ether) was first synthesized in 1968 and its clinical use reported in 1971. The initial development was slow because of some apparent toxic effects, which were found later to be caused by flawed experimental

design. After its first use in volunteers in 1981, further work was delayed again because of problems of biotransformation and stability with soda lime. The drug has been available for general clinical use since 1990.

Physical Properties

It is non-flammable and has a pleasant smell. The blood/gas partition coefficient of sevoflurane is 0.69, which is about half that of isoflurane (1.43) and closer to those of desflurane (0.42) and nitrous oxide (0.44). The MAC value of sevoflurane in adults is between 1.7 and 2% in oxygen and 0.66% in 60% nitrous oxide. The MAC, in common with other volatile agents, is higher in children (2.6% in oxygen and 2.0% in nitrous oxide) and neonates (3.3%) and it is reduced in the elderly (1.48%). It is stable and is stored in amber-coloured bottles. In the presence of water, it undergoes some hydrolysis and this reaction also occurs with soda lime.

Advantages

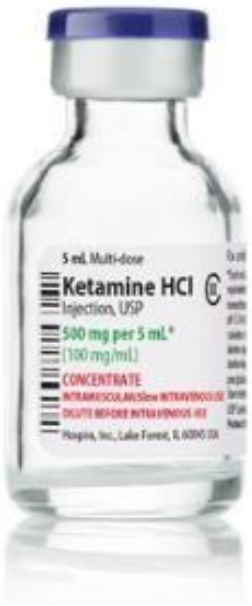
- smooth, fast induction
- rapid recovery
- ease of use, requiring conventional vaporizers (particularly when compared with desflurane).

Disadvantages

· production of potentially toxic metabolites in the body (more a theoretical problem)· instability with carbon dioxide absorbers · relative expense.

1.4 KETAMINE HYDROCHLORIDE

This is a phencyclidine derivative and was introduced in 1965. It differs from other i.v. anaesthetic agents in many respects, and produces dissociative anaesthesia rather than generalized depression of the CNS.



Physical Characteristics and Presentation

Ketamine is soluble in water and is presented as solutions of 10mgmL–1 containing sodium chloride to produce isotonicity and 50 or 100mgmL–1 in multidose vials which contain benzethonium chloride 0.1mgmL–1 as preservative. The pH of the solutions is 3.5–5.5. The pKa of ketamine is 7.5.

Pharmacokinetics

Only approximately 12% of ketamine is bound to protein. The initial peak concentration after i.v. injection decreases as the drug is distributed, but this occurs more slowly than with other i.v. anaesthetic agents. Metabolism occurs predominantly in the liver by demethylation and hydroxylation of the cyclohexanone ring; among the metabolites is norketamine, which is pharmacologically active. Approximately 80% of the

injected dose is excreted renally as glucuronides; only 2.5% is excreted unchanged. The elimination half-life is approximately 2.5h. Distribution and elimination are slower if halothane, benzodiazepines or barbiturates are administered concurrently.

After IM injection, peak concentrations are achieved after approximately 20min.

Adverse Effects

- Emergence delirium, nightmares and hallucinations
- Hypertension and tachycardia – this may be harmful in previously hypertensive patients and in those with ischaemic heart disease
- Prolonged recovery
- Salivation – anticholinergic premedication is essential
- Increased intracranial pressure
- Allergic reactions – skin rashes have been reported.

Indications

- The high-risk patient.
-
- Difficult locations.
- Analgesia and sedation.
- Developing countries.

Absolute Contraindications

- Airway obstruction – although the airway is maintained better with ketamine than with other agents, its patency cannot be guaranteed. Inhalational agents should be used for induction of anaesthesia if airway obstruction is anticipated.
- Raised intracranial pressure.

Chapter 2

OTHER MEDICATIONS USED DURING ANESTHESIA

2.1 INTRODUCTION

The aim of general anesthesia is to ensure patient safety, reduce patient suffering, and allow optimal conditions for the surgeon to perform the operation. The patient properties that facilitate this are unconsciousness, amnesia, analgesia, and often paralysis. No single agent reliably produces all these properties safely. Often, an operating room pharmacy will prepare a standardized tray containing medications from each class (induction agent, neuromuscular blocker, antiemetics, etc.). Medications are the cornerstone of an anesthetic, and as an anesthesia technician, you will need a thorough understanding of the wide variety of medications used by the anesthesia provider. This may seem overwhelming, but in fact, many have already been discussed in this text along with the organ systems they affect:

This chapter will provide more comprehensive coverage of the fundamental intravenous medications used to induce anesthesia: the sedative-hypnotics, the opioids, and the neuromuscular blockers. Adjunct pain medications will be discussed, as well as anti-nausea medications.

Fig. Sample anesthesia tray, replenished for each case, with drugs from each common class of non-controlled medication.

2.2 BENZODIAZEPINES

This class of medications reduces anxiety and induces a sense of calm and wellbeing by stimulating the GABAA receptor in the central nervous system (CNS). Benzodiazepines also cause amnesia, one of the principal components of an anesthetic. Midazolam is used most commonly by anesthesiologists, and is often administered intravenously just before heading to the operating room. It can also be given orally to pediatric patients prior to IV placement and induction of general anesthesia. Benzodiazepines are also very effective antiseizure medications and are the first line of treatment for an active seizure. Other drugs in this class include lorazepam (Ativan) and diazepam (Valium).

2.3 DEXMEDETOMIDINE

Dexmedetomidine is a highly selective blocker of alpha-2 adrenergic receptors. This causes decreased release of epinephrine and norepinephrine, resulting in a decrease in heart rate and blood pressure. Dexmedetomidine blocks these receptors in

an area of the brain responsible for arousal; this results in sedation. Dexmedetomidine also blocks these receptors in part of the spinal cord that transmits pain, resulting in analgesia.

A key property of dexmedetomidine is its ability to produce sedation without significant respiratory depression. It is thus popular for awake fiberoptic intubation and for ICU patients weaning from the ventilator. Dexmedetomidine also has an analgesic effect and can be administered as an adjunct to general anesthesia to reduce opioid administration, particularly in patients with morbid obesity and obstructive sleep apnea.

2.4 ANALGESICS

Medications that reduce pain are called analgesics. Anesthetic medications vary in their analgesic properties. Benzodiazepines and propofol are not analgesic. Volatile anesthetics provide some analgesia, but patients will often respond to a painful stimulus in the form of increased heart rate and blood pressure unless an analgesic is administered. Ketamine and dexmedetomidine provide significant analgesia. The most profoundly analgesic systemic medications, however, are the opioids.

2.5 OPIOIDS

Opioids are a family of medications that include the naturally occurring opiates (codeine and morphine) as well as synthetically produced ones such as fentanyl, hydromorphone (Dilaudid), and oxycodone. They are the most widely used medications for the treatment of pain and are particularly effective at treating acute pain. Opioid receptors are found throughout the body, primarily in the brain, spinal cord, peripheral neurons, and GI tract.

Opioids with a rapid onset are given commonly in anesthesia. These include fentanyl, alfentanil, sufentanil, and remifentanil. Opioids with a short half-life may be given by infusion. Opioids with a slower onset and longer half-life, such as morphine and hydromorphone, may be given when a more predictable or longer duration of action is desired, in the recovery unit, or on the postoperative floor.

Fig. Fentanyl, a short-acting opioid.

Fig. Morphine, a long-acting opioid.

Fig. Hydromorphone, another long-acting opioid.

The reliable analgesia achieved by opioids comes with adverse effects. One of the most important is respiratory depression. Opioids decrease the brain's response to circulating carbon dioxide, the body's primary respiratory drive. This causes patients to breathe less frequently. Opioids are also a potent stimulant of nausea and vomiting. Opioids decrease sympathetic activity, which can lower blood pressure and heart rate. Muscle rigidity can occur with higher doses of opioids often given at induction of anesthesia. This can severely impair the ability to mask ventilate a patient. Opioids also can cause urinary retention and constipation.

2.6 NON OPIOIDS ANALGESICS

Opioid analgesics have significant adverse effects, not the least of which is that they are addictive. In addition to their sedating effects, they are less effective than other pain relief methods at relieving pain when patients move, and they reduce appetite: the net result is that patients tend not to get up, move around, and eat—all of which delays surgical recovery. Thus, anesthesia and surgical practice is moving away from opioid based pain relief to the use of multiple, less-toxic interventions together—a concept known as multimodal analgesia. Not only does it produce better analgesia, multimodal analgesia can reduce opioid administration and its secondary adverse effects.

2.6.1 Acetaminophen

Acetaminophen, commonly known under the brand name of Tylenol, is one of the most common analgesic and antipyretic medications in the world. Unlike aspirin and other Nonsteriodal anti-inflammatory drugs (NSAIDs) acetaminophen has virtually no anti-inflammatory properties. The mechanism of action is unknown. Acetaminophen is often given with other nonopioid analgesics that work through different mechanisms. It produces pain relief on its own, but also has “synergy” with other pain medications in some studies, it cuts the amount of opioid pain medicine patients use in half.

Acetaminophen is most conveniently given orally with a small sip of water before an operation; an intravenous formulation is currently very costly. A rectal suppository is also available, and in common pediatric use. Liver toxicity is a potential adverse effect, thus proper attention to doses is required.

2.6.2 Nonsteroidal Anti-inflammatory Drugs

NSAIDs are some of the most common medications administered in the world for the common ailments, aches, and pains that occur in everyday life. There are many drugs from this class on the market, including many that can be obtained over the counter. NSAIDs work by inhibiting enzymes known as COX-1 and COX-2. The effect diminishes production of prostaglandins, and in turn reduces pain, inflammation, and fever if present. The most commonly used medications in this class, including the over-the-counter NSAIDs like ibuprofen, are nonselective and block both enzymes. Ketorolac is commonly used perioperatively and is the most effective drug in this class. More recently, medications selective for COX-2 only have been developed, the most common used perioperatively is celecoxib (Celebrex).

NSAIDs can cause adverse effects in multiple systems so they must be carefully administered, particularly in surgical patients who may be vulnerable to these effects. Nonselective COX inhibitors can cause decreased platelet function, GI ulceration and bleeding, renal dysfunction, inhibition of bone healing, and bronchospasm. Selective COX-2 inhibitors have improved adverse effect profiles.

2.7 NEUROMUSCULAR BLOCKING AGENTS AND REVERSAL AGENTS

Muscle paralysis is achieved using medications that interfere with transmission of information at the nicotinic acetylcholine (ACh) receptor at the junction where nerves and muscle cells interact. This interaction is covered in detail in Chapter 14, Neuromuscular Anatomy and Physiology. Neuromuscular blocking agents (NMBAs) are generally grouped into two categories depending on whether they “depolarize” the membrane of

the affected muscle cell or not. Thus, the groups are appropriately titled depolarizing and nondepolarizing NMBAs.

Succinylcholine (SCh) is the only currently used depolarizing NMBA. It is essentially two acetylcholine molecules bound together. SCh binds the nicotinic ACh receptor causing the muscle to fire. This causes widespread uncoordinated muscle cell contraction called fasciculations, which can be seen 30-60 seconds after an intubating dose of succinylcholine is given. This large-scale firing leads to desensitization of the receptor to further stimulus, resulting in paralysis. SCh provides excellent intubating conditions, as it has the fastest onset and shortest duration of all the available NMBAs.

There are a number of adverse effects to consider when administering SCh. Perhaps the most potentially dangerous is a transient rise in potassium concentration of about 0.5 mEq/L. This rise can be substantially more when the patient has excess immature nicotinic ACh receptors, which can occur in patients with stroke, burns, or spinal cord injuries. Pediatric patients with muscular dystrophies are also at higher risk of hyperkalemia after SCh administration. Muscle aches are common after SCh administration, occasionally even causing more discomfort than the postoperative surgical pain. The SCh-caused muscle contractions also cause transient increases in pressure in multiple body compartments. Increased intraocular pressure makes SCh controversial in open injuries of the eye. SCh also causes increased intracranial pressure, a consideration in emergency neurosurgery.

The nondepolarizing NMBAs can be divided into two groups based on their chemical structure, the aminosteroids and benzyl isoquinoliniums. (Just as with the amide and ester local anesthetics, the two classes of drugs can be distinguished by the spelling of their names: aminosteroids are “-curoniums” and benzyl isoquinoliniums are “curiums”). Vecuronium and rocuronium are the most commonly used nondepolarizing NMBAs in the United States. Both have an “intermediate duration,” (i.e., shorter than the older, “long-acting” NMBAs pancuronium and curare but longer than SCh), minimal metabolites, and are hemodynamically stable. Rocuronium also has the advantage of a fast onset, allowing its use (in high doses) for rapid sequence inductions, with good intubating conditions in less than 60 seconds. The benzyl isoquinoliniums include curare (d-tubocur rine, yes, originally derived from South American arrow poisons!) atracurium, and cis-atracurium. Cis-atracurium is the only benzyl isoquinolinium in widespread clinical use in the United States; atracurium and curare are limited by histamine release and adverse effects (hypotension, bronchospasm). Cis-atracurium is, however, widely used. Like vecuronium and rocuronium, it is of intermediate duration. Unlike both these drugs, it undergoes nonenzymatic degradation and does not rely on the liver or kidney for elimination. This property makes it very useful in patients with failure of these organs.

2.8 NEUROMUSCULAR BLOCKING REVERSAL AGENTS

Adequate muscle strength is imperative [required] upon emergence from anesthesia, particularly when the patient will be extubated. Acetylcholinesterase (AChAse) inhibitors, such as neostigmine, are used to reverse neuromuscular blockade. AChAse inhibitors increase the concentration of ACh in the neuromuscular junction, outcompeting the remaining nondepolarizing NMBA and restoring tone and strength.

The rise in ACh is not limited to the neuromuscular junction, but is widespread throughout the body. Excessive concentrations of acetylcholine at autonomic sites cause signs of cholinergic toxicity including bradycardia, bronchospasm, and salivation. (See Chapter 13, Autonomic Nervous System.) Luckily, the muscarinic acetylcholine receptor at autonomic sites is structurally and functionally different from the nicotinic receptor found at the neuromuscular junction. The muscarinic receptor can be blocked by administering a muscarinic antagonist such as glycopyrrolate or atropine. This prevents the cholinergic side effects, while allowing the increased concentrations of ACh to work on the nicotinic receptors at the neuromuscular junction. Secondary effects of glycopyrrolate and other muscarinic blockers include tachycardia and reduced secretions.

2.9 ANTI EMATICS

Postoperative nausea and vomiting (PONV) is a common problem after general and even regional anesthesia. PONV can cause significant distress to the patient, extend the stay in the recovery area, delay discharge in ambulatory settings, and result in unplanned admissions. Research into effective prevention and treatment has reduced, but not eliminated, PONV. Just as with analgesia, a multimodal approach, using interventions on multiple different pathways at the same time, treats but probably does not eliminate nausea completely. Several interventions (including modifications to anesthesia and pain relief techniques) can only be done for prevention; once nausea and vomiting have begun, available tools are more limited.

Scopolamine, an anticholinergic medication, is effective for nausea and vomiting, but its preferred route is a transdermal patch, and its effect takes 1.5-2 hours to begin. Thus, it is best applied preop. Dexamethasone, a glucocorticoid steroid, is an effective preventative but, curiously, does not work for treatment of nausea and vomiting once it has begun. Medications that antagonize the serotonin 5-HT3 receptor are a mainstay of PONV prevention and treatment, as they are effective and have few side effects. These include medications such as ondansetron and granisetron. Dopamine antagonists are also commonly used medications to treat PONV. Droperidol, haloperidol, metoclopramide, promethazine, and prochlorperazine can all be given for PONV

prevention and treatment. All share (as do the 5-HT3 blockers) a concern regarding provocation of arrhythmias in susceptible patients; it is controversial how significant this may be at the low doses used for PONV treatment. The FDA placed a “black box warning” on droperidol, the most commonly used and well-researched anesthesia PONV drug, in 2001, cautioning against its use without careful monitoring. Promethazine and prochlorperazine are effective but sedating. Metoclopramide’s efficacy for PONV is not as well established.

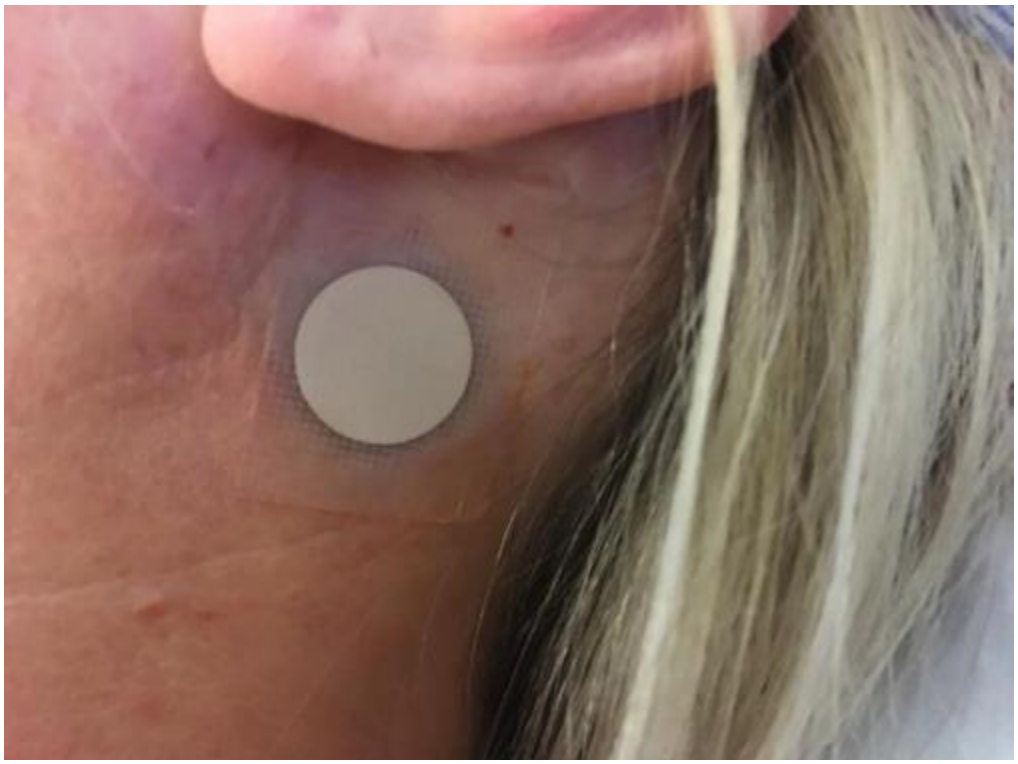


FIG. Scopolamine, a skin patch applied preop as part of a prevention plan for postoperative nausea and vomiting.

Fig. Dexamethasone, a steroid that helps prevent, but does not treat, PONV.

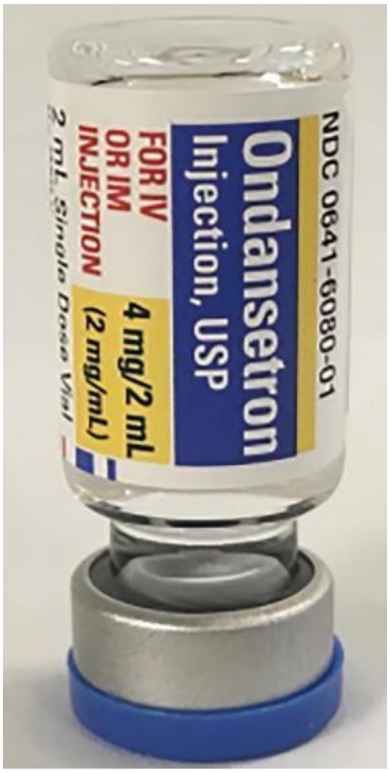


Fig. Ondansetron, an antiemetic.

Chapter 3

INTRAVENOUS FLUIDS

3.1 INTRODUCTION

This chapter addresses several key questions related to intravenous fluids and its therapy during anesthesia and surgery:

1. Why do surgical patients require intravenous access?
2. What are the complications of fluid therapy?
3. What fluids are available for use during anesthesia?
4. How much fluid should be given during anesthesia and surgery?
5. How do anesthesiologists monitor fluid therapy?

3.2 WHY DO SURGICAL PATIENTS REQUIRE INTRAVENOUS ACCESS?

Nearly all patients who require anesthesia for a surgical or diagnostic procedure must have intravenous access, and most also require intravenous fluid therapy. Intravenous access is necessary in the event that anesthetic drugs and fluids are required. Because the volumes and types of intravenous fluids that are administered have specific benefits and can cause complications, it is appropriate to consider administration of intravenous fluids as equivalent to the administration of drugs.

3.2.1 Maintenance fluid replacement

Intravenous fluids are necessary to replace pre-existing fluid deficits and ongoing losses of blood and fluid. During the perioperative period, a patient's intravascular volume is in flux. Before scheduled surgical procedures, patients are usually asked to abstain from oral intake for 6-8 hours to permit their stomachs to empty. This decreases the risk of regurgitation or vomiting of gastric contents and aspiration of those contents into the lungs. Often, with case delays and rearrangement of the surgical schedule, patients are NPO (nil per os, "nothing by mouth") for more than 12 hours. Patients gradually lose both water and electrolytes, such as sodium and potassium (Table 3.1). In patients who are not able to eat or drink, replacement of those losses is referred to as maintenance fluid replacement.

Table 3.1. Calculation of Maintenance Requirements for Water, Sodium, and Potassium

Water: 2,500 mL/d/70 kg	1. 4:2:1 rule—4 mL/kg/h for the first 10 kg of body weight, 2 mL/kg/h for the second 10 kg of body weight, and 1 mL/kg/h for all additional kilograms of body weight 2. 100:50:20 rule—100 mL/kg/d for the first 10 kg of body weight, 50 mL/kg/d for the second 10 kg of body weight, and 20 mL/kg/d for all additional kilograms of body weight
Sodium (Na^+)	1.0 mEq/kg/d (70 mEq/2,500 mL or approximately 28 mEq/L)
Potassium (K^+)	0.75 mEq/kg/d (50 mEq/2,500 mL or approximately 20 mEq/L)

3.2.2 WHAT ARE THE COMPLICATIONS OF FLUID THERAPY?

Complications associated with perioperative fluid therapy can be life threatening, such as

- shock (poor perfusion and oxygen supply to all the tissues of the body)
- pulmonary edema (fluid in the lungs).

However, from a patient's perspective, even the short-term, less serious symptoms can be distressing or delay recovery. Complications can arise from inadequate fluid administration or from excessive administration (Table 3.2).

Table 3.2. Risks of Fluid Administration

	Too Little Fluid	Too Much Fluid
Life threatening	Shock and lactic acidosis Acute renal failure Multisystem organ failure	Pulmonary edema Cardiac failure Airway edema
Not immediately life threatening	Thirst Drowsiness Dizziness Postoperative nausea and vomiting Pain	Peripheral edema Periorbital edema Impaired gut function Impaired wound healing

3.2.3 WHAT FLUIDS ARE AVAILABLE FOR USE DURING ANESTHESIA?

Intravenous fluids are classified as

1. Crystalloid solutions
2. Colloid solutions

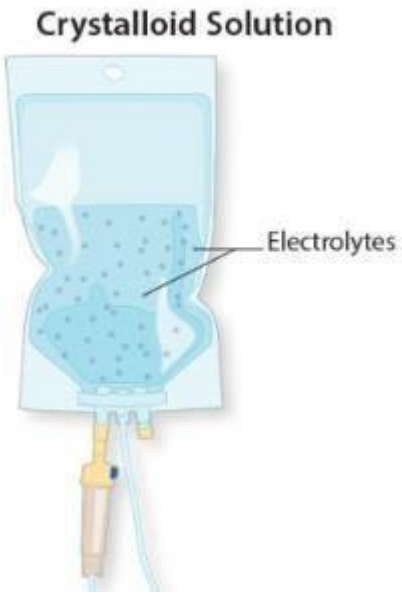
Crystalloid solutions

- A crystalloid solution is an aqueous solution of low molecular weight salts, such as sodium chloride (table salt).
- Crystalloids are generally considered the primary resuscitation fluid.
- Crystalloid solutions quickly escape blood vessels and equilibrate with extravascular, extracellular fluid, so only a small fraction of infused crystalloid produces sustained expansion of plasma volume.
- The most commonly used crystalloid solutions are 0.9% saline (often colloquially and incorrectly called "normal" saline) and lactated Ringer solution, a balanced salt solution that contains small amounts of electrolytes other than sodium and chloride (Table 3.3)

Table 21.3. Contents of Crystalloid Solutions

	Sodium (mEq/L)	Chloride (mEq/L)	Potassium (mEq/L)	Calcium (mEq/L)	Lactate (mEq/L)	pH
Extracellular fluid	140	108	4.5	2.0	5.0	7.3
0.9% Saline	154	154	—	—	—	5.6
Lactated Ringer solution	130	109	4.0	3.0	28	6.6
Plasma-Lyte	140	98	5.0	—	—	7.4

- Intravenous solutions are also prepared with glucose. Such mixtures are usually avoided during the perioperative period, unless a patient is at risk for intraoperative hypoglycemia (such as a patient who has recently received insulin). Increased serum glucose levels can lead to several unwanted complications. These solutions are reserved for maintenance fluids, at slow rates, in hospitalized patients who are NPO.



- The advantages of crystalloid solutions over colloids are that they are inexpensive and equally effective at expanding intravascular volume, although much larger volumes are required to attain equivalent intravascular volume expansion.

Colloid solutions

- Colloid solutions are high molecular weight substances, such as large proteins or large complex sugars, suspended in crystalloid solutions.
- Because of these large molecules, colloid solutions remain inside of blood vessels and maintain plasma volume more effectively than crystalloid solutions.
- Colloid solutions are more commonly used for fluid resuscitation in patients with severe fluid deficits, while infrequently used for surgery of limited duration or extent. For example, colloids may be infused in the patient with massive hemorrhage while awaiting blood products for transfusion or in the resuscitation of a burn patient with low serum albumin levels or large protein losses.
- They can also be used to supplement fluid administration when large volumes of crystalloids have already been infused.
- Colloids seem like the ideal replacement fluid, however, they are expensive and, surprisingly, science has never shown that they produce better results.
- Blood-derived colloids include human serum albumin (commonly available in 5% and 25% concentrations dissolved in 0.9% saline) and plasma protein fraction. Both of

these solutions carry a low, but definitive risk of transmitting blood-borne pathogens, such as hepatitis and other viral diseases.

- Synthetic colloid solutions include dextrose starches (dextrans), gelatins (not available in the United States), and hydroxyethyl starches. Gelatin is a synthetic protein, and dextran and HES are synthetic starches (complex long-chain sugars). These offer lower risks of infection and are much lower cost than albumin. However, they have been associated with allergy, blood clotting problems, and kidney injury and are thus now used with great caution.

No strong clinical evidence establishes the superiority of either crystalloids or colloids. Both colloid and crystalloid solutions can be used effectively for fluid resuscitation. A large clinical trial in critically ill patients recently failed to identify any effect of the choice of colloid or crystalloid on mortality.

3.2.4 HOW MUCH FLUID SHOULD BE GIVEN DURING ANESTHESIA AND SURGERY?

The anesthesia provider must evaluate and replace preexisting fluid deficits and ongoing fluid losses during surgery. By definition, patients who arrive in the operating room NPO for scheduled surgery have slight deficits of water and electrolytes. Intravascular volume will also be slightly decreased. For example, if a 70-kg healthy patient who has been NPO for 12 hours has a water deficit of approximately 1,250 mL and a sodium deficit of approximately 35 mEq, or about the amount of sodium in 250 mL of lactated Ringer solution. The water deficit reduces total body water, normally 60% of total body weight, from 42 L to slightly less than 41 L. Plasma volume (which together with red blood cells constitutes blood volume) would be reduced from approximately 3.0 to 2.95 L (i.e., by 50 mL). In adults, such small blood volume deficits are unlikely to be physiologically important; however, in infants and smaller children, deficits due to NPO requirements for surgery are proportionately greater.

Disease processes and drug treatments also contribute to preoperative volume deficits. For example, a patient who has been in a motor vehicle crash may have suffered large internal or external blood loss and may still be bleeding. A patient who has been vomiting may have lost fluid and electrolytes and also may have been unable to drink fluids. A patient with bowel obstruction may lose large quantities of fluid into the blocked intestine.

In addition to preoperative fluid deficits and surgical fluid loss, the anesthesia provider must also account for evaporative losses and accumulation of edema (swelling) in surgically manipulated tissue. The type of surgical procedure, and the associated exposure of internal tissue, governs the amount of evaporative losses and tissue edema that require consideration (Table 3.4). This volume of fluid is customarily administered as 0.9% saline or lactated Ringer solution.

Table 3.4. Fluid Losses during Surgery due to Evaporation and Edema Formation in the Surgical Site

Degree of Tissue Exposure	Additional Fluid Requirement
Minimal (e.g., hernia repair)	0-2 mL/kg
Moderate (e.g., mammoplasty, cholecystectomy)	2-4 mL/kg
Extreme (e.g., bowel resection, prostatectomy)	4-8 mL/kg

3.3 Estimation of blood loss

During surgery, ongoing losses of fluid and blood are monitored. The anesthesiologist must estimate the blood loss in the surgical field.

- One can measure the blood that has been collected in the surgical suction container (taking into account the amount of irrigating solution that has been added to the field by the surgeons)
- Assess the number of laparotomy pads and sponges, along with the blood accumulated on them
- Visually inspect the field regularly to detect bleeding into the wound or under the surgical drapes.
- Following serial hemoglobin and hematocrit levels is also helpful. The clinical signs mentioned previously (increased heart rate, decreased blood pressure, decreased urine output, etc.) are late signs of hypovolemia.
- By closely monitoring the blood loss throughout the procedure, one is able to continually replace the lost fluid, rather than “get behind” and expose a patient to the risks of insufficient fluid administration.

The anesthesia provider thus evaluates each patient’s volume status, taking into account existing deficits, ongoing losses due to evaporation and movement of fluid into swollen and injured tissues, and blood loss, and estimates the need for fluid therapy.

3.4 HOW DO ANESTHESIOLOGISTS MONITOR FLUID THERAPY?

If too much fluid is bad, and too little fluid is bad, how does the anesthesia provider determine how much the patient needs? Assessment of intravascular volume can be made from the physical examination, laboratory data, and invasive monitoring. These evaluations are continually analyzed as volume resuscitation continues.

An adequate history and physical examination can provide helpful information. Did the patient require a bowel preparation prior to surgery? Did the patient have vomiting or diarrhea? Has the patient experienced massive trauma or a burn? Several other diseases can cause intravascular fluid to redistribute to other "compartments" in the body and must be considered (sepsis, ascites, bowel anomalies, adult respiratory distress syndrome). Physical examination signs that are suggestive of dehydration include dry mucous membranes, faint peripheral pulses, decreased urine output, and low blood pressure accompanied by a rapid heart rate (Table 3.5).

Table 3.5. Physical Examination Signs of Dehydration

	Mild	Moderate	Severe
Blood pressure	Normal	Slightly decreased	Decreased
Heart rate	Normal/increased	Increased (>100 beats/min)	Markedly increased (>120 beats/min)
Mental status	Awake, alert	Lethargic	Obtunded
Mucous membranes	Dry	Very dry	Parched
Urinary output	Mildly decreased	Decreased	Markedly decreased

There are several laboratory measurements that aid in determining a patient's fluid status. These include the hematocrit, serum sodium, creatinine, and blood urea nitrogen (BUN) levels. Serial arterial blood gases provide information regarding how well tissues are being perfused by circulating oxygen in the blood (as measured by the arterial pH, the arterial oxygen pressure [PaO2], and the base deficit). The urine can also be examined by assessing the urinary specific gravity, urine sodium, and urine chloride concentrations. Visual inspection of the urine can also help the anesthesiologist determine how "dry" the patient is, with a low volume and dark color suggesting highly concentrated urine and a dehydrated patient.

Invasive hemodynamic monitoring may also assist in assessing intravascular volume status. One of the simplest and most effective ways anesthesia providers do this is with analysis of the arterial line waveform, which may display a large "pulse pressure variation" (PPV) in a hypovolemic patient who is being mechanically ventilated. A normal PPV is less than about 13; many monitors will automatically calculate this value.

Another way of assessing volume status is via a central venous pressure (CVP) catheter or a pulmonary arterial catheter; these have risks associated with placement

CVP is measured from the tip of a central venous catheter that is properly placed at the junction of the superior vena cava and the right atrium. CVP measurements provide information regarding pressure of blood in the cava and right atrium; this may be, but is not always, related to the volume of blood available to the right side of the heart. CVP monitoring must be assessed in conjunction with clinical signs. Trends in the CVP are often more useful than single measurements. Normal values range from 0 to 12 mm Hg and are affected by many things besides volume so that a single CVP value can represent almost any volume status; a change in CVP, however, may well be due to a change in volume status

A pulmonary arterial pressure catheter can be placed through central venous access into the pulmonary artery to assess volume status when the patient has right heart dysfunction, as it can measure the filling pressure of the left side of the heart, as well as measuring cardiac output via thermodilution. Clinical correlation between data from pulmonary arterial catheterization and other diagnostic data is critical.

Recently, transthoracic echocardiography and transesophageal echocardiography (TEE) have become useful tools in evaluating volume status. A skilled clinician can assess the filling, emptying, and contractile function of the heart in real time with the use of these devices.

Several noninvasive devices to measure cardiac output (which, in turn, depends on the volume filling the heart) are marketed as well, primarily those which provide monitoring of the arterial pulse waveform contour and those which measure the aortic Doppler waveform, either transesophageally or transthoracically. Other technologies utilize CO2 monitoring and thoracic bioimpedance. As an anesthesia technician, you may encounter one or more of these devices used in your institution to achieve cardiac output–based fluid goals.

Recently, clinical research has investigated the influence of different perioperative fluid administration strategies on outcomes, including nausea, vomiting, infection, pulmonary complications, and return of bowel function. These studies have addressed the general question of whether fluid administration should be relatively liberal or conservative, as well as which measurement techniques work best. In patients undergoing outpatient surgery, administration of larger amounts of fluid (approximately 1 L) of balanced salt solution reduces nausea, vomiting, and pain in comparison to administration of smaller amounts of fluid (30-100 mL of balanced solution). In patients

undergoing laparoscopic surgery, administration of 2-3 L of crystalloid fluid is associated with less nausea and vomiting than administration of 750 mL to 1 L of crystalloid. In contrast, for patients undergoing major open abdominal surgery, such as colon resection, conservative fluid strategy seemed to have a better outcome. The administration of approximately 1 L (plus replacement of blood loss) resulted in a more prompt return of bowel function and fewer tissue complications than the administration of 3-4 L, as would have been the standard of care 10 years ago. Similarly in the technology area, “goal-directed” fluid therapy using noninvasive cardiac output devices to guide fluid management has produced equivocal results in surgical outcomes. So far, no device or single measurement alone determines the “right” amount of fluid during surgery. In the future, evidence-based clinical guidelines will no doubt be available for intraoperative fluid therapy in particular types of patients undergoing specific procedures.

Chapter 4

BLOOD TRANSFUSION

4.1 INTRODUCTION

Transfusion of blood products is a common occurrence during surgery. Anesthesia technicians may be called upon to retrieve blood products, help check them in, and help administer them. Transfusion of incompatible blood products to a patient can cause serious patient injury, and anesthesia technicians should be familiar with basic transfusion medicine. This chapter provides an introduction to the different types of blood products, what makes them compatible or incompatible with a patient, how they should be administered, and the potential complications or adverse reactions from the transfusion of blood products.

4.2 BLOOD TYPES

The different blood types (blood groups) and their relationship to the immune system are the basis of transfusion science. Blood types are inherited and represent contributions from both parents. A total of 30 human blood group systems are now recognized by the International Society of Blood Transfusion (ISBT). A blood type is a classification of blood based on the presence or absence of inherited antigenic substances on the surface of red blood cells (RBCs). Antigens, which may be proteins, carbohydrates, glycoproteins, or glycolipids, are present on the cellular membrane of RBCs and are also secreted to plasma and body fluids. Antigens determine the blood group type.

4.2.1 ABO Blood Groups

In 1900, Karl Landsteiner, Austrian biologist and physician, discovered the ABO blood groups for which he received the 1930 Nobel Prize in Medicine and Physiology. The ABO antigen system is the most important determinant of blood type grouping in transfusion medicine. The two major RBC antigens are known as A and B. The blood groups are A, B, AB, and O, where O is when the RBCs lack both A and B antigens. People with AB blood type have RBCs that have both antigens. People with RBCs that only have the A or B antigen are blood types A and B, respectively.

ABO compatibility

ABO compatibility remains the major safety consideration of blood product transfusions (Table 4.1). Compatibility means that the recipient does not recognize the blood transfusion as foreign. Immune systems of virtually all individuals produce antibodies directed against antigens they do not have (anti-B antibodies in type A individuals, anti-A antibodies in type B individuals, and anti-A and anti-B antibodies in type O individuals). The process whereby foreign antigens from blood groups cause production of antibodies directed against them in the recipient is called alloimmunization. This concept is extremely important to the understanding of transfusion medicine. If a recipient patient receives blood that has antigens foreign to the recipient, the recipient can mount a massive immune reaction (allergic reaction) against the foreign blood. These reactions are particularly severe if the recipient has preformed antibodies (a primed immune system) against the foreign antigen. This type of reaction is akin to an anaphylactic reaction except that the foreign antigen is the transfused blood. Humans form antibodies to A or B antigens in the first years of life if they do not have them on their own RBCs. This is thought to be triggered by the exposure to environmental antigens

(food, bacteria, virus, etc.). Thus, humans are usually “primed” against ABO-incompatible blood. A reaction to ABO-incompatible blood is called an acute hemolytic transfusion reaction and is fatal in about 10% of cases. The recipient may not only manifest symptoms of an anaphylactic reaction (low blood pressure, fever, bronchospasm) from the immune mediators released but also suffer because his or her immune system attacks the foreign blood cells, causing them to hemolyze (rupture) and release free hemoglobin into the bloodstream. The primary cause of transfusing ABO-incompatible units (incorrect blood type) is clerical errors in patient identification or errors in sample labeling.

Table 4.1 ABO Compatibility Chart

ABO Compatibility		Donor			
		A	B	O	AB
Recipient	A	Yes	No	Yes	No
	B	No	Yes	Yes	No
	O	No	No	Yes	No
	AB	Yes	Yes	Yes	Yes

(Rh) blood group system

The second most important blood group system is the rhesus (Rh) system. Rh positivity is indicated by the presence of D antigen in the membrane of the RBC; D antigen is absent in Rh (D)-negative individuals. About 15% of people are Rh (D)-negative.

Unlike the ABO system, Rh (D)-negative individuals do not produce anti-Rh (D) antibodies until they are exposed to Rh (D)-positive blood. When an Rh (D)-negative individual is exposed to Rh (D)-positive cells, sensitization occurs and the immune system can produce anti-D alloantibody. Any subsequent exposure to Rh (D)-positive blood can result in a severe adverse reaction. Sensitization can occur by transfusion or during pregnancy. Even in emergencies, Rh (D)- positive blood should not be given to Rh

(D)negative patients to avoid sensitization. Typically, type O Rh (D)–negative blood (O neg) is stored by hospitals for emergency transfusion because of its near-universal safety for patients with untyped blood due to its lack of AB or Rh (D) antigens.

4.2.2 Other systems

About 30 other blood group systems exist in addition to the ABO and Rh (D) systems: Lewis, I system, P system, MNScU system, Kell protein, and the Duffy and Kidd antigens. These antigens can be present on RBCs and result in incompatibility, but these are not necessarily tested for in every patient because they are extremely rare, extremely common, or compatibility can be ensured by providing warmed blood (above 30°C).

Patients who have received multiple transfusions over the course of their life are at higher risk of developing antibodies, and it may be more difficult to find a compatible unit. Procuring a compatible unit can take extra time and may result in a surgical delay.

4.3 COMPATIBILITY TESTING

Before any RBC unit is given to a patient, it undergoes several different tests to ensure that it is compatible with the recipient. The tests are separate from the testing done for diseases such as hepatitis and human immunodeficiency virus (HIV). Screening of potential donors and rigorous testing of donated units have largely eliminated the risk of units containing HIV, hepatitis, and other infections.

4.3.1 Type and screen

The first test, known as the type and screen, is done on donated blood before releasing the unit. This test determines the blood type—A, B, AB, O, and Rh (D)positive/Rh (D)-negative. This testing is also done on the patients to determine their blood type as well as screen for the presence of antibodies to A or B and antibodies against other antigens known to cause hemolytic reactions. The test is performed by mixing the sample blood (the patient’s blood or the donated blood) with a solution containing antibodies against the antigen being screened. For example, if one wants to determine if a patient has A antigen on his or her RBCs (he or she is blood type A or AB), a blood sample from him or her is mixed with a solution containing anti-A antibodies. If the resulting mixture agglutinates (clumps), it is because the antibodies have bound to cells with the A antigen. A second phase of the test involves mixing the patient’s plasma with commercially available O-negative RBCs that have approximately 20 different antigens that can cause a hemolytic reaction.

If the patient's plasma does not agglutinate these cells, the screen is negative. If the patient's plasma does react to the cells, the patient possesses at least one antibody to a significant antigen and the screen is positive. If the screen is positive, further testing must be done to identify the antibody and to locate blood units that lack the antigen.

4.3.2 Cross match

The second test, the crossmatch, checks the patient's blood against a specific donor unit for errors in ABO type. If the screen portion of the type and screen test was negative, the crossmatch consists of matching the patient with compatible donor blood. No real "test" is performed. The crossmatch involves matching the paperwork for the donor unit and the recipient. This step can even be performed by automated vending style machines that scan barcodes from the patient ID and the donor unit. If the patient's screen was positive, a serologic crossmatch test is performed. This test is conducted by mixing the patient's serum with a specific donor unit that has been selected because it lacks the antigens that the patient has antibodies against identified during the screen. A crossmatch is only necessary when the patient receives RBCs, as opposed to plasma or other blood derivatives.

4.3.3 Plasma Compatibility

Compatibility of plasma is different from that of whole blood or RBCs. Plasma from an AB blood type donor can be transfused to a recipient of any blood group. This is because the AB donor plasma lacks A or B antibodies and will not react with the recipient's RBCs even if he or she has the A or B antigen. Type O recipients already have A and B antibodies and can receive plasma from any blood type. The only problem is plasma from a type O donor. This donor has A and B antibodies in the plasma, and it cannot be given to a type A, B, or AB recipient.

In emergency situations when RBC transfusion is immediately needed, abbreviated testing methods are necessary. Type-specific blood is always essential (unless the donor unit is O), but there are abridged versions of the crossmatch: partial crossmatch checks for the most severe errors (ABO-Rh (D) blood type) and takes less than 10 minutes; un-crossmatched blood is less risky in previously un-transfused patients.

4.4 INDICATIONS FOR TRANSFUSION

Indications of blood transfusion can be summed up as:

- Anemia.
- Major Surgical Operation.
- Accidents resulting in considerable blood loss.
- Cancer patients requiring therapy.
- Women in childbirth and newborn babies in certain cases.
- Patients of hereditary disorders like Haemophilia and Thalassemia.
- Severe burn victims.

The American Association of Blood Banks (AABB) guidelines, published in 2012 (http://www.aabb.org/resources), stress the importance of considering symptoms and expected surgical blood loss as well as the Hb concentration in making the decision to transfuse. The AABB recommendations for red cell transfusion after surgery are as follows:

- Consider transfusion if Hb 8 gm/dl or less.
- Transfuse if symptomatic of anaemia – chest pain, orthostatic hypotension or tachycardia unresponsive to fluid resuscitation, or congestive heart failure.
- The same thresholds can be safely applied to patients with stable cardiovascular disease.

Patients who are not actively bleeding should be transfused with a single unit of red cells and then reassessed before further blood is given.

4.5 COMPLICATIONS AND ADVERSE REACTIONS

There are a variety of adverse reactions that come from a few basic sources:

- Contaminated or infected blood (e.g., HIV or hepatitis)
- Incompatible blood
- Problems related to the infusion of RBCs (e.g., transfusion-related lung injury, volume overload, electrolyte disturbances, and coagulopathy).

Table 4.2 summarizes some of the possible complications of transfusions. As soon as an acute adverse reaction to a transfusion is suspected, the first step is to stop the transfusion and call the blood bank. The three most common causes of transfusion related deaths are hemolytic transfusion reactions, septic transfusions, and transfusion related lung injury. Acute hemolytic transfusion reaction is caused by type incompatible blood,

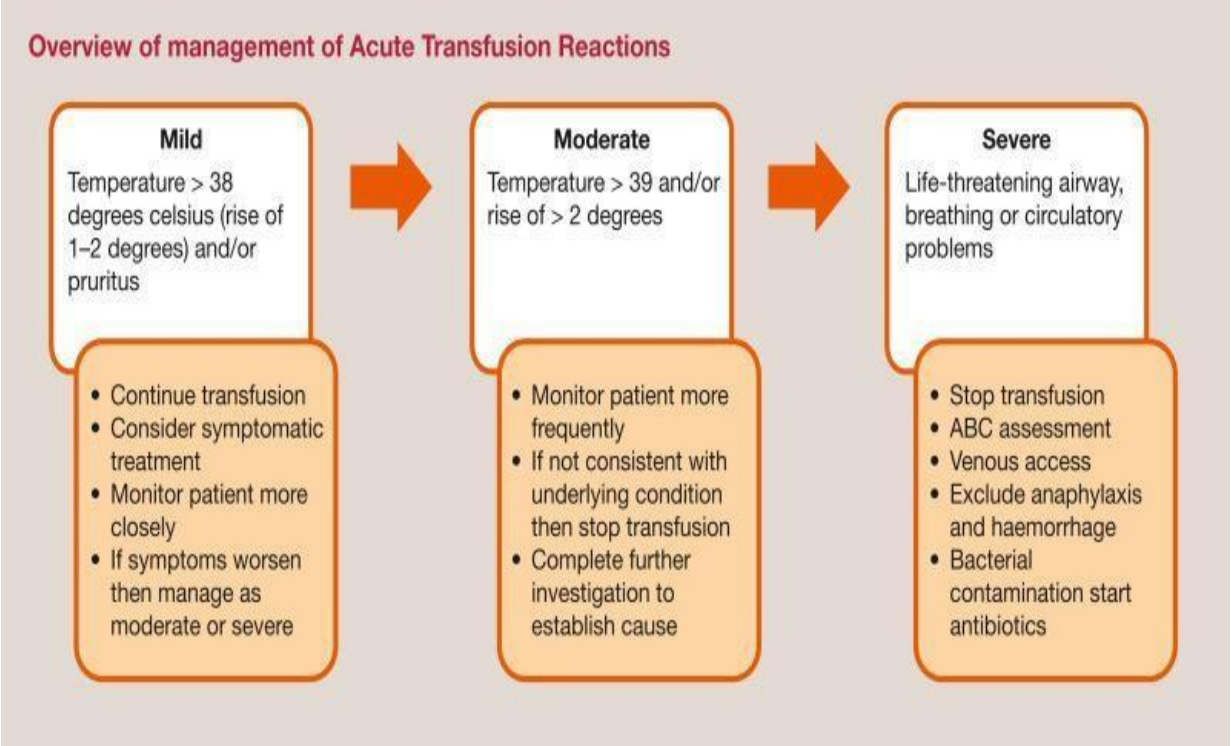
typically due to human error at some point between issuing the unit and transfusion. The body mounts an immune response to the offending blood, which can lead to severe coagulation issues, kidney failure, and even death.

Table 4.2. Other Adverse Reactions

Immune-mediated reactions
Delayed hemolytic transfusion reaction
Febrile nonhemolytic transfusion reaction
Allergic reaction
Anaphylactic reaction
Alloimmunization—sensitization to antigens present in transfusion
Posttransfusion purpura—caused by platelets' transfusion
Transfusion-associated graft vs host disease—immunocompromised at greatest risk
Transfusion-related acute lung injury—causes pulmonary edema
Nonimmunologic reactions
Hypothermia, fluid overload, electrolyte toxicity, iron overload, and others
Infections
Transfusion-associated viral infections: HIV type 1; hepatitis B, C, and G virus; cytomegalovirus (CMV); and other viral and bacterial infections

Other complications are more common during a massive transfusion (loss of at least one times the patient's blood volume):

- Hypocalcemia: The citrate used to preserve blood can bind calcium in the patient, leading to hypocalcemia. This is more common when the units are transfused quickly (>1 unit in 5 minutes), it is a massive transfusion, or the patient has liver dysfunction and has difficulty in metabolizing citrate. Monitoring of blood calcium levels will guide treatment with calcium.
- Coagulopathy: Packed RBC units contain minimal platelets or plasma clotting factors. Patients who receive large transfusions will require replacement of platelets and clotting factors to avoid a dilutional coagulopathy.
- Hyperkalemia: Typical blood units contain less potassium than normal blood; as the blood is stored, the RBCs release potassium. Rapid or large transfusions of RBC units, particularly older units, can lead to a significant potassium increase in the recipient.
- TRALI: transfusion-related lung injury is characterized by the acute onset of the noncardiogenic pulmonary edema following administration of the blood products. It is typically associated with the transfusion of plasma components like thrombocytes and fresh frozen plasma, less likely with RBC transfusion.



HANDLING, VERIFICATION, AND STORAGE

Retaining the oxygen-carrying capacity of blood throughout its shelf life is one of the primary problems of blood storage. Over time, red cells lose their capacity to carry the same amount of oxygen as they could when fresh. It is hard to standardize the capacity of each unit because each starts from a different level and degrades at different rates.

Different products are added to each unit to prolong the shelf life. One of the most common is citrate-phosphate-dextrose-adenine (CPDA-1):

- citrate is an anticoagulant
- phosphate is added as a buffer
- dextrose provides an energy source for the red cells
- adenine allows the cells to make adenosine triphosphate (ATP), a common cellular energy source.

AS-1 (Adsol), AS-3 (Nutricel), and AS-5 (Optisol) are similar to CPDA-1 with slight variations.

Storing the units between 1°C and 6°C slows down the metabolic processes of the red cells, but glycolysis (RBCs do not use the citric acid cycle because they lack mitochondria) still converts glucose to lactate. This accumulation of lactate lowers the

pH of the unit and alters the intra- and intercellular concentrations of sodium and potassium. The lower pH also contributes to the decreased oxygen-carrying capacity of the RBCs. If several units of plasma or packed red cells are needed in the operating room, they are often kept in a cooler or bucket with ice; the ice and blood product should be separated by a towel or other barrier to prevent the blood from freezing. The formation of ice crystals damages the RBCs. Another important reason to keep the blood cold in the operating room is to be able to return it to the blood bank if it is not used.

ADMINISTRATION

Each unit must be checked at the bedside before transfusing.

Checklist before transfusion

- double check the information on the unit, it accompanies paper work, and the patient information. This procedure will vary from institution to institution, but typically requires two people, and often one of them must be licensed (e.g., a physician or a nurse)
- Commonly, the unit number, expiration date, blood type, and some type of patient identifier are used (Fig. 4.1). This is a role that the anesthesia technician frequently participates in.
- Blood products are typically warmed before being given to a patient. This is often achieved with in-line heated tubing. The majority of these units utilize heated water, which is circulated outside an inner set of tubing through which the blood product or fluid is administered. As mentioned earlier, heating units above 30°C can reduce the risk of some complications. Additionally, blood is typically stored at 4°C, and transfusing cold units to a patient could rapidly lower his or her body temperature.

  Platelets are the exception and are stored at room temperature.

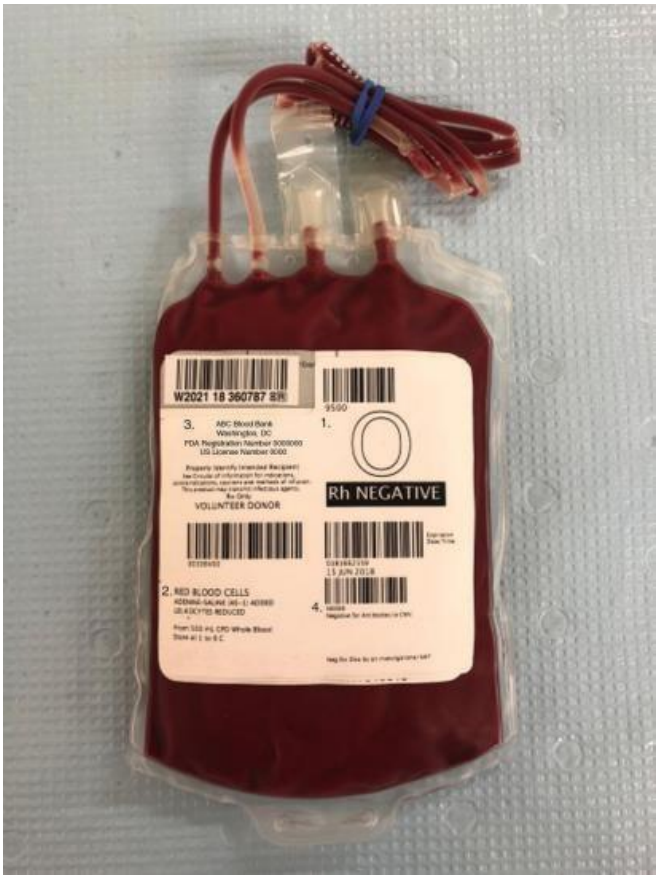


FIGURE 4.1. Transfusion unit of red blood cells (RBCs). 1. Blood type: 0 Rh (D)-negative, 2. RBCs transfusion unit—leuko-reduced, 3. Unit number, 4. Expiration date.

Blood products are typically administered through special blood administration tubing (Fig. 4.2). The vast majority of blood administration sets have an in-line filter to remove cellular debris and coagulated proteins. Most filters are designed for transfusion of two to four RBC units before they should be changed. Refer to the product information for specific guidelines. Some practitioners prefer to attach a separate blood filter to the spike. After multiple units have been transfused, the filter can be replaced without having to replace the entire tubing setup. Again, refer to the product information for the number of RBC units that can be transfused before filter replacement is recommended, as some filters allow up to 10 units. The tubing used in operating rooms usually has a Y connector with two separate “spikes” so that a unit or fluid can be prepared on one spike while the other is being actively used for transfusion or fluid administration. Typical blood administration sets used in the operating room also include a squeezable chamber that allows pumping the fluid or blood products to speed administration. Pediatric blood sets can come equipped with a chamber (buretrol) in which the provider can measure a

specific amount of blood product or fluid. This allows more precise administration of fluids or blood products, which can be critical in pediatric patients.

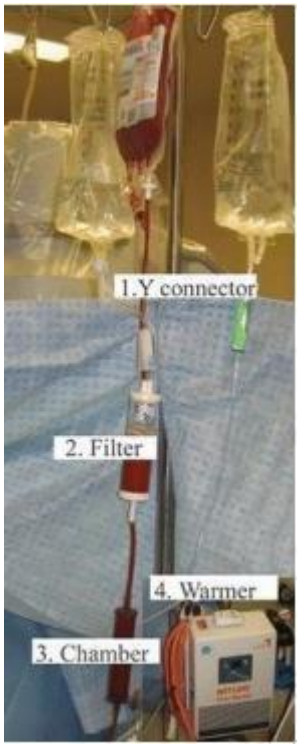


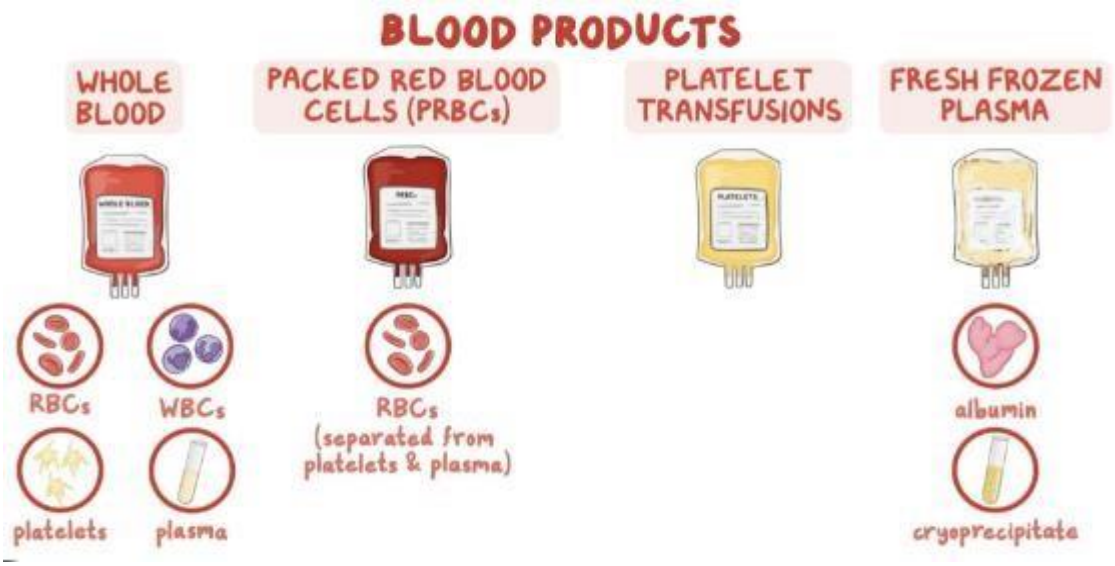
FIGURE 4.2. Blood administration tubing. 1. Y connector. 2. Filter —holds debris and particles. 3. Chamber—blood can be pumped by squeezing the chamber to speed delivery. 4. Warmer—blood is warmed by passing through the hot-line system. It is routine practice to draw blood samples for laboratory testing of the patient's blood before and after transfusion. Testing includes hematocrit and other values like electrolytes, glucose level, coagulation studies, etc., and it is often the anesthesia technician's responsibility to run these samples if the operating room suites have their own blood gas analyzer machine.

4.6 DIFFERENT BLOOD PRODUCTS

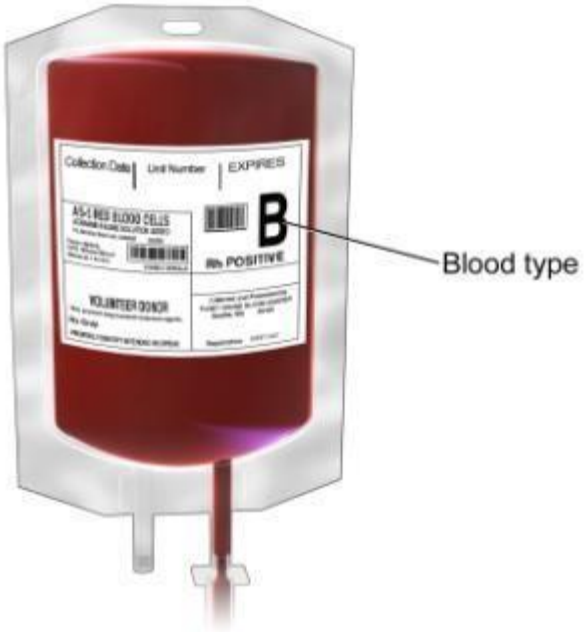
While blood donations typically consist of whole blood, or blood containing all of its normal components (RBCs, plasma, etc.), they are routinely processed into several different products for clinical use. Today, whole blood is not readily available for transfusion and instead serves as the basis for further processing. Because the different components in blood differ in density, blood banks use centrifugation to separate the different parts, resulting in different blood products (see Table 4.3).

Table 4.3. Different Blood Products

Product	Storage Temperature (°C)	Volume (mL)	Supplies	Hematocrit
Whole blood	4	500	Oxygen-carrying capacity, blood volume	40
PRBC packed red blood cells	4	300	Oxygen-carrying capacity, increases hematocrit 3%	70
Fresh frozen plasma	4	200	Factor V and VIII deficiencies	—
Cryoprecipitate	4	10	Factor VIII and fibrinogen	—
Platelets	Room temperature	50-200	Increases platelet count 5,000-10,000/μL	—
Prothrombin complex Kcentra	4	—	Factors II, VII, IX, and X, protein C and protein S	—



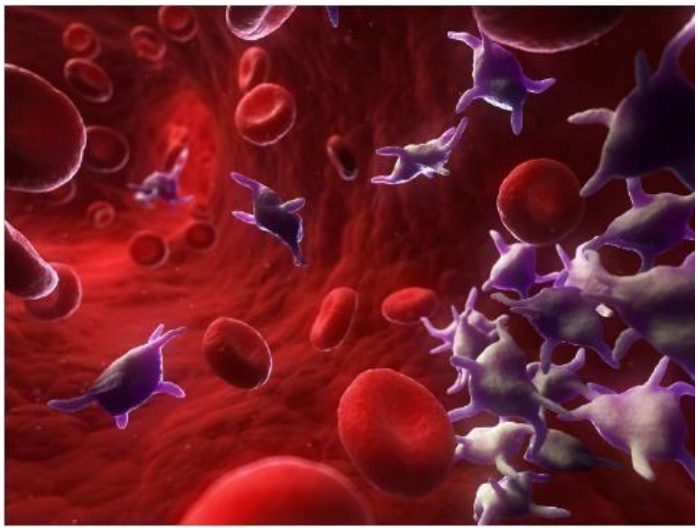
4.6.1 Packed red blood cells (PRBCs): PRBCs provide additional oxygen carrying capacity because of the hemoglobin they contain. Transfusion of white blood cells (leukocytes) increases the risk of infection because they suppress the immune system of the recipient. Transfusion of leukocytes can also sensitize the recipient to leukocyte antigens. For these reasons, PRBC units have the vast majority of leukocytes removed by filtration (leukoreduction). The hematocrit value of a PRBC is 70%. When RBCs are separated from whole blood, plasma and other blood components are retained for other use.



Labeled Blood Bag

4.6.2 Fresh frozen plasma (FFP): FFP contains plasma proteins including factors V and VIII, which are needed for effective blood clotting. FFP replaces the coagulation factors lost with bleeding and can also be used as a reversal agent for anticoagulation drugs like warfarin, in treatment of immunodeficiencies, in antithrombin III deficiency, in massive blood transfusion, and in other coagulation system deficiencies. FFP does increase the circulating volume but should never be used as primary volume expander. FFP is stored frozen and after thawing it must be used between 24 hours and 5 days, which will be indicated on the expiration date printed on the bag. Because of the time it takes to thaw stored FFP, there can be a delay in receiving FFP after it has been requested. As mentioned above, FFP must be compatible with the recipient's ABO-Rh (D) type.

4.6.3 Platelets: Platelets play a vital role in how the body forms blood clots. Platelets can be collected either from whole blood donations or separately from platelet specific donations. Because they are stored at room temperature (never place them in the refrigerator with other blood products), they present a higher risk of bacterial contamination. Indications for platelet transfusion vary but are guided by the patient's platelet count (provided by the laboratory) and the extent of surgical bleeding. It is not necessary to provide ABO-compatible platelets.



4.6.4 Cryoprecipitate: Cryoprecipitate contains high concentrations of clotting factor VIII, vWF and fibrinogen and is used to treat clotting factor deficiencies including hemophilia A. Cryoprecipitate also contains other clotting factors and plasma proteins. Cryoprecipitate should be filtered when administered, and it must be used within 6 hours of thawing.

Cryoprecipitate is usually administered as ABO compatible, but it is not too important since the concentration of antibodies in cryoprecipitate is extremely low.

4.6.5 Prothrombin complex: Prothrombin complex concentrate contains vitamin K–dependent coagulation factors II, VII, IX, and X and antithrombotic protein C and protein S. Administration of the prothrombin complex concentrate increases plasma levels of all components. It is used to reverse the effect of vitamin K antagonist warfarin, to treat factor IX deficiency, hemophilia B, and other bleeding disorders.

Chapter 5

CARDIAC ARREST & BASIC LIFE SUPPORT

5.1 INTRODUCTION

Cardiac arrest occurs when the heart is unable to provide sufficient blood flow to oxygenate the heart and the brain. The heart may or may not have some remaining electrical or mechanical activity, but it is insufficient to produce blood flow or a blood pressure. An awake patient will lose consciousness and stop breathing normally. A cardiac arrest in the perioperative setting is a critical event that will require the coordinated efforts of a team to give the patient the best chance to survive. During a resuscitation, the anesthesia technician must know his or her potential roles on the resuscitation team, what the priorities of the resuscitation are, and what equipment or support the team will require. The American Heart Association has produced national guidelines for the care of patients with cardiac arrest. These are developed via extensive literature review, updated every 5 years, and taught via certification in basic life support (BLS) and advanced cardiac life support (ACLS), which requires renewal every 2 years. BLS covers basic airway management, rescue breathing, cardiopulmonary resuscitation (CPR) with chest compressions, and use of an automated external defibrillator (AED) (Fig. 58.1). ACLS for cardiac arrest includes advanced airway skills, CPR, AEDs and manual defibrillation, heart rhythm diagnosis, and treatment with medications (Fig. 58.2). Your institution may require you, as an anesthesia technician, to be certified in BLS or even ACLS. Use of these evidence-based team approaches ensures that all teams share a common set of expectations and should provide all cardiac arrest patients the highest-quality care

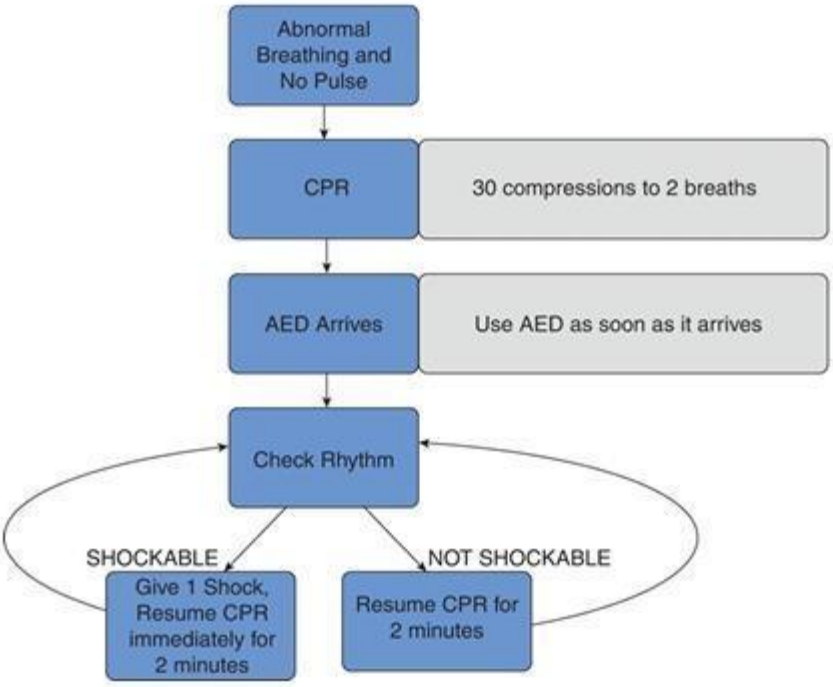


Fig. 2015 Adult BLS algorithm

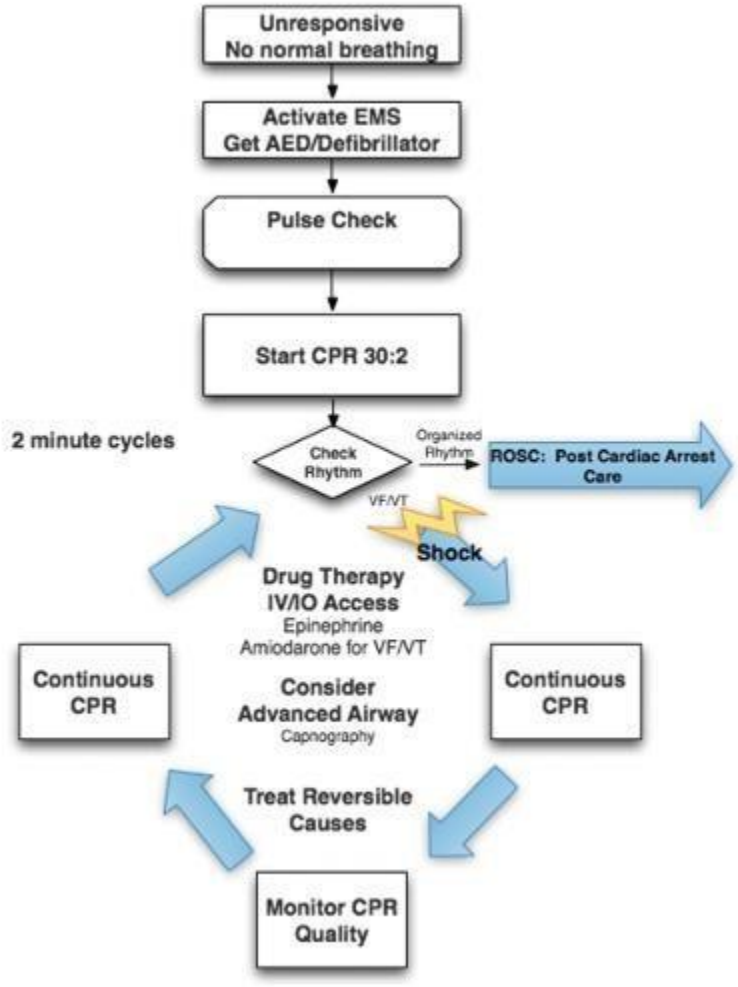


Fig. 2015 Simplified Adult ACLS pulseless arrest algorithm

5.2 PRE-ARREST: INITIAL RESPONSE

Health care providers may call for assistance or even call a full code when a patient's condition is deteriorating. This initiates a formal protocol that rapidly summons resources to the bedside and plans team behavior, even if there is not yet a need for BLS or ACLS. Initial team priorities include the following:

- Bring the code cart (defibrillator, resuscitation drugs, airway equipment).
- Apply defibrillator/pacing pads.
- Provide adequate oxygenation and ventilation.

- Via the endotracheal tube and anesthesia ventilator if in place ○ Via face mask or even bag/valve/mask ventilation if not (or supraglottic airway [SGA] if in place)

- Assess for intubation. Is this an airway emergency? If so, the AT should assemble airway equipment, including difficult airway cart and cricothyrotomy kit.
- Assess for adequate vascular access. If necessary, prepare vascular access equipment.
- Providers may request a transesophageal echocardiography (TEE) or TTE machine and a skilled TEE operator such as a cardiac anesthesiologist.
- Assess the need for additional help or expertise.

5.3 SIGNS OF CARDIAC ARREST

The hallmark of cardiac arrest is the loss of a palpable pulse; CPR should start at that point. A pulse oximeter waveform can be a clue to loss of pulse, but is not a substitute for palpation of the pulse, usually at the carotid. An intra-arterial waveform is a better marker but is still subject to artifact: it is very useful, however, to follow during a resuscitation and is often placed in a near-arrest situation or after return of spontaneous circulation (ROSC). Other signs of perfusion that the provider will be looking for are:

- Loss of respiratory efforts and consciousness in a previously conscious patient
- Arrhythmias (very disorganized heartbeats) or very slow or fast heart rates
- Low end-tidal CO2 (this is a sign of very low flow of CO2-containing blood to the lungs)

5.4 CARDIAC ARREST: INITIAL RESPONSE (BASIC LIFE SUPPORT)

Once cardiac arrest is diagnosed, BLS should be initiated. BLS emphasizes effective chest compressions (“press hard and fast”) and prompt defibrillation. Because there are multiple causes of cardiac arrest in the perioperative setting, the specific equipment and tasks that need to be performed in advanced resuscitation will vary, but the initial goal is to establish effective circulation, maintain ventilation, and shock if necessary.

Operating room team priorities will include the following:

- Turn off anesthetics if applicable and administer 100% oxygen. Assess and confirm the airway including attachments of the breathing circuit.
- Hold surgery if possible/place patient supine and expose the chest.
- Begin high-quality chest compressions; the quality of chest compressions is critical to a successful outcome. Goals of effective compressions include:
- In adults, a compression rate of 100 compressions/min with a depth of 2 inches, allowing complete chest recoil.
- Rotate person giving CPR every 2 minutes to maintain vigor of compressions.

- Minimize interruptions (<10 seconds between compressors or for pulse checks).
- Lower the operating room bed, and arrange a step stool if necessary for proper positioning.
- Elbows should be straight and the heel of the hand on the sternum, just above the xiphoid.
- Monitor for EtCO2 greater than 20 mm Hg.
- If an arterial line is present, assess arterial waveform for pulsatility; if not, a pulse may be palpable with compressions.
- Bring the code cart (defibrillator, resuscitation drugs, airway equipment).
- Apply the defibrillator pads as soon as possible and deliver a shock, if appropriate. · Deliver additional shocks as indicated in the ACLS guidelines.
- Prepare to initiate ACLS drug therapy, epinephrine +/− amiodarone.

5.5 CARDIAC ARREST: SECONDARY RESPONSE

Once initial resuscitation steps are underway (BLS and ACLS), the priority will turn to determining the underlying cause of the cardiac arrest and treating appropriately. The possible underlying cause of the cardiac arrest will dictate which procedures or equipment become a priority. Common to many situations are:

- Advanced vascular access: The patient may need a central line (for central delivery of vasoactive medications), a large-bore introducer, peripheral IV, or rapid infusion device (if the arrest was hypovolemic). Most patients who undergo cardiac arrest, unless it is very brief, will need an arterial line.
- A pressure transducer bag made up.
- Blood gas sampling to assess ventilation, oxygenation, perfusion, acid base status, electrolyte, and glucose abnormalities.
- Pumps and vasoactive medications.
- Transport. Will the patient need to go to the OR, to the ICU, to the cath lab? What monitors, pumps, and other devices will the patient need for transport? What will the receiving team need to prepare? Situations to consider include the following:
- **Arrhythmia:** Patients with a prior history of cardiac rhythm problems can be at an increased risk for arrhythmias during surgery due to the sympathetic stress of surgery, interactions with anesthetic medications, electrolyte imbalances, or the disease condition for which the patient is having surgery. The priorities for treatment will mostly involve treatment recommendations for defibrillation/cardioversion and drug delivery according to the ACLS guidelines. Be prepared to obtain and process arterial or venous blood gas samples to assess for electrolyte or glucose abnormalities, that providers may request infusions of

amiodarone, potassium, and insulin and bolus doses of calcium and magnesium, and that patients may need to travel with defibrillators with pacing capability.

- **Myocardial Infarction:** Insufficient blood flow to even a portion of the heart (myocardial ischemia) can cause myocardial cell death (myocardial infarction). Either myocardial ischemia or infarction can cause a lethal cardiac arrhythmia resulting in cardiac arrest. TEE may be useful in diagnosis for these patients and can show regional wall motion abnormalities or depression of overall heart function. Other clues are ST elevation on the EKG or new-onset chest pain in the awake patient prior to cardiac arrest. The initial treatment for these patients will usually follow the ACLS guidelines. The patient may have to be intubated during the resuscitation. The patient may require vasopressors, antiarrhythmics, or anticoagulants via infusion, and vascular access equipment and infusion equipment should be readily available. They may also require an intra-aortic balloon pump.
-
- **Difficult or Failed Airway:** Severe hypoxemia associated with inability to maintain an airway and oxygenate the blood can rapidly lead to cardiac ischemia, arrhythmias, (particularly bradycardia), and cardiac arrest. Respiratory causes of cardiac arrest are particularly common in children, and resuscitation protocols for children differ from adult protocols, in part to reflect this. Although one initial response will be to treat the arrhythmia, resuscitation will not ultimately be successful until an airway is established and the blood can be reoxygenated. Equipment that needs to be immediately available will include additional laryngoscopes and blades, an oral airway, a supraglottic airway (SGA), a flexible bronchoscope and video laryngoscope, and an intubating stylet or bougie. If an airway cannot be immediately established, the anesthesia provider may wish to prepare for a surgical airway. It will be necessary to prepare an emergency percutaneous cricothyrotomy kit and/or jet ventilation or equipment for open emergency tracheostomy. Although the necessary equipment may be on the code cart or immediately available, it takes time to set up. It is critical for the anesthesia technician to anticipate what equipment may be necessary and have it ready in case it is asked for. If the patient is in the operating room and a surgeon is available or en route, the circulating nurse and surgical technologist may be involved in setup of the surgical airway equipment while the AT sets up or assists with the anesthesiologist’s emergency equipment.
- **Hypovolemia or Hemorrhage**: Severe hypovolemia can readily cause a cardiac arrest. Once the initial resuscitation steps are underway, be prepared for obtaining additional vascular access and delivering fluid resuscitation. If hemorrhage is the

cause, be prepared to send a sample to the blood bank, to obtain blood or blood products and to initiate rapid transfusion. A rapid infuser, such as Belmont or Level 1, should be brought to the room and set up immediately. Be prepared to have a pressure transducer bag for an arterial line placement. Arterial blood gas (ABG) machine should be ready and with the proper daily calibration. During massive transfusion, patients can quickly become cold; hypothermia will then interfere with blood clotting. Thus, make sure fluid warmers are in place and working appropriately right away. Have additional help available to be able to send multiple ABGs, check blood, retrieve medications, etc.

- **Cardiac Arrest Associated with Regional Anesthesia:** Many different kinds of regional anesthesia can result in an unintended high or total spinal block if too much local anesthetic reaches the spinal fluid. Rarely, this can occur with a planned spinal anesthetic. Any other regional procedure with needle placement near the spine (epidural, interscalene, even eye blocks, which are near the cerebrospinal fluid in the front of the brain) can result in unintended injection of the local anesthetic into the spinal fluid.

  Early signs of high or total spinal may be:

  - weakness in the upper extremities
  - shortness of breath
  - nausea ○ anxiety.

This can then progress to difficulty with ventilation, respiratory arrest, bradycardia, hypotension, loss of consciousness, or even cardiac arrest. After the initial resuscitation steps, patients will require intubation, ventilation, and cardiovascular support. Fluids and vasopressors (phenylephrine, epinephrine, vasopressin, etc.) may be needed. A promptly recognized total spinal usually responds easily to supportive care (intubation, ventilation, and vasopressor support) and does not progress to an emergency; an unrecognized one is fatal. The most serious complication after regional block or epidural anesthesia injected with local anesthetics is cardiac arrest following an unintended intravascular injection Cardiac arrest due to local anesthetic toxicity can be particularly hard to treat because it causes dysrhythmias that are less responsive to traditional ACLS maneuvers such as defibrillation and antiarrhythmic drugs. An infusion of lipid (20% lipid emulsion) has been demonstrated to reduce the amount of local anesthetic interfering with cardiac cells. Patients may then be successfully resuscitated even after prolonged performance of CPR. If a cardiac arrest is precipitated by local anesthetic toxicity, the anesthesia

technician should immediately locate the lipid infusion. Most institutions maintain this important drug in their regional anesthesia carts.

- **Anaphylaxis**: Major allergic reactions to drugs or other agents can release large amounts of histamine into the circulation and can produce cardiac arrest. Many of these patients will require intubation due to airway swelling, bronchospasm, or cardiovascular collapse.
    - **Treatment:** The mainstay of treatment for anaphylaxis is epinephrine. Epinephrine is the most potent cardiac stimulant, a bronchodilator that can counteract bronchoconstriction and a vasopressor that can counteract the severe vasodilation that occurs with anaphylaxis. Other treatments include antihistamines, fluid administration, steroids, and bronchodilators. Anesthesia technicians encountering anaphylaxis should make epinephrine immediately available, as well as the other treatments.

Airway equipment, including advanced airway equipment for patients with difficult airways from swelling, may be required. The AT should be prepared to send relevant labs (such as serum tryptase and histamine) and be prepared to transport these patients to the intensive care unit with monitoring, infusions, and ongoing large-volume fluid resuscitation.

Section II

ADVANCED EQUIPMENT AND TECHNOLOGY

Chapter 6

INVASIVE MONITORS

6.1 INTRODUCTION

Invasive hemodynamic monitoring is the collection and analysis of quantitative and qualitative data of cardiopulmonary function. It is crucial to anesthetic management during major surgery with large fluid shifts or that of critically ill patients. Fluid-filled monitoring systems attached to intravascular catheters are used for continuous measurement of arterial and intracardiac pressures, as well as for obtaining intermittent or continuous cardiac output measurement.

6.2 INVASIVE ARTERIAL BLOOD PRESSURE MONITORING COMPONENTS

1. An indwelling Teflon arterial cannula (20 or 22 G) is used. The cannula has parallel walls to minimize the effect on blood flow to the distal parts of the limb. Cannulation can be achieved by directly threading the cannula (either by direct insertion method or a transfixation technique) or by using a modified Seldinger technique with a guidewire to assist in the insertion as in some designs.
2. A column of bubble-free heparinized or plain 0.9% normal saline at a pressure of 300 mmHg, incorporating a flushing device.
3. Via the fluid column, the cannula is connected to a transducer. This in turn is connected to an amplifier and oscilloscope. A strain gauge variable resistor transducer is used.
4. The diaphragm (a very thin membrane) acts as an interface between the transducer and the fluid column.

Fig. BD Flow switch arterial cannula. Note the on–off switch valve.

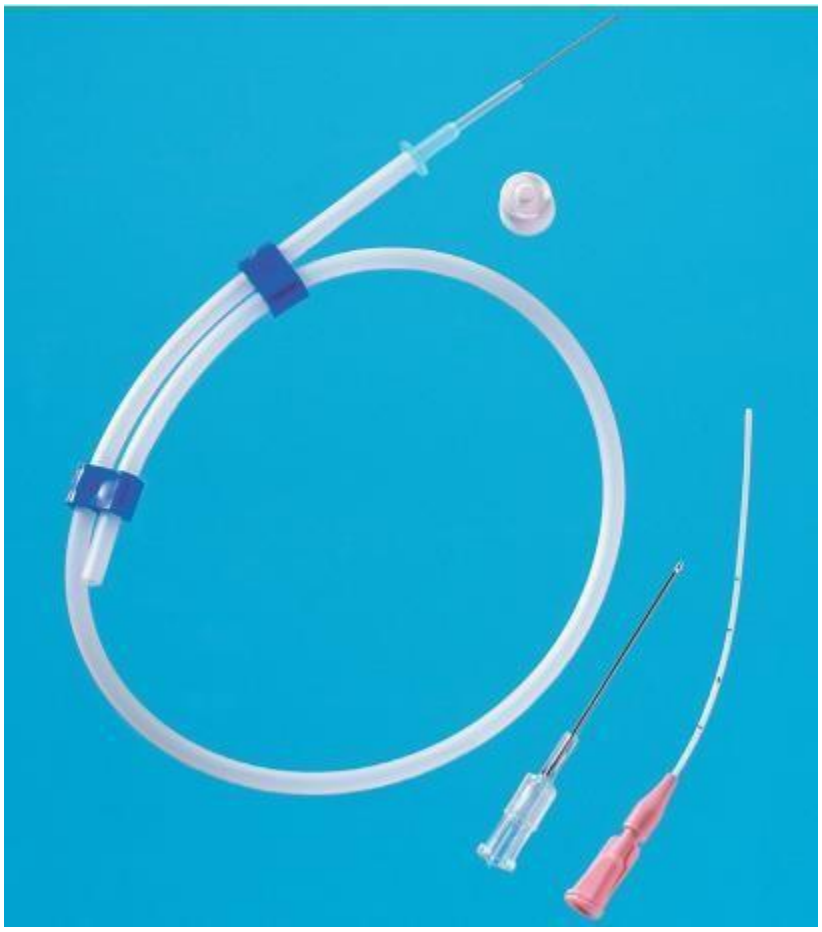


Fig. Argon Careflow arterial cannula with its guidewire.

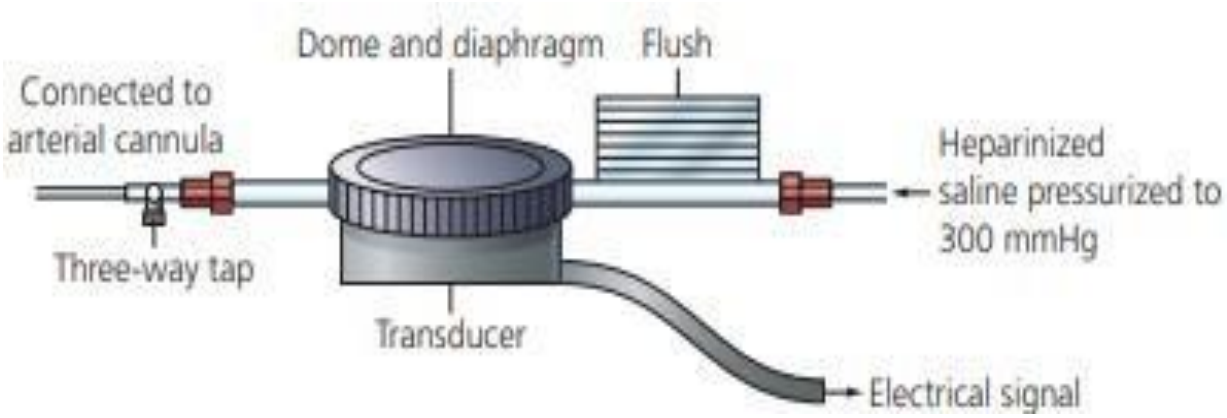


Fig. Components of a pressure measuring system.

5. The pressure transducer is a device that changes either electrical resistance or capacitance in response to changes in pressure on a solid-state device. The moving part of the transducer is very small and has little mass.

Fig. Smith's Medex reusable pressure transducer.

6.2.1 Problems in practice and safety features

- The arterial pressure waveform should be displayed in order to detect damping or resonance. The monitoring system should be able to apply an optimal damping value of 0.64.

    a. **Damping** is caused by dissipation (wasting) of stored energy. Anything that takes energy out of the system results in a progressive diminution of amplitude of oscillations. Increased damping lowers the systolic and elevates the diastolic pressures with loss of detail in the waveform. Damping can be caused by an air bubble (air is more compressible in comparison to the saline column), clot or a highly compliant, soft transducer diaphragm and tube.

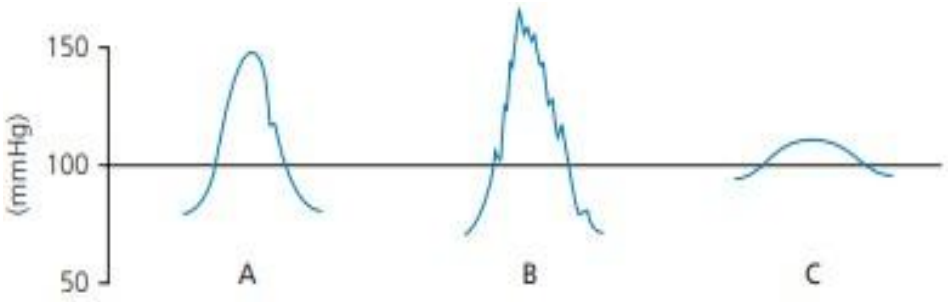


Fig. Arterial pressure waveform. (A) Correct, optimally damped waveform. (B) Underdamped waveform. (C) Overdamped waveform.

b. **Resonance** occurs when the frequency of the driving force coincides with the resonant frequency of the system. If the natural frequency is less than 40 Hz, it falls within the range of the blood pressure and a sine wave will be superimposed on the blood pressure wave. Increased resonance elevates the systolic and lowers the diastolic pressures. The mean pressure should stay unchanged. Resonance can be due to a stiff, non-compliant diaphragm and tube. It is worse with tachycardia.

- To determine the optimum damping of the system, a square wave test (fast flush test) is used. The system is flushed by applying a pressure of 300 mmHg (compress and release the flush button or pull the lever located near the transducer). This results in a square waveform, followed by oscillations:

  a. in an optimally damped system, there will be two or three oscillations before settling to zero

  b. an overdamped system settles to zero without any oscillations.

  c. an underdamped system oscillates for more than three to four cycles before settling to zero.

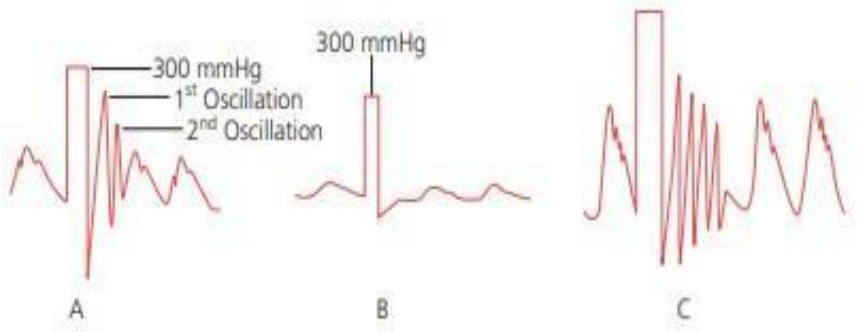


Fig. The square wave test (fast flush test). (A) Optimally damped system. (B) Overdamped system. (C) Underdamped system.

- The transducer should be positioned at the level of the right atrium as a reference point that is at the level of the midaxillary line. Raising or lowering the transducer above or below the level of the right atrium gives error readings equivalent to 7.5 mmHg for each 10 cm.
- Ischaemia distal to the cannula is rare but should be monitored for. Multiple attempts at insertion and haematoma formation increase the risk of ischaemia.
- Arterial thrombosis occurs in 20–25% of cases with very rare adverse effects such as ischaemia or necrosis of the hand. Cannulae in place for less than 24 h very rarely cause thrombosis.
- There is risk of bleeding due to disconnection.
- Inadvertent drug injection causes distal vascular occlusion and gangrene. An arterial cannula should be clearly labelled.
- Local infection is thought to be less than 20%. Systemic infection is thought to be less than 5%. This is more common in patients with an arterial cannula for more than 4 days with a traumatic insertion.
- Arterial cannulae should not be inserted in sites with evidence of infection and trauma or through a vascular prosthesis.

- Periodic checks, calibrations and re-zeroing are carried out to prevent baseline drift of the transducer electrical circuits. Zero calibration eliminates the effect of atmospheric pressure on the measured pressure. This ensures that the monitor indicates zero pressure in the absence of applied pressure, so eliminating the offset drift (zero drift). To eliminate the gradient drift, calibration at a higher pressure is necessary. The transducer is connected to an aneroid manometer using a sterile tubing, through a three-way stopcock and the manometer pressure is raised to 100 and 200 mmHg. The monitor display should read the same pressure as is applied to the transducer.

6.3 CENTRAL VENOUS CATHETERIZATION AND PRESSURE (CVP)

The CVP is the filling pressure of the right atrium. It can be measured directly using a central venous catheter. The catheter can also be used to administer fluids, blood, drugs, parenteral nutrition and sample blood. Specialized catheters can be used for haemofiltration, haemodialysis and transvenous pacemaker placement.

The tip of the catheter is usually positioned in the superior vena cava at the entrance to the right atrium. The internal jugular, subclavian and basilic veins are possible routes for central venous catheterization. The subclavian route is associated with the highest rate of complications but is convenient for the patient and for the nursing care.

The Seldinger technique is the common and standard method used for central venous catheterization regardless of catheter type. The procedure should be done under sterile conditions

6.3.1 Components

Pressure Transducer

- A similar measuring system to that used for invasive arterial pressure monitoring (catheter, heparinized saline column, transducer, diaphragm, flushing device and oscilloscope system). The transducer is positioned at the level of the right atrium.
- A measuring system of limited frequency range is adequate because of the shape of the waveform and the values of the central venous pressure. **Fluid Manometer**
- A giving set with either normal saline or 5% dextrose is connected to the vertical manometer via a three-way tap. The latter is also connected to the central venous catheter.

- The manometer has a spirit level side arm positioned at the level of the right atrium (zero reference point). The upper end of the column is open to air via a filter. This filter must stay dry to maintain direct connection with the atmosphere.
- The vertical manometer is filled to about the 20-cm mark. By opening the three way tap to the patient, a swing of the column should be seen with respiration. The CVP is read in cm H_2O when the fluid level stabilizes.
- The manometer uses a balance of forces: downward pressure of the fluid (determined by density and height) against pressure of the central venous system (caused by hydrostatic and recoil forces)

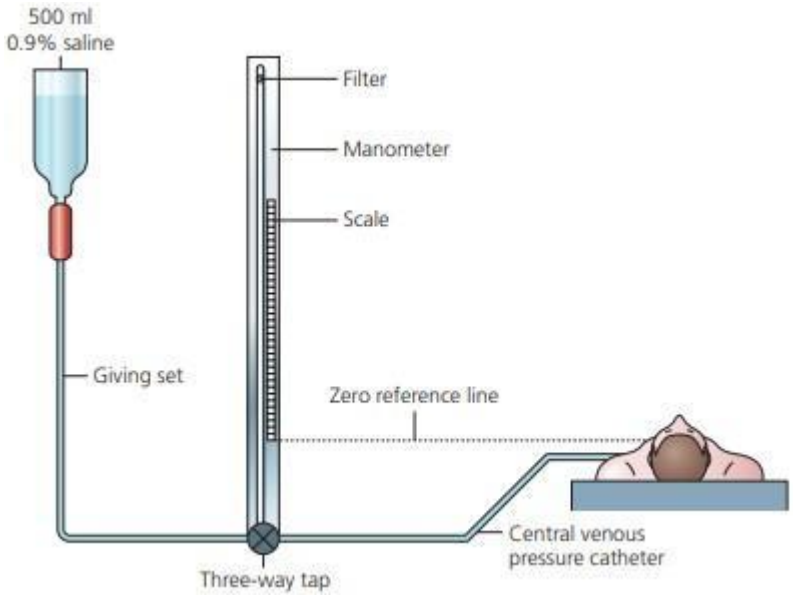


Fig. Measurement of CVP using a manometer. The manometer's fluid level falls until the height of the fluid column above the zero reference point is equal to the CVP.

6.4 CATHETERS

There are different types of catheters used for central venous cannulation and CVP measurement. They differ in their lumen size, length, number of lumens, the presence or absence of a subcutaneous cuff and the material they are made of. The vast majority of catheters are designed to be inserted using the Seldinger technique although some are designed as 'long' intravenous cannulae (cannula over a needle).

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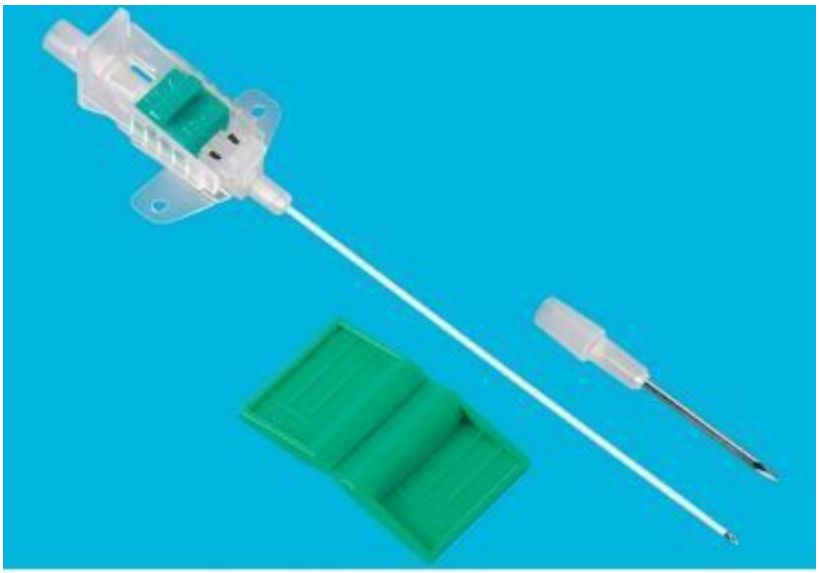

Fig. Argon cannula over a needle central line.

Antimicrobial-coated catheters have been designed to reduce the incidence of catheter related bloodstream infection. These can be either antiseptic coated (e.g. chlorhexidine/silver sulfadiazine, benzalkonium chloride, platinum/ silver) or antibiotic coated (e.g. minocycline/rifampin) on either the internal or external surface or both. The antibiotic-coated central lines are thought to be more effective in reducing the incidence of infection.

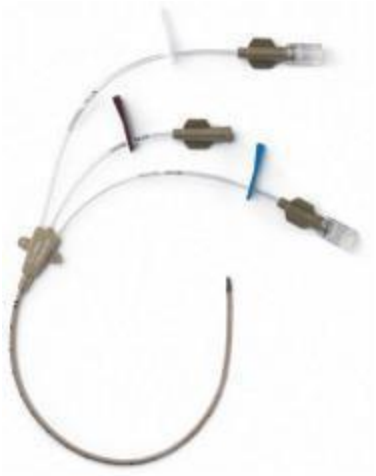

Fig. Smith's Medex silver impregnated triple lumen central venous catheter. Both the inside and outside surfaces are impregnated with silver.

Multilumen catheter

- The catheter has two or more lumens of different sizes, e.g. 16 G and 18 G.

Paediatric sizes also exist.

The different lumens should be flushed with heparinized saline before insertion.

- Single and double lumen versions exist.
- Simultaneous administration of drugs and CVP monitoring is possible. It does not allow the insertion of a pulmonary artery catheter.
- These catheters are made of polyurethane. This provides good tensile strength, allowing larger lumens for smaller internal diameter.

Fig. An adult triple lumen catheter

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6.4.1 LONG CENTRAL CATHETERS/ PERIPHERALLY INSERTED CENTRAL CATHETERS (PICC)

- These catheters, 60 cm in length, are designed to be inserted through an introducing cannula via an antecubital fossa vein, usually the basilic vein.
- They are used when a central catheter is required in situations when it is undesirable to gain access via the internal jugular or the subclavian veins, for example during head and neck surgery or prolonged antibiotic therapy. They are made of soft flexible polyurethane or silicone.

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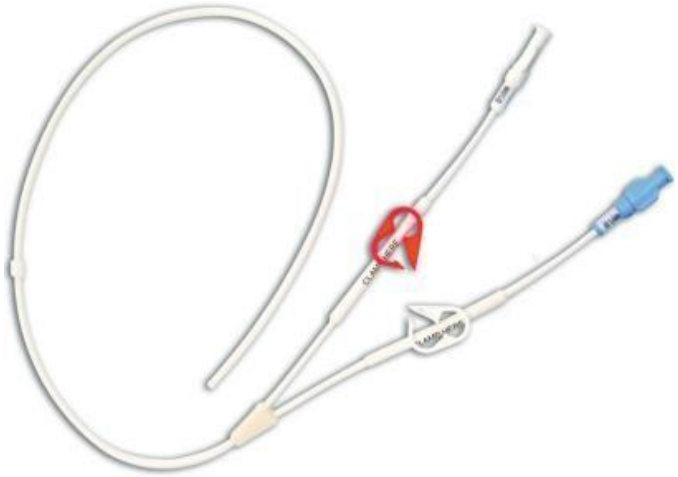

Fig. A double lumen long-term Hickman catheter. Note the Dacron cuff.

6.4.2 Dialysis catheters

These are large-calibre catheters designed to allow high flow rates of at least 300 mL/min. They are made of silicone or polyurethane. Most of them are dual lumen with staggered end and side holes to prevent admixture of blood at the inflow and outflow portions reducing recirculation.

Problems in practice and safety features

- Inaccurate readings can be due to catheter blockage, catheter inserted too far or using the wrong zero level.
- Pneumohaemothorax (with an incidence of 2–10% with subclavian vein catheterization and 1–2% with internal jugular catheterization), trauma to the arteries (carotid, subclavian and brachial), air embolism, haematoma and tracheal puncture are complications of insertion.
- Sepsis and infection are common complications with an incidence of 2.8–20%. Staphylococcus aureus and Enterococcus are the most common organisms.
- A false passage may be created if the guidewire or dilator are advanced against resistance. The insertion should be smooth.
- There may be cardiace complications such as self-limiting arrhythmias due to the irritation caused by the guidewire or catheter. Gradual withdrawal of the device is usually adequate to restore normal rhythm. More serious but unusual complication such as venous or cardiac perforation can be lethal.

Catheter-related venous thrombosis is thought to be up to 40% depending on the site, the duration of placement, the technique and the condition of the patient.

- Micro shock. A central venous catheter presents a direct pathway to the heart muscle. Faulty electrical equipment can produce minute electrical currents (less than 1 ampere) which can travel via this route to the myocardium. This can produce ventricular fibrillation (VF) if the tip of the catheter is in direct contact with the myocardium. This very small current does not cause any adverse effects if applied to the body surface, but if passed directly to the heart, the current density will be high enough to cause VF, hence the name micro shock.

6.5 TEMPERATURE MONITORS

Monitoring a patient's temperature during surgery is a common and routine procedure. Different types of thermometers are available.

6.5.1 THERMISTOR

Components

1. A small bead of a temperature-dependent semiconductor.
2. Wheatstone bridge circuit.

Mechanism of action

- The thermistor has electrical resistance which changes non-linearly with temperature. The response is made linear electronically. This property allows them to accurately measure temperature to an order of 0.1°C.
- It can be made in very small sizes and is relatively cheap to manufacture.
- It is mounted in a plastic or stainless steel probe making it mechanically robust, and it can be chemically sterilized.
- It is used in PA catheters to measure cardiac output.
- In the negative thermal conductivity thermistors, such as cobalt oxide, copper oxide and manganese oxide, the electrical resistance decreases as the temperature increases. In the positive thermal conductivity thermistors, such as barium titanate, the electrical resistance increases with the temperature. Problems in practice and safety features Thermistors need to be stabilized as they age.

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6.5.2 INFRARED TYMPANIC THERMOMETER:

COMPONENTS:

1. A small probe with a disposable and transparent cover is inserted into the external auditory meatus.
2. The detector (which consists of a series of thermocouples called a thermopile).

Mechanism of action

- The detector receives infrared radiation from the tympanic membrane.
- The infrared signal detected is converted into an electrical signal that is processed to measure accurately the core temperature within 3 s.
- The rate of radiation by an object is proportional to temperature to the fourth power.

Problems in practice and safety features

1. Non-continuous intermittent readings.
2. The probe has to be accurately aimed at the tympanic membrane. False low readings from the sides of the ear canal can be a problem.
3. Wax in the ear can affect the accuracy.

6.5.3 THERMOCOUPLES

These are devices that make use of the principle that two different metals in contact generate a voltage, which is temperature dependent.

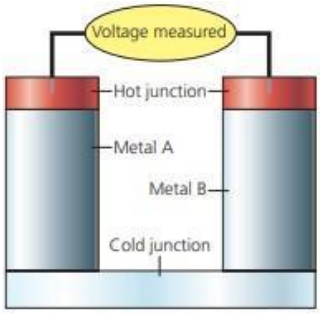


Fig. Thermocouple.

Components

1. Two strips of dissimilar metals (0.4–2-mm diameter) of different specific heats and in contact from both ends. Usually copper-constantan (copper with 40% nickel) junctions are used.

2. A galvanometer.

Mechanism of action

- One junction is used as the measuring junction whereas the other one is the reference. The latter is kept at a constant temperature.
- The metals expand and contract to different degrees with change in temperature producing an electrical potential that is compared to a reference junction. The current produced is directly proportional to the temperature difference between the two junctions, i.e. there is a linear relationship between voltage and temperature.
- The voltage produced is called the Seebeck effect or thermoelectric effect.
- The measuring junction produces a potential of 40 µV per °C. This potential is measured by an amplifier.
- They are stable and accurate to 0.1°C.
- If multiple thermocouples are linked in series, they constitute a thermopile. This is done to improve their sensitivity

Sites for temperature probes

Body core temperature can be measured using different sites:

1. Rectal temperature does not accurately reflect the core temperature in anaesthetized patients. During an operation, changes in temperature are relatively rapid and the rectal temperature lags behind.
2. Oesophageal temperature accurately reflects the core temperature with the probe positioned in the lower oesophagus (at the level of the left atrium). Here the probe is not affected by the cooler tracheal temperature.

Fig. Oesophageal/rectal temperature probe.

3. Tympanic membrane temperature is closely associated with brain temperature. It accurately reflects core temperature, compared with lower oesophageal temperature. Thermocouple and thermistor probes as well as the infrared probe can be used.

Fig. Tympanic membrane temperature probe.

Fig. Tympanic membrane thermometer.

4. Bladder temperature correlates well with the core temperature when there is a normal urine output.

Fig. Smith's Medical bladder catheter with a temperature probe.

5. Skin temperature, when measured with the core temperature, can be useful in determining the volemic status of the patient.

Fig. Skin temperature probe.

· The axilla is the best location for monitoring muscle temperature, making it most suitable for detecting malignant hyperthermia.

Chapter 7

PAIN CONTROL TECHNIQUES AND REGIONAL ANAESTHESIA

7.1 Patient controlled analgesia (PCA)

PCA represents one of the most significant advances in the treatment of postoperative pain. Improved technology enables pumps to accurately deliver boluses of opioid when a demand button is activated by the patient.

It is the patient who determines the plasma concentration of the opioid, this being a balance between the dose required to control the pain and that which causes side effects. The plasma concentration of the opioid is maintained at a relatively constant level with the dose requirements being generally smaller.

Components

1. A pump with an accuracy of at least ±5% of the programmed dose.
2. The remote demand button connected to the pump and activated by the patient.
3. An anti-siphon and backflow valve.

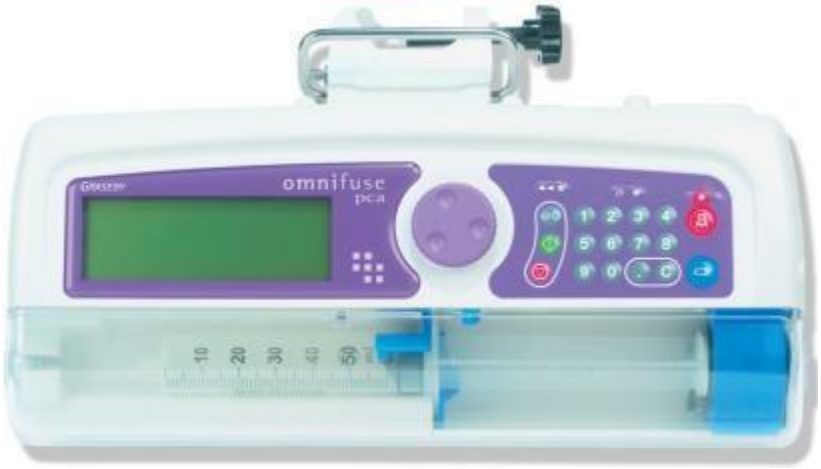


Fig. The Graseby Omnifuse PCA pump.

7.2 Modes of administration

Different modes of analgesic administration can be employed:

- patient controlled on-demand bolus administration (PCA)
- continuous background infusion and patient controlled bolus administration.

7.2.1 Variable settings

The initial programming of the pump must be tailored for the individual patient.

Different variable settings on a PCA device are:

- The mode of administration
- the amount of analgesic administered per bolus
- the ‘lock-out’ time (i.e. the time period during which the patient is prevented from receiving another bolus despite activating the demand button)
- the duration of the administration of the bolus and the maximum amount of analgesic permitted per unit time

7.2.2 Important features

- Some designs have the capability to be used as a PCA pump for a particular variable duration then switching automatically to a continuous infusion as programmed.
- The history of the drug administration including the total dose of the analgesic, the number of boluses and the number of successful and failed attempts can be displayed.
- The devices have memory capabilities so they retain their programming during syringe changing.
- Tamper-resistant features are included.
- Some designs have a safety measure where an accidental triggering of the device is usually prevented by the need for the patient to make two successive presses on the hand control within 1 second.
- PCA devices operate on mains or battery.
- Different routes of administration can be used for PCA, e.g. intravenous, intramuscular, subcutaneous or epidural routes.
- Alarms are included for malfunction, occlusion and disconnection.
- Ambulatory PCA pumps are available allowing patient’s mobilization during use.

Fig. The CADD Legacy portable PCA.

7.2.3 Problems in practice and safety features

1. The ability of the patient to co-operate and understand is essential.
2. Availability of trained staff to program the device and monitor the patient is vital.
3. In the PCA mode, the patient may awaken in severe pain because no boluses were administered during sleep.
4. Some PCA devices require special giving sets and syringes.
5. Technical errors can be fatal.

7.3 SYRINGE PUMPS

These are programmable pumps that can be adjusted to give variable rates of infusion and also bolus administration. They are used to maintain continuous infusions of analgesics (or other drugs). The type of flow is pulsatile continuous delivery and their accuracy is within ±2–5%. Some designs can accept a variety of different size syringes. The power source can be battery and/or mains.

7.3.1 Prevention and precautions to use

It is important to prevent free flow from the syringe pump. Anti-siphon valves are usually used to achieve this. Inadvertent free flow can occur if the syringe barrel or plunger is not engaged firmly in the pump mechanism. The syringe should be securely clamped to the pump. Syringe drivers should not be positioned above the level of the patient. If the pump is more than 100 cm above the patient, a gravitational pressure can be generated that overcomes the friction between a non-secured plunger and barrel. Siphoning can also occur if there is a crack in the syringe allowing air to enter.

Some pumps have a ‘back-off’ function that prevents the pump from administering a bolus following an obstruction due to increased pressure in the system. An anti-reflux valve should be inserted in any other line that is connected to the infusion line. Anti-reflux valves prevent backflow up the secondary and often lower pressure line should a distal occlusion occur and they avoid a subsequent inadvertent bolus

Fig. The Graseby 2000 syringe pump.

7.4 EPIDURAL NEEDLES

Epidural needles are used to identify and cannulate the epidural space. The Tuohy needle is widely used in the UK.

Fig. 18-G Tuohy needle. Note the 1 cm markings along its shaft.

7.4.1 Components

1. The needle is 10 cm in length with a shaft of 8 cm (with 1-cm markings). A 15cm version exists for obese patients.
2. The needle wall is thin in order to allow a catheter to be inserted through it.
3. The needle is provided with a stylet introducer to prevent occlusion of the lumen by a core of tissue as the needle is inserted.
4. The bevel (called a Huber point) is designed to be slightly oblique at 20° to the shaft, with a rather blunt leading edge.
5. Some designs allow the wings at the hub to be added or removed.
6. The commonly used gauges are either 16 G or 18 G.

7.4.2 How it works?

- The markings on the needle enable the anaesthetist to determine the distance between the skin and the epidural space. Hence the length of the catheter left inside the epidural space can be estimated.
- The shape and design of the bevel enable the anaesthetist to direct the catheter within the epidural space (either in a cephalic or caudal direction).

Fig. Detail of a spinal needle introduced through a Tuohy needle (top); an epidural catheter passing through a Tuohy needle (bottom).

- The bluntness of the bevel also minimizes the risk of accidental dural puncture.
- Some anaesthetists prefer winged epidural needles for better control and handling of the needle during insertion.
- A paediatric 19-G, 5-cm long Tuohy needle (with 0.5-cm markings), allowing the passage of a 21-G nylon catheter, is available.

7.5 Combined Spinal–Epidural Technique

A combined spinal–epidural technique is possible using a 26-G spinal needle of about 12 cm length with a standard 16-G Tuohy needle. The Tuohy needle is first

positioned in the epidural space then the spinal needle is introduced through it into the subarachnoid space. A relatively high pressure is required to inject through the spinal needle because of its small bore. This might lead to accidental displacement of the tip of the needle from the subarachnoid space leading to a failed or partial block. To prevent this happening, in some designs, the spinal needle is ‘anchored’ to the epidural needle to prevent displacement.

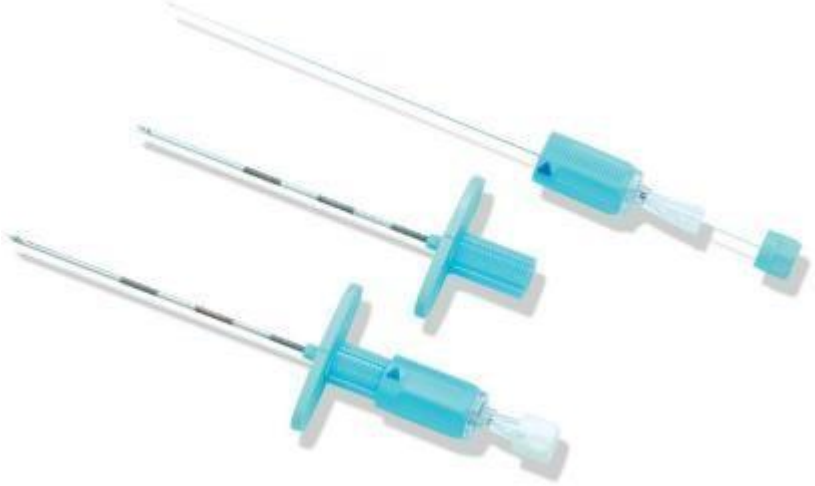


Fig. The Portex CSEcure combined spinal-epidural device. The spinal needle (top); the epidural needle (middle); the spinal needle inserted and ‘anchored’ to the epidural needle (bottom).

7.5.1 Problems in practice and safety features

- During insertion of the catheter through the needle, if it is necessary to withdraw the catheter, the needle must be withdrawn simultaneously. This is because of the risk of the catheter being transected by the oblique bevel.
- In accidental dural puncture, there is a high incidence of postdural headache due to the epidural needle’s large bore (e.g. 16 G or 18 G).
- Wrong route errors: in order to avoid administering drugs that were intended for intravenous administration, all epidural bolus doses are performed using syringes, needles and other devices with safer connectors that cannot connect with intravenous Luer connectors.

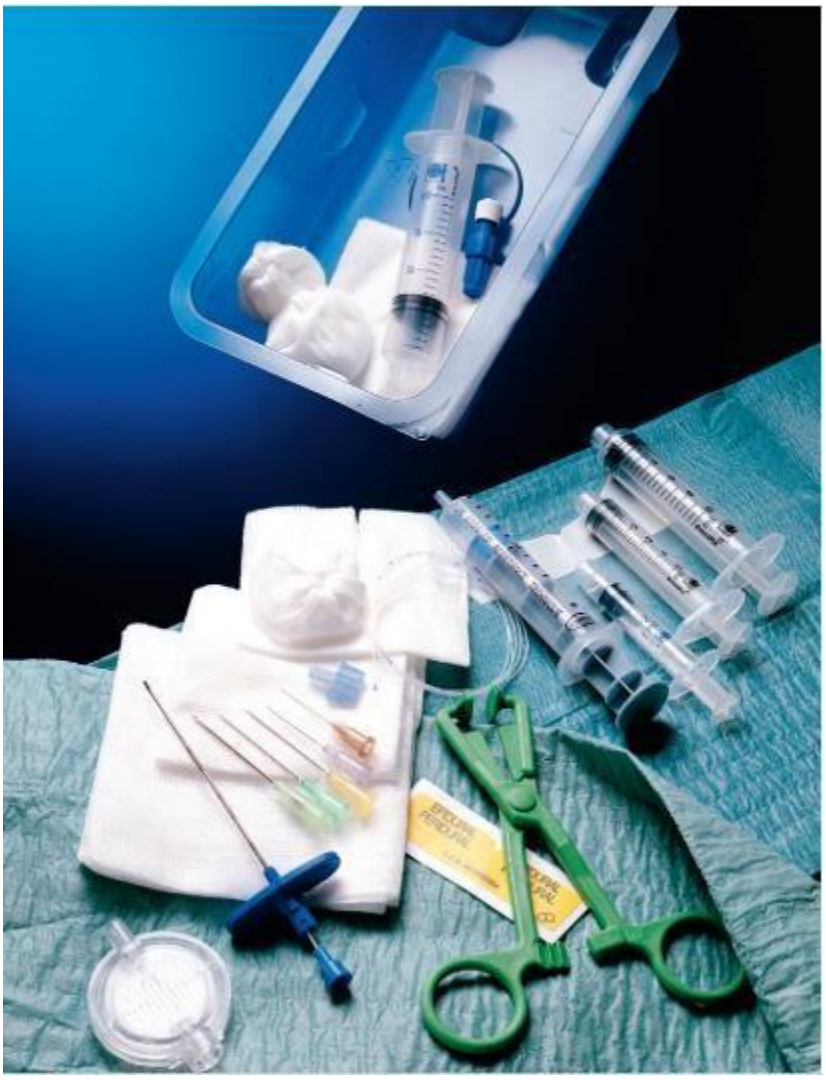


Fig. The Portex epidural set containing Tuohy needle, loss of resistance syringe and a range of other syringes and needles, epidural catheter and filter, drape, swabs and epidural catheter label.

7.5.2 The Catheter Components

1. 90-cm transparent, malleable tube made of either nylon or Teflon and biologically inert. The 16-G version has an external diameter of about 1 mm and an internal diameter of 0.55 mm.
2. The distal end has two or three side ports with a closed and rounded tip in order to reduce the risk of vascular or dural puncture (see Fig). Paediatric designs, 18 G or 19 G, have closer distal side ports.
3. Some designs have an open end.

4. The distal end of the catheter is marked clearly at 5-cm intervals, with additional 1cm markings between 5 and 15 cm.
5. The proximal end of the catheter is connected to a Luer lock and a filter (Fig. 12.10).
6. In order to prevent kinking, some designs incorporate a coil-reinforced catheter.
7. Some designs are radio-opaque. These catheters tend to be more rigid than the normal design. They can be used in patients with chronic pain to ensure correct placement of the catheter.

7.5.3 How to use?

- The catheters are designed to pass easily through their matched gauge epidural needles.
- The markings enable the anaesthetist to place the desired length of catheter within the epidural space (usually 3–5 cm).
- There are catheters with a single port at the distal tip. These offer a rather sharp point and increase the incidence of catheter-induced vascular or dural puncture.
- An epidural fixing device can be used to prevent the catheter falling out. The device clips on the catheter. It has an adhesive flange that secures it to the skin. The device does not occlude the catheter and does not increase the resistance to injection.

Fig. Smith's Portex LockIt Plus epidural catheter fixing device.

7.5.4 Problems in practice and safety features

- The patency of the catheter should be tested prior to insertion.
- The catheter can puncture an epidural vessel or the dura at the time of insertion or even days later.

- The catheter should not be withdrawn through the Tuohy needle once it has been threaded beyond the bevel as that can transect the catheter. Both needle and catheter should be removed in unison.
- It is almost impossible to predict in which direction the epidural catheter is heading when it is advanced.
- Once the catheter has been removed from the patient, it should be inspected for any signs of breakage. The side ports are points of catheter weakness where it is possible for the catheter to break. Usually, if a portion of the catheter were to remain in the patient after removal, conservative management would be recommended.
- Advancing the catheter too much can cause knotting.

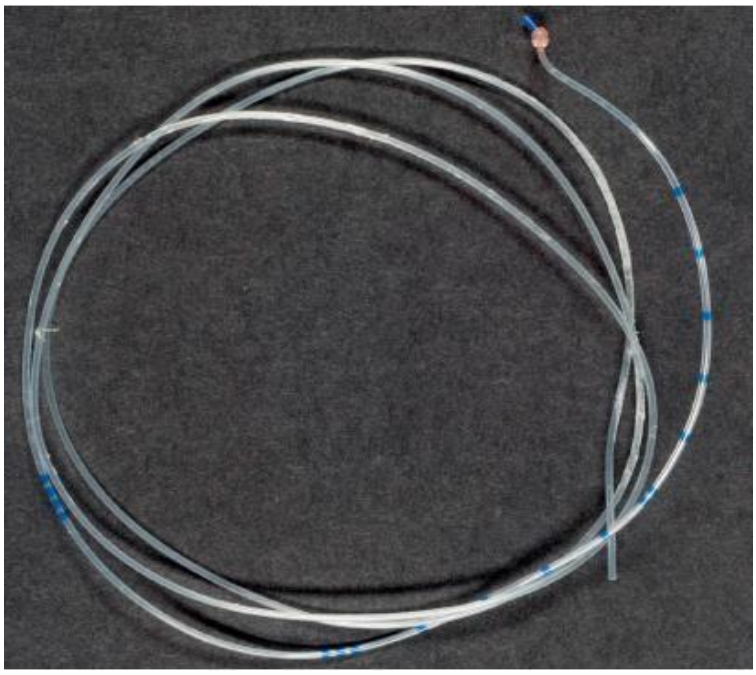


Fig. An epidural catheter with a knot near its tip.

7.5.5 The Filter

The hydrophilic filter is a 0.22- micron mesh which acts as a bacterial, viral and foreign body (e.g. glass) filter with a priming volume of about 0.7 mL. It is recommended that the filter should be changed every 24 h if the catheter is going to stay in situ for long periods.

Fig. Portex epidural catheter and filter. Note the markings up to 20 cm.

7.5.6 Loss of Resistance Device or Syringe

The syringe has a special low-resistance plunger used to identify the epidural space by loss of resistance to either air or saline. Plastic and glass versions are available.

7.6 SPINAL NEEDLES

These needles are used to inject local anaesthetic(s) and/or opiates into the subarachnoid space. In addition, they are used to sample cerebrospinal fluid (CSF) or for intrathecal injections of antibiotics and cytotoxics.

7.6.1 Components

1. The needle's length varies from 5 to 15 cm; the 10-cm version is most commonly used. They have a transparent hub in order to identify quickly the flow of CSF.
2. A stylet is used to prevent a core of tissue occluding the lumen of the needle during insertion. It also acts to strengthen the shaft. The stylet is withdrawn once the tip of the needle is (or is suspected to be) in the subarachnoid space.

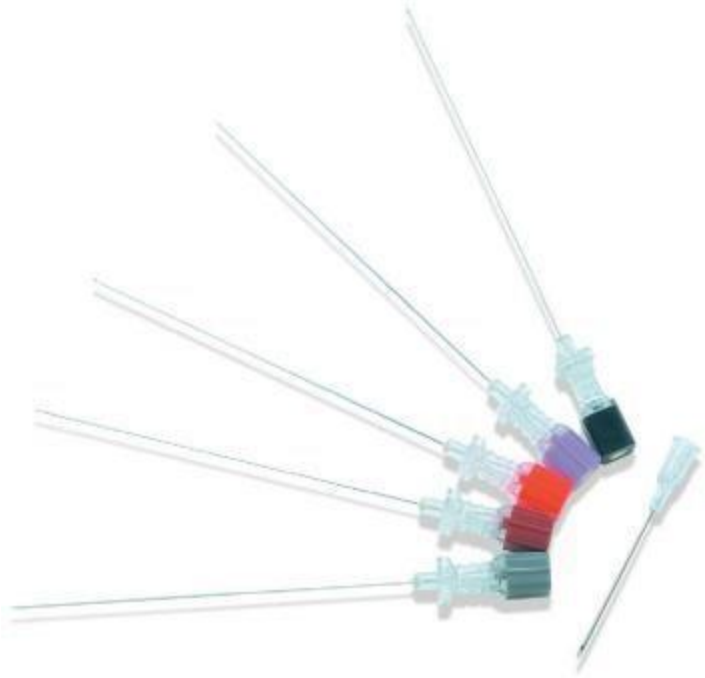


Fig. Different size spinal needles with their introducer. From left; 27 G (grey), 26 G (brown), 25 G (orange), 24 G (purple) and 22 G (black).

3. Spinal needles are made in different sizes, from 18 G to 29 G in diameter. 32-G spinal needles have been described but are not widely used.
4. The 25-G and smaller needles are used with an introducer which is usually an 18G or 19-G needle.
5. There are two designs for the bevel. The cutting, traumatic bevel is seen in the Yale and Quincke needles. The non-cutting, atraumatic pencil point, with a side hole just proximal to the tip, is seen in the Whitacre and Sprotte needles.

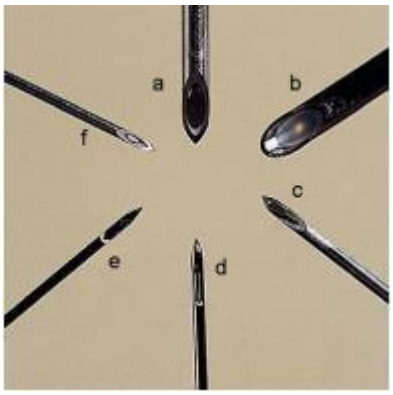


Fig. Bevel design of (A) 18-G Quincke; (B) 16-G Tuohy; (C) 22-G Yale; (D) 24-G Sprotte; (E) 25-G Whitacre; (F) 25-G Yale.

Fig. Pencil-shaped Whitacre bevel (left) and cutting Quincke bevel (right).

6. A 28-G nylon, open-ended microcatheter can be inserted through a Crawford spinal needle (23 G). A stylet inside the catheter is removed during the insertion. This allows top-ups to be administered. The priming volume is 0.03 mL with a length of 910 mm. A 0.2-micron filter is attached to the catheter.

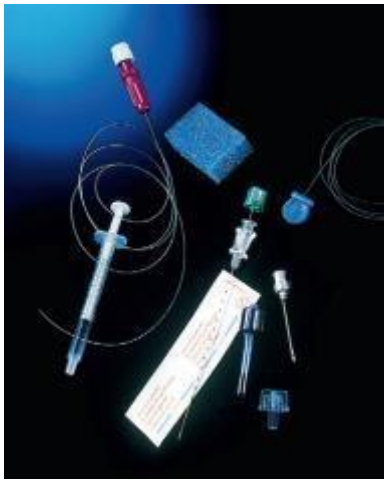


Fig The Portex spinal microcatheter set.

7.6.2 Mechanism of action

- The large 22-G needle is more rigid and easier to direct. It gives a better feedback feel as it passes through the different tissue layers.
- The CSF is slower to emerge from the smaller sized needles. Aspirating gently with a syringe can speed up the tracking back of CSF.
- Continuous spinal anaesthesia can be achieved by inserting 3–4 cm of the 28-G spinal microcatheter into the subarachnoid space.

7.6.3 Problems in practice and safety features

Wrong route errors:

- in order to avoid administering drugs that were intended for intravenous administration, all spinal (intrathecal) bolus doses and lumbar puncture samples are performed using syringes, needles and other devices with safer connectors that cannot connect with intravenous Luer connectors.

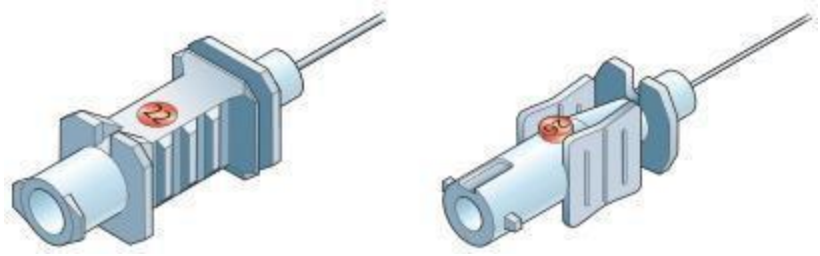


Fig. The standard Luer connection spinal needle (left) and the Portex Correct Inject spinal needle designed to avoid wrong route error injections.

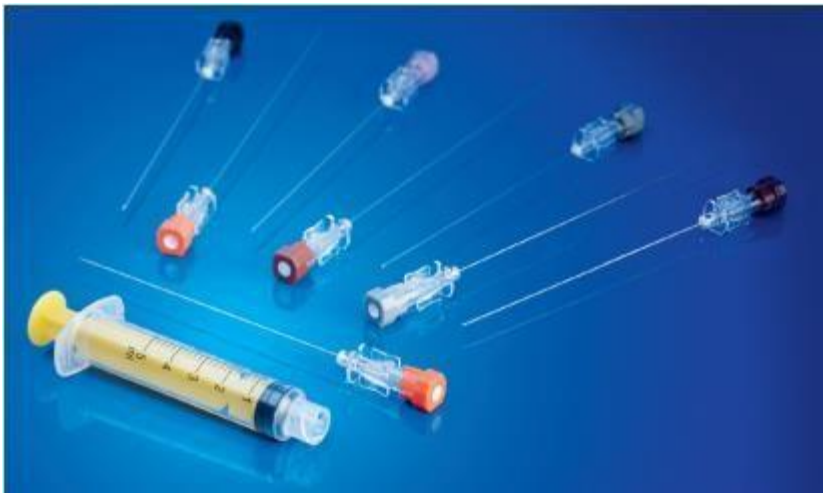


Fig. Portex Correct Inject range of spinal needles and syringe designed to avoid wrong route error injections.

Dural headache

- The incidence of dural headache is directly proportional to the gauge of the needle and the number of punctures made through the dura and indirectly proportional to the age of the patient. There is a 30% incidence of dural headache using a 20-G spinal needle, whereas the incidence is reduced to about 1% when a 26-G needle is used. For this reason, smaller gauge spinal needles are preferred.
- The Whitacre and Sprotte atraumatic needles separate rather than cut the longitudinal fibres of the dura. The defect in the dura has a higher chance of sealing after the removal of the needles. This reduces the incidence of dural headache.
- Traumatic bevel needles cut the dural fibres, producing a ragged tear which allows leakage of CSF. Dural headache is thought to be caused by the leakage of CSF
- The risk of dural headache is higher during pregnancy and labour, day-surgery patients and those who have experienced a dural headache in the past.

Spinal microcatheters

- They are difficult to advance.
- There is a risk of trauma to nerves.
- Cauda equina syndrome is thought to be due to the potential neurotoxicity from the anaesthetic solutions rather than the microcatheter.

7.7 NERVE BLOCK NEEDLES

These needles are used in regional anaesthesia to identify a nerve plexus or peripheral nerve.

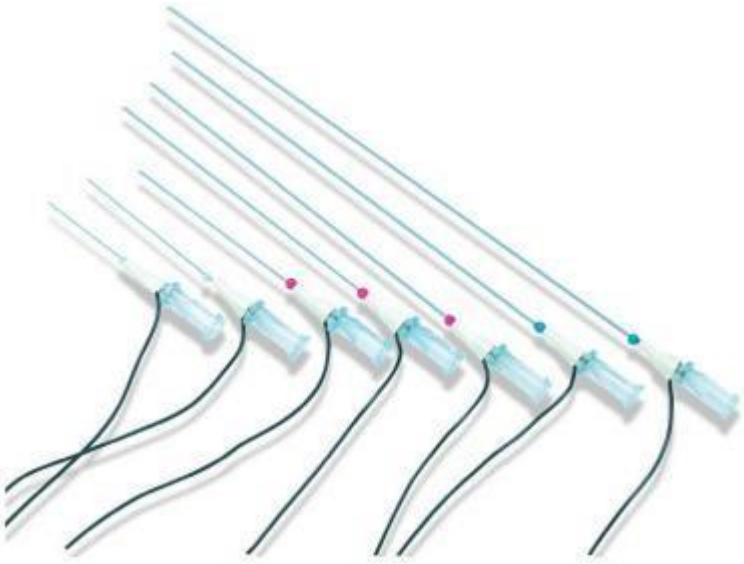


Fig. A range of Smiths Medical insulated peripheral nerve block needles of different lengths.

7.7.1 Components

1. They are made of steel with a Luer-lock attachment.
2. They have short, rather blunt bevels in order to cause minimal trauma to the nervous tissue. The bluntness makes skin insertion more difficult. This can be overcome by a small incision.
3. The needles have transparent hubs which allow earlier recognition of intravascular placement while performing blocks.
4. A side port for injecting the local anaesthetic solution is found in some designs.
5. The needles are connected to a nerve stimulator to aid in localizing the nerve using an insulated cable to prevent leakage of current.
6. 22-G size needles are optimal for the vast majority of blocks. There are different lengths depending on the depth of the nerve or plexus. Some suggested length needles for common blocks are: a) interscalene block: 25–50 mm b) axillary block: 35–50 mm c) psoas compartment block: 80–120 mm d) femoral nerve block: 50 mm e) sciatic nerve block (depending on the approach): 80–150 mm.
7. A pencil-shaped needle tip with a distal side hole for injecting local anaesthetic drugs is available.

Nerve block needles can either be insulated with an exposed tip or non-insulated.

7.8 ULTRASOUND GUIDANCE IN REGIONAL ANAESTHESIA

This more recent technique uses ultrasound control to locate the nerves/plexuses. It is thought that a higher success rate can be achieved when it is used, with lower complication rates.

More specially designed needles for the use of ultrasound are available allowing a better reflection of the ultrasound waves. The needles have echogenic laser markings to facilitate better needle visualization with minimal acoustic shadowing.

Fig. B Braun Contiplex S Ultra designed for improved echogenicity.

Most nerve blocks need ultrasound frequencies in the range of 10–14 MHz. Many broadband ultrasound transducers with a bandwidth of 5–12 or 8–14 MHz can offer excellent resolution of superficial structures in the upper frequency range and good penetration depth in the lower frequency range.

The true echogenicity of a nerve is only captured if the sound beam is oriented perpendicularly to the nerve axis. This can be achieved best with linear array transducers

with parallel sound beam emission rather than with sector transducers. The latter are characterized by diverging sound waves, such that the echotexture of the nerves will only be displayed in the centre of the image.

The linear probes are most often used for the majority of peripheral blocks. The curved arrays are used for deep nerve structures (lower frequency is required). Smaller footprint probes are useful for smaller infants and children and for certain uses such as very superficial blocks (e.g. ankle blocks).

Chapter 8

ADDITIONAL EQUIPMENT USED IN ANESTHESIA

8.1 SUCTION APPARATUS

Suction apparatus is vital during anaesthesia and resuscitation to clear the airway of any mucus, blood or debris. It is also used during surgery to clear the operating field of either blood or fluid.

8.1.1 Components

Suction apparatus consists of

- A source of vacuum
- a suction unit
- a suction tubing

The source of vacuum can be either piped vacuum or electrically or manually operated units. Piped vacuum is the most commonly used source in many operating theatres.

The suction unit consists of a reservoir jar, bacterial filter, vacuum control regulator and a vacuum gauge. The reservoir jar is graduated so that the volume of aspirate may be estimated. It contains a cut-off valve. The cut-off valve has a float that rises as the fluid level increases and shuts off the valve when the reservoir jar is full. This prevents liquid from the suction jar entering the suction system. There is a bacterial filter between the cut-off valve and the suction control unit to prevent air that has been contaminated during passage through the apparatus infecting the atmosphere when it is blown out. The filter also traps any particulate or nebulized matter. Filters should be changed at regular intervals.

The vacuum regulator adjusts the degree of vacuum. The vacuum is indicated on the pressure gauge. This is normally marked in mmHg or kPa. The needle on the gauge goes in an anticlockwise direction as the vacuum increases. Suction units can achieve flows of

FIG. Suction apparatus.

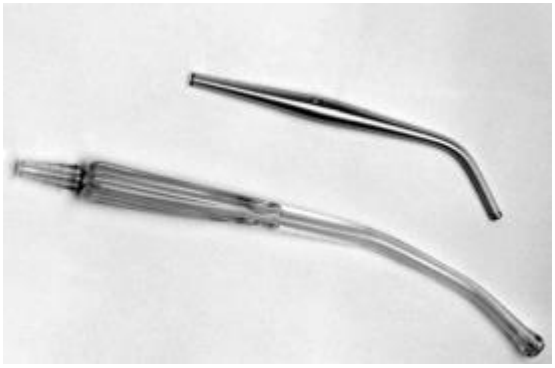


FIG. Yankauer suction catheters. Paediatric (above) and adult apparatus.

greater than 25Lmin–1 and a vacuum of greater than 67 kPa. However, flows and vacuum as high as these are seldom necessary and can cause harm if used inappropriately, particularly in children. The suction reservoir jar is connected to the patient via a suction tubing and either a Yankauer hand piece or suction catheters.

8.2 CONTINUOUS POSITIVE AIRWAY PRESSURE (CPAP)

CPAP is a spontaneous breathing mode used in the intensive care unit, during anaesthesia and for patients requiring respiratory support at home. It increases the functional residual capacity (FRC) and improves oxygenation. CPAP prevents alveolar collapse and possibly recruits already collapsed alveoli. **Components**

1. A flow generator producing high flows of gas, or a large reservoir bag may be needed.

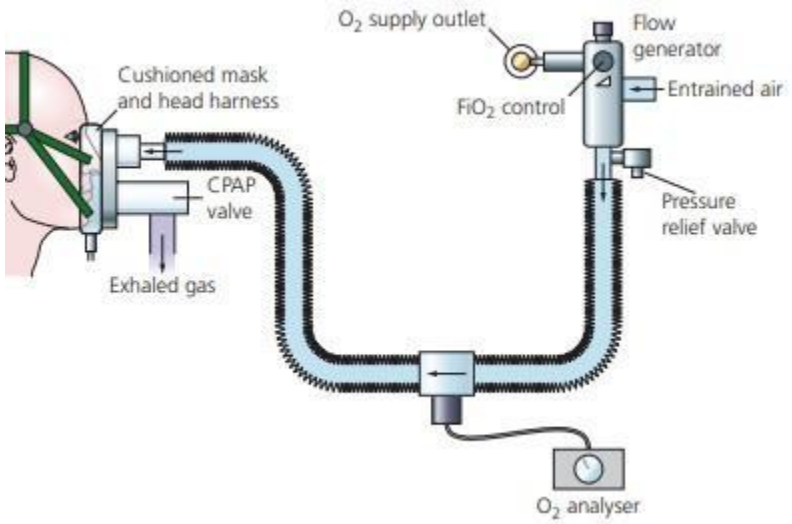


Fig. A CPAP breathing system set-up.

2. Connecting tubing from the flow generator to the inspiratory port of the mask. An oxygen analyzer is fitted along the tubing to determine the inspired oxygen concentration.
3. A tight-fitting mask or a hood. The mask or hood has both inspiratory and expiratory ports. A CPAP valve is fitted to the expiratory port.
4. If the patient is intubated and spontaneously breathing, a T-piece with a CPAP valve fitted to the expiratory limb can be used.

8.3 Problems in practice and safety features

1. CPAP has cardiovascular effects similar to PEEP but to a lesser extent. Although the arterial oxygenation may be improved, the cardiac output can be reduced. This may reduce the oxygen delivery to the tissues.
2. Barotrauma can occur.
3. A loose-fitting mask allows leakage of gas and loss of pressure.
4. A nasogastric tube is inserted in patients with depressed consciousness level to prevent gastric distension.
5. Skin erosion caused by the tight-fitting mask. This is minimized by the use of soft silicone masks or protective dressings or by using a CPAP hood. Rhinorrhoea and nasal dryness can also occur.
6. Nasal masks are better tolerated but mouth breathing reduces the effects of CPAP.

8.4 INTRAVENOUS GIVING SETS

These are designed to administer intravenous fluids, blood and blood products.

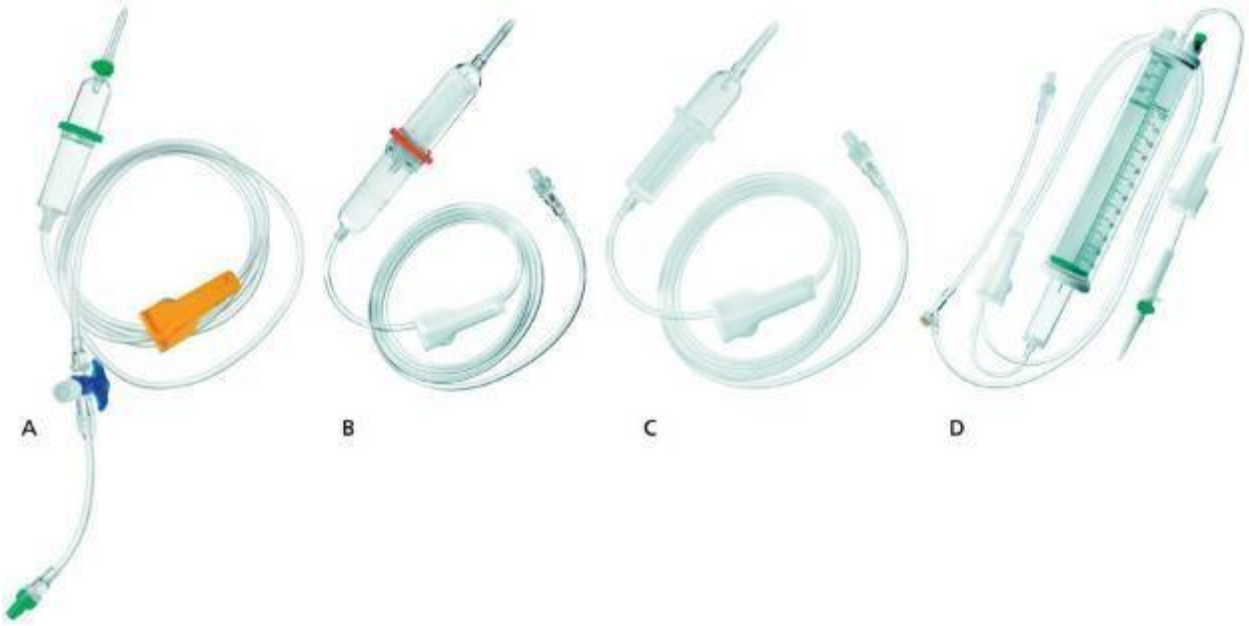


Fig. Intravenous giving sets: (A) Intrafix Safe Set adult fluid set; (B) SangofixB blood adult giving set with a 200-µm filter; (C) Sangopur adult blood set with a 40-µm filter; (D) Dosifix paediatric fluid set with burrette.

8.5 Components

1. Adult giving set:
    a. A clear plastic tube of about 175 cm in length and 4 mm in internal diameter. One end is designed for insertion into the fluid bag whereas the other end is attached to an intravascular cannula with a Luer-lock connection.
    b. Blood giving sets have a filter with a mesh of about 150–200 µm and a fluid chamber. Giving sets with finer mesh filter of about 40 µm are available.
    c. Some designs have a one-way valve and a three-way tap attachment or a rubber injection site at the patient's end. The maximum size needle used for injection should be 23 G.
    d. A flow controller determines the drip rate (20 drops of clear fluid is 1 mL and 15 drops of blood is 1 mL).
2. Paediatric set:
    a. In order to attain accuracy, a burette (30–200 mL) in 1 mL divisions is used to measure the volume of fluid to be infused. The burette has a filter, air inlet

and an injection site on its top. At the bottom, there is a flap/ball valve to prevent air entry when the burette is empty.

b. There are two flow controllers: one is between the fluid bag and the burette and is used to fill the burette; the second is between the burette and the patient and controls the drip rate. An injection site should be close to the patient to reduce the dead space.
c. Drop size is 60 drops per 1 mL of clear fluid. A burette with a drop size similar to the adult's version (15 drops per mL) is used for blood transfusion.
d. 0.2-micron filters can be added in line to filter out air and foreign bodies, e.g. glass or plastic particles. Infusion-related thrombophlebitis can be reduced by the use of these filters.

8.6 Intravenous cannulae

Intravenous cannulae are made of plastic. They are made by different manufacturers with different characteristics.

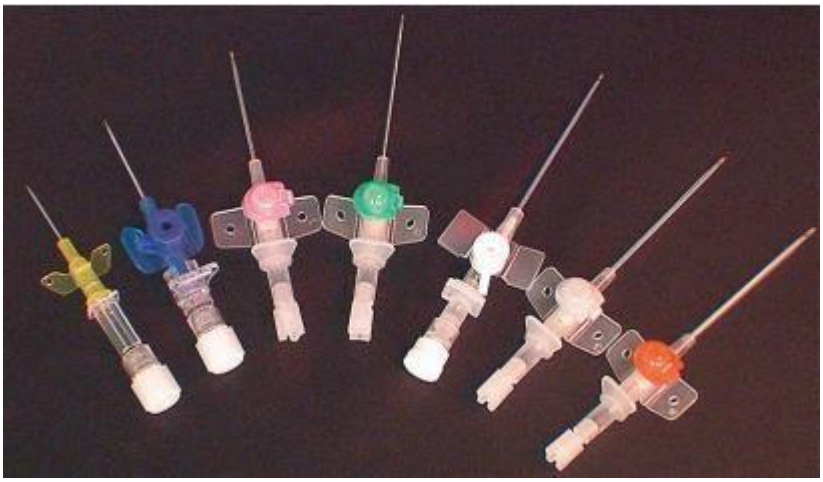


Fig. A range of intravenous cannulae.

Intravenous cannulae can be either with or without a port. Some designs offer protection against the risk of needle stick injuries, covering the sharp needle tip with a blunt end.

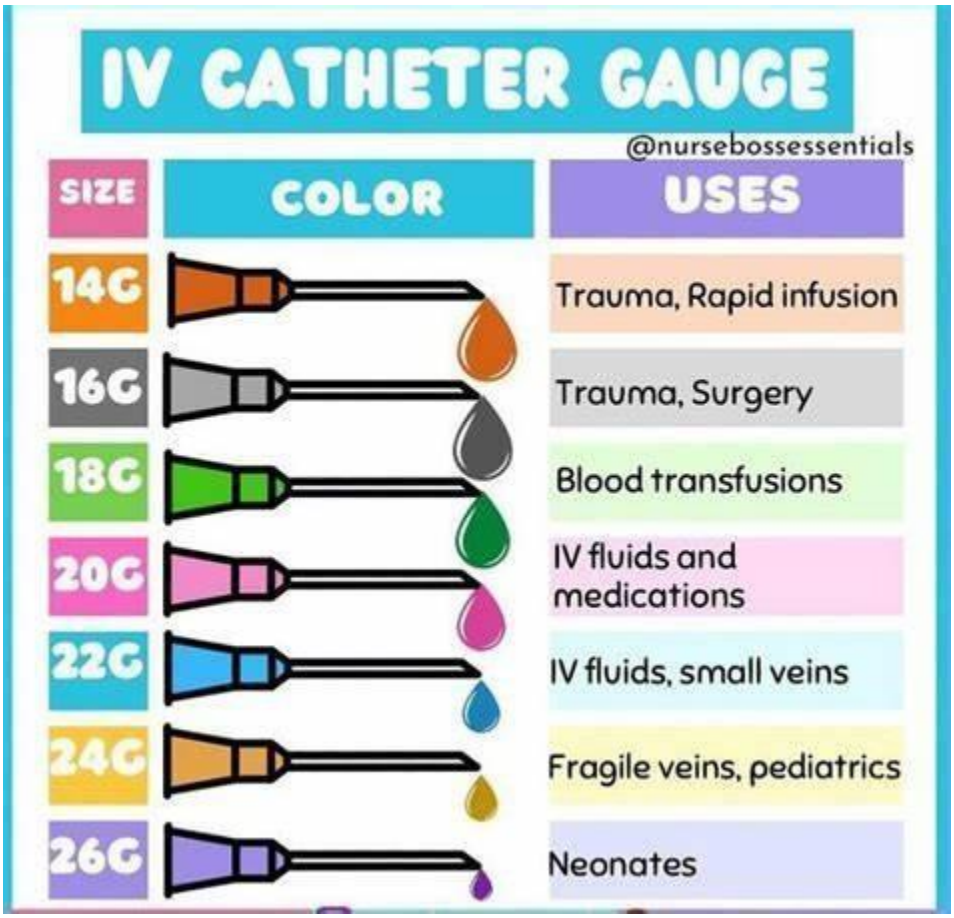

IV CATHETER GAUGE
@nursebossessentials
SIZE
COLOR
USES
14G
Trauma, Rapid infusion
16G
Trauma, Surgery
18G
Blood transfusions
20G
IV fluids and medications
22G
IV fluids, small veins
24G
Fragile veins, pediatrics
26G
Neonates

Fig. Smith's Medical Protective Acuvance cannula designed to reduce the risk of needle stick injury.

More recent designs are the 'closed and integrated' cannulae. A 'closed' system may offer better protection against bacterial exposure than conventional 'open' ports. As the blood does not naturally escape from the catheter hub, these devices further minimize the risk of exposing the clinician to blood during the insertion procedure.

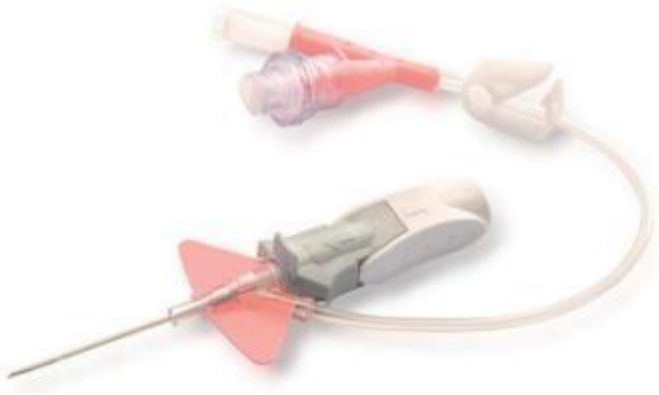


Fig. The BD Nexiva IV closed and integrated cannula.

Using distilled water at a temperature of 22°C and under a pressure of 10 kPa, the flow through a 110-cm tubing with an internal diameter of 4 mm is as follows: 20 G: 40–80 mL/min. 18 G: 75–120 mL/min. 16 G: 130–220 mL/min. 14 G: 250–360 mL/min.

8.7 BLOOD WARMERS

These are used to warm blood (and other fluids) before administering them to the patient. The aim is to deliver blood/fluids to the patient at 37°C. At this temperature, there is no significant haemolysis or increase in osmotic fragility of the red blood cells. There are various designs with the coaxial fluid/blood warmer devices are most popular. A coaxial tubing is used to heat and deliver the fluids to the patient. The outside tubing carries heated sterile water. The inside tubing carries the intravenous fluid. The sterile water is heated to 40°C and stored by the heating case. The water is circulated through the outside tubing. The intravenous fluid does not come in contact with the circulating water. The coaxial tubing extends to the intravenous cannula reducing the loss of heat as fluid is exposed to room temperature.

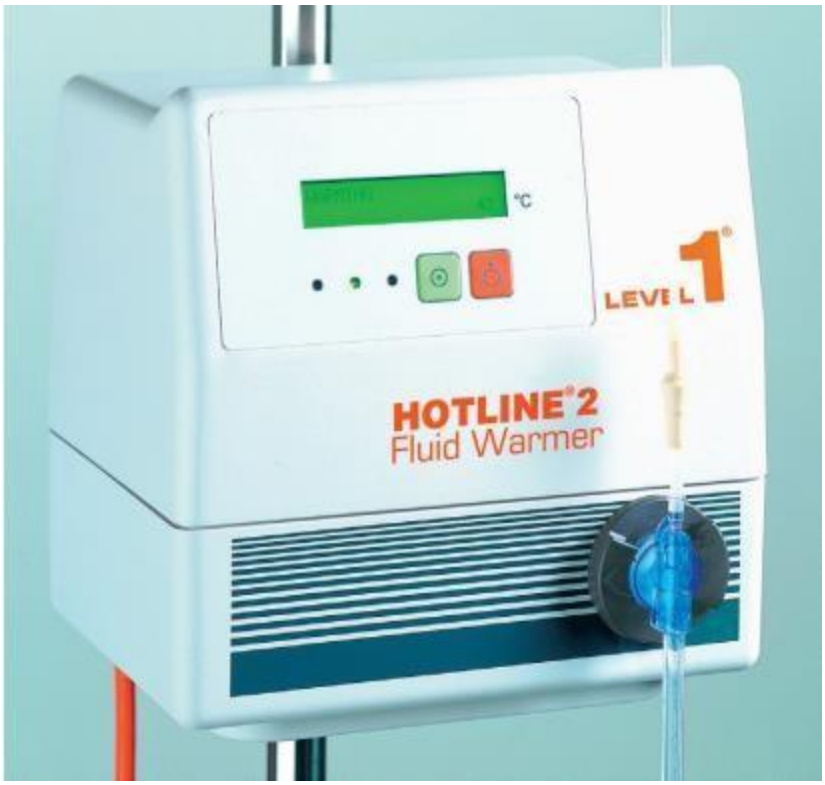


Fig. The Hotline 2 Fluid Warmer.

For patients requiring large and rapid intravenous therapy, special devices are used to deliver warm fluids. Fluids are pressurized to 300 mmHg and warmed with a countercurrent recirculation fluid at a temperature of 42°C.

Fig. Smiths Medical Level 1 H 1200 fast fluid warmer with an integrated air detector.

8.8 FORCED-AIR WARMERS.

These devices are used to maintain the temperature of patients during surgery. They have been found to be effective even when applied to a limited surface body area. They consist of a case where warm ambient air is pumped at variable temperatures between 32 and 37°C. The warm air is delivered via a hose to a thin-walled channeled bag positioned on the patient's body. There are different bags available depending on which part of the body is covered (e.g. upper or lower body). A thermostat to prevent overheating controls the temperature of the warm air. Cooling versions also exist for surgery where body temperature >37°C is desirable, e.g. neurosurgery.

8.9 DEFIBRILLATOR

This is a device that delivers electrical energy to the heart causing simultaneous depolarization of an adequate number of myocardial cells to allow a stable rhythm to be established. Defibrillators can be divided into the automated external defibrillators (AEDs)

and manual defibrillators. AEDs offer interaction with the rescuer through voice and visual prompts.

Fig. The Zoll Pro AED.

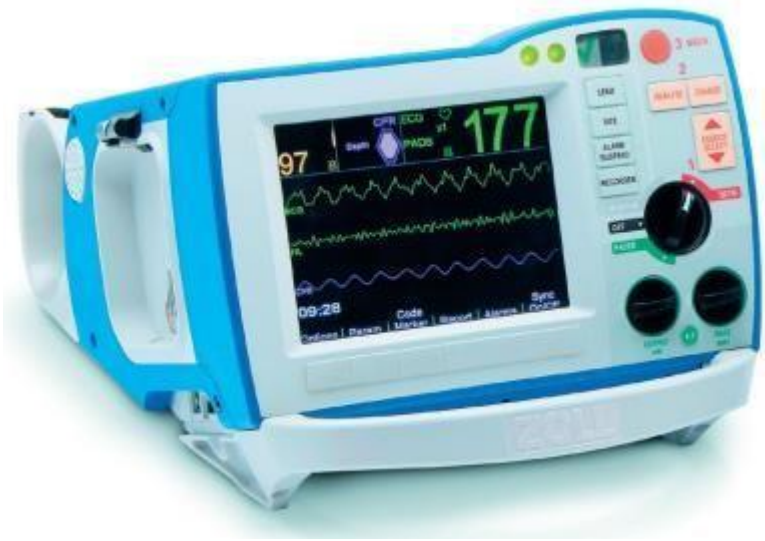


Fig. The Zoll R manual defibrillator.

8.9.1 Components

1. The device has an on/off switch, Joules setting control, charge and discharge buttons.
2. Paddles can be either external (applied to the chest wall) or internal (applied directly to the heart). The external paddles/pads are usually 8–8.5 cm in size.

8.9.2 Mechanism of action

- DC energy rather than AC energy is used. DC energy is more effective causing less myocardial damage and being less arrhythmogenic than AC energy. The lower the energy used, the less the damage to the heart.

- Transformers are used to step up mains voltage from 240 V AC to 5000–9000 V AC. A rectifier converts it to 5000 V DC. A variable voltage step-up transformer is used so that different amounts of charge may be selected. Most defibrillators have internal rechargeable batteries that supply DC in the absence of mains supply.

  This is then converted to AC by means of an inverter, and then amplified to 5000 V DC by a step-up transformer and rectifier.

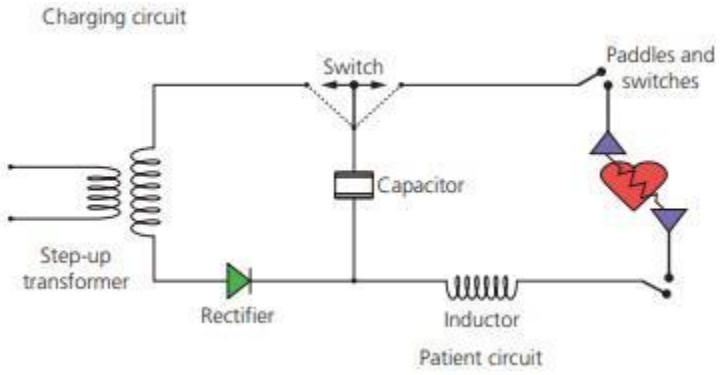


Fig. Defibrillator electric circuit.

- The DC shock is of brief duration and produced by discharge from a capacitor. The capacitor stores energy in the form of an electrical charge, and then releases it over a short period of time. The current delivered is maintained for several milliseconds in order to achieve successful defibrillation. As the current and charge delivered by a discharging capacitor decay rapidly and exponentially, inductors are used to prolong the duration of current flow.
- The external paddles/pads are positioned on the sternum and on the left midaxillary line (fifth–sixth rib). An alternative placement is one paddle positioned anteriorly over the left precordium and the other positioned posteriorly behind the heart. Firm pressure on the paddles is required in order to reduce the transthoracic impedance and achieve a higher peak current flow. Using conductive gel pads helps in reducing the transthoracic impedance. Disposable adhesive defibrillator electrode pads are currently used instead of paddles, offering hands-free defibrillation.
- Most of the current is dissipated through the resistance of the skin and the rest of the tissues and only a small part of the total current (about 35 A) flows through the heart. The impedance to the flow of current is about 50–150 Ohms; however, repeated administration of shocks in quick succession reduces impedance.
- Waveform:

- Monophasic defibrillators deliver current that is unipolar (i.e. one direction of current flow). They are not used in modern practice as they were likely to have waveform modification depending on transthoracic impedance (e.g. larger patients with high transthoracic impedance received considerably less transmyocardial current than smaller patients).
- Biphasic defibrillators deliver a two-phased current flow in which electrical current flows in one direction for a specified duration, then reverses and flows in the opposite direction for the remaining milliseconds of the electrical discharge. Biphasic defibrillators can either be biphasic truncated exponential (BTE) or rectilinear biphasic (RLB). Biphasic defibrillators compensate for the wide variations in transthoracic impedance by electronically adjusting the waveform magnitude and duration to ensure optimal current delivery to the myocardium, irrespective of the patient's size.
  - Monophasic vs biphasic performance: as can be seen in the highest part of the current waveform is known as the 'peak current' when the most current is flowing. Note the difference in height (amps) between the monophasic peak current and the biphasic peak current. Too much peak current during the shock can injure the heart. It's the peak current, not energy that can injure the heart. The goal of defibrillation is to deliver enough current to the heart to stop the lethal rhythm but with a low peak current to decrease risk of injury to the heart muscle.

- For internal defibrillation, the shock delivered to the heart depends on the size of the heart and the paddles.
- Some designs have an ECG oscilloscope and paper recording facilities. DC defibrillation can be synchronized with the top of the R-wave in the treatment of certain arrhythmias such as atrial fibrillation.
- The implantable automatic internal defibrillator (Fig. 13.24) is a self-contained diagnostic and therapeutic device placed next to the heart. It consists of a battery and electrical circuitry (pulse generator) connected to one or more insulated wires. The pulse generator and batteries are

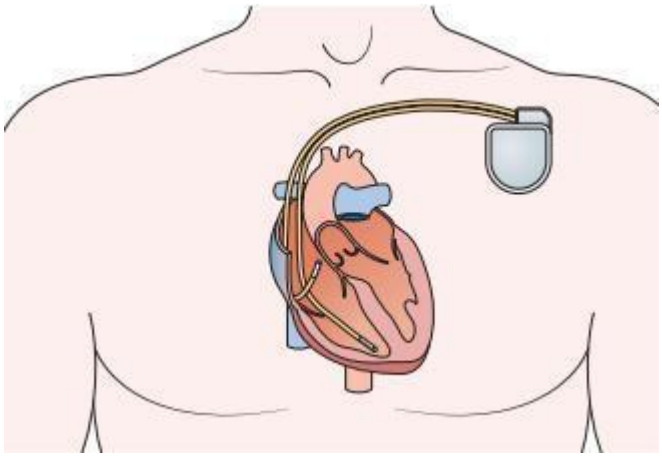


Fig. Implantable cardiac defibrillator.

sealed together and implanted under the skin, usually near the shoulder. The wires are threaded through blood vessels from the implantable cardiac defibrillator (ICD) to the heart muscle. It continuously monitors the rhythm, and when malignant tachyarrhythmias are detected, a defibrillation shock is automatically delivered.

ICDs are subject to malfunction due to internal short circuit when attempting to deliver an electrical shock to the heart or due to a memory error. Newer devices also provide overdrive pacing to electrically convert a sustained ventricular tachycardia, and 'back-up' pacing if bradycardia occurs. They also offer a host of other sophisticated functions (such as storage of detected arrhythmic events and the ability to do 'non-invasive' electrophysiologic testing).

Problems in practice and safety features

1. Skin burns.
2. Further arrythmias.

8.10 CHEST DRAIN

Used for the drainage of air, blood and fluids from the pleural space.

Fig. Seldinger chest drainage kit.

8.10.1 Components

1. A drainage tubing with distal ports.
2. An underwater seal and a collection chamber of approximately 20-cm diameter.

8.10.2 Mechanism of action

- An air-tight system is required to maintain a sub-atmospheric intrapleural pressure. The underwater seal acts as a one-way valve through which air is expelled from the pleural space and prevented from re-entering during the next inspiration. This allows re-expansion of the lung after a pneumothorax and restores haemodynamic stability by minimizing mediastinal shift.
- Under asepsis, skin and subcutaneous tissues are infiltrated with local anaesthetic at the level of the fourth–fifth intercostal space in the midaxillary line. The chest wall is incised and blunt dissection using artery forceps through to the pleural cavity is performed. Using the tip of the finger, adherent lung is swept away from the insertion site.
- The drain is inserted into the pleural cavity and slid into position (usually towards the apex). The drain is then connected to an underwater seal device.
- Some designs have a flexible trocar to reduce the risk of trauma.
- The drainage tube is submerged to a depth of 1–2 cm in the collection chamber. This ensures minimum resistance to drainage of air and maintains the underwater seal even in the face of a large inspiratory effort.

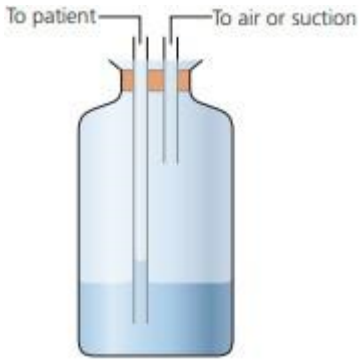


Fig. Chest drain underwater seal.

- The collection chamber should be about 100 cm below the chest as sub atmospheric pressures up to −80 cm H2O may be produced during obstructed inspiration.
- A Heimlich flutter one-way valve can be used instead of an underwater seal, allowing better patient mobility.
- Drainage can be allowed to occur under gravity or suction of about −15–20 mmHg may be applied.

8.10.3 Problems in practice and safety features

1. Retrograde flow of fluid may occur if the collection chamber is raised above the level of the patient. The collection chamber should be kept below the level of the patient at all times to prevent fluid being siphoned into the pleural space.
2. Absence of oscillations may indicate obstruction of the drainage system by clots or kinks, loss of sub-atmospheric pressure or complete re-expansion of the lung.
3. Persistent bubbling indicates a continuing bronchopleural air leak.
4. Clamping a pleural drain in the presence of a continuing air leak may result in a tension pneumothorax.

Chapter 9
EQUIPMENT AND MACHINE CHECKOUT

9.1 INTRODUCTION

The delivery of a safe anesthetic in modern-day practice begins with a checkout of the anesthesia machine. Improper or lack of inspection of anesthetic equipment prior to use has been associated with several significant incidents. Failure to check equipment clearly results in an increased risk of operative morbidity and mortality. With the large variety of anesthetic delivery systems available today, it is critical to understand the basic components of the system so that malfunctions can be detected prior to use or when failure occurs during use. Moreover, regular testing may lead to improved preventive maintenance and enhanced familiarity with the equipment. This chapter focuses on the fundamental components of the anesthesia machine checkout. Specific issues related to unique anesthesia delivery systems should be resolved by referring to the appropriate manufacturers' operator manuals.

9.2 HISTORY OF MACHINE CHECKOUT RECOMMENDATIONS

In 1993, a joint effort between the American Society of Anesthesiologists (ASA) and the U.S. Food and Drug Administration (FDA) resulted in the 1993 FDA Anesthesia Apparatus Checkout Recommendations. This simplified the initial 1986 preuse checkout and made it more user-friendly. At the time, the 1993 checklist focused on components that were immediately dangerous for patients and mechanisms that failed more regularly. This checklist was applicable to most commonly available anesthesia machines. Nevertheless, despite the recognized importance of an anesthesia machine checkout, available evidence suggests that the 1993 recommendations were neither well understood nor reliably used by anesthesia providers.

Moreover, because of recent and ongoing fundamental changes to the various anesthesia machine designs, the 1993 FDA preuse checklist may no longer be universally applicable to all anesthesia delivery systems. As more machines incorporate electronic checkouts, the user must determine which portions are automatically checked and which portions require manual checks. In such cases, the anesthesia care provider must be aware that the electronic machine check may not be a comprehensive preanesthesia checkout. The user should follow the equipment manufacturer's recommended preuse checklist, utilizing both its automated and manual components: for example, the machine may check the high and low-pressure systems for leaks and the electronics of the

ventilator, but the user may still need to perform manual checks of the pressure release of the breathing circuit, of auxiliary oxygen, and of backup emergency ventilation.

As a result, in 2005, the ASA's Committee on Equipment and Facilities, in conjunction with the American Association of Nurse Anesthetists (AANA) and the American Society of Anesthesia Technologists and Technicians (ASATT), began to develop a revised preuse checklist that was designed to be more workstation specific. These recommendations were published in 2008 and were intended to eventually replace the 1993 FDA Anesthesia Apparatus Checkout Recommendations. Rather than a checklist with specific instructions on how to perform each test, these new guidelines elaborate on specific systems and subsystems that must be evaluated. It is ultimately up to the user, along with the anesthesia machine manufacturer, to determine the actual mechanisms and/or specific checks that should be used to accomplish these subsystem evaluations. Appropriate personalized checkout procedures may need to be developed for individual machines and practices.

The 1993 Anesthesia Apparatus Checkout Recommendations placed all of the responsibility for the preuse checkout on the anesthesia provider. The new 2008 recommendations identify certain aspects of the preanesthesia checkout that may be performed by a qualified anesthesia technician or a biomedical technician.

Redundancy in the critical aspects of the checkout process makes it more likely that problems will be identified prior to use for a patient. Nevertheless, regardless of the additional support of technicians, the anesthesia care provider is ultimately responsible for the proper function of all equipment used to deliver anesthesia care.

The anesthesia machine, in addition to the maintenance schedule imposed by the manufacturer, undergoes a detailed checkout of all its functions at the beginning of every day. In addition, prior to every case, the provider performs a shorter checkout of the machine's most essential functions and those most likely to have developed a temporary interruption between cases (breathing circuit integrity; adequate suction; adequate anesthesia gas in vaporizer, gas flows, and working monitors) but does not repeat in detail all the gas pipeline and electrical power checks performed in the morning unless the machine has been moved or there is some other reason to think they may have been altered.

9.3 ANESTHESIA MACHINE CHECKOUT PROCEDURE

As stated earlier, the goal of the preanesthesia checkout is to allow for the safe delivery of anesthesia care. Requirements for safe delivery of anesthetic care include the following:

- Reliable delivery of oxygen at any appropriate concentration up to 100%
- Reliable means of positive pressure ventilation
- Availability of functional backup ventilation equipment
- Controlled release of positive pressure from the breathing circuit
- Anesthesia vapor delivery (if intended as part of the anesthetic plan)
- Adequate suction
- Means to conform to standards for patient monitoring

The new guidelines for the preanesthesia checkout procedures consist of 15 items. These items must be performed as part of a complete preanesthesia checkout on a daily basis (**items that must be completed prior to each procedure are in bold**). The 15 items are as follows:

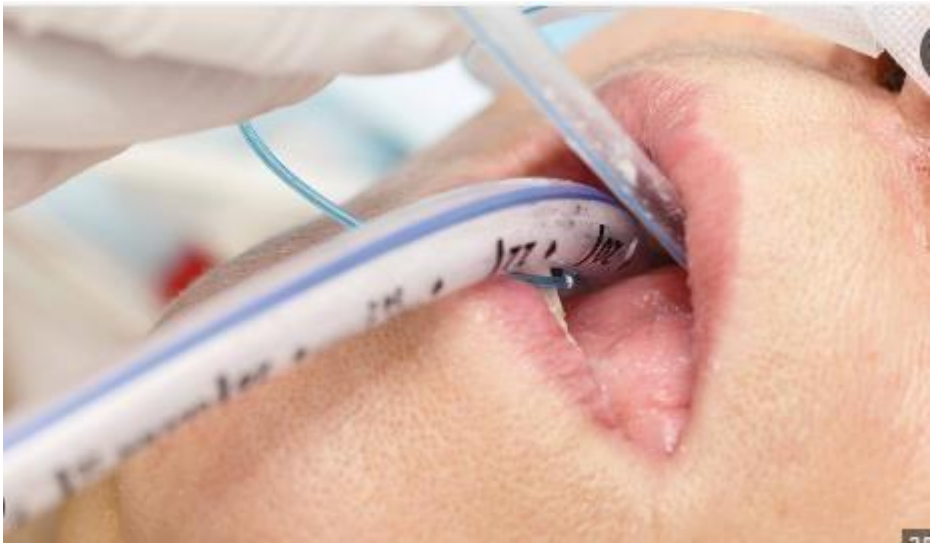
1. Verify that auxiliary oxygen cylinder and self-inflating manual ventilation device are available and functioning.

   Anesthesia ventilator failure resulting in the inability to provide patient ventilation is rare but can occur at anytime. For those situations where the problem cannot be immediately identified or corrected, a manual ventilation device (e.g., bag valve mask) may be necessary to provide positive pressure ventilation until the problem is resolved. As a result, a self-inflating manual ventilation device and an auxiliary oxygen cylinder should be available and checked for proper function at each anesthesia setting.

   In addition, the oxygen cylinder should have a regulator, and a device to open the cylinder valve should be present. A full E cylinder of oxygen has a pressure of about 2,000-pound-force per square inch gauge (psig), which is equivalent to around 625 L of oxygen. After checking the oxygen cylinder pressure to ensure adequate supply, the cylinder should be stored with the valve closed in order to prevent unintended leakage or drainage of oxygen.

Fig. self-inflating manual ventilation device

2. **Verify that patient suction is adequate to clear the airway.** The immediate ability to clear airway secretions or gastric contents is essential for safe anesthetic care. Inability to visualize the glottic opening and therefore delay in timely acquisition of a secure airway can be dangerous and possibly fatal. Aspiration of gastric contents can cause prolonged intubation and airway complications.



Adequate strength of the suction can be tested by occluding the suction tubing orifice with the underside of a thumb and determining if the weight of the suction tubing can be supported at waist height. Prior to anesthesia, adequate suction

should be checked, and a rigid suction catheter (e.g., Yankauer) should be available on the machine.

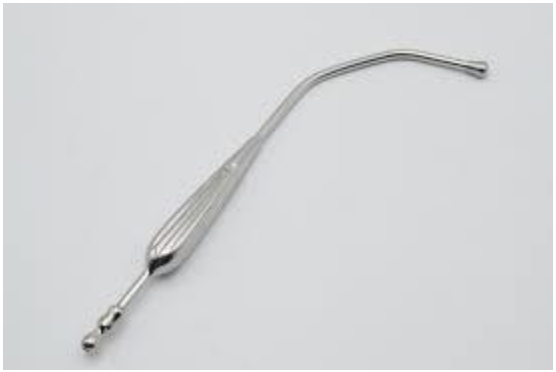
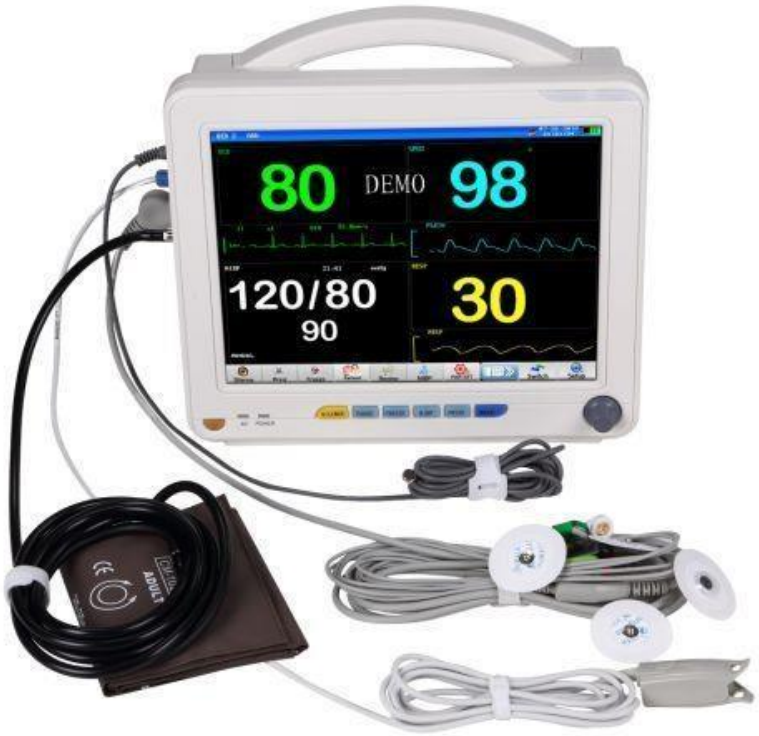


Fig. Yankauer suction tubing

3. Turn on the anesthesia delivery system and confirm that AC power is available. AC power and the availability of backup battery power should be confirmed prior to the delivery of anesthesia. Visual indicators of the power systems exist on most anesthesia delivery systems. These should be confirmed as should appropriate connection of the power cord to a working AC power source. If the AC power is not confirmed, complete system shutdown is at risk when battery power is unknowingly depleted. Desflurane vaporizers, if used, should be checked for adequate electrical power source as well.

4. **Verify availability of required monitors and check alarms.** The patient's oxygenation, ventilation, circulation, and temperature should be continually evaluated according to the ASA's Standards for Basic Anesthetic Monitoring. Verification of the availability and proper function of the appropriate monitoring supplies should be performed prior to each anesthetic. Examples of necessary equipment include, but are not limited to, blood pressure cuffs, pulse oximetry probes, electrocardiogram (ECG) leads, and capnography. Moreover, the appropriate audible or visual alarms that would indicate problems with, or disruption of, patient oxygenation, ventilation, circulation, and temperature should be intact. It is prudent for the anesthesiology technician to turn off the monitors and then turn them back on between cases to be sure that alarms are reset to default values as designed by each individual institution.



5. Verify that pressure is adequate on the spare oxygen cylinder mounted on the anesthesia machine.

   Spare oxygen cylinders are mounted on anesthesia machines in the event that central oxygen supply is lost. Anesthesia machines require oxygen not only to provide oxygen to the patient but often to power pneumatically driven ventilators. The pressure of the oxygen cylinders should be checked to ensure an acceptable amount of backup oxygen is available. The oxygen cylinder valves should be closed after verification in order to prevent unrecognized depletion of the cylinder due to pressure fluctuations in the machine during mechanical ventilation or in the event of actual pipeline supply failure.

   Rarely, the cylinder is intended to be the primary oxygen source. In these cases, if the ventilator is pneumatically driven, then the oxygen cylinder supply may be depleted quickly. As a result, manual or spontaneous ventilation may be more appropriate in order to maximize the duration of oxygen supply. On the other hand, the duration of oxygen supply for electrically powered or piston-driven ventilators depends only on total fresh oxygen gas flow.

Verify that the piped gas pressures are ≥50 psig. Since there are many scenarios that may cause disruption of gas delivery from a central source, pressure in the piped gas supply should be checked at minimum once per day in order to ensure that adequate pressure is available for proper function of the anesthesia machine. If the pipeline hoses have been disconnected in order to move the anesthesia machine at any point during the day, the hoses should be reconnected to the central pipeline supply, and the fittings should be examined for firm connections without audible leaks. The pipeline pressure should be 50-55 psi.

7. **Verify that vaporizers are adequately filled and, if applicable, that the filler ports are tightly closed. (Provider completes prior to each procedure; technician can complete daily.)**

   Adequate supply of volatile anesthetics is requisite for vapor-based anesthetics in order to reduce the likelihood of inadequate anesthesia and recall under anesthesia. In addition, many vaporizers do not have low-agent alarms, so checking prior to usage is important. After filling the vaporizers, filler ports should be adequately tightened to prevent unrecognized leakage, especially for older vaporizers that do not have systems that automatically close after completion of refilling. Vaporizers should also be secured so that they cannot tilt or be lifted from their mounts.

8. Verify that there are no leaks in the gas supply lines between the flowmeters and the common gas outlet. **(If the vaporizer has been changed, this should be rechecked prior to use.)**

   The low-pressure component of the anesthesia machine circuit is located between the flow control valves and the common gas outlet. The leak test checks the integrity of the anesthesia machine in this part of the circuit. The components located within this area are subject to breaking and developing leaks. Leaks in the low-pressure circuit can cause leakage of oxygen from the inspired gas and delivery of a hypoxic gas mixture. Likewise, leakage of inhaled anesthetic can result in the patient receiving much less gas anesthetic than is indicated on the machine vaporizer, which places the patient at risk for awareness under anesthesia. In addition, each individual vaporizer must be turned on in order to check for leaks within each vaporizer or at the mount, and it is especially important to recheck this test whenever a vaporizer is changed.

   Several different methods have been used to check the low-pressure circuit for leaks. One reason for the large number of methods is that the internal design of various machines differs considerably. The clearest example is the difference between most GE Healthcare/Datex-Ohmeda and Dräger Medical workstations. Most GE Healthcare/Datex-Ohmeda workstations have a check valve near the

common gas outlet, whereas Dräger Medical workstations do not. The presence or absence of this check valve may determine which preoperative leak test is indicated. The check valve is located downstream from the vaporizers and upstream from the oxygen flush valve, and it is open in the absence of back pressure. Gas flow from the manifold moves a rubber flapper valve off its seat, thereby allowing the gas to proceed freely to the common outlet. The valve closes when back pressure is exerted on it, preventing the flow of gas back into the machine and through a leak. Examples of back pressure that can cause the check valve to close are oxygen flushing, peak breathing circuit pressures generated during positive pressure ventilation, and the use of a positive pressure leak test. Typically, the low-pressure circuit of anesthesia workstations without an outlet check valve can be tested with a positive pressure leak test (e.g., with Dräger Medical machine). When performing a positive pressure leak test, the operator generates positive pressure in the low-pressure circuit by using flow from the anesthesia machine or from a positive pressure bulb to detect a leak. One common test is the retrograde fill test, which is performed by closing the adjustable pressure-limiting (APL) valve and occluding the patient port. Oxygen flow or flush is used to fill and distend the reservoir bag, and flow is adjusted so that a pressure of 30 cm H2O on the manometer is maintained in the breathing system. No more than 350 mL/min flow should be necessary to maintain a steady pressure. When complete, the pressure should be relieved by opening the APL valve, not by opening the patient port. Relieving the pressure by opening the patient port could cause CO2 absorbent dust to enter the system. Notably, the retrograde fill test checks both the low-pressure part of the machine and the breathing circuit and does not isolate the source of the leak. In addition, it is not very sensitive to small leaks.

Machines with check valves must be tested with a negative pressure leak test (e.g., GE Healthcare/Datex-Ohmeda machine). When performing a negative pressure leak test, the operator creates negative pressure in the low-pressure circuit by using a suction bulb to detect leaks. In order to do this, the machine's master switch, flow control valves, and vaporizers should all be initially turned off. The suction bulb is attached by tubing and an adapter to the common fresh gas outlet, and the bulb is squeezed repeatedly until it is fully collapsed. This creates a vacuum in the low-pressure system. If the bulb stays collapsed for at least 10 seconds, the system is free of leaks, but if the bulb reinflates during this period, a leak is present. The test is repeated with each vaporizer individually turned to the "on" position because leaks inside the vaporizer can be detected only when the vaporizer is turned on. The negative pressure leak test is the most sensitive leak test, as it can detect leaks as small as 30 mL/min. This test is used to be

considered the universal leak test since it works for machines with or without a check valve, but unfortunately, many new machines do not have accessible common gas outlets. Most machines whose low-pressure system cannot be tested via the common gas outlet are tested for low-pressure leaks electronically. For specific instructions, the appropriate anesthesia machine manual should be referenced, as there are many machines that have automated checks and/or variations to these procedures.

9. Test scavenging system function.

    To prevent room contamination by anesthetic gases, a functional scavenging system is necessary. The connections between the anesthetic machine and the scavenging system must be checked daily to ensure integrity of the scavenging system. The anesthesia technician should be particularly careful to remember to attach the scavenging system to the evacuation system when moving anesthesia machines to out-of-operating room (OR) locations of care. There are various scavenging system designs that may require that an adequate vacuum level be present. On active systems (e.g., full vacuum), vacuum pressure can be modulated by the screw valve. Most modern scavenging systems have positive and negative pressure relief valves. The positive pressure relief valve allows exhaled gases to be released into the OR in the event of inadequate vacuum (usually occurs in an active system when someone inadvertently closes the screw valve). The negative pressure relief valve prevents suction in an active vacuum system from affecting airway pressure in the breathing circuit for the patients. As these valves are important to protect the patient from pressure fluctuations coming from the scavenging system, they must be checked daily. The checks on these valves can be quite complex; therefore, the anesthesia technician should receive specific troubleshooting training from the hospital biomedical engineers and refer more complicated problems to them directly.

10. Calibrate or verify calibration of the oxygen monitor and check the low-oxygen alarm.

    Calibration of the oxygen sensor is critical for safe patient care. Continuous monitoring of the inspired oxygen concentration helps prevent the delivery of a hypoxic gas concentration to patients. The oxygen monitor is crucial to detect any changes in the oxygen supply.

    Oxygen sensor calibration should occur at least once per day. Some anesthesia machines are self-calibrating. For these machines, they should be verified to read 21% when sampling room air. The oxygen sensor calibration can be performed by an anesthesia provider or anesthesia technician. If more than one oxygen monitor is present, the primary sensor that will be relied upon for oxygen monitoring during patient care should be the one checked.

The low–oxygen concentration alarm should also be checked at this time. This is done by setting the low-oxygen alarm above the measured oxygen concentration and confirming that an audible alarm is generated. Detailed oxygen sensor calibration instructions can be found in the specific anesthesia machine's operator manual.

11. **Verify that carbon dioxide absorbent is not exhausted.**

A circle breathing system relies on the removal of carbon dioxide to prevent rebreathing of carbon dioxide by the patient. There is a characteristic color change in the carbon dioxide absorbent, depending on the particular absorbent being used, that indicates depletion of the absorbent. When this color change occurs, it is a visual reminder that the absorbent must be replaced. Some newer absorbents change color when they become desiccated. If the carbon dioxide absorbent is exhausted or desiccated, it should be changed.

It is possible that the carbon dioxide absorbent loses its ability to remove carbon dioxide without producing a color change. For example, an exhausted desiccated absorbent may return to its original color after a period of rest. Capnography, which must be used in every anesthetic, is helpful in indicating the need to replace the absorbent. When the inspired carbon dioxide concentration is detected to be greater than 0, this indicates that the patient is rebreathing carbon dioxide and that the absorbent may be used up and, therefore, needs to be replaced. When replacing carbon dioxide absorbent canisters, it is important to install them correctly. Incorrectly installed carbon dioxide absorbent canisters are a common source of leaks within the anesthesia machine.

12. **Breathing system pressure and leak testing.** The breathing system leak test must be performed on the components that will be used during a particular anesthetic. If any portion of the circuit is changed after completing the leak test, the leak test must be performed again to ensure the integrity of the breathing system. The purpose of this test is to ensure that adequate pressure can be generated and maintained in the breathing system during assisted ventilation. Adequate pressure is usually considered to be greater than or equal to 30 cm H_2O. This test also checks the ability to relieve pressure in the breathing circuit with the APL valve during manual ventilation.

To manually check the breathing system for leaks, the APL valve is closed and the patient port is occluded at the Y-piece. The oxygen flush valve is used to instill 30-cm H_2O pressure into the breathing circuit. If the circle system is free of leaks, the value on the pressure gauge should not decrease. Of note, newer machines may have automated testing that can be used to detect leaks. Additionally, they can also determine the compliance of the breathing system. Once adequate pressure is obtained in the circle system, it can be released by completely opening

the APL valve. This step can test for proper functioning of the APL valve, ensuring that it entirely relieves the pressure in the circle system.

13. Verify that gas flows properly through the breathing circuit during both inspiration and exhalation. Although checking the breathing system for pressure and leaks is important, this test does not assess the function of the unidirectional inspiratory and expiratory valves. The presence of the valves can be assessed visually. To test for proper function of the unidirectional inspiratory and expiratory valves, first remove the Y-piece from the circle system. Next, breathe through the two corrugated hoses separately. The valves should be present, and they should move appropriately. The person performing the test should be able to inhale but not exhale through the inspiratory limb and able to exhale but not inhale through the expiratory limb. At the completion of this test, the breathing circuit should be changed to a fresh circuit prior to attaching the anesthesia machine to the patient. This flow test can also be performed by attaching a breathing bag to the Y-piece and using the ventilator. In addition, capnography can also be useful to detect an incompetent valve. For example, an incompetent inspiratory valve should be considered in situations of high (>0) inspired carbon dioxide concentration.
14. Document completion of the checkout procedures. A printed copy of the preanesthesia checkout procedures should be retained near or in the anesthesia machine since an organized and systematic list may result in improved fault detection over memory alone. Moreover, a pictorial checklist may be helpful as it can be simpler to follow than a typewritten list. Documentation of checkout procedure completion should be performed and may be important in the case of an adverse incident, as omission of the checkout can by cited as evidence of substandard care. Dates and times of certain checkout procedures may be recorded automatically by some computerized checkout systems, but those that are not automatically recorded should be manually documented by the individual who performs the checkout procedure.

    Record keeping is important to provide supporting evidence that the equipment is being appropriately maintained. Should service be necessary, this record may also be helpful for service representatives who come to repair the equipment, for providing a reminder to check on the repair that was done, and for referencing at a later date what was repaired and who performed the repair. A log of malfunctions may also help to determine if a particular piece of equipment warrants replacement. This record should be retained, should an adverse outcome lead to litigation.
15. **Confirm ventilator settings and evaluate readiness to deliver anesthesia care. (Anesthesia provider should perform this.)** Prior to starting each anesthetic, the completion of the preanesthesia checkout procedures should be

verified as well as the availability of essential equipment. Ventilator settings should be confirmed and pressure limit settings used as a secondary backup to prevent barotrauma once positive pressure ventilation is used. Specifically, the presence and functionality of appropriate monitors, the capnogram, and oxygen saturation by pulse oximetry should be checked. Proper flowmeter and ventilator settings, placement of the ventilator switch to manual, and adequate filling of the vaporizers should also be ensured before initiation of an anesthetic.

The delivery of safe anesthetic care in modern practice begins with a thorough evaluation of the anesthetic delivery system being used. Anesthesia providers, along with trained anesthesia technicians and biomedical technicians, must have a thorough understanding of the fundamental components of the anesthesia machine. Thus, malfunctioning components can be repaired or replaced to decrease the potential for patient injury. These preanesthesia machine checks should be documented not only for maintenance records but also for medical-legal reasons. With the great variation in anesthesia machine design, it is important to always refer to the specific anesthesia machine manufacturer's instruction manual for more detailed information and instruction.

9.4 AUTOMATED CHECKOUTS

With increasing automaticity in the current environment, anesthesia providers and technologists must not become complacent with automated checks built into modern anesthesia machines. Depending on the machine, it may not be an adequately comprehensive checkout. As machines transition to more automated checkouts, the operator is removed one step further from the actual machine check process, which may reduce familiarity with how the machine actually works. It has been shown in simulation with conventional anesthesia machines that known equipment failures may not be properly diagnosed and managed even by experienced anesthesia providers, as failures become less common and, paradoxically, providers become less adept at rapidly identifying them. This is where a well-trained anesthesia technologist, familiar with the essential engineering functions of the machine, becomes especially vital.

After an anesthesia machine check has passed inspection, malfunctions and failures may still occur. Over the years, there have been multiple reports of automated checks passing inspection, only to discover a malfunction after the start of an anesthetic. Anesthesia equipment may malfunction while providing an anesthetic if a mechanical (or an electronic or software) failure. Anesthesia equipment is frequently moved during a surgical procedure, and this may cause an inadvertent disconnection in tubing or wires on the anesthesia machine. Anesthesia providers and technologists must be ready to act quickly to troubleshoot the problem.

Anesthesia technologists are a critical part of the anesthesia team. Their expertise with the anesthesia machine is invaluable. Even well-trained and prepared anesthesia providers require assistance in checking the anesthesia machine for malfunctions and for troubleshooting them when problems actually occur.

9.5 SUMMARY

The anesthesia machine is a critical component of safety in the OR. Failures of the anesthesia machine function have the potential to cause significant patient injuries if undetected or if backup fail-safes are not properly deployed. Proper maintenance and a thorough checkout procedure can identify many machine problems before they have a chance to cause patient problems. Multiple organizations and the anesthesia machine manufacturers have been instrumental in devising detailed anesthesia machine checkout procedures. Each machine will have a unique checkout procedure detailed by the manufacturer; all checkout procedures, however, share common concerns. This chapter presents an overview of the machine checkout procedure that should be customized for each anesthesia machine according to the manufacturer.

Section III

EMERGENCIES

Chapter 10

ANAPHYLAXIS

10.1 INTRODUCTION

An acute, severe, hypersensitivity reaction to a trigger such as an antibiotic or latex.

- Allergic reactions may be triggered by a variety of **factors i**ncluding drugs (e.g., penicillin), foods (e.g., shellfish), insect stings and chemicals (Table 10-1).
- There is a wide spectrum of severity ranging from a **harmless** skin rash (urticaria), to **potentially fatal** airway obstruction (laryngeal oedema) and **full blown** anaphylaxis (hypotension, bronchospasm).
- Anaphylaxis is much more common in adults than children.

TABLE 10-1 Causes of Anaphylactic and anaphylactoid reactions.

Anaphylactic reactions[1] **against polypeptides**	· Venoms (Hymenoptera, fire ant, snake, jellyfish) · Airborne allergens (pollen, molds, danders) · Foods (peanuts, milk, egg, seafood, grain) · Enzymes (trypsin, streptokinase, chymopapain, asparaginase) · Heterologous serum (tetanus antitoxin, antilymphocyte globulin, antivenin) · Human proteins (insulin, corticotropin, vasopressin, serum and seminal proteins) · Latex
Anaphylactic reactions against hapten carrier	· Antibiotics (penicillin, cephalosporins, sulfonamides) · Disinfectants (ethylene oxide, chlorhexidine) · Local anesthetics (procaine)

Anaphylactoid reactions[2]	· Polyionic solutions (radiocontrast medium, polymyxin B) · Opioids (morphine, meperidine) · Hypnotics (propofol, thiopental) · Muscle relaxants (rocuronium, succinylcholine, cis-atracurium) · Synthetic membranes (dialysis) · Nonsteroidal anti-inflammatory drugs (NSAIDs) · Preservatives (sulfites, benzoates) · Protamine · Dextran · Steroids · Exercise · Idiopathic (unknown)

[1] Anaphylaxis (or Anaphylactic reactions) is an exaggerated response to an allergen (e.g., antibiotic) that is mediated by a type I hypersensitivity reaction.

[2] Anaphylactoid reactions resemble anaphylaxis but do not depend on IgE antibody interaction with antigen.

10.2 Clinical Manifestations

Despite differing mechanisms, anaphylactic and anaphylactoid reactions typically are clinically indistinguishable and equally life-threatening. (Table 10-2) summarizes important manifestations of anaphylactic reactions.

TABLE 10-2 Clinical Manifestations of Anaphylaxis.

Organ System	Signs and Symptoms
Cardiovascular	Hypotension,[1] tachycardia, arrhythmias
Pulmonary	Bronchospasm,[1] cough, dyspnea, pulmonary edema, laryngeal edema, hypoxia
Dermatological	Urticaria,[1] facial edema, pruritus

[1]Key signs during general anesthesia.

10.3 Pathophysiology

Massive release of leukotrienes, histamine, and prostaglandins in response to release of IgE from mast cells, causing potentially life-threatening

- Vasodilation
- myocardial suppression · Bronchoconstriction.

10.4 Differential Diagnosis

- Sepsis
- Nonimmunologic drug reaction (“Red man syndrome” with vancomycin, Morphine-induced histamine release)

10.5 Immediate Management

- Ensure an adequate airway
- Supplemental O_2 (FiO_2 100% if severe)
- Establish large-bore IV access
- Treat bronchospasm (inhaled albuterol 4–8 metered doses)
- Search for triggering agent If mild anaphylaxis:
- Epinephrine 0.1 mg-0.5 SQ or IV every 10–20 minutes
- Hydrocortisone 100 mg IV

- Diphenhydramine 50 mg IV (H_1 antagonist)
- H_2 antagonist (e.g., famotidine 20 mg IV)

If severe anaphylaxis with hypotension, above interventions plus

- Consider intubation and mechanical ventilation
- Arterial blood gas for pH, $PaCO_2$ and PaO_2 assessment
- Fluid resuscitates with isotonic crystalloid or colloid solutions
- Epinephrine infusion (titrate to systolic blood pressure)
- If cardiac arrest occurs, administer epinephrine 1 mg IV and begin advanced cardiac life support (ACLS).
- Perform a careful evaluation of agents administered immediately prior to the anaphylactic event, including antibiotics, latex, and neuromuscular blocking agents.
- Inform the surgeons; consider terminating the procedure

10.6 Diagnostic Studies

Clinical diagnosis—no diagnostic studies

10.7 Subsequent Management

- Careful review of the patient's chart for known drug allergies
- Prominently document the event to prevent another exposure to the same drug
(if identifiable)
- If the trigger is unknown, consider skin testing **Risk Factors**
- History of atopy (genetic tendency to develop allergic diseases)
- History of allergic rhinitis
- Prior exposure to certain drugs (e.g., intravenous contrast)

Prevention

- Review each patient's chart carefully for known allergies.
- Limit the use of latex products in the operating room.

Special Considerations

- Neuromuscular blocking agents are the most common trigger in the perioperative period.
- Latex and perfume allergy (undisclosed) is probably more common than is reported in the medical literature.

Chapter 11

BURN, HEMORRHAGE AND SHOCK

11.1 BURN

Burns represent a unique but common traumatic injury. Temperature and duration of heat contact determine the extent of burn injury. Children (because of a high body surface area to body mass ratio) and the elderly (whose thinner skin allows deeper burns from similar thermal insult) are at greater risk for major burn injury.

11.2 Classification of Burns

Burns are classified as first, second, or third degree.

11.2.1 First-degree Burns

- First-degree burns are injuries that do not penetrate the epidermis.
- For example: sunburns and superficial thermal injuries.
- These burns heal spontaneously.
- Fluid replacement for these burns is not necessary.

11.2.2 Second-degree Burns

- Second-degree burns are partial-thickness injuries (superficial or deep) that penetrate the epidermis, extend into the dermis for some depth.
- These are associated with blistering.
- Fluid replacement therapy is indicated for patients with second-degree burns when more than 20% of total body surface area (TBSA) is involved.
- Skin grafting also may be necessary in some cases of second-degree burns, depending upon size and location of the wounds.

11.2.3 Third-degree Burns

- Third-degree burns are those in which the thermal injury penetrates the full thickness of the dermis.
- Nerves, blood vessels, lymphatic channels, and other deep structures may have been destroyed, creating a severe, but insensate, wound (although surrounding tissue may be very painful).

- Debridement and skin grafting are nearly always required for recovery of patients from third-degree burns.

Fourth-degree burns involve muscle, fascia, and bone.

11.1.3 Rule of Nine

Major burns (a second- or third-degree burn) involve >20% TBSA. The *rule of nines* (Fig 11-1) is utilized to estimate burned surface area as a percentage of total body surface area (TBSA).

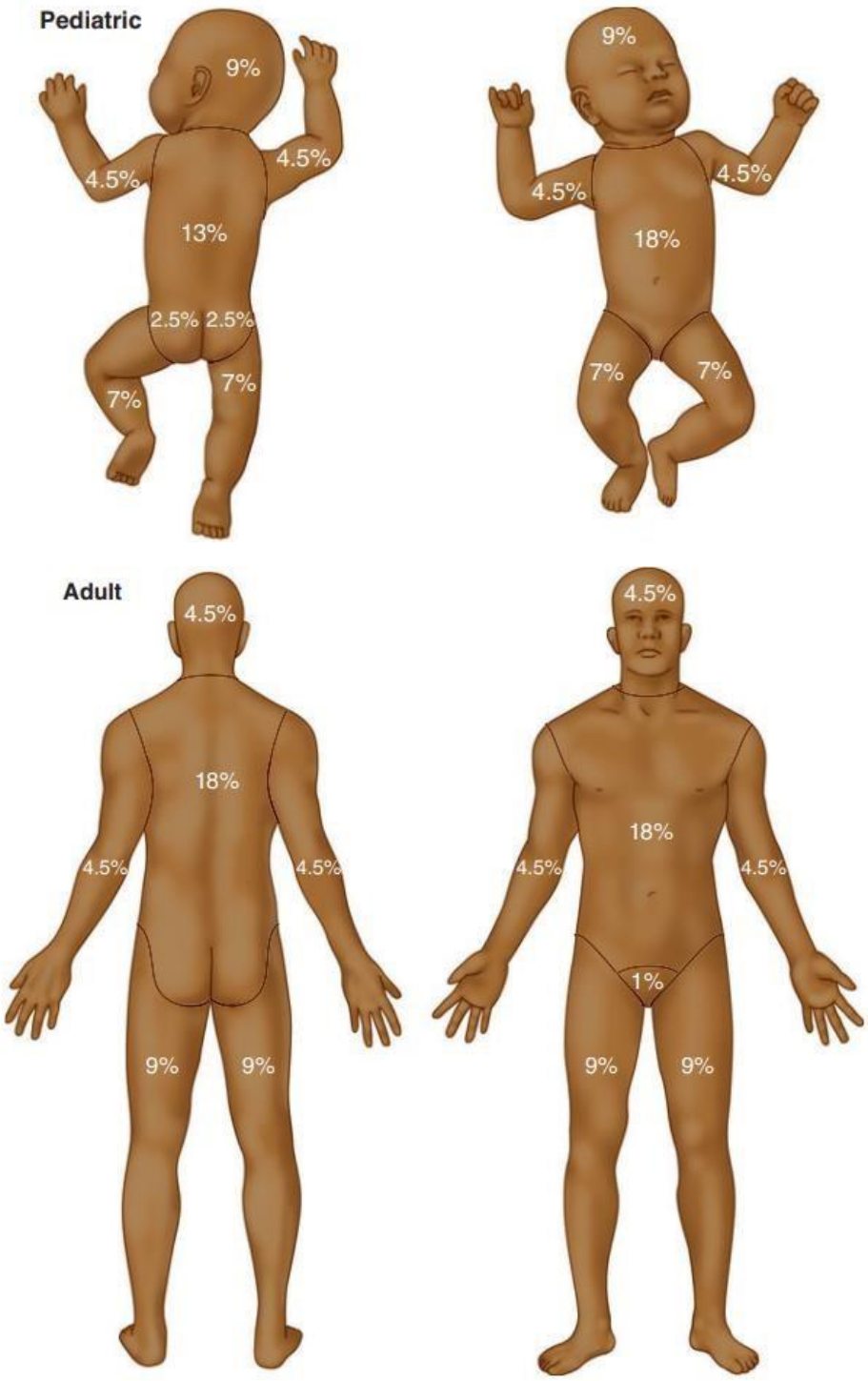


Fig. 11-1. The rule of nines.

11.1.4 Presentation

- Thermal trauma after exposure to flames in an enclosed space

- Thermal trauma after airplane, motor vehicle, or industrial accidents
- Chemical burns after industrial accidents
- Partial-thickness burns are red, blanch when touched, and heal spontaneously.
- Full-thickness burns do not blanch and are insensate.
- Airway injury from smoke inhalation present with dyspnea and airway obstruction (airway injury may not be immediately apparent)

11.1.5 Pathophysiology

Severe burns cause multiple systemic reactions, including release of interleukins and tumor necrosis factor, resulting in immunosuppression, sepsis, multiple organ failure, and protein catabolism. Hypoxemia may result from lung injury, atelectasis, and airway edema. Extensive fluid loss from the injury and massive fluid shifts may cause hypovolemic shock.

Major burns induce a unique hemodynamic response. Cardiac output declines by up to 50% within 30 minutes in response to massive vasoconstriction, inducing a state of normovolemic hypoperfusion (burn shock).

11.1.6 Immediate Management

- Immediately administer 100% O_2 by facemask in patients with a patent airway.
- Secure the airway with an endotracheal tube. Awake fiberoptic intubation with topical anesthesia is preferred in patients with severe facial or airway injury, but other techniques may be considered.
- After intubation, maintain a high FiO_2 due to the risk of carbon monoxide (CO) toxicity.
- Begin aggressive fluid resuscitation in patients with burns greater than 15% total body surface area (TBSA).
- Crystalloid resuscitation is preferred in the first 24 hours following burn injury. · Estimate requirements according to the Parkland formula: Fluid Requirements = TBSA burned (%) × Weight (kg) × 4 mL · Estimate requirements according to the modified Brooke protocol: Fluid Requirements = TBSA burned (%) × Weight (kg) × 2 mL

- Administer 1/2 of total requirement in first 8 hours; give second half over next 16 hours.
- Fluid management is guided by urine output, CVP, or pulmonary artery pressures.

- In both protocols, an amount equal to half the volume administered in the first 24 h is infused in the second 24-h period following injury, with continued attention to maintaining urine output.
- Both formulas use urine output as a reliable indicator of fluid resuscitation, targeting (adult) urine production of 0.5–1.0 mL/kg/h as indications of adequate circulating volume. If adult urine output exceeds 1.0 mL/kg/h, the infusion is slowed.
- Th e formula for fluid resuscitation of children is the same as that for adults, but children weighing less than 30 kg should receive 5% dextrose in Ringer's lactate as their resuscitation fluid and target urine output should be 1.0 mL/kg/h.
- The target urine output for infants younger than 1 year of age is 1–2 mL/kg/h.
- If cyanide toxicity is suspected, administer sodium thiosulfate, sodium nitrate 3% solution, and hydroxy-cobalamin.
- If a chemical burn is suspected, use caution to prevent contamination of unit or staff.

11.1.7 Diagnostic Studies

- Surface area can be estimated by the Rule of Nines (Fig 11-1) · Blood electrolytes
- Arterial blood gas, including co-oximetry to determine carboxyhemoglobin level · Lactic acid level (lactic acidosis may indicate cyanide poisoning from burning plastics).

11.1.8 Subsequent Management

- Maintain normothermia. Use warming blankets, forced-air warmers, fluid warmers as necessary. Keep the room temperature as high as possible.
- Use topical antibiotics to prevent infection.
- Consider hyperbaric oxygen therapy if the patient is stable, a pressure chamber is available, and severe CO poisoning is suspected.

11.1.9 Risk Factors

Fires in the operating room due to electrocautery or lasers. "Fire resistant" plastic drapes will burn in the presence of O_2 and release toxic smoke.

11.1.10 Special Considerations

- Full-thickness burns appear white, waxy, or leatherlike and may be confused with unburnt skin. Full thickness burns do not bleed.

- Succinylcholine is generally safe to use within the first few hours after a burn, but after that must be avoided for 12 months after the burn injury.
- Resistance to nondepolarizing neuromuscular blocking agents may occur for up to 10 weeks postinjury.

11.2 HEMORRHAGE

11.2.1 Definition

Escape of blood outside the blood vessels or cardiac chambers. (loss of blood from circulation)

11.2.2 Classification

The American College of Surgeons' (ACS) identifies **four classes** of hemorrhage (Table 11-1).

Class I hemorrhage

- Class I hemorrhage is the volume of blood that can be lost without hemodynamic consequence.
- The heart rate does not change and the blood pressure does not decrease in response to losing this volume of blood.
- In most circumstances, this volume represents less than 15% of circulating blood volume.
- The typical adult has a blood volume equivalent to 70 mL/ kg. A 70-kg adult can be presumed to have nearly 5 L of circulating blood. Children are considered to have 80 mL/kg and infants, 90 mL/kg blood volume.
- Intravenous fluid is not required if the bleeding is controlled, as in brief, controlled bleeding encountered during an elective surgical procedure.

Class II hemorrhage

- Class II hemorrhage is the volume of blood, that, when lost, prompts sympathetic responses to maintain perfusion.
- This usually represents 15–30% of circulating blood volume.
- The diastolic blood pressure will increase (a reflection of vasoconstriction) and the heart rate will increase to maintain cardiac output.
- Intravenous fluid or colloid is usually indicated for blood loss of this volume.
- Transfusions may be required if bleeding continues, suggesting progression to class III hemorrhage.

Class III hemorrhage

- Class III hemorrhage represents the volume of blood loss (30–40% of circulating blood volume) that consistently results in decreased blood pressure.
- Compensatory mechanisms of vasoconstriction and tachycardia are not sufficient to maintain perfusion and meet the metabolic demands of the body.
- Metabolic acidosis will be detected on arterial blood gas analysis.
- Blood transfusions are necessary to restore tissue perfusion and provide oxygen to tissues.
- The patient may transiently respond to fluid boluses given in response to hemorrhage; however, if bleeding persists or given time for the fluid bolus to redistribute, the blood pressure will decline.
- Surgeons should be advised when this pattern persists, particularly during elective surgical cases where the development of shock is not expected.
- Class III hemorrhage may prompt an intervention such as a damage control procedure.

Class IV hemorrhage

- Class IV hemorrhage represents life-threatening hemorrhage.
- When more than 40% of circulating blood volume is lost, the patient will be unresponsive and profoundly hypotensive.
- Rapid control of bleeding and aggressive blood-based resuscitation (i.e., damage control resuscitation) will be required to prevent death.
- Patients experiencing this degree of hemorrhage will likely develop a trauma-induced coagulopathy, require massive blood transfusion, and experience a high likelihood of death.

TABLE 11-1 The ACS Advance Trauma Life Support classification of hemorrhagic shock severity

Severity of Hypovolemia	**Class 1**	**Class 2**	**Class 3**	**Class 4**
Blood loss	<750 ml (0-15%)	750-1500 ml (15-30%)	1500-2000 ml (30-40%)	>2000 ml (>40%)
Pulse rate (per minute)	<100	>100	>120	>140
Blood pressure	Normal	Normal	Decreased	Decreased

Pulse pressure (mmHg)	Normal	Decreased	Decreased	Decreased
Respiratory rate (per minute)	14-20	20-30	30-40	>40
Urine Output (ml/h)	>30	20-30	5-15	Anuric (absent urine output)
Central nervous system	+/- slightly anxious	Mildly anxious	Confused	Lethargic

11.2.3 Presentation

- Symptoms depend on the etiology (cause) of the hemorrhage. Gastrointestinal blood loss is typically painless, while blood loss associated with trauma is associated with pain due to the injury.
- Perturbation of vital signs depends on the degree of blood loss.
- Severe shock is notable for cool, moist, pallid or cyanotic skin. Mental status changes progress from anxiety to confusion to lethargy as the degree of shock progresses. Tachypnea progresses as the patient's spontaneous minute ventilation increases in order to meet increased metabolic demands. Oliguria (decreased urine output) is caused by renal hypoperfusion. · The source of hemorrhage may or may not be visible on external examination.

11.2.4 Pathophysiology

Hemorrhagic shock (that occurs in case of massive hemorrhage) is a complex spectrum of events:

- Acute massive blood loss resulting in circulatory collapse
- Ischemia-reperfusion injury
- Inflammatory and anti-inflammatory responses
- Multiple organ dysfunction

11.2.5 Immediate Management

- Intubate the trachea and initiate mechanical ventilation.
- Establish large-bore peripheral and central IV access.

- Transfuse with packed red blood and factors cells as indicated. Consider activating the massive transfusion protocol.
- Interstitial resuscitation with crystalloid solutions (e.g., lactated Ringer's solution or normal saline solution)
- If laboratory studies are not feasible, current recommendations for massive transfusion therapy in traumatic injury include minimal use of crystalloid solutions and use of plasma, PRBCs and platelets in a 1:1:1 ratio. Administer cryoprecipitate for continued microvascular bleeding.
- If time permits, identify and correct specific deficits with serial PT, PTT, INR, fibrinogen and platelet count.

11.2.6 Differential Diagnosis

Includes other causes of acute circulatory collapse:

- Hypovolemic shock of non-hemorrhagic etiology (bowel obstruction, pancreatitis)
- Vasodilatory/distributive shock
- Obstructive shock
- Cardiogenic shock

11.2.7 Diagnostic Studies

- **Gastrointestinal source:** endoscopy, interventional radiology, nuclear medicine.
- **Traumatic source:** CT scan, focused abdominal sonography in trauma (FAST), diagnostic peritoneal lavage, immediate operative intervention in hemodynamically unstable patients.
- **Obstetric source:** usually apparent on physical examination or ultrasound examination.

11.2.8 Subsequent Management

Prompt surgical control of the bleeding is of paramount importance. Continue resuscitation postoperatively or post-procedure.

The goals of resuscitation are optimization of preload, cardiac performance, blood pressure, oxygen delivery and end-organ perfusion. No single parameter is universally applicable to every patient. Therefore, multiple endpoints should be optimized:

- Clinical endpoints (heart rate, respiratory rate, blood pressure, urine output, level of consciousness, pulse pressure)
- Cardiac output measurement
- Metabolic parameters (lactate, base deficit)
- Regional perfusion (gastric tonometry, sublingual capnography, near-infrared spectroscopy)

11.2.9 Risk Factors

- **GI source:** Advanced age, comorbidities.
- **Traumatic source:** Young age (injury is the leading cause of death for persons younger than 44 years of age in the US) and lifestyle issues.
- **Obstetric source:** Postpartum hemorrhage is largely due to uterine atony

11.2.10 Prevention

Prevention of shock and resultant hypoperfusion and organ dysfunction is dependent on early control of the bleeding and appropriate resuscitation, regardless of the etiology of the hemorrhage.

Special Considerations

Massive blood transfusion is usually defined as the complete replacement of the patient's entire blood volume—10 units PRBC—in a 24-hour period.

Role of recombinant-activated factor VII (rFVIIa)**:** · Approved in the US only for bleeding associated with hemophilia.

- Significant off-label use has been noted, including for the reversal of the coagulopathy of trauma.
- Generates a thrombin peak, which in turn leads to formation of a fi brin plug.
- May not be efficient in acidosis (consider biochemical correction of acidosis prior to administration).
- Hypothermia has little effect on efficacy.

11.3 SHOCK Definition

Shock is a clinical syndrome characterized by inadequate tissue perfusion, leading to organ dysfunction.

11.3.1 Classification of Shock

Shock is characterized in various ways based on etiology (cause).

- **Hypovolemic/hemorrhagic** (acute hemorrhage of any etiology, peritonitis, pancreatitis, bowel obstruction)

  Hemodynamic parameters: low cardiac output with compensatory vasoconstriction causes high systemic vascular resistance, low central venous pressures, likely to improve with intravenous fluid boluses

  Clinical features: The patient may be hypotensive, tachycardiac and peripherally cold due to vasoconstriction.

- **Vasodilatory/distributive** (**sepsis**, adrenal insufficiency, high spinal cord injury, liver failure, anaphylaxis)

  Hemodynamic parameters: Initially compensation occurs as a supranormal cardiac output is achieved by a rise in heart rate, cardiac output may fall in later stages of septic shock and at this stage may be little response seen in hemodynamic parameters on administration of intravenous fluid boluses. *Clinical features:* Usually includes tachycardia with bounding pulses and the patient may be flushed and be warm to touch. Hypotension and pyrexia (or hypothermia) may be present.

- **Cardiogenic** (myocardial infarction, tamponade, arrhythmias)

  Hemodynamic parameters: Cardiac index less than 2.2L/min/m^2, low SvO_2 despite adequate preload and associated with signs of hypoperfusion. Echocardiogram is extremely useful in making diagnosis.

  Clinical features: Hypotension, vasoconstriction and raised JVP (jugular venous pressure)

- **Obstructive** (pulmonary embolus, pneumothorax)

  Hemodynamic parameters: low cardiac output, high systemic vascular resistance, high central venous pressures. It is unlikely to respond to fluid boluses. Echocardiogram is extremely useful in making diagnosis.

  Clinical features: Hypotension, vasoconstriction and raised JVP (jugular venous pressure), pulsus paradoxus may be seen.

- **Traumatic** (a combination of hemorrhage, ischemia, reperfusion, activation of proinflammatory cascades)

11.3.2 Immediate Management

- Intubate the trachea and initiate mechanical ventilation.
- Increase FiO_2 to maintain adequate oxygenation.
- Establish large-bore peripheral IV access or central venous access.
- Begin aggressive resuscitation with IV fluids. Transfuse with packed red blood cells (PRBCs) if indicated (i.e., for hemorrhagic shock)
- Promptly identify and correct coagulopathy, thrombocytopenia, and platelet dysfunction
- Support blood pressure with vasopressors if indicated (septic shock).
- Consider epinephrine infusion starting at 0.03–0.05 mcg/kg/min.
- Begin broad-spectrum empiric or culture-directed antibiotic therapy (septic shock)

11.3.3 Subsequent Management

- Communication with the surgical team is essential.
- Anesthetic management should focus on maintaining oxygenation and perfusion. Induction agents are chosen based on the patient's volume status and medical condition. Consider etomidate (0.3 mg/kg IV) if the patient is hemodynamically unstable. Sympathomimetic agents (e.g., ketamine) may cause profound hypotension in critically ill patients who have high circulating levels of endogenous catecholamines (hypovolemia from sepsis or hemorrhage; myocardial dysfunction; pain). Hypotension is exacerbated by the transition from spontaneous ventilation to positive pressure mechanical ventilation.
- Instability during the surgical procedure is most likely due to surgical manipulation and the patient's underlying pathophysiology. A narcotic based technique is associated with less vasodilation and negative inotropy and may be appropriate for hemodynamically unstable patients and those with underlying myocardial dysfunction. Pay close attention to the patient's volume status, body temperature (fluid warmer, forced hot air blanket, elevated room temperature, humidification of the mechanical ventilation circuit), and prevention of positioning injuries.

11.3.4 Prevention

Early diagnosis (either by physical examination) prior to development of shock may prevent significant hypoperfusion, associated end organ dysfunction, complications, and death.

Chapter 12

LOCAL ANESTHETIC TOXICITY

12.1 INTRODUCTION

Local anesthetics (LA) are drugs commonly used in anesthesia for the control of pain and/or as the primary anesthetic for surgical procedures. When taking the proper safety precautions, these drugs are generally very safe. Despite this, adverse events from local anesthetic administration do occur. Local anesthetic toxicity is predominantly encountered in situations where large doses of LA are commonly administered (i.e., regional nerve blocks and epidurals). Toxicity from LA occurs from being improperly dosed at the intended site of administration or from unintentional vascular injection directly into the bloodstream. Symptoms of excessive systemic doses range from mild to life-threatening central nervous system and cardiac disturbance.

Local anesthetic systemic toxicity (LAST), albeit rare, can be a devastating complication leading to brain injury and death. Early identification and treatment can significantly reduce the morbidity and mortality of LAST; therefore, it is an emergency that requires multiple team members familiar with its management. The anesthesia technician is a valuable team member who should be able to anticipate, locate, and recognize the special medications and equipment needed to treat LAST. The technician should also be prepared to assist in resuscitation, from the specialized protocols of LAST to the basics of prolonged CPR, in the event of a cardiac arrest.

12.2 PREPARATION AND PREVENTION

Preparation is tremendously important for managing LAST. Adequate planning includes checking and stocking emergency medications and equipment for airway management and IV access. These items should be readily available in locations (including mobile carts) where LA are commonly administered. A common practice is to assemble a Local Anesthetic Toxicity Kit (Table 12.1) for ease of administration. Regularly checking equipment and replacing expired medications is imperative.

Table 12.1. Sample Contents of Local Anesthetic Toxicity Kit

Medication	Quantity
Amiodarone 50 mg/mL—3 mL	2
Intralipid 20%—250 mL	2
Propofol 10 mg/mL—20 mL	1
Succinylcholine 20 mg/mL—10 mL	1
Epinephrine 10 µg/mL—10 mL	1
Epinephrine 100 µg/mL—10 mL	1
Etomidate 2 mg/mL—10 mL	1

Primary prevention is critical: many strategies exist.

- Patients are monitored with American Society of Anesthesia (ASA) standard monitors during and immediately after placement.
- The lowest dose of local anesthetic needed for anesthesia or pain relief is used.
- Techniques for careful identification that the needle or catheter is not in a blood vessel may be possible (perhaps nerve stimulation or ultrasound).
- Incremental injection of 3-5 mL of local anesthetic at a time, with careful aspiration in between each injection looking for blood, and careful observation of the patient to ask about symptoms of local anesthetic toxicity.
- Epinephrine, which causes an increase in heart rate and blood pressure if injected intravascularly, can be added to the local anesthetic solution.
- Local anesthetics with lower cardiotoxicity can be used, such as levobupivacaine or ropivacaine.

12.3 DETECTION Monitors

The proper monitoring modalities are important for the detection of LAST. The recommended monitors consist of the following:

- Standard ASA monitors
- Monitor the patient during and after injection of local anesthetic (toxicity can present as late as 30 minutes post injection)
- Frequent communication with the patient to elicit symptoms of toxicity

12.4 Symptoms

Classically, the presenting symptoms of LAST have been described as neurologic, followed by cardiac manifestations. Nonetheless, there is a small portion of patients who present with isolated cardiovascular signs.

12.5 Neurological toxicity

The typical progression of symptoms is of CNS excitement which contain:

- Circumoral numbness
- Tongue paresthesia
- Agitation
- Tinnitus
- metallic taste
- abrupt psychological change

Excitement is followed by seizures then CNS depression which may lead to

- coma
- respiratory arrest

12.6 Cardiovascular toxicity

Cardiovascular toxicity usually manifests later in the continuum.

Initially, cardiac toxicity can be hyperdynamic and the symotoms include:

- hypertension
- tachycardia
- ventricular arrhythmias

This is followed by cardiac depression and the symptoms include

- hypotension
- bradycardia

- conduction block
- asystole

Simultaneous presentation of CNS and cardiac toxicity can occur, and vigilance for atypical presentations must be practiced. Some LA, notably bupivacaine, are more likely to present with cardiovascular toxicity alone. Midazolam can also blunt the CNS toxicity of LA, so that cardiovascular toxicity may be the presenting sign of LAST.

Timing of symptoms can be variable. They can range from immediate, as expected with direct vascular injection, to delayed (>15 minutes after injection).

12.7 TREATMENT

The treatment of LAST includes

- airway management
- circulatory support
- limiting toxic effects of the local anesthetic.

12.7.1 Airway Management

- Airway management is of utmost importance, because prevention of hypoxia and acidosis may prevent further progression or even limit the severity of symptoms.
- These maneuvers include oxygen supplementation, bag-mask ventilation, and/or endotracheal intubation depending on the clinical situation.
- Seizures should be controlled early because they worsen hypoxia and acidosis. Benzodiazepines are ideal for the treatment of seizures because they have less potential for circulatory depression than other hypnotics (i.e., propofol).
- Help should be called for as soon as possible, and preparations should be made to provide Basic Life Support (BLS) and Advanced Cardiac Life Support (ACLS)
- Effective chest compressions are critical as these patients are often in a non-perfusing rhythm, and the arrhythmias associated with LAST can be resistant to standard ACLS treatment.
- Rescuers should rotate so that compressions remain effective. Resuscitation that is not responsive to standard treatment should include cardiopulmonary bypass (CPB), just as is initiated in preparation for heart surgery. It is a bridging therapy until the local anesthetic has had time to clear. CPB can take significant time to initiate, so it is reasonable to alert the closest facility capable of providing CPB as early as possible.

In the event of cardiac arrest, it is important to recall that ACLS is modified in LAST. ACLS is a broad protocol for any cause of cardiac arrest, primarily researched in the out of hospital setting. In LAST, the cause of the cardiac arrest is known, and rapid, cause-

specific treatment should start right away; treatments known to be harmful in this setting should be withheld. For example, lidocaine (a local anesthetic) is a treatment for arrest caused by cardiac ischemia and is a part of standard ACLS—it would be the wrong treatment for someone whose arrhythmia was caused by too much local anesthetic! (Table 12.2). The administration of lipid emulsion is also essential to successful ACLS in LAST and should be considered at the first symptoms of LAST after airway management has occurred.

Table 12.2. ACLS Modifications for Treatment of LAST

Reduced doses of epinephrine
Avoid vasopressin
Avoid calcium channel blockers and β-adrenergic receptor blockers
Treat ventricular arrhythmias with amiodarone and avoid lidocaine

12.7.2 Lipid Emulsion Therapy

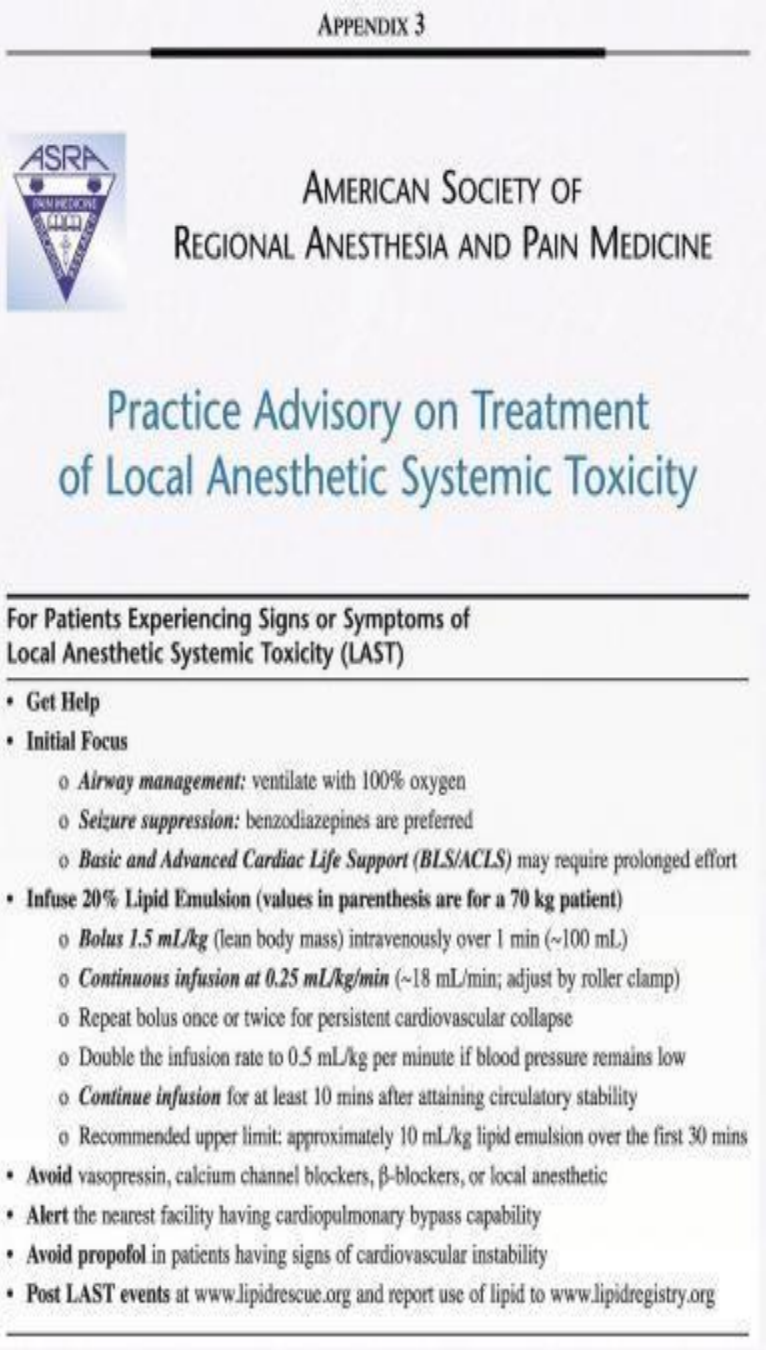
A 20% lipid infusion is an intravenous lipid emulsion that has been demonstrated to be effective in restoring circulation after LAST has occurred. Lipid emulsion is a critical intervention for LAST and an essential modification to standard ACLS and should be given as early as possible. It's mechanism is thought to scavenge local anesthetic from high blood flow organs, increases cardiac performance and act as a cardioprotective agent. Lipid emulsion is a low-risk treatment for LAST and should be readily available where local anesthetics are regularly administered (i.e., OR, regional or obstetric anesthesia carts). Propofol contains a small amount of lipid compared to 20% lipid emulsion therapy and therefore cannot serve as a substitute. Lipid emulsion therapy should be continued despite the restoration of circulation because cardiovascular depression can persist or recur after treatment.

12.8 SUMMARY

Local anesthetic systemic toxicity treatment is an uncommon but serious emergency. The key to management includes appropriate preparation. The role of the anesthesia technician is therefore critical in saving lives of otherwise low-risk patients for

routine, often ambulatory procedures. Preparing the anesthetizing areas with the correct drugs and equipment can make the difference between life and death. When LAST occurs, prompt treatment relies on symptom detection with the proper monitors, and the prompt availability of an intralipid and resuscitation kit. The presentation can vary based on dose and route of administration as well as patient factors. The symptoms range from neurologic to cardiovascular effects with variable severity. Treatment must follow quickly, which involves airway management, control of seizures, and cardiac support. In the event of cardiac arrest, ACLS protocols must be modified, and lipid emulsion infusions can be lifesaving. The anesthesia technician must be intimately familiar with the management of LAST in order to effectively participate as a member of the resuscitative team.

In 2008, the American Society of Regional Anesthesia and Pain Medicine (ASRA) convened its second practice advisory panel on LAST. The 2010 executive summary of that panel's findings can be downloaded for free from the ASRA Web site (www.asra.com). A key component of the ASRA practice advisory was the creation of a treatment checklist, a copy of which can also be obtained from the ASRA Web site.



APPENDIX 3

AMERICAN SOCIETY OF
REGIONAL ANESTHESIA AND PAIN MEDICINE

Practice Advisory on Treatment of Local Anesthetic Systemic Toxicity

For Patients Experiencing Signs or Symptoms of Local Anesthetic Systemic Toxicity (LAST)

- **Get Help**
- **Initial Focus**
  - ***Airway management:*** ventilate with 100% oxygen
  - ***Seizure suppression:*** benzodiazepines are preferred
  - ***Basic and Advanced Cardiac Life Support (BLS/ACLS)*** may require prolonged effort
- **Infuse 20% Lipid Emulsion (values in parenthesis are for a 70 kg patient)**
  - ***Bolus 1.5 mL/kg*** (lean body mass) intravenously over 1 min (~100 mL)
  - ***Continuous infusion at 0.25 mL/kg/min*** (~18 mL/min; adjust by roller clamp)
  - Repeat bolus once or twice for persistent cardiovascular collapse
  - Double the infusion rate to 0.5 mL/kg per minute if blood pressure remains low
  - ***Continue infusion*** for at least 10 mins after attaining circulatory stability
  - Recommended upper limit: approximately 10 mL/kg lipid emulsion over the first 30 mins
- **Avoid** vasopressin, calcium channel blockers, β-blockers, or local anesthetic
- **Alert** the nearest facility having cardiopulmonary bypass capability
- **Avoid propofol** in patients having signs of cardiovascular instability
- **Post LAST events** at www.lipidrescue.org and report use of lipid to www.lipidregistry.org

Fig. American Society of Regional Anesthesia and Pain Medicine Practice Advisory for Local Anesthetic and Regional Toxicity.

MISCELLANEOUS

Setting up a sterile tray

- Set a Mayo stand and tray directly to the right of the scrub nurse or scrub physician who will be assisting in the surgery.
- Put on sterile gloves while setting up for any surgical procedure
- Cover the Mayo stand and instrument tray with a sterile drape by carefully opening each side . Avoid touching the inside of the surgical drape where the instruments will be placed.
- Open carefully the basic sterile instrument setup for the surgical procedure that is being done . Once the package is opened you will need to re-glove in order to place the individual instruments on the tray.
- Look at expiration dates, sterilization indicators and packaging before opening and placing any injections, surgical instruments or equipment needed for procedure
- Place the instruments carefully, handle first on the sterile drape using proper sterile technique. Make sure that nothing contaminates the sterile field or the sterile instruments.

Setting up arterial and CVP lines

Equipment

- Pressure bag able to inflate to 300 mmHg
- 500 mls Saline solution with iv additives label:
  Consider adding 500 iu heparin (document in chart if given)
- 2 ml syringe and green needle
- 1000 units refrigerated heparin
- Two pressure modules with pressure cables
- Single or dual transducer set (comes complete with giving set) with transducer holder (if available)
- Suitable cardiac monitor with invasive pressure monitoring
- Gloves
- Narrow width tape

Procedure

- Gather equipment together
- Test integrity of pressure bag (check for leaks, breaks, faulty dial…)
- Insert the pressure modules into the cardiac monitor by clicking them in place (for CVP monitoring, both modules will still be required)
- Apply gloves; check and draw up the heparin (complete an iv additives label)
- Add the heparin to the saline solution and shake well
- Attach the transducer set and prime
- Connect the saline to the pressure bag
- Ensure the saline rests in the centre of the pressure bag
- Inflate the pressure bag as it rests on the counter
- Inflate to 300 mmHg
- Hang the pressure bag up
- Take care to close all ports open to air when priming lines
- 'Squeeze' the fluid through the lines (using the blue and red flush valves)

Arterial line

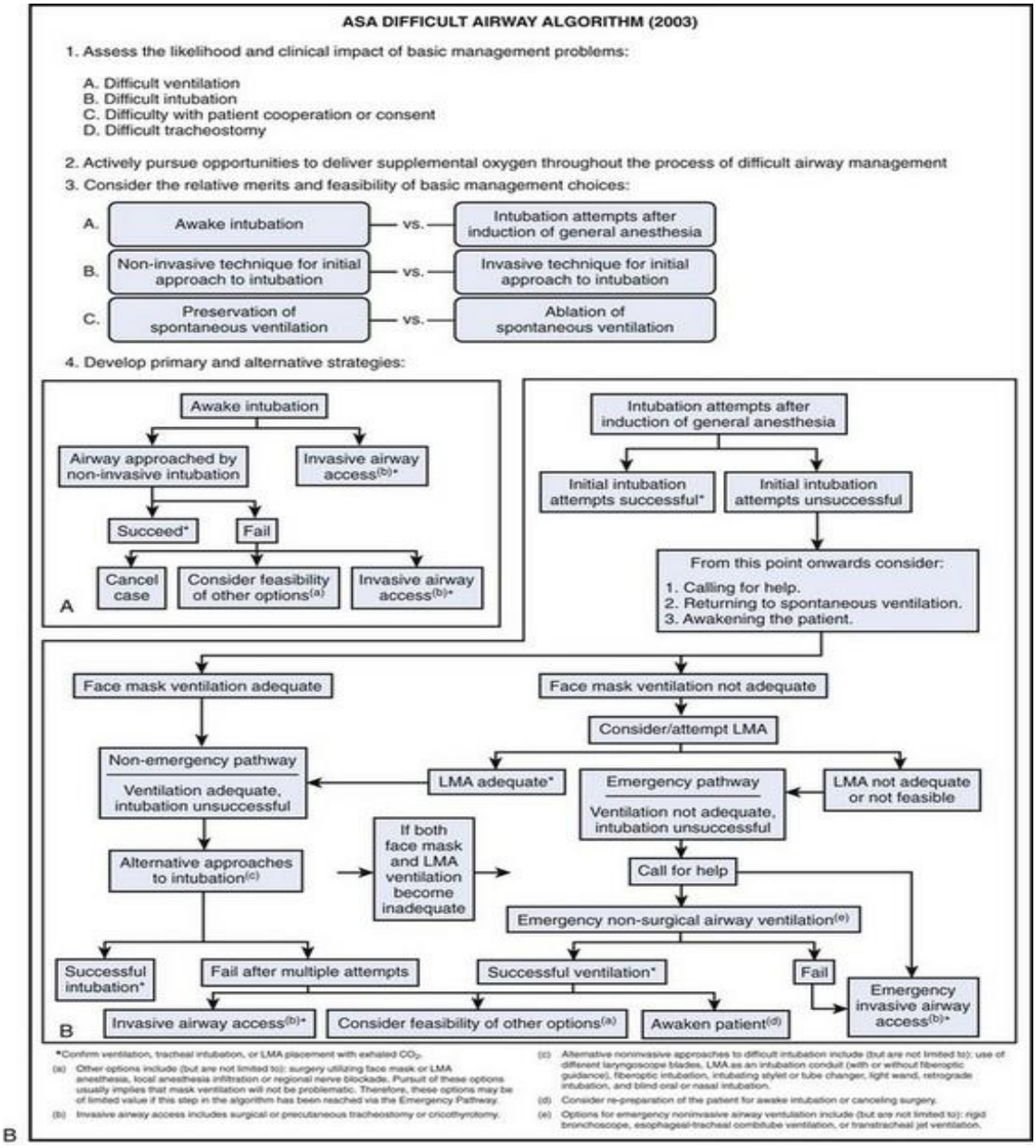
- Connect the arterial tubing (red) to the patient’s arterial access
- Connect the white pressure cable inserted in the first pressure module to the white transducer cable
- To zero, turn the white tap ‘off’ to the patient, i.e., in the direction of the patient, and open the orange port to air

- Press zero on the arterial pressure module. Hold it down until the monitor states ‘arterial zero done’
- Turn the white tap ‘off’ to air and close the orange port
- Ensure arterial access site remains visible at all times
- Using tape and a red pen, label the arterial line clearly with the letters 'ART'

CVP line

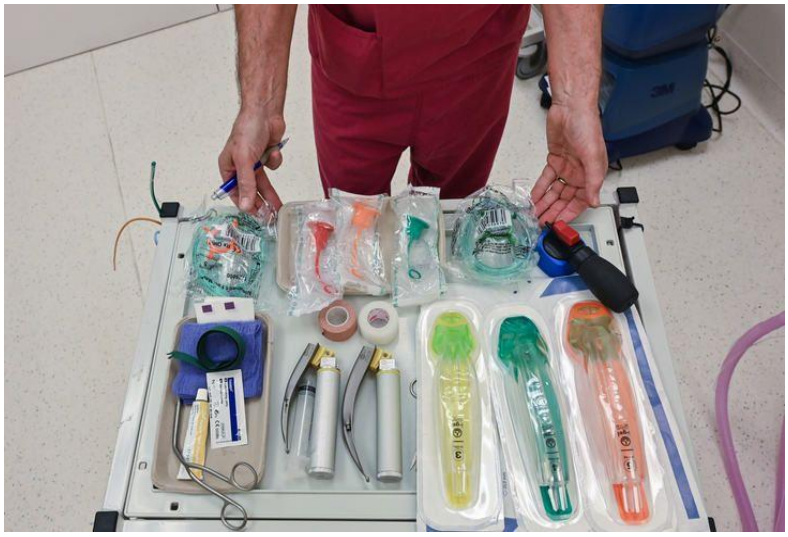
- Ensure CVP line stitched in, flushed through and secured with tegaderm
- Connect the white CVP connector to monitor
- Connect the grey pressure cable inserted in the second pressure module to the white transducer cable
- To zero, turn the white tap ‘off’ to the patient and open the orange port to air
- Press zero on the CVP pressure module. Hold it down until the monitor states ‘CVP zero done’
- Turn the white tap ‘off’ to air and close the orange port
- Secure the ‘red’ and ‘blue’ flush valves at the level of the patient’s right atrium
- Tape them, if necessary to the patient's mid upper arm, at the level of the heart
- Re-zero
- Re-zero the arterial and/or central venous pressures if:
- The equipment becomes disconnected in any way
- The patient’s position alters
- Monitoring is transferred to a different machine, i.e., for transfers

Difficult Airway Algorithm



Difficult airway trolley/ cart

An anesthesia technician is a healthcare professional who assists anesthesiologists in administering anesthesia to patients. They are responsible for preparing and maintaining anesthesia equipment, monitoring patients' vital signs, and assisting with airway management. In the context of difficult airway management, the American Society of Anesthesiologists (ASA) has published practice guidelines that provide recommendations for the management of difficult airways. These guidelines suggest that the anesthesia technician should be prepared to assist with airway management when a difficult airway is encountered. The technician should also ensure that the necessary equipment for managing a difficult airway is available .



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