Anesthesia and Pain Relief in Trauma Patients

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6.1 Fundamentals

6.1.1 Introduction and General Pathophysiology

Analgesia and anesthesia have long posed a medical challenge that goes far beyond the administration of analgesics and anesthesia. The basic analgesics of *personal attention and positioning* should be used by everyone, including physicians.

Pain does not cause mental suffering alone; beyond that, it has a negative effect on the body, especially in the respiratory and cardiocirculatory systems, that can delay healing. When pain leads to shallow breathing and the inability to cough up phlegm, atelectasia can develop and lead to pneumonia, while activation of the sympatho-adrenergic system with tachycardia, increase in blood pressure, peripheral vasoconstriction, and greater myocardial oxygen demand can cause myocardial ischemia.

Concern is often expressed that analgesia could obscure the diagnosis; however, if the medical history is documented carefully and symptoms are ordered systematically, this concern is unfounded and should be relegated to the past.

The hope that the postoperative stress reaction could be clearly reduced with suitable pain therapy has not been fulfilled. It has been found that pain therapy that is subjectively judged to be excellent is not necessarily related to a decrease in endocrine stress parameters [1]. At least in the immediate postoperative period, and surely in the preclinical phase as well, pain is not the decisive stressor, and freedom from pain is not to be equated with freedom from stress. There are apparently stressors other than pain that dominate. These humoral and neural stressors originate in traumatized tissue with the liberation of a number of mediator substances that cannot be neutralized even with the best peripheral or central blockade; this nonetheless does not cast any doubt on the value of pain therapy.

Analgesia does not require any further justification and can considerably improve the patient's respiratory and cardiovascular situation as a whole. In contrast, anesthesia is not a simple entity and is only indicated when, upon careful review, the patient's condition is seen to absolutely demand it.

6.1.2 Endocrine Stress Reaction and Stress Concepts

Current stress concepts are based on the work of Claude Bernard, Walter Cannon, and Hans Selye. Claude Bernard (1813–1873) coined the term *milieu*

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Fig. 6.1 Stress and stress reaction. *ACTH* adrenocorticotropic hormone, *ADH* antidiuretic hormone
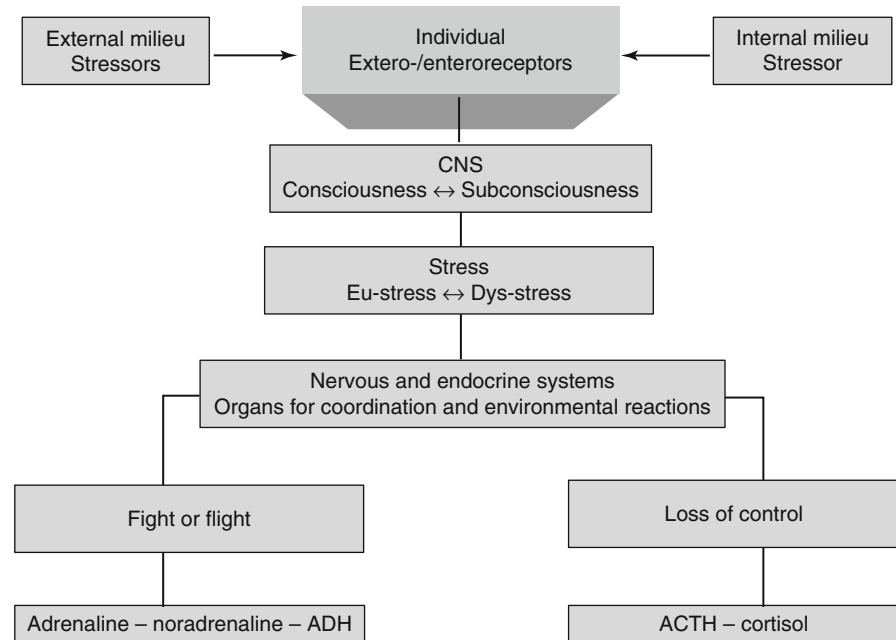
intérieur. Walter Cannon (1871–1945) provided the definition of homeostasis and developed the concept of "fight or flight," with emphasis on the role of the catecholamines [2]. Hans Selye (1907–1982) characterized stress as a "general adaptation syndrome," distinguished between "eu-stress" and "dys-stress," and emphasized the role of the glucocorticoids [3, 4].

Selye separated the stress reaction into three sequential phases:
- The alarm reaction (corresponding to Cannon's "fight or flight")
- Phase of resistance
- Phase of exhaustion

J. Henry took the endocrine-metabolic aspects into consideration and saw a dual stress response with two main components [5] that were not necessarily sequential but could run a parallel course (Fig. 6.1); this response is described as follows:
- Activation of the sympathoadrenergic system with release of catecholamines is an immediate and active response to the stressor; release of the antidiuretic hormone is also a component of the immediate stress response.
- In contrast, activation of the hypothalamus-hypophysis-adrenal cortex axis focuses on tolerance and adaptation and, with a protracted course, is to be seen as a sign of loss of control in the confrontation with the stressor.

Later concepts emphasize organic responses to interior and exterior stressors that serve to maintain homeostasis [6]. A distinction is made between homeostatic systems in the narrow sense (e.g., oxygen concentration in the blood, body temperature) that allow changes only within very narrow limits and so-called allostatic systems that allow a wider range of responses. This is described as "maintaining stability through change (allostasis)" [7]. Allostasis as a basic function of bodily stress modulation thus reacts quickly via the sympathoadrenergic system and slowly via the hypothalamus-hypophysis-adrenal cortex axis.

> Afferent neuronal stimuli and humoral factors are of great importance in modulating the surgical stress reaction.

The neural facilitation of the stress response to tissue trauma is explained by afferent impulses from nociceptive, somatosensitive, and sympathetic pathways, whereby the relative importance of the somatosensitive nervous system in comparison with the sympathetic nervous system remains unclear. The nociceptive afferents modify the neuroendocrine function of the hypothalamus and trigger the endocrine surgical stress response. A stress reaction can, however, also be induced by physiological factors (e.g., anxiety).

Humoral mediators of the stress response include prostaglandins, bradykinin, substance P, histamine, and serotonin, which are released upon tissue trauma. Among the mediators secreted by macrophages, interleukin-1 and tumor necrosis factor-α are especially important [8].

> For all trauma patients – as for all critically ill patients – the effects of analgesia and anesthesia on the stress reaction must be borne in mind. The acutely injured patient is in the "fight or flight" stage, directly confronted with the trauma-induced stressors. Because the life-preserving stress reaction depends on catecholamines, the effect of anesthetics on the sympathoadrenergic system demands special attention. The goal of anesthesia and analgosedation is to preserve the stress reaction without suppression or overactivity.

6.1.3   Basic Concepts in Anesthesiology

6.1.3.1 Definitions

The following definitions will be used [9]:

- *Anesthesia* is the Greek-Neo Latin word for insensitivity or lack of sensation and applies not only to the *state* of an iatrogenically induced reversible insensitivity that aims to make an intervention possible, but also to a medical *procedure* to induce such a state.
- *General anesthesia* affects the entire organism, while *local anesthesia* is limited to particular areas.
- *Narcosis* (Greek: torpor) is general anesthesia with central exclusion of pain and consciousness induced by anesthetics. "Narcosis" is synonymous with "general anesthesia" and extends the term anesthesia to cover exclusion of consciousness or hypnosis while simultaneously disallowing the term partial narcosis that is sometimes used (e.g., for spinal anesthesia).
- "Narcosis" expands the term "anesthesia," while "analgesia" limits it to the pain component by excluding sensitivity to position, touch, and temperature. *Analgesia* eliminates sensitivity to pain, producing painlessness. By definition, a properly "narcotized" patient cannot sense pain, therefore the term "analgesia" in the context of narcosis is problematic. Because a lack of specific inhibition of the nociceptive system in a narcotized patient is mentally and physically noxious, "antinociception" for specific blockade of
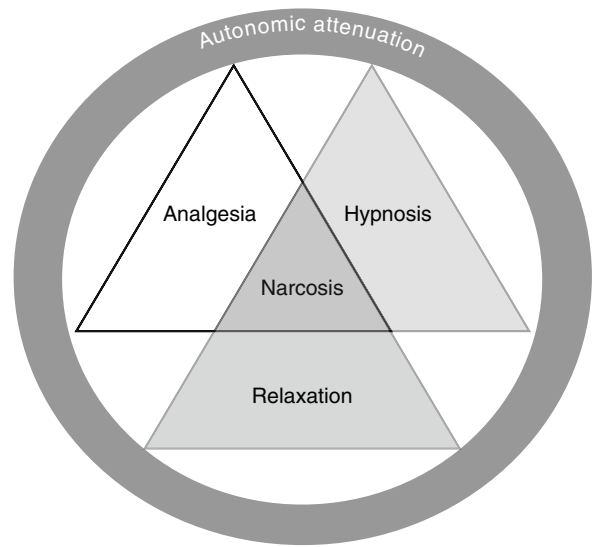
Fig. 6.2 Components of anesthesia – the specific effect sought should always be borne in mind

the nociceptive system [10] is an essential component of adequate general anesthesia.
- Local anesthesia, or more precisely, local insensitivity, with its sequence of sympathetic, sensory, and when necessary, motor blockade, is more than analgesia and thus a form of anesthesia. Local anesthesia implies regional exclusion of pain in the area of the nerve endings or nerve tracts without affecting consciousness.

6.1.3.2 Components of Anesthesia

Traditionally, anesthesia comprises three main components (Fig. 6.2):
- Analgesia as *exclusion of pain* only
- Hypnosis as *loss of consciousness*
- *Attenuation of autonomic nervous activity*

Muscle relaxation is a further component that is usually required for surgery. A clear distinction cannot be made between analgesia and hypnosis, as the respective medications interact. Analgesia and hypnosis have attenuation of autonomic nervous activity in common as an additional effect.

6.1.4   Anesthetics and Anesthetic Procedures

Anesthetics, in the broader sense, are all those medications that are specifically used to induce general or local anesthesia. The substances in their main groups

Fig. 6.3 Schematic of anesthesia techniques
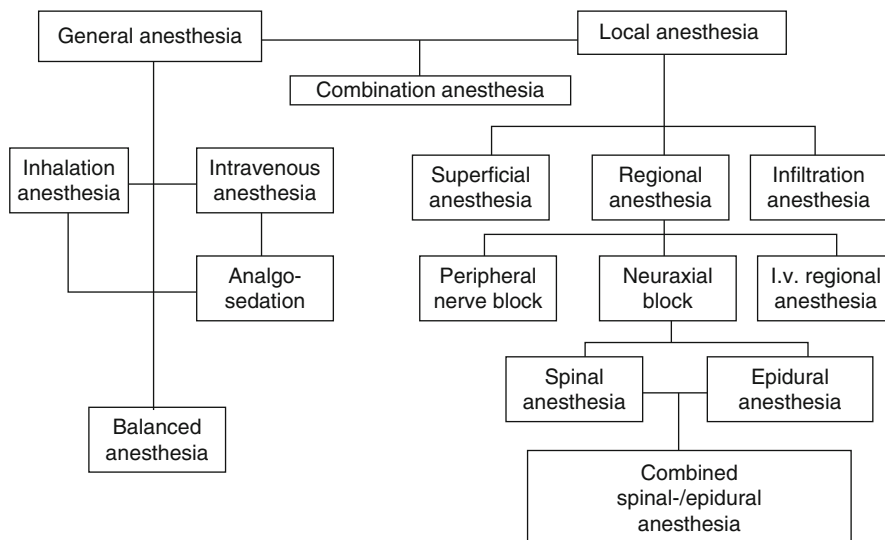
of *general anesthetics* and *local anesthetics* are classified according to their mode of application:

- Local anesthetics are applied to the immediate vicinity of their target area.
- General anesthetics are classified according to their mode of application as inhalation and intravenous anesthetics.
- Inhalation anesthetics include volatile substances such as sevoflurane or desflurane and the gases nitrous oxide (laughing gas, N_2O) and xenon.
- Intravenous, better described as injectable anesthetics, because some may be administered intramuscularly, are the hypnotics, sedatives and analgesics, and ketamine.
- Because of their lack of analgesic and hypnotic effect, muscle relaxants can only be considered as anesthetics in the widest sense.

The classification of anesthetic procedures [9] depends on their mode of administration, as shown in Fig. 6.3.

6.2 Preclinical Analgesia and Anesthesia

6.2.1 Medications

6.2.1.1 General

For medical and logistic reasons, only a few tried and true medications are suitable for the preclinical situation (Table 6.1). In regard to the administration of anesthesia, the concrete effect (analgesia or sedation) that is desired of the respective medication must be taken into account (Fig. 6.2).

> During embryogenesis and the early fetal period (until about the 16th week of gestation), analgesics and anesthetics are contraindicated except in special cases.

- Metamizole is contraindicated in the first and third trimesters; in the second trimester it is limited to exceptional cases. Inhibition of prostaglandin can lead to early closure of ductus arteriosus in the newborn.
- Morphine, fentanyl, and esketamine (the latter only in high doses >1 mg/kg body weight (BW)) administered to the mother shortly before birth can cause respiratory depression in the newborn.
- Midozalam administered to the mother in high doses shortly before birth can cause "floppy infant" syndrome with flaccid muscle tone and respiratory depression.
- Use of analgesics and anesthetics during lactation is generally not at issue because the mother's illness will usually preclude nursing.

> The abbreviation RAD used below indicates the recommended dose for an adult weighing about 75 kg, but the dose should be adjusted to the individual patient.

Table 6.1 Medications for analgesia, sedation, and anesthesia

Medication	Indications	Dosage	RAD
Metamizole	Analgesic for mild to moderate pain	6–12.5 (–30) mg/kg BW IV	0.5–1.0 (–2.5) g IV
Morphine	Analgesic for severe pain	0.05–0.1 mg/kg BW IV	4–8 mg IV
Fentanyl	Highly potent analgesic for TIVA with ventilation	Analgesia with spontaneous respiration (*cave* respiratory depression) 0.6–1.8 µg/kg BW IV	0.05–0.15 mg IV
		For analgosedation 3–15 µg/kg BW/h	up to 1.0 mg/h
		Initial TIVA 1.25–3.75 µg/kg BW IV	0.1–0.3 mg IV
Esketamine	Analgesic and anesthetic	Analgesia 0.125–0.25 mg/kg BW IV (if needed 0.25–0.5 mg/kg BW i.m.)	10–20 mg IV (if necessary 20–40 mg i.m.)
		Analgosedation with spontaneous respiration 0.3–0.5 mg/kg BW/h	25–40 mg/h IV
		Analgosedation of ventilated patients 0.3–1.5 mg/kg BW/h	25–125 mg/h
		Anesthesia 0.5–1.0 mg/kg BW IV (if necessary 2.5 mg/kg BW IM)	40–80 mg IV (if necessary 200 mg IM)
Etomidate	Induction for generally stable patients	0.25–0.5 mg/kg BW IV	20–40 mg IV
Midazolam	Standard sedative for sedation, analgosedation and TIVA	Sedation 0.03–0.1 mg/kg BW IV	Boluses of 1–2 mg
		Induction 0.1–0.2 mg/kg BW IV	7.5–15 mg IV
Succinylcholine	Depolarizing muscle relaxant for rapid induction	1.0–1.5 mg/kg BW IV	100 mg IV

RAD recommended dose for an adult of about 75 kg body weight (BW), that should be adjusted for each individual case, *TIVA* total intravenous anesthesia

6.2.1.2 Metamizole

Metamizole is a pyrazolone derivative for analgesia for mild to moderate pain (e.g., soft-tissue injuries) with antipyretic and anti-inflammatory properties.

The effect begins within a few minutes and lasts for about 2 h. Side effects (SE) are decrease in blood pressure and tachycardia (that can lead to shock) when administered too quickly intravenously (IV).
- The single analgesic dose is 6–12.5 mg/kg BW IV (RAD 0.5–1.0 g).
- For severe pain, up to 30 mg/kg BW (2.5 g) is injected IV.

6.2.1.3 Morphine

Morphine is the standard analgesic for extremely severe pain.

The effect sets in within minutes and lasts about 4 h. SEs are respiratory depression, nausea, vomiting,

and release of histamine with hypotonia. Naloxone is available as a specific antagonist.
- The individual dose is 0.05–0.1 mg/kg BW IV (RAD 4–8 mg).

6.2.1.4 Fentanyl

Fentanyl is a highly potent synthetic µ-receptor antagonist for profound analgesia with total IV anesthesia (TIVA) and airway management. It can also be used for analgesia with spontaneous breathing; however, that is off-label use.

It is effective within 2–3 min and remains so for 20–40 min. The most important SE is a potentially life threatening respiratory depression. Naxolone is the specific antagonist.
- For analgesia with spontaneous breathing, 0.6–1.8 µg/kg BW (RAD 0.05–0.15 mg) are administered IV in selected cases (*cave* respiratory depression, if necessary use breathing on command or a breathing mask).
- Depending on the patient's general condition, 1.25–3.75 µg/kg BW (RAD 0.1–0.3 mg) is injected IV.

6.2.1.5 Esketamine and Ketamine

Esketamine is the dextrorotatory isomer of the ketamine racemate. It has double the analgesic and anesthetic potency of the racemic mixture as well as a higher elimination rate with shorter time to awakening. The dosages for esketamine are given below; if the ketamine racemate is used, the dose should be doubled.

> Esketamine is an anesthetic with a strong analgesic and weak hypnotic effect. Depending on the dosage, it can be used for analgesia and analgosedation as well as anesthesia that, because of its typical effects, is termed dissociative anesthesia.

Centrally modulated activation of the sympathetic nervous system by the substance increases blood pressure, heart rate (HR), and myocardial oxygen demand. Spontaneous breathing is usually maintained. After IV administration and after a circulation time, the analgesic effect sets in and persists for some 15 min. After intramuscular (IM) injection, the effect commences within 2–5 min and lasts for up to 30 min.

No relevant increase in intracranial pressure (ICP) is to be expected with controlled normoventilation. SE include dreams and nightmares and hypersalivation. It is contraindicated for patients with hypertension, coronary heart disease, preeclampsia, and eclampsia. Dream reactions and excessive circulatory effects can be diminished or avoided with combination with midazolam; atropine combats hypersalivation.

- For *analgesia*, 0.125–0.25 mg/kg esketamine (RAD 10–20 mg) is injected IV; this can be followed by half of the initial dose when necessary. If there is no venous access, 0.25–0.5 mg/kg BW (RAD 20–40 mg) can be injected IM.
- For *analgosedation with spontaneous breathing*, 0.3–0.5 mg/kg BW/h esketamine (RAD 25–40 mg/h) is injected IV by pump, or an IV drip with 0.5 mg esketamine/ml (1 ml = 20 drops) can be used. Dosage depends on the effect. For sedation, midazolam is administered in a lower fractionated dose (1–2 mg) IV, or alternatively, 0.03 mg/kg BW/h (RAD 2.5 mg/h) is administered by injection pump. Before the IV drip is started, adequate analgesia and sedation should be secured with IV boluses of esketamine and when needed, midazolam.

- For *monoanesthesia* for rapid sequence induction (RSI) of patients in a poor general condition (e.g., in shock) 0.5 mg/kg BW (RAD 40–50 mg) is injected IV.
- For *TIVA*, esketamine is typically combined with midazolam. For RSI as is often necessary for emergency medical service, depending on the patient's condition, first up to 0.1 mg/kg BW midazolam (RAD 8 mg) is injected IV, followed by 0.5–1.0 mg/kg BW esketamine (RAD 40–80 mg) and when necessary, 1.5 mg/kg BW succinylcholine (RAD 100 mg). When necessary, further injection of half of the initial dose of esketamine can be administered, but further midazolam injections are seldom required.
- For *IM induction* – as a last resort when there is no venous access – about 2.5 mg/kg BW esketamine (RAD 200 mg) is injected along with 0.01 mg/kg BW atropine (up to 0.5 mg). Anesthesia sets in within a few minutes and a venous access should be established immediately.
- For management of special cases of uncooperative patients, 1.25–2.5 mg/kg BW esketamine (RAD 100–200 mg) is injected IM to enable venipuncture and further measures.

6.2.1.6 Etomidate

> Etomidate is an induction hypnotic without analgesic potency or significant effect on the circulatory system. It is used for induction in generally stable patients.

- For induction, depending on the patient's general condition 0.25–0.5 mg/kg BW (RAD 20–40 mg) is injected IV. Typically, fentanyl or esketamine is also needed.

> Etomidate suppresses cortisol (hydrocortisone) synthesis in the adrenal cortex for approximately 1 day and a single dose increases mortality in septic patients.

6.2.1.7 Midazolam

> Midazolam is a benzodiazepine with sedating, anxiolytic, and amnestic effects. It is used alone as a sedative or in combination with esketamine or fentanyl for TIVA.

The effect is seen quickly and usually lasts about 30 min. There can be severe respiratory depression in elderly patients and those in poor general condition. With careful dosage, cardiovascular SEs should not be expected. Flumazenil is the specific antagonist.

- For *sedation*, a fractionated IV dose of small boluses of 1–2 mg is administered until the patient slurs his or her speech or is sleepily responsive (total dose 0.03–0.1 mg/kg BW).
- For *induction*, 0.1–0.2 mg/kg BW (RAD 7.5–15 mg) is injected combined with esketamine or fentanyl.

6.2.1.8 Succinylcholine

Succinylcholine is a depolarizing muscle relaxant that has the shortest onset time (30–45 s) and the shortest action time of all muscle relaxants.

SEs are sinus bradycardia, other arrhythmias (especially in children), triggering of malignant hyperthermia (MH), and increased serum potassium. Because of the danger of hyperkalemia, it is contraindicated in patients with neuromuscular diseases (particularly extensive paralysis), longer immobilization, and predisposition to MH.

- Dosage is 1.0–1.5 mg/kg BW IV (RAD 100 mg).

6.2.1.9 Vecuronium

Vecuronium is a moderately long-acting, nondepolarizing muscle relaxant without any particular SEs that is easily stored in its dry form.

- The initial dose is 0.1 mg/kg BW IV (RAD 8 mg) followed by 0.025 mg/kg BW IV (RAD 2 mg) if necessary.

Other moderately long-acting relaxants are also suitable. In all, the indications for these substances are limited (e.g., prevention of bucking and pressing in spite of adequate anesthesia with head injury).

6.2.2 Practical Procedure

6.2.2.1 General Aspects of Analgesia and Anesthesia

In the preclinical situation, analgesia or anesthesia should be applied conservatively and only after careful consideration. Anxious nontreatment and noncritical overtreatment are both to be avoided.

The following basic aspects should be borne in mind:
- Whenever possible, all medications should be administered by drip through a safe venous access.
- Analgesics should be *titrated*, beginning with one half the normal dose or less, depending on the patient's condition. With opioids and benzodiazepines, the physician should wait until the medication takes effect before administering additional doses, and this requires patience. Anesthesia in contrast generally requires *RSI with high doses* of anesthetics to preclude defensive reflexes and movements.
- All patients who have received analgesics and sedatives generally are given oxygen (at least 5 l/min via a face mask or nasal tube).
- The patient must be closely observed for state of consciousness, skin color and perfusion, and breathing pattern; blood pressure measurement, pulse oxymetry, ECG, and with ventilated patients, capnometry/capnography are essential.

6.2.2.2 Preclinical Anesthesia

Any emergency physician should be able to intubate a profoundly unconscious patient, but induction of anesthesia in a patient who is breathing spontaneously – to improve oxygenation and for analgesia when required – should be decided on a case-to-case basis [11]. Anesthesia is generally only considered with vital or urgent indication: the patient is not fasting, is in poor condition, and must be treated by limited personnel and equipment in unfamiliar surroundings. This is true to a lesser extent for analgesic treatment protocols.

Induction of anesthesia in emergencies

1. **Prepare and check material**
   - Anesthetics
   - Bag valve mask and oxygen source
   - Suction
   - Laryngoscope
   - Tube (e.g. ID 7.5 mm) with guide wire and Luer lock-tip syringe

2. **Preparing the patient**
   - Secure venous (if necessary intraosseous) access
   - Take advantage of every opportunity for preoxygenation
   - Apply ECG and pulse oxymeter, measure blood pressure
   - Check the situation for intubation, inspect oral cavity

3. **Depending on the patient's condition, rapid i.v. injection of**
   - Up to 0.1 mg/kg BW midazolam (RAD up to approx. 8.0 mg);
     no midazolam when the patient is in very poor condition
   - 0.5 – 1.0 mg/kg BW esketamine (RAD 40–80 mg);
     low dose for patient in very poor condition
   - If necessary, 1.5 mg/kg BW succinylcholine (RAD 100 mg)

4. **Rapid orotracheal intubation as soon as relaxation sets in**
   - Mouth can be opened with little resistance

5. **Block tube and ventilate**
   - Check tube position (auscultation of the epigastrium and thoracic flanks, capnometry/-graphy)
   - Fix tube securely

6. **Administer oxygen (FiO$_2$ 1.0)**

7. **Monitor patient**
   - Pulse, ECG, pulse oxymeter, blood pressure, capnometry/-graphy

9. **Have anesthetics at hand for further administration**

Fig. 6.4 Rapid sequence induction (RSI) of emergency anesthesia. *FiO$_2$* inspiratory oxygen fraction

The physician must be attentive and cautious. If intubation is expected to be difficult or the physician is inexperienced, alternative airway management (especially larynx tube) should be considered, or the patient should be turned on his or her side and given oxygen with a face mask. Alternative measures for securing the airway that require experience include laryngeal tube and mask; coniotomy should be the last resort.

The practical procedure for obligatory *RSI* of anesthesia (Fig. 6.4) – for which temporary respiration with a face mask should be avoided whenever possible – is as follows [12]:

- Carefully check the material and lay it out; a powerful vacuum pump should be available.
- From the onset, use every possibility for preoxygenation and give the patient oxygen at a high flow with a mask. Washing nitrogen out of the lung (denitrogenization) and filling the intrapulmonary oxygen reserve creates an important safety margin.

- Briefly check the situation for intubation and inspect the oral cavity if possible (Is the mouth open far enough? Is the uvula visible? Is there bleeding or vomiting?)
- A Magill tube with a 7.5 mm inner diameter is standard. It should have a guidewire so that the curve of the tube can be corrected without delay if needed.
- Depending on the patient's condition, anesthesia is induced with up to 0.1 mg/kg BW midazolam (RAD up to 8.0 mg), 0.5–1.0 mg/kg BW esketamine (RAD 40–80 mg), and 1.5 mg/kg BW succinylcholine (RAD 100 mg) IV. Etomidate (dose 0.2–0.3 mg/kg BW IV; RAD 15–20 mg) or a similar substance can be used instead of midazolam, and fentanyl (dose 1.25–3.75 µg/kg BW IV; RAD 0.1–0.3 mg) instead of esketamine.
- Midazolam and etomidate are not administered to patients in very poor condition and manifestly in shock; they should only receive esketamine in a dose of approximately 0.5 mg/kg BW (RAD 40–50 mg) IV. In this case, succinylcholine can be dispensed with.
- Hyperextension of the head should be kept at a minimum for intubation. An assistant stabilizes the cervical spine with the hands on both sides of the neck. Any neck support device should be opened but left in place.
- After intubation, the patient is ventilated – regardless of the arterial oxygen saturation by pulse oxymeter (SpO_2) – with an inspiratory oxygen fraction (FiO_2) of 1.0 and a positive end expiratory pressure (PEEP) of about 5 mbar.
- After intubation, the end tidal CO_2 partial pressure is monitored with capnometry/capnography (target value 35–40 mmHg), but this value will be limited in polytrauma patients by inadequate perfusion and abnormal gas exchange in the lungs.

> At first, many severely injured patients do not need any further analgesics or sedatives after induction of anesthesia their sensitivity to pain is apparently diminished. In these patients, the life-preserving endocrine stress reaction must not be suppressed by unsuitable administration of anesthetics.

With clinical signs of insufficient anesthesia (furrowed brow, tearing, defensive movements), anesthesia is optimized with injection of one half of the initial dose of esketamine or an opioid such as fentanyl (in boluses of approximately 0.2 mg); there can also be further sedation with midazolam as needed.

When *pneumothorax* is suspected in a ventilated patient, immediate insertion of a thorax drain (approximately 24 Ch.) is indicated. As hematothorax cannot be diagnosed with certainty on a clinical basis alone; a drain (approximately 28 Ch.) should be inserted pre-hospital only when ventilation cannot be otherwise secured (e.g., with increasing respiratory pressure and decreasing SpO_2) and not prophylactically.

> The endotracheal tube, vascular accesses, and drains should be securely fixed in place to prevent dislocation.

When possible, a history of previous illness and surgeries, as well as current medications being used should be sought so that effects and interactions of medications can be taken into consideration.

> Inadequate *documentation* of the pre-hospital care can have serious medical and legal consequences.

In particular, the neurological status (Glasgow Coma Score [GCS], pupil status, motor function of the individual extremities) should be assessed before induction of anesthesia, as well as the nature and localization of pain before administration of analgesia. Other important parameters are the courses of blood pressure and HR, and the volume requirement. Anesthetics and other medications administered should be documented carefully [11].

6.2.2.3 Special Aspects with of Polytrauma Patients

Unconscious and deeply somnolent patients (GCS persistently <9) should generally be intubated and ventilated. Endotracheal intubation and ventilation will assure optimal oxygenation (FiO_2 normally

1.0) and at the same time protect the airway from aspiration. Analgesia is less important because polytrauma patients often have little need for pain medication.

> Oxygenation – not pain relief – is the first aim of anesthesia for polytrauma patients.

- *RSI* of patients with manifest hypovolemic shock – whenever possible after preoxygenation – is with esketamine and if necessary, with succinylcholine; thereafter, the patient is ventilated (FiO$_2$ 1.0 und PEEP 5 mbar).
- Maintenance of anesthesia depends mainly on the blood pressure. With hemodynamic instability, often no further anesthetics are needed. The patient is monitored carefully; when there are signs of insufficient depth of anesthesia (see above), minimal doses of esketamine and midazolam are injected when necessary. If the patient is hemodynamically stable (e.g., after volume replacement), fentanyl and midazolam are administered in small, and then increasing doses; a muscle relaxant (e.g., Vecuronium) is given only when necessary and the depth of anesthesia is sure to allow it.
- To preserve clotting function, normothermia [13, 14] and avoiding acidosis [15–17] have high priority; the negative effects of these parameters on coagulation are often underestimated.

6.2.2.4 Trapped Patients

> With a *trapped person* – as long as there is no free access to the patient – anesthesia and even analgesia must be avoided as far as possible.

- For analgesia, esketamine is administered in the smallest dose IV (alternatively IM) if necessary.
- An unavoidable anesthesia is performed as RSI with esketamine, and if necessary, succinylcholine.

6.2.2.5 Traumatic Brain Injury

> Before induction, the neurological situation should be assessed and documented.

- As isolated traumatic brain injury is often not accompanied by hypovolemic shock. RSI with etomidate, and if necessary succinylcholine, is indicated.
- In cardiovascular stable patients, anesthesia is optimized with enough fentanyl and midazolam to prevent coughing and bucking with increase in ICP. Vecuronium can be added as a muscle relaxant.

6.2.2.6 Other Trauma

- When a fracture needs to be reduced, small doses of esketamine (up to 0.5 mg/kg BW IV) work well, usually after sedation with midazolam. Esketamine works faster than fentanyl or morphine and there is a less risk of respiratory depression.
- With burns, analgosedation with esketamine and midazolam often suffices. Induction, also with those two drugs, should be considered with care and will possibly be best administered after hospital admission, (e.g., with inhalation trauma or a burned body surface of more than 20 %).

6.2.2.7 Procedure with Children

Children preferably receive small doses of esketamine, or even morphine. Anesthesia is performed – as in adults – with esketamine, fentanyl and midazolam.

6.3 Anesthesia After Hospital Admission

6.3.1 Receiving the Patient

> Correct oral and written procedures for handover of the patient by the emergency physician is imperative. The emergency physician informs all the admitting physicians simultaneously, and not separately. The emergency physician's protocol can be completed, or additions made to it after the handover.

Basic anesthesiological care in the shockroom includes [18]:

- With intubated patients, the position of the endotracheal tube should be checked immediately by inspection of thoracic movement and auscultation (epigastrium and thorax flanks); this is to be repeated every time the patient's position is changed.
- Any vascular accesses should be checked for correct position, particularly with regard to backflow.
- An anesthesia protocol should begin immediately, as well as documentation of at least blood pressure, HR, and SpO_2 at the time of handover.
- Sufficient venous accesses (at least two large-lumen peripheral venous accesses) are essential.
- When possible, a multi-lumen central venous catheter (CVC) with high flow rate should be inserted to allow sufficient volume therapy as well as measurement of central venous pressure (CVP) and central venous sO_2. The catheter must be inserted in the subclavian or internal jugular vein because the femoral vein does not allow valid measurement of CVP and central venous sO_2.
- The *CVP* [11] provides valuable information on the volume status as well as right ventricular preload and compliance. The general target value is 5–10 mm Hg. A lesser value points to a lack of volume, while the target value does not guarantee sufficient preload. Higher values may be required to optimize cardiac output (e.g., with chronic right heart load). In ventilated patients, PEEP should also be taken into account, although it does not follow the CVP in a linear-additive manner.
- Arterial puncture for *invasive arterial pressure measurement* to monitor circulation beat by beat and for arterial blood gas analysis (BGA) should be performed as soon as possible but without substantially delaying patient care that is more essential. Arterial measurement does not improve a state of shock, therefore, the cause should better be sought. Respiratory fluctuations in the arterial pressure curve indicate a lack of volume.

Even if urgent action is called for, the *basic rules of hygiene* must be followed. This is especially true for insertion of a thorax drain or a CVC with Seldinger technique. Trauma patients are potentially immune compromised and must not be subjected to any avoidable additional antigen load.

6.3.2 Intrahospital Transport

Patient transportation within the hospital often entails gaps in surveillance and other risks such as inadequate ventilation or dislocation of vascular accesses.

- Transportation within the hospital demands careful clinical and technical surveillance (see above); furthermore, the patient must be protected from chill.
- Patient ventilation with FiO_2 of 1.0 and PEEP of about 5 mbar should be monitored regularly. As oxygen toxicity takes hours to become relevant, an FiO_2 of 1.0 is harmless and is an important safety factor with disconnections.
- Keeping an eye on the clock prevents unnecessary time loss.

6.3.3 Continuation of Anesthesia

Further anesthesiological procedure [18] is limited here to the basics:

- Anesthesia is maintained – with an eye on the target blood pressure value – with midazolam (RAD some 5 mg) and fentanyl (RAD some 0.2 mg) IV as needed. Particularly for patients with a head injury, a relaxant is indicated to prevent an increase in ICP as a result of coughing or bucking. Because of their pronounced sympatholytic effect, propofol and remifentanil are to be avoided in unstable patients.

- Continuous treatment of shock with volume replacement and blood components is imperative.
- Administration of catecholamines should only be considered in special situations, for example, when lack of volume cannot otherwise be brought under control.
- Until proven otherwise, it is to be assumed that the spine of the polytrauma patient is unstable; this should be kept in mind particularly when the patient is repositioned.

Other measures during in-hospital transport, diagnostic procedures, and subsequent care in the operating room include:

- Regular checks of the position of the tube
- Regular auscultation of the thorax
- Monitoring of respiratory pressures
- Monitoring of the circulatory situation and urine output
- Regular checks of pupil reaction
- Avoiding chilling and active rewarming of the patient as needed

6.3.4 Contribution of Anesthesia to Overall Care

In the individual case, the following factors can help to optimize overall care, whereby all the parameters in the previous and expected course are to be considered and included in the total clinical setting (trauma, age, previous illnesses, etc.) [18].

- Assessing pulmonary function and gas exchange, particularly FiO_2 and BGA throughout the course.
- Evaluating circulatory function, taking into account previous IV drip and transfusion requirements, as well as the course of blood pressure, HR, and CVP. The target is a systolic arterial pressure (SAP) >90 mm Hg with an HR <100/min; in head injury patients the SAP should be >120 mm Hg. Further critical values are a pH (preferably taken from a CVC) <7.25, a base excess (also preferably taken from a CVC) of −6 mmol/l or worse, a lactate

concentration >2.5 mmol/l, and central venous sO2 < 70 %.

- Evaluation of renal function: hourly urine output <0.5 ml/kg BW is critical.
- Evaluation of coagulation status: critical signs are a decrease in thrombocytes, Quick value, antithrombin (AT) and fibrinogen concentration over 50 % and doubling of prothrombin time (PTT) or thrombin time (TT).
- Measuring body core temperature; values <35 °C are critical, especially for coagulatory function.

6.4 Perioperative and Postoperative Pain Therapy

6.4.1 Fundamentals

Efficient perioperative and postoperative pain therapy [19] can only be achieved with clear organizational guidelines and orientation to objectifiable standards such as the exemplary German-language guideline, S3-Leitlinie "Behandlung akuter perioperativer und posttraumatischer Schmerzen" [20].

Immediate postoperative pain therapy is often ordered by the anesthesiologist using the anesthesia protocol and begun in the recovery room. This makes sense as the anesthesiologist can best evaluate the transition from intra- to postoperative analgesia and the initial success of the latter. When the patient is transferred from the recovery room, responsibility for the continuation and monitoring of the treatment ordered by the anesthesiologist passes to the nursing staff. The primary physician is responsible for any changes in the suggested treatment plan.

The respective surgical service is responsible for the general perioperative (not immediately postoperative) analgesic care of patients, possibly with the support of a consultant, or with treatment directed by an acute pain service. In any case, responsibility for treatment during this phase must be clarified.

The most important ground rules for practical analgesic care are:

- Before surgery, the patient should be informed of possible postoperative pain and its treatment. This can be

Table 6.2 Mode of application, dosage, and main indication of common analgesics for postoperative pain therapy

Medication	Application	Dosage	Main indication
Acetylsalicylic acid	Oral; IV	RAD 0.5–1.0 g every 6–8 h MDD 3 g	Mild to moderate pain, fever
Paracetamol	Oral, rectal	RAD 0.5–1.0 g every 6–8 h MDD 50 mg/kg BW	Mild to moderate pain, fever
Diclofenac	Oral, rectal, (IM)	RAD 50 mg (−100 mg) every 8–12 h; single dose of 75 mg IM MDD 100 mg (initial 150 mg)	Rheumatic and nonrheumatic pain
Metamizol	Oral, rectal, IV, (IM)	MDD 0.5–1.0 g (20–40 drops) every 6–8 h (up to 2.5 g IV) MDD 5 g	Acute moderate and severe pain, colic, high fever
Tramadol	Oral, IV, (IM, s.c.)	RAD 50–100 mg or 20–40 drops MDD 400 mg or more	Moderate to severe pain
Tilidin-naloxone	Oral	RAD 50–100 mg or 20–40 drops every 4–8 h MDD 600 mg	Severe and very severe pain
Morphine	IV (s.c., IM)	RAD 5–10 mg IV, 10–20 mg IM or s.c. every 4–6 h MDD no information available	Severe and most severe pain

RAD Recommended dose for an adult of about 75 kg body weight (BW), *MDD* maximum daily dose, *s.c.* subcutaneous

done by the anesthesiologist or the surgeon, if the former is involved in the postoperative pain therapy.
- Pain is a subjective matter that is to be evaluated regularly by the patient himself/herself as it can be misestimated by physicians or nursing staff.
- The visual analog scale (VAS) has proved useful for evaluating subjective pain intensity. The patient selects a value between 0 and 100 (or 10) on a 10 cm long ruler indicating degree of pain from "no pain" to "unbearable pain." The VAS is especially useful for objectifying treatment success.
- In individual cases and beside clinical surveillance of the patient, a technical monitoring (ECG, pulse oxymeter, blood pressure) is necessary even on the ward to register respiratory depression due to opioids, systemic effects of local anesthetics (LA) or rostral diffusion of neuraxial anesthesia. It may also be necessary to administer oxygen. With extreme pain, at least the initial analgesia should be given in the intermediate care unit.

6.4.2   Systemic Analgesia

Systemic analgesia is fast and uncomplicated and is the basis of perioperative analgesia.

6.4.2.1   General

The substances used in systemic perioperative pain therapy can basically be given IV, IM, orally, sublingually, rectally, or transdermally. The following general rules should be observed:
- For fast treatment and to avoid incalculable resorption phenomena, analgesics are best administered via a drip using a reliable venous access; other modes of administration are usually used for chronic pain.
- First, a bolus injection is given for fast pain relief. The injections should be titrated, depending on the patient's condition to avoid under- or overdosage. Patience is needed with opioids in particular, to let the full effect develop and avoid overhasty further injections.
- The initial freedom from pain is maintained with an IV drip or further scheduled doses to avoid analgesic gaps.

6.4.2.2   Medications

Analgesics are classified as non-opioids and opioids. The analgesics that are commonly used to treat postoperative pain are listed in Table 6.2, with information on mode of application, dosage, and main indication.

Non-opioids mainly work peripherally and were previously called "peripheral analgesics." Their analgesic effect is based mainly on inhibition of prostaglandin (PG) synthesis in traumatized tissue but they

also have some additional central effects. Their SEs are clear. With short-term use, gastrointestinal disorders such as bleeding and activation of ulcers can occur, along with asthma attacks in predisposed persons, resulting from the absence of bronchodilating PG. Relative contraindications are kidney and liver disease as well as hemorrhagic diathesis. Use in the third trimester of pregnancy is normally contraindicated because inhibition of PG synthesis can lead to premature closure of the ductus arteriosus and bradytocia; acetylsalicylic acid (ASS) and diclofenac can increase the risk of bleeding in mother and child.

> The non-opioid analgesics are the basis of perioperative pain treatment and are indicated for mild to moderate pain. Because of their limited analgesic potency, they are often combined with opioids. They are valued mainly for their antipyretic and anti-inflammatory effects, particularly when the musculoskeletal system is involved.

Opioids are natural or synthetic morphine derivatives, acting on various opioid receptors. Because these receptors can also be expressed in traumatized tissue, the term "central analgesics" is not completely correct. The opioids used in perioperative pain control are usually morphine agonists that mainly affect the μ-opioid receptor; their effect is felt within a few minutes and persists for several hours. Typical SEs are respiratory depression, emesis, constipation, and miosis, which currently cannot be separated from the analgesic effect. Morphine agonists can be displaced at the receptor by naloxone and so antagonized.

> As extremely potent analgesics, opioids basically fulfill all clinical requirements. Their main SE is respiratory depression. As the transition from sufficient analgesia to relative overdosage with dangerous respiratory depression is insidious ("silent death"), experience as well as careful surveillance and monitoring of the patient are essential.

6.4.2.3 Patient-Controlled Analgesia

> Patient-controlled analgesia (PCA) is a method in which the patient controls his/her own pain medication using an injection pump programmed by the physician. This can have optimal results with a patient who is willing and able to understand and apply the technique.

After titrated boluses have produced adequate analgesia, the physician programs the injection pump with the amount of the single dose (bolus) and the interval between doses (blocking period); a maximum dose can also be programmed. The injection pump is then connected to the patient. Although many opioids are suitable, it is suggested that only medications in regular use at the particular institution be used; the following is an example:

- Fill a 50 ml-syringe with 50 mg morphine in 50 ml NaCl 0.9 %; 1 ml of this solution corresponds to 1 mg morphine
- Bolus 2,5 ml (= 2,5 mg)
- Blocking time 15 min
- Maximal dose 25 mg morphine in 4 h

If satisfactory analgesia has not been achieved within an hour, the bolus dose is increased; if that does not produce the desired effect, the second step is to decrease the blocking time. It may be necessary to add non-opioids such as diclofenac or metamizole. Additional opioids may be administered in exceptional cases only, with the agreement of the physician responsible for PCA and surveillance of the vital functions (*cave* respiratory depression).

Metoclopramide is often prescribed for prophylaxis and treatment of nausea and vomiting. If the venous access for the PCA is also used for a gravity drip, this should have a backup valve to prevent accidental infusion of the opioid into the drip solution. Naloxon should be immediately on hand to quickly counteract an opioid overdose.

6.4.3 Regional Analgesia and Anesthesia

6.4.3.1 Fundamentals

> Regional analgesia or anesthesia is usually highly effective and avoids the SEs of higher doses of systemic opioids.

In addition to the actual analgesic effects, regional administration has the following *general advantages*:

- The patient's general well-being is largely uncompromised.
- Sympathicolysis resulting from LA improves perfusion in the affected area.
- Thoracal and lumbal peridural analgesia or anesthesia considerably attenuates the systemic endocrine stress response – in this case the sympathoadrenergic reaction in particular [1] – that is of particular benefit to patients with cardiocirculatory and metabolic disorders (coronary heart disease, diabetes mellitus).

Regional administration can occur preoperatively (e.g., 3-in-1 block with fractures in the hip area), intraoperatively for anesthesia and subsequent analgesia (e.g., blockade of the axillary plexus), or postoperatively. Planning should be anticipatory, in close cooperation with the specialists involved.

The catheter technique allows the blockade to be continued over a longer period of time so that once-only blockades are the exception. LA are used for analgesia and, in the vicinity of the spinal cord, opioids. The substances are given as a bolus or a continuous drip.

With both the catheter technique and once-only blockade, the first injection should be administered by the physician who performed the puncture. Subsequent bolus injections should also be administered by the physician. Continuous infusion with an injection pump helps to avoid analgesic gaps and further allows patient control, especially for patient-controlled epidural analgesia. Axillary plexus blockade and others can also be continued on a patient-controlled basis.

Regional techniques are absolutely contraindicated with infections in the area of the planned puncture. Peripheral nerve damage or other neurological diseases (e.g., diabetic neuropathy) are *relative contraindications* because patients can potentially blame persistent neurological symptoms on previous regional anesthesia. If necessary, the patient should be referred to a neurologist for documentation of the initial status.

There are no particular hemostasiological prerequisites for *peripheral blockades*, including axillary plexus anesthesia, except with clinically manifest coagulation disorders. This is not the case, however, *in neuraxial techniques* with spinal anesthesia or analgesia, epidural anesthesia or analgesia (EDA), and combined spinal and epidural anesthesia. There is the danger of intraspinal bleeding with subsequent neurological damage and even paraplegia, so that the relevant national [21] and international [22] guidelines should be followed.

6.4.3.2 Substances

The *LA* used for spinal and peripheral blockades differ in their time to take effect and duration of effect.

- Mepivacaine and prilocaine are LA with medium-term effect (up to 2 h) that are mainly used intraoperatively and seldom for postoperative pain treatment. Although it forms methemoglobin, prilocaine is less toxic than mepivacaine and penetrates tissue particularly well.
- Bupivacaine and ropivacaine have a longer effect (5–6 h and more) but take longer to take effect. Ropivacaine is less cardiotoxic, and in low concentrations allows better distinction of sensory and motor blockade (differential blockade).

After a catheter has been inserted, a fast-acting substance is often injected to check for correct positioning. A long-acting substance is then used, generally for the above-mentioned differential blockade. With painful measures (physiotherapy, etc.) the blockade can be reinforced with a short-acting LA.

Opioids are used alone or in combination with an LA for catheter EDA (C-EDA). Sufentanil and morphine are approved for EDA; morphine is also approved for intrathecal application with spinal anesthesia, but this does not play any particular role in normal perioperative pain therapy.

Metamizole and diclofenac are usually used as co-medication for insufficient analgesia.

6.4.3.3 Procedures and Indications

Of the many possible options for postoperative regional analgesia, only the most important are mentioned here:

- Thoracal C-EDA is normally used for thoracotomy and serial rib fractures.
- Lumbal C-EDA is used with extensive laparotomies (with the addition of morphine for adequate rostral diffusion), as well as for pain in the pelvic area and lower extremities.
- Continuous blockade of the axillary plexus is usually via the axillary or vertical infraclavicular plexus (VIP) access and allows analgesia in the entire upper extremity. VIP also eliminates pain in the shoulder joint.

- The 3-in-1 block is indicated particularly for knee operations (including endoprosthetic procedures), as well as for hip fractures and hip joint replacement.
- A foot block is an option for pain in the mid- and forefoot.
- The ulnar, radial, and median nerves can also be targeted for blockade.

6.4.3.4 Surveillance and Monitoring

Because of the danger of primary or secondary displacement of the catheter and the SEs that can follow, every continuous regional anesthesia should be carefully monitored.

With all regional techniques, there can be unnoticed intravasal application of LA either *primarily* during the bolus application or *secondarily* after displacement of the infusion catheter, with systemic signs such as tingling and paresthesia in nonsupplied areas (often perioral), cardiac arrhythmias, clouded consciousness, and convulsions; ultimately there can be coma and cardiocirculatory failure.

- Accidental intrathecal LA administration through an epidural catheter leads to ascending paresis with the danger of respiratory failure ("total spinal anesthesia").
- Unnoticed intrathecal administration of opioids is more insidious, with central respiratory depression and "silent death."

Care of patients with neuraxial analgesia via C-EDA poses problems because of the danger of secondary intrathecal or intravasal dislocation of the catheter. In many hospitals, C-EDA with LA and opioids is limited to intensive or intermediate care conditions. In the ward, only LA (and no opioids) are used and patients should be monitored every 15–30 min.

The following rules apply for the surveillance of the regional techniques ("pain catheter") used for postoperative pain treatment (for basic information see the section "Organization and general practice"):

- There should be written orders for every patient that include the substances to be administered, dosage intervals, procedure for insufficient effects, and the intervals for replacing filters and syringes.
- These orders should be discussed, preferably daily, with the acute pain service as well as with the medical and nursing team and changed when necessary.
- When dressings are changed, punctures and catheter insertion points should be examined for any erythema, swelling, or secretion.

- Catheters that have not been used for 2 days and that are not expected to be needed further (no physiotherapy, etc., scheduled) are to be removed.
- The acute pain service should always be consulted in any cases of doubt until neurology has completely normalized and the puncture site is painless. Furthermore, the patient should be evaluated daily by a physician or a specially trained member of the acute pain service.

Patients with C-EDA for pain therapy should be monitored carefully. New symptoms, especially one-sided neurological ones and pronounced back pain, indicate an intraspinal space-occupying process (bleeding, abscess) and demand immediate clarification using magnetic resonance imaging (MRI) (computed tomography, if MRI is not available).

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