Recommended practice for out-of-hospital emergency anaesthesia in adults

Statement from the Out-of-Hospital Emergency Anaesthesia Working Group of the Emergency Medicine Research Group of the German Society of Anaesthesiology and Intensive Care

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Abbreviations
ARDS  Acute Respiratory Distress Syndrome
AWMF  Working Group of Scientific Medical Associations
BMI  Body Mass Index
EAST  Eastern Association for the Surgery of Trauma
ECG  Electrocardiogram
EMS  Emergency Medical Service

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Abstract

Emergency anaesthesia is an important therapeutic measure in out-of-hospital emergency medicine. The associated risks are considerably higher than those of in-hospital anaesthesia. The primary objectives of emergency anaesthesia are hypnosis, analgesia, oxygenation and ventilation through airway management. The secondary objectives of emergency anaesthesia are amnesia, anxiolysis, the reduction of oxygen consumption and respiratory work, the protection of vital organs and the avoidance of secondary myocardial and cerebral damage. A critical evaluation of the indications for out-of-hospital emergency anaesthesia must take into consideration patient, case and provider-related factors.

1.1 Rationale, frequency and indication

1.1.1 Rationale

Emergency anaesthesia, airway management and ventilation are important therapeutic measures in emergency medicine. In physician-based emergency medical service (EMS) systems, every EMS-physician – irrespective of his or her specialty – should be able to safely induce emergency anaesthesia in patients with various injury patterns, clinical pictures and risks despite adverse conditions outside the hospital. This leads to the question of what procedure should be recommended for out-of-hospital anaesthesia under complex conditions and what anaesthetic drugs should be used, especially with regard to different groups of patients. It must also be taken into consideration that the induction and performance of out-of-hospital anaesthesia is in many respects more difficult than routine anaesthesia in operating theatres or ICUs in hospitals.

The following recommendations of the German Society of Anaesthesiology and Intensive Care Medicine (DGAI) have been prepared for EMS-physicians.
Tables 2 and 3 provide an overview of the indications for out-of-hospital emergency anaesthesia, which takes into consideration patient, case and physician-related factors. Indications for emergency anaesthesia can be broadly divided into acute respiratory insufficiency (e.g. hypoxia due to decreased oxygenation or impaired oxygen transport, respiratory rate < 6 or > 29 breaths per min) and contraindications to or failure of noninvasive ventilation. Unconsciousness/neurological deficit with risk of pulmonary aspiration. Major trauma in association with haemodynamic instability, SBP < 90 mmHg or hypoxia at levels of SpO₂ < 90% despite oxygenation or cranioencephalic trauma with GCS < 9.

1.1.3 Frequency

According to the German minimal emergency data record data base (82,000 ground deployments of emergency physicians of Baden-Württemberg, Germany, and the AirRescue Information and Communication Systems data base (47,000 air rescue missions, Germany), every emergency physician induces out-of-hospital anaesthesia every 2 weeks in air rescue missions and every 1.4 months in ground deployments. Out-of-hospital anaesthesia is induced in approximately 3 to 5% of all EMS missions and in 4 to 7% of EMS missions to children (age < 18 years). 8–12

1.1.4 Indications for emergency anaesthesia

Emergency anaesthesia must often be induced in unconscious (Glasgow coma scale, GCS < 9), uncooperative, severely injured or critically ill patients with a full stomach and unstable cardiopulmonary conditions. Emergency anaesthesia is, in most cases, necessary for airway management. An exception to this rule are patients undergoing cardiopulmonary resuscitation who require airway management first and, if necessary, emergency anaesthesia later once spontaneous circulation has returned. Indications for emergency anaesthesia can be found in critically ill or injured patients with cardiopulmonary or neurological diseases, trauma patients, and patients who are intoxicated or have markedly impaired conscious level with a reduction in protective reflexes (GCS < 9) and a high risk of pulmonary aspiration. 13,14 This does not include rapidly reversible causes of impaired consciousness (e.g. hypoglycaemia) or conditions in which the GCS does not correlate with the extent of the loss of protective reflexes (e.g. stroke with aphasia or dementia). Patients with markedly impaired consciousness (GCS < 9) require emergency anaesthesia to tolerate airway management. 15,16

If emergency anaesthesia is required, personnel must take into account the guideline on out-of-hospital airway management 17 by the DGAI as well as the information on emergency anaesthesia, airway management and ventilation in the German S3 guideline on treatment of major agreement of 12 out of 14 participants (> 85%) could be reached.

1.1.2 Methods

The recommendations contain measures based on the latest scientific findings, which ensure the appropriate provision of out-of-hospital emergency anaesthesia for critically ill or injured patients under various circumstances (e.g. infrastructure, specific situation, patient condition, individual capabilities, knowledge and experience of the physician).

To achieve consensus on scientific findings and current practice of out-of-hospital emergency anaesthesia, the Emergency Medicine Research Group of the DGAI invited 14 anaesthesiologists experienced in out-of-hospital emergency medicine from German and Austrian medical centres to participate in an Out-of-Hospital Emergency Anaesthesia Working Group. After constructing a frame of contents, single topics were distributed to teams of authors to review relevant literature and provide a first version of the text.

In a second step, these results were assembled and a three-round digital Delphi study was conducted to reach consensus.

The Delphi technique is a structured approach of debating by experts to converge a discussion towards group consensus, which was initially developed in the 1950s for complex problems exceeding the analytical capabilities of a single person. 6

Working group opinion was fed back after each Delphi round to allow the participants to revise their previous opinions and so converge towards group consensus. Recommendations were approved with consensus when

Table 1

<table>
<thead>
<tr>
<th>Overview of the recommendations for out-of-hospital emergency anaesthesia</th>
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<tr>
<td>Critical evaluation of the indications for out-of-hospital emergency anaesthesia, which takes into consideration patient, case and physician-related factors. Rapid sequence induction with preoxygenation, standardisation of out-of-hospital anaesthesia procedures and preparation of anaesthetic/emergency drugs and airway equipment and ventilator, standard monitoring, vascular accesses, drug administration, temporary removal of cervical collar and manual in-line stabilisation during intubation, airway management and checking of correct tube placement using capnography. Preoxygenation for every spontaneously breathing emergency patient for at least 3 to 4 min using 12 to 15 l/min of oxygen and a tight-fitting face mask with reservoir or demand valve or noninvasive ventilation (CPAP). Standardised preparation of anaesthetics and emergency drugs, bag valve mask with reservoir or demand valve, including mask, endotracheal tube and cuff inflation syringe, introducer and fixation, alternative airway devices, stethoscope, checking of suction, ventilation and standard monitoring devices, including capnography. Standard monitoring for out-of-hospital anaesthesia comprises ECG, (automatic) blood pressure monitoring, pulse oximetry and capnography. Capnography to check tube placement, disconnection and displacement and to indirectly monitor haemodynamics. Before anaesthesia is induced, at least two peripheral venous catheters should be placed (if possible).</td>
</tr>
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</table>

CPAP Continuous Positive Airway Pressure Ventilation.

and paramedics in two-tier physician-staffed EMS systems. Table 1 shows a list of the central points of these recommendations.

Table 2

<table>
<thead>
<tr>
<th>Indications for out-of-hospital emergency anaesthesia</th>
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<tbody>
<tr>
<td>Acute respiratory insufficiency (hypoxia and/or respiratory rate &lt; 6 or &gt; 29 breaths per min) and contraindications to or failure of noninvasive ventilation. Unconsciousness/neurological deficit with risk of pulmonary aspiration. Major trauma in association with haemodynamic instability, SBP &lt; 90 mmHg or hypoxia at levels of SpO₂ &lt; 90% despite oxygenation or cranioencephalic trauma with GCS &lt; 9.</td>
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*If causes are not rapidly reversible.*
trauma (S3-Guideline on Treatment of patients with severe and multiple injuries. German Trauma Society. 2011 www.awmf.org Nr. 012-019 Assessed 29 September 2015). Indications for, planning of and performance of emergency anaesthesia are influenced by the following factors:

- Training, experience and routines of the emergency physician and paramedics
- Out-of-hospital environment (e.g. illumination, space, weather)
- Time and type of transport (ground, air ambulance)
- Circumstances surrounding airway management and (foreseeable) intubation problems (e.g., expected difficult airways of emergency patients with sufficient spontaneous breathing).

The EMS-physician must not only consider the situation of the patient but must also critically assess his own skills when deciding to perform out-of-hospital emergency anaesthesia. Emergency anaesthesia is an invasive measure, poses a lethal risk, places special requirements on performance, monitoring and complication management. Before inducing emergency anaesthesia, the EMS-physician must consider disadvantages and possible complications (e.g., vomiting, pulmonary aspiration, airway displacement, cardiovascular depression, allergic reaction) and analyse the risks and benefits. In addition, the skills of the EMS-physician and the paramedics as well as relevant team factors must also be considered. Unlike junior hospital doctors, EMS-physicians usually cannot request direct support from a medical specialist or a senior physician. Several incidents have been reported in which severe complications were caused by a lack of experience in out-of-hospital emergency anaesthesia. Mistakes are easily made by inexperienced personnel. Guidelines and standard operating procedures must define clear procedures to provide less experienced emergency teams with a standardised approach to out-of-hospital anaesthesia. Given the life-threatening risks for the patient, it is crucial that all EMS-physicians know the procedures for inducing and performing out-of-hospital anaesthesia. The Association of Anaesthetists of Great Britain and Ireland requests that physicians inducing out-of-

### 1.2 Special features of out-of-hospital emergency anaesthesia

Emergency anaesthesia induced in the ICU, in the emergency department, and especially outside the hospital is associated with a high level of difficulty. According to Timmermann et al., these multifactorial risk-increasing conditions can be categorised as physician, patient and case-related factors.

#### 1.2.1 Patient-related factors

Patient-related factors complicating the induction and performance of emergency anaesthesia include a full stomach, injury of the airway, restricted mobility of the cervical spine (preexisting, on account of trauma or immobilisation), cardiopulmonary or other disorders because of preexisting diseases and/or injuries, a poor venous state and long-term medication.

- Full stomach: Out-of-hospital emergency patients must be assumed to have a full stomach. To reduce the risk of aspiration in adults, rapid sequence induction is the technique of choice. This involves rapid anaesthetic induction and airway management without intermittent ventilation. This has a considerable influence on the choice of anaesthetic. The DGAI Paediatric Anaesthesia Scientific Working Group recommends intermittent ventilation in paediatric patients to avoid hypoxia during rapid sequence induction.

- Difficult vascular access: If possible, early insertion of two peripheral venous catheters is recommended during out-of-hospital anaesthesia in critically ill or severely injured patients to always have a second access available during induction (e.g. in case of extravasation).

- Haemorrhagic shock: Blood loss is underestimated in many patients (e.g., major trauma, internal haemorrhaging). Medical personnel must take into account that in such cases the number of red blood cells is critically reduced and patients must be carefully preoxygenated. Tests have shown that animals with severe haemorrhagic shock had oxygen saturation (SpO2) of less than 70% after only 1 to 2 min of apnoea despite preoxygenation. If emergency anaesthesia is induced in patients with severe haemorrhagic shock at the scene of the accident, sudden hypotension may occur, which is extremely difficult to correct.
1.2.2 Case-related factors
Position of the patient: Trapped patients or patients in a confined area should be treated first by inducing appropriate analgesia and sedation and by maintaining spontaneous breathing. Rescued patients should then be appropriately positioned for anaesthesia induction and airway management. The best conditions outside hospital can be provided in an ambulance car.  

Equipment constraints: Clinical physicians have a wide range of equipment, devices and drugs at their disposal. In an out-of-hospital environment, however, the selection of equipment and drugs is considerably limited. 

Urgency: Depending on the condition of the patient, out-of-hospital anaesthesia must often be induced as quickly as possible. Medical personnel in an out-of-hospital environment must, therefore, have a high level of experience to ensure patient safety.

1.3 Preparation, performance and monitoring of emergency anaesthesia
On account of the risks and hazards of out-of-hospital emergency anaesthesia, standardised procedures are necessary to avoid complications. Personnel performing out-of-hospital emergency anaesthesia must, therefore, take the following points into consideration:

1. Thorough evaluation and examination of the patient
2. Critical verification of the indications for out-of-hospital emergency anaesthesia
3. Optimisation of patient condition through preoxygenation, haemorrhage control and infusion (if necessary)
4. Standardised procedures for the preparation and performance of out-of-hospital emergency anaesthesia
5. Management of complications

1.3.1 Critical verification of the indications for out-of-hospital emergency anaesthesia
Information provided in sections 1.1.3 and 1.2 must be taken into account in this context. The decision to induce out-of-hospital anaesthesia must be communicated to the entire emergency team. The team must discuss the best location to induce anaesthesia, the tasks of each team member, the drugs selected and other important issues. A common approach must be agreed upon, which ideally is based on a standardised procedure.

1.3.2 Preparation of out-of-hospital anaesthesia
Rapid sequence induction is performed to induce emergency anaesthesia. The goal is to rapidly and effectively bring about a state of unconsciousness in which airway management and ventilation are tolerated. This procedure involves the administration of a sedative followed by muscle relaxant. Analgesic drugs may be administered prior to or immediately after these two substances or after the airway is secured. Ventilation must be ensured after anaesthesia induction. Drugs must be filled into syringes and labelled beforehand. Airway management equipment must be prepared and checked for functionality (Table 4).

1.3.3 Performance and procedure of out-of-hospital emergency anaesthesia
Table 5 and Figs. 1 and 2 provide an overview of the phases of out-of-hospital emergency anaesthesia.

After paramedics prepare the drugs and the equipment for airway management and ventilation as instructed by the EMS-physician, preoxygenation is initiated as soon as the EMS-physician decides to induce emergency anaesthesia (Fig. 1). To prevent desaturation during anaesthesia induction and airway management or to prolong the time until the oxygen saturation level decreases (apnoeic tolerance), spontaneously breathing emergency

Table 4 Standardised preparation of emergency anaesthesia equipment

<table>
<thead>
<tr>
<th>Equipment</th>
</tr>
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<tbody>
<tr>
<td>Syringes must be filled with anaesthetic and emergency drugs; labels must indicate the name and concentration of the drug</td>
</tr>
<tr>
<td>Bag valve mask with reservoir or demand valve and appropriate mask</td>
</tr>
<tr>
<td>Appropriate endotracheal tube, including inflation cuff syringe and introducer, tube fixation and stethoscope</td>
</tr>
<tr>
<td>Alternative airway device</td>
</tr>
<tr>
<td>Suction, ventilation and capnography devices must be checked for completeness and functionality</td>
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</tbody>
</table>

Table 5 Standardised performance of out-of-hospital anaesthesia

<table>
<thead>
<tr>
<th>Procedure</th>
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<tbody>
<tr>
<td>Critically evaluate indications for emergency anaesthesia</td>
</tr>
<tr>
<td>Inform all team members about indications for emergency anaesthesia</td>
</tr>
<tr>
<td>Optimise out-of-hospital conditions (e.g. transport of patient into ambulance, head position)</td>
</tr>
<tr>
<td>Immediately begin preoxygenation of spontaneously breathing patients</td>
</tr>
<tr>
<td>Prepare drugs and airway management equipment (Table 2)</td>
</tr>
<tr>
<td>Monitor patient (place ECG electrodes, SPO2, automatic NIBP, have capnograph ready)</td>
</tr>
<tr>
<td>Place and fix two peripheral venous catheters with continuous infusions (if possible)</td>
</tr>
<tr>
<td>Perform rapid sequence induction</td>
</tr>
<tr>
<td>If necessary, remove cervical collar and start manual in-line stabilisation</td>
</tr>
<tr>
<td>Call out drugs, indicating the active agent and dose; consecutive administration</td>
</tr>
<tr>
<td>Wait for loss of consciousness and muscle relaxation</td>
</tr>
<tr>
<td>Airway management without intermittent ventilation in normoxic patients</td>
</tr>
<tr>
<td>Confirm tube placement (capnography, auscultation, insertion depth)</td>
</tr>
<tr>
<td>If necessary, stop manual in-line stabilisation and close cervical collar</td>
</tr>
<tr>
<td>Perform continuous monitoring, including continuous capnography and adjust ventilation device</td>
</tr>
<tr>
<td>Maintain and monitor anaesthesia</td>
</tr>
<tr>
<td>Recognise and treat problems regarding vital signs</td>
</tr>
<tr>
<td>Manage complications if necessary</td>
</tr>
</tbody>
</table>

NIBP, noninvasive blood pressure.
patients are given oxygen for 3 to 4 min, whenever possible. Preoxygenation must be performed only with 100% oxygen through a face mask or the tightly fitting bag valve mask, each with an oxygen reservoir (at least 12 to 15 l/min of oxygen). A demand valve or noninvasive ventilation (NIV) may be used, which are even more effective and require less oxygen. A face mask without reservoir is not sufficient for preoxygenation even at the highest possible flow rates.

During preoxygenation, optimal monitoring is ensured, and syringes are filled with anaesthetic and emergency agents according to the instructions of the emergency physician. Standard monitoring includes ECG (3-lead ECG: heart rate and rhythm), capnography, continuous automatic blood pressure (BP) monitoring (at least every 3 min), and pulse oximetry (heart rate and SpO₂). The German Interdisciplinary Association for Intensive Care and Emergency Medicine (DIVI) recommends the use of standardised self-adhesive syringe labels to avoid confusion in critical situations. (DIVI-recommendation for labelling syringes in intensive care and emergency medicine 2012)

To prevent regurgitation, the upper body should be elevated (but kept in line) if there is no contraindication (e.g. spinal immobilisation in trauma patients or a haemodynamically unstable patient).

After the venous accesses are checked, anaesthesia is induced according to agreed team approaches and procedures. The paramedics confirm the names and doses of the drugs (in ml or mg) requested by the physician. The drugs are then administered. At this point, the cervical collar of patients with neck immobilisation is opened while ensuring manual in-line stabilisation provided by an assistant. After the patient has lost consciousness and the muscle relaxant has an effect, the airway is then secured. In adult patients, airway management is usually performed without intermittent ventilation. In some cases, intermittent ventilation may be necessary to maintain oxygenation despite the increased risk of aspiration (e.g. severe respiratory insufficiency). The application of cricoid pressure (Sellick’s manoeuvre) is no longer recommended on account of a lack of evidence about its positive effects and because of potential problems at the tube site. The cuff of the endotracheal tube or the supraglottic airway (SGA) device (e.g.
laryngeal mask, laryngeal tube) is inflated immediately after insertion, placement is confirmed and the device is fixed.

In out-of-hospital environments, two procedures are used to verify endotracheal tube placement for intubation: visually via direct laryngoscopy or video laryngoscopy, and via capnometry/capnography. German standard DIN EN 1789 stipulates that all ambulances must have equipment for monitoring end-tidal carbon dioxide. This equipment must be used. Capnography provides vital information about ventilation and thus about the placement of the tube or SGA device. In addition, end-tidal carbon dioxide monitoring indicates acute changes in cardiac output earlier than other out-of-hospital methods. Continuous capnography also can detect the displacement, disconnection or kinking of the endotracheal tube. As unrecognised oesophageal intubation can have devastating consequences, correct tube placement must be confirmed using capnography (100% sensitivity). This does not, however, rule out over-insertion of the tube (endobronchial intubation). Bilateral breath sounds and chest movement can confirm the correct depth (measured from the teeth: women: approximately 20 to 21 cm, men: approximately 22 to 23 cm). Continuous standard monitoring must be ensured during the entire duration of anaesthesia to adequately monitor vital signs and respond to any changes.

1.3.4 Management of complications and problems

Out-of-hospital anaesthesia involves many risks. Complications must, therefore, be quickly identified and knowledgeably managed and eliminated.

Insufficient depth of anaesthesia: If laryngospasms or bronchospasms occur during induction or if the patient resists airway management, attempts to intubate must be interrupted. Anaesthesia must be deepened or muscle relaxants must be administered. Resistance, laryngospasms and bronchospasms usually cease once anaesthesia is deepened. Intermittent ventilation during rapid sequence induction in adults is possible as hypoxia is more dangerous than aspiration.

Hypotension: Temporary hypotension occurs in 7 to 18% of all cases of out-of-hospital anaesthesia. Continuous automated oscilometric BP measurement is, therefore, vital. Patients with acute hypovolaemia have an increased risk of hypotension. Treat hypotension with fluids, calcium, adrenaline, noradrenaline or, if necessary, adrenaline. The relevant drugs must be prepared before anaesthesia is induced. Fluid imbalances must be corrected through appropriate intravenous infusions. Heart failure should also be considered as a differential diagnosis, particularly in patients who have preexisting conditions.

Allergic reactions: In rare cases, some drugs may release histamine and/or cause allergic reactions. In the event of allergic reactions, the usual treatment is to avoid or stop using the allergy-triggering agent and, depending on the reaction, administer glucocorticoids, H1/H2 antagonists, fluids and adrenaline (intravenously).

Bleeding in oral, nasal and pharyngeal cavities and aspiration: Out-of-hospital anaesthesia involves a 14 to 20% higher risk of bleeding/secrections in oral, nasal and pharyngeal cavities as well as gastric-content aspiration than anaesthesia induced in hospital. When performing modified rapid sequence induction, personnel should have an operational suction device available at all times.

Hypoxia: In out-of-hospital anaesthesia, hypoxia occurs in 5 to 18% of all cases. Even short-term hypoxia increases mortality by a factor of 2.6 in patients with traumatic brain injury (TBI). In many cases, hypoxia persists over a longer period and incidence increases especially during rapid sequence induction. To ensure ideal conditions, the patient should be properly preoxegenated. Hypoxia may occur especially on account of failed or prolonged airway management.

Limited mouth opening: Before anaesthesia is induced, it should be verified that the mouth can open sufficiently (width of two fingers, if possible). If the mouth does not open sufficiently, the indications for anaesthesia induction must be critically evaluated. If a mechanical problem is the cause of limited mouth opening, personnel may try to insert a SGA device. If this cannot be done quickly, the patient should be carefully ventilated through a mask as an interim measure. As a last resort, emergency cricothyrotomy must be performed (part of the ‘forward strategy’ below). Difficult airway management: Please refer to the DGAI recommendations for out-of-hospital airway management and other literature. In the operating theatre, the incidence of a life-threatening ‘cannot ventilate, cannot intubate’ situation is approximately 0.4%. This figure is much higher in out-of-hospital situations. These rare complications may lead to the death of a patient in a very short time. In clinical anaesthesia and during elective surgery, a return to spontaneous breathing is an option in such cases. This is rarely successful, however, even if succinylcholine has been used, which has a short duration of action. If muscle relaxation has been induced by rocuronium, sugammadex can be used for reversal (within 3 to 4 min), which is faster than spontaneous recovery from the effects of succinylcholine. This option remains theoretical only, however, and is not considered in the algorithms for out-of-hospital airway management and anaesthesia (‘forward strategy’).

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The management of unexpected difficult airways in the out-of-hospital phase also follows the recommendation for out-of-hospital airway management of the DGAI. Apart from a proper assessment of tube placement, the stages of airway management escalate from mask ventilation to the use of SGAs, and, if required, surgical procedures to ensure sufficient oxygenation.

The best way to minimise the risk of an unexpected difficult airway is the early identification of patients who have difficult airways. For this reason, indicators of a difficult airway play a crucial role prior to induction of anaesthesia (Table 6). If several of these indicators are present, the induction of anaesthesia should be critically assessed. In some cases, a risk–benefit analysis will lead to the conclusion that anaesthesia must be avoided. Wherever possible, assistance should be requested or, if spontaneous breathing can be maintained, the patient should be transferred to a hospital notified in advance. A subjective inspection of patient physical characteristics helps experienced anaesthesiologists to assess a difficult airway. To obtain optimal intubation conditions in such cases, the authors recommend using a muscle relaxant when inducing anaesthesia, especially because a return to spontaneous breathing is only a theoretical option if the indication is correct (‘forward strategy’).

Alternative airway management options: The percentage of cases in which primary endotracheal intubation is not possible is significantly higher in out-of-hospital emergency medicine than in a hospital setting. As it can be assumed that out-of-hospital emergency patients have full stomachs, alternative airway management methods must be swiftly performed if primary intubation is not possible. It is essential to create optimal initial conditions (sufficient preoxygenation) and to use an introducer. The BURP (backward–upward–rightward pressure) manoeuvre and placing the patient in the improved Jackson’s position with the patient’s head elevated on a pillow causing an anterior movement of the skull (so called ‘sniffing position’), are two simple drug-free options for optimising the visualisation of the glottis. Although correct placement of the endotracheal tube can usually be achieved by making several intubation attempts, the risk of complications increases with each additional intubation attempt. If the glottic view is poor (according to Cormack/Lehane classification), personnel must check whether muscle relaxation has been induced. If not, this should be performed.

If endotracheal intubation is not possible, a quick and priority-oriented approach is necessary to prevent hypoxia and thus long-term damage to the patient:

1. Ensure oxygenation (target: SpO$_2$ $\geq$ 90%); the first measure is careful mask ventilation, with two assistants if required, also for patients with potentially full stomachs
2. If the laryngeal inlet is difficult to access, intubation catheters, if available, can be helpful in tracheal intubation.
3. SGA devices (e.g., laryngeal mask, laryngeal tube) should be used if the vocal cord level is not visible; their early use reduces the complication rate in airway management.
4. Video laryngoscopes can be used to facilitate laryngoscopy. The success rate of video laryngoscopic intubation is described as high for the induction of anaesthesia in the operating room and in out-of-hospital settings, but the time needed to ensure airway management is sometimes longer, and lower success rates in non-standard situations have been reported in some publications.
5. If the measures stated above should fail, emergency cricothyrotomy must be performed as a last resort to ensure sufficient oxygenation; the success rate and incidence of this procedure remain unclear, however.
6. All personnel performing these procedures must have sufficient practice, training and experience in the use of (airway) devices and their application.

### 1.4 Anaesthesia procedures for common emergency situations

#### 1.4.1 Severe trauma/major trauma
With respect to the induction and management of emergency anaesthesia, several important factors are encountered in major trauma patients:

1. Adverse situations (e.g., patient trapped in vehicle, construction site)
2. Difficult vascular access caused by hypovolaemia, hypothermia and vasoconstriction
3. Latent/acute/peracute hypovolaemia caused by haemorrhaging with circulatory instability
4. Lack of oxygen carriers with risk of hypoxia
5. Injuries complicating airway management

**Hypovolaemic/circulatory instability:** Major trauma patients often have latent hypovolaemia caused by haemorrhaging. This hypovolaemia can initially be compensated for or concealed by compensatory mechanisms (primarily in healthy young patients) or long-term medication (e.g., $\beta$-blockers in elderly patients). As many anaesthesiologists have the side-effect of...
of cardiovascular depression, significant hypotension may occur after the induction of anaesthesia. If latent hypovolaemia is likely, it is recommended that fluids be given prior to the induction of anaesthesia. As it has only a minor depressive effect on the circulatory system, ketamine is particularly suited for inducing emergency anaesthesia in obviously hypovolemic patients. Simultaneous use of catecholamines (e.g. 10 μg i.v. bolus dose of noradrenaline) may be necessary. Permissive hypotension (the tolerance of low normal BP levels) only applies to patients without TBI who have bleeding from noncompressible penetrating injuries. Recent studies have shown that mortality rates increase not only among patients with TBI but also among patients suffering from blunt trauma without TBI when SBP is below 110 mmHg.64

Reduced oxygenation: Trauma patients often exhibit hemorrhaging and reduced haemoglobin levels. In such situations, prehospital monitoring of oxygenation is suitable only to a limited degree, as pulse oximetry only measures levels of oxygenated haemoglobin. In order that physically dissolved oxygen can be used for the effective arterial oxygen content, major trauma patients should be ventilated with 100% oxygen until admission to the resuscitation room and subsequent arterial blood gas analysis in accordance with relevant guidelines. Respiration settings should comply with intensive medical requirements [maximum tidal volume: 6 ml kg−1 ideal body weight, initial respiratory frequency: 12 to 16/min, PEEP: 5 to 10 cmH2O (NB, Nota Bene: a tendency towards hypotension is associated with hypovolaemia as a result of reduced venous return), I : E ratio of 1:1 to 1:1.5].

Patient with cardiovascular failure: In general, there are two possible approaches for dealing with major trauma and haemodynamic instability:

(1) Approach 1: Titrated dosage of hypnotics in the lower range of the recommended dose to avoid further adverse effects on the cardiocirculatory stability of the major trauma patient but induction of anaesthesia under full relaxation.
(2) Approach 2: Ketamine-based anaesthetic, which does not depress circulation.

Both approaches require complete muscle relaxation, particularly in cases with concomitant TBI, to ensure optimum intubation conditions and to avoid intracranial pressure (ICP) peaks because of coughing or pressing. Both approaches have advantages and disadvantages. Although light anaesthesia always involves the risk of awareness, the use of ketamine increases the heart rate, BP and cardiac output (and thus the myocardial oxygen demand) of the patient.13 These considerations suggest that a mixed anaesthetic is the best choice. Table 7 shows a proposed standard procedure for inducing out-of-hospital emergency anaesthesia in cases of severe trauma (major trauma) and includes a selection of suitable anaesthetics.

### 1.4.2 Isolated neurotrauma, stroke, intracranial bleeding

Emergency anaesthesia is necessary for airway management in patients with isolated neurotrauma, a stroke or intracranial bleeding, particularly against the backdrop of impaired states of consciousness associated with an increased risk of hypoxia and aspiration. Table 8 shows a proposed standard procedure for inducing out-of-hospital emergency anaesthesia in cases of isolated neurotrauma, stroke or intracranial bleeding and includes a selection of suitable anaesthetics. General procedures

#### Table 7 Emergency anaesthesia for seriously injured patients

<table>
<thead>
<tr>
<th>Example: Road accident, 26-year-old man, SBP 100 mmHg, HR 110/min, S\textsubscript{O\textsubscript{2}} 86%, weight approximately 70 kg, traumatic brain injury, thoracic trauma, open fracture of the right femur, fracture of the left upper ankle joint, trapped in vehicle when emergency physician arrived</th>
</tr>
</thead>
<tbody>
<tr>
<td>Analgesiation for technical rescue with maintained spontaneous breathing:</td>
</tr>
<tr>
<td>3 mg midazolam i.v. + 25 mg esketamine i.v. (if necessary, repetitive administration of 10 mg every 20 min) + fluids via a suitable intravenous infusion solution</td>
</tr>
<tr>
<td>Anaesthesia preparation and preoxygenation</td>
</tr>
<tr>
<td>Anaesthesia induction(\uparrow) 200 mg thiopental or 7 mg midazolam or 100 mg propofol i.v. + 100 mg esketamine or 0.2 mg fentanyl or 20 μg sufentanil i.v. + 70 to 100 mg rocuronium or 100 mg succinylcholine i.v.</td>
</tr>
<tr>
<td>Airway management(\uparrow) if necessary, enhancement of anaesthesia with 3 to 5 mg midazolam i.v.</td>
</tr>
<tr>
<td>Anaesthesia maintenance(\uparrow) 3 to 5 mg midazolam i.v. (repeated approximately every 20 min) + 20 mg esketamine (repeated approximately every 20 min) or 0.15 mg fentanyl i.v. (repeated approximately every 20 min) + 20 mg rocuronium i.v. (repeated every 20 min)</td>
</tr>
</tbody>
</table>

\(\uparrow\) i.v., intravenous (ly). \(\downarrow\) Cardiovascular support with noradrenaline, 10 μg bolus dose administered according to target SBP or by a syringe pump. |
include elevating the upper body and immobilising the head in a neutral position. Appropriate BP management, normoventilation as well as the prevention of hypoxia, hypotension, coughing and pressing are important criteria for inducing anaesthesia in patients with isolated neurotrauma, a stroke or intracranial bleeding. Noradrenaline should be available for fractionated intravenous administration in 10 μg bolus doses during the induction phase and, if required, should subsequently be applied by a syringe pump.

Neurotrauma: As a surrogate parameter of cerebral perfusion pressure, BP is accepted as a decisive prognostic factor in cases of neurotrauma. Automatic BP monitoring at close intervals is required for this purpose. Although we are currently unable to specify exact BP target ranges, a SBP of 90 mmHg is considered to be the absolute lower limit.\textsuperscript{55} Even short phases below this critical limit can increase mortality in cases of neurotrauma. Significantly higher levels are more desirable, for example, an arterial mean pressure of 90 mmHg or a SBP above 120 mmHg.\textsuperscript{55,56}

Stroke/intracranial bleeding: In an out-of-hospital environment, it is impossible to differentiate between cerebral ischaemia and bleeding. In the penumbra surrounding the infarction core, cerebral blood flow is reduced and autoregulation is ineffective. The survival of nerve cells thus directly depends on systemic BP, and drops in BP should be prevented by all means in the acute phase.\textsuperscript{57,58} A systolic target of 180 mmHg and a diastolic target of 100 to 105 mmHg are recommended for patients with preexisting hypertension. For patients without a history of hypertension, lower target values are recommended (SBP/DBP: 140 to 180/90 to 100 mmHg). Systolic values above 220 mmHg and diastolic values above 120 mmHg should be lowered carefully.\textsuperscript{57,58} If signs of an ICP crisis (e.g., unequal pupils, Cushing reflex) persist after anaesthesia induction, an enhancement of anaesthesia (e.g., by thiopental or propofol bolus), administration of mannitol or short-term hyperventilation can be performed in accordance with the recommended guidelines.

### 1.4.3 The high-risk cardiac patient

Emergency anaesthesia may be necessary for a high-risk cardiac patient on account of acute cardiac failure (e.g., pulmonary oedema) or if a patient with a preexisting heart condition is involved in another emergency (e.g., trauma). If deterioration of oxygenation occurs (e.g., left ventricular failure with consecutive pulmonary oedema), the possibility of NIV under careful sedation must be considered for preoxygenation before anaesthesia is induced.\textsuperscript{59} Particularly in the event of deterioration of oxygenation, a spontaneously breathing high-risk cardiac patient should be extensively preoxygenated. Preference should be given to anaesthetics such as midazolam, etomidate, fentanyl and sufentanil that have little effect on the cardiovascular system (changes in inotropic state, preload and afterload; Table 9).\textsuperscript{59} High-risk cardiac patients often require catecholamines for circulatory support during anaesthesia induction and subsequently during the maintenance of anaesthesia when sympatho-adrenergic stimulation has ceased. Noradrenaline or adrenaline should be available for fractionated intravenous administration in 10 μg bolus doses during the induction phase and, if required, should be subsequently administered by a syringe pump.

### 1.4.4 Patients with respiratory insufficiency

There are many different reasons why patients with respiratory insufficiency may require out-of-hospital emergency anaesthesia. The underlying disorders

---

### Table 9  Emergency anaesthesia for high-risk cardiac patients

<table>
<thead>
<tr>
<th>Example: 76-year-old man, known three-vessel coronary disease, weight: approximately 70 kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variation 1: hypertensive crisis and consecutive pulmonary oedema, SBP 190 mmHg, HR 110/min, S\textsubscript{PO\textsubscript{2}} 84%, failure of noninvasive ventilation</td>
</tr>
<tr>
<td>Variation 2: cardiogenic shock, SBP 80 mmHg, HR 150/min, S\textsubscript{PO\textsubscript{2}} 83%</td>
</tr>
<tr>
<td>Variation 1\textsuperscript{a}:</td>
</tr>
<tr>
<td>Anaesthesia preparation and preoxygenation</td>
</tr>
<tr>
<td>Anaesthesia induction</td>
</tr>
<tr>
<td>+ 20 mg etomidate i.v.</td>
</tr>
<tr>
<td>+ 70 to 100 mg rocuronium or 70 mg succinylcholine i.v.</td>
</tr>
<tr>
<td>Airway management</td>
</tr>
<tr>
<td>Anaesthesia maintenance</td>
</tr>
<tr>
<td>+ 3 to 5 mg midazolam i.v. (repeated approximately every 20 min)</td>
</tr>
<tr>
<td>Variation 2\textsuperscript{a}:</td>
</tr>
<tr>
<td>Anaesthesia preparation during preoxygenation with 100% O\textsubscript{2} through tightly fitting face mask with oxygen reservoir for 3 to 4 min or continuation of noninvasive ventilation</td>
</tr>
<tr>
<td>Anaesthesia induction</td>
</tr>
<tr>
<td>+ 7 mg midazolam i.v.</td>
</tr>
<tr>
<td>+ 70 to 100 mg rocuronium or 70 mg succinylcholine i.v.</td>
</tr>
<tr>
<td>Airway management</td>
</tr>
<tr>
<td>Anaesthesia maintenance</td>
</tr>
<tr>
<td>+ 3 to 5 mg midazolam i.v. (repeated approximately every 20 min)</td>
</tr>
</tbody>
</table>

\textsuperscript{a}Circulatory support with noradrenaline, 10 μg bolus doses administered according to target SBP, syringe pump where required.
Table 10  Emergency anaesthesia for patients with respiratory insufficiency

<table>
<thead>
<tr>
<th>Example: 75-year-old woman with increasingly productive greenish sputum for 5 days at home, fever, left basal crackles, suspected pneumonia, SBP 140 mmHg, HR 110/min, SpO2 84%, weight: approximately 70 kg, increasing drowsiness in spite of noninvasive ventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaesthesia option 1:</td>
</tr>
<tr>
<td>----------------------</td>
</tr>
<tr>
<td>Anaesthesia induction</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Airway management</td>
</tr>
<tr>
<td>Anaesthesia maintenance</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Anaesthesia option 2:</th>
<th>Anaesthesia preparation, including preoxygenation with 100% O2 through tightly fitting face mask with oxygen reservoir for 3 to 4 min or continuation of noninvasive ventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaesthesia induction</td>
<td>+ 35 to 100 mg esketamine i.v.</td>
</tr>
<tr>
<td></td>
<td>+ 7 mg midazolam i.v.</td>
</tr>
<tr>
<td></td>
<td>+ 70 to 100 mg rocuronium or 100 mg succinylcholine i.v.</td>
</tr>
<tr>
<td>Airway management</td>
<td>If necessary, enhancement of anaesthesia with 3 to 5 mg midazolam i.v.</td>
</tr>
<tr>
<td>Anaesthesia maintenance</td>
<td>20 mg esketamine i.v. (repeated approximately every 20 min)</td>
</tr>
<tr>
<td></td>
<td>+ 3 to 5 mg midazolam i.v. (repeated approximately every 20 min)</td>
</tr>
</tbody>
</table>

i.v., intravenous (ly). *Circulatory support with noradrenaline, 10 μg bolus doses administered according to target SBP, syringe pump where required.

Comprise acute obstructions (e.g. asthma, COPD), acute oxygenation impairments (e.g. pulmonary oedema) and/or ventilation disorders (e.g. hypercapnia). Common risk factors in this group of patients are preexisting pulmonary and cardiovascular diseases, old age, nicotine abuse, a worsening general condition associated with a chronic course of disease and acute infections. If ventilation disorders are present, assisted ventilation (and possibly NIV) after appropriate analgesia and sedation (e.g. morphine) may be required during the preoxygenation and induction phases.32,60 Substances with a short onset time should be used for inducing anaesthesia (Table 10).31,61 Ideally, muscle relaxants should be used when inducing anaesthesia.62 It is advisable to induce deep anaesthesia using bronchodilatory/antiobstructive drugs (e.g. propofol, ketamine) that do not cause respiratory irritation, relax the smooth respiratory muscles, and do not lead to a release of histamines.63,64 Thiopental, atracurium, mivacurium and pancuronium should not be used on account of their side-effects.

1.5 Drugs for emergency anaesthesia

Depending on the location, a great variety of hypnotics, analgesics and muscle relaxants are stocked in rescue assets.55–67 When drugs are selected for inducing and maintaining anaesthesia, the physician’s knowledge of handling these substances, their availability and pharmacological properties as well as patient characteristics should be considered. Drugs with optimum pharmacokinetic and pharmacodynamic properties for emergency anaesthesia are characterised by a fast onset, a short duration of action, minor/no haemodynamic effects, minor/no adverse effects and rapid reversibility.13 This article provides an overview of the drugs most commonly used to induce and maintain emergency anaesthesia. Particularly in cases of critically ill or severely injured patients as well as those with unstable cardiopulmonary status, any drugs used for anaesthesia should be administered carefully or titrated to effect to avoid undesired hypotension or cardiac decompensation up to cardiovascular arrest.

1.5.1 Hypnotics

Propofol: Propofol (2,6-diisopropylphenol) has a purely hypnotic effect and has become the most commonly used hypnotic induction agent in hospitals.68 Apart from respiratory depression, propofol can also lead to a drop in BP owing to its negative inotropic effect and reduced peripheral vascular resistance (NB: reduction of cerebral perfusion pressure in case of TBI).59–71 These undesired effects are increased in hypovolaemic patients. Particular care should thus be taken when treating patients with cardiovascular insufficiency and/or hypovolaemia.69,72 Propofol is suitable for rapid sequence induction. This has, however, only been demonstrated in patients with stable circulation.72 Propofol is described as an alternative to barbiturate anaesthesia in controlling status epilepticus.73 Like barbiturates, propofol reduces cerebral blood flow and thus leads to a reduction in ICP, including in cases of isolated TBI. Owing to the narrow therapeutic range of propofol, its dosage depends on comorbidity and the opioid dose used. It should, therefore, only be applied by experienced physicians.68 Owing to the short half-life of propofol, repeated administration or alternative medication is required to maintain anaesthesia. Propofol infusion syndrome is not relevant to emergency medicine. Table 11 provides an overview of the most important characteristics of propofol.

Etomidate: Etomidate has a purely hypnotic effect. Haemodynamic stability and good intubation conditions are some of most convincing arguments in favour of using etomidate to induce anaesthesia.57 There are, however, numerous studies in which ketamine is rated as...
etomidate may cause adverse effects such as pain, myoclonus, and dyskinesia. In particular, pain on injection, histamine release, and increased ICP may occur, especially in cases of hypovolaemia, multiple organ failure, and increased ICP, with a higher mortality risk.

**Table 11: Midazolam**

<table>
<thead>
<tr>
<th>Dosage</th>
<th>Mechanism of action</th>
<th>Side-effects</th>
<th>Special characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaesthesia induction: 0.15 to 0.25 mg kg⁻¹ bodyweight i.v.; Onset: after 15 to 45 sec; Offset: after 1 to 3 min</td>
<td>Not entirely clear, hypnotic effect</td>
<td>Nausea and vomiting, mild respiratory depression, localised pain on injection, myoclonus</td>
<td>Reduced cortisol synthesis (11β-hydroxylase) even after a single bolus dose, with particular risk in case of sepsis and trauma (e.g. ARDS), multiple organ failure, longer hospital stay, increase in ventilation days, longer ICU stays (higher mortality), store at room temperature (below 25°C), protect from light</td>
</tr>
</tbody>
</table>

Midazolam can thus be considered an equivalent alternative to etomidate as a hypnotic used to induce and maintain anaesthesia in trauma patients. In addition, midazolam has a significantly longer half-life than etomidate. Midazolam should, however, always be combined with opioids or ketamine. Table 13 gives an overview of the most important characteristics of midazolam.

**Table 12: Etomidate**

<table>
<thead>
<tr>
<th>Dosage</th>
<th>Mechanism of action</th>
<th>Side-effects</th>
<th>Special characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaesthesia induction: 0.15 to 0.3 mg kg⁻¹ bodyweight i.v.; Onset: after 15 to 45 sec; Offset (half-life): after 3 to 12 min</td>
<td>Not entirely clear, hypnotic effect partly mediated through GABAergic mechanism</td>
<td>Nausea and vomiting, mild respiratory depression, localised pain on injection, myoclonus</td>
<td>Reduced cortisol synthesis (11β-hydroxylase) even after a single bolus dose, with particular risk in case of sepsis and trauma (e.g. ARDS), multiple organ failure, longer hospital stay, increase in ventilation days, longer ICU stays (higher mortality), store at room temperature (below 25°C), protect from light</td>
</tr>
</tbody>
</table>

Fentanyl and sufentanil are the opioids of choice in emergency anaesthesia. Different opioids have different degrees of analgesic, sedative, and antitussive effects. The side-effects of opioids include respiratory depression, sedation, bradycardia, hypotensive cardiovascular disorders, emesis, pruritus, bronchospasm, sweating, spasms of the bile and pancreatic ducts, constipation and miosis. Once it has been decided to perform out-of-hospital anaesthesia induction.

**Table 13: Propofol**

<table>
<thead>
<tr>
<th>Dosage</th>
<th>Mechanism of action</th>
<th>Side-effects</th>
<th>Special characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaesthesia induction: (1) 1.5 to 2.5 mg kg⁻¹ bodyweight i.v.; Anaesthesia maintenance: 3 (4) to 6 (12) mg kg⁻¹ bodyweight i.v.; or bolus dose of 0.25 to 0.5 mg kg⁻¹ bodyweight i.v.; Onset: after 15 to 45 s; Offset: after 5 to 10 min</td>
<td>GABA receptor agonist</td>
<td>Respiratory depression, apnoea, drop in blood pressure (negatively inotropic, reduced peripheral vascular resistance), especially in case of hypovolaemia, arousal phenomena, localised pain on injection, histamine release</td>
<td>Minor bronchodilatory effect, favourable in case of TBI and increased ICP, Store at room temperature (below 25°C), protect from light</td>
</tr>
</tbody>
</table>

GABA, Gamma-Aminobutyric Acid; ICP, intracranial pressure; i.v., intravenous (ly); TBI, traumatic brain injury. Reproduced with permission from.13

ARDS, acute respiratory distress syndrome; i.v., intravenous (ly). Reproduced with permission from.13

**Table 14: Thiopental**

<table>
<thead>
<tr>
<th>Dosage</th>
<th>Mechanism of action</th>
<th>Side-effects</th>
<th>Special characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaesthesia induction: (1) 1.5 to 2.5 mg kg⁻¹ bodyweight i.v.; Onset: after 15 to 45 sec; Offset (half-life): after 3 to 12 min</td>
<td>Not entirely clear, hypnotic effect partly mediated through GABAergic mechanism</td>
<td>Nausea and vomiting, mild respiratory depression, localised pain on injection, myoclonus</td>
<td>Reduced cortisol synthesis (11β-hydroxylase) even after a single bolus dose, with particular risk in case of sepsis and trauma (e.g. ARDS), multiple organ failure, longer hospital stay, increase in ventilation days, longer ICU stays (higher mortality), store at room temperature (below 25°C), protect from light</td>
</tr>
</tbody>
</table>

Thiopental: Thiopental is a barbiturate that has been used to induce anaesthesia in emergency medicine for many years (Table 14). This hypnotic agent is characterised by a quick onset and good reflex depression and depth of anaesthesia. Thiopental helps to reduce ICP (e.g. use in trauma patients with or without TBI). However, because of its vasodilator and negatively inotropic properties, thiopental may cause hypotension, particularly in patients with preexisting hypovolaemia. Volume management adjusted to the individual patient’s condition is recommended as a preventive measure; vasopressors can be used to provide compensation. Another relevant side-effect that must be mentioned is thiopental-induced histamine release, which, in extreme cases, may lead to bronchial obstruction.

**1.5.2 Analgesics**

Fentanyl and sufentanil are the opioids of choice in emergency anaesthesia. Different opioids have different degrees of analgesic, sedative, and antitussive effects. The side-effects of opioids include respiratory depression, sedation, bradycardia, hypotensive cardiovascular disorders, emesis, pruritus, bronchospasm, sweating, spasms of the bile and pancreatic ducts, constipation and miosis. Once it has been decided to perform out-of-hospital anaesthesia induction.
emergency anaesthesia, there are no absolute contraindica-
tions. This also applies to the strict indication during
pregnancy and nursing. Morphine or piritramide are not
recommended for inducing anaesthesia.

Fentanyl: Fentanyl can be used for analgesia as well as
anaesthesia induction and control. In small titrated doses,
it can also be used for analgesia alone while the patient
continues to breathe spontaneously (NB: close respira-
.tory monitoring; Table 15).

Sufentanil: This opioid has the highest affinity for
µ-receptors. Sufentanil can be administered both as a
bolus dose and with a syringe pump (Table 16). However,
it has not been approved for use as a pure analgesic
without intubation anaesthesia. Its range of out-of-
hospital applications is, therefore, limited.

Ketamine: Ketamine plays a special role in emergency
medicine, as, depending on the dose, this substance can
be used both for analgesia and for complete induction and
maintenance of anaesthesia. Ketamine causes dissoci-
ative anaesthesia, which is associated with catalepsy,
amnesia and analgesia. Depending on the dose, the
patient’s protective reflexes and spontaneous breathing
are not affected. Adverse effects include arousal and
nightmares, which makes concomitant medication with
a benzodiazepine obligatory. Sensitivity to sound and
hypersalivation may also occur. Particularly patients
who are trapped or difficult to reach may benefit
from analgesia and sedation based on ketamine and a
benzodiazepine, because spontaneous breathing and
circulatory stability are maintained in most cases. It
should be noted that, apart from racemic ketamine, the
S-enantiomer esketamine is available with considerably
different dosage recommendations. The most important
characteristics of esketamine are summarised in
Table 17, those of ketamine in Table 18.

### Table 13  Midazolam

<table>
<thead>
<tr>
<th>Dosage</th>
<th>Mechanism of action</th>
<th>Side-effects</th>
<th>Special characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaesthesia induction: 0.15 to 0.2 mg kg⁻¹ bodyweight i.v.; anaesthesia maintenance: 0.03 to 0.2 mg kg⁻¹ bodyweight i.v.; onset: after 60 to 90 s; Offset (half-life): after 1 to 4 h</td>
<td>Binding to the α-subunit of the GABA receptor causes prolonged opening of chloride channels, thus enhancing the effect of the inhibitory CNS transmitter GABA</td>
<td>Paradoxical arousal; NB: combination with alcohol (increased effect of alcohol), respiratory failure when combined with opioids</td>
<td>NB: dosage errors because of confusion when 5 mg/5 ml (=1 mg/ml) ampoules and 15 mg/3 ml (=5 mg/ml) ampoules are stored together, storage: protect from light</td>
</tr>
</tbody>
</table>

CNS, central nervous system; GABA, gamma-aminobutyric acid; i.v., intravenous (ly). Reproduced with permission from.¹³

### Table 14  Thiopental

<table>
<thead>
<tr>
<th>Dosage</th>
<th>Mechanism of action</th>
<th>Side-effects</th>
<th>Special characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaesthesia induction: 3 to 5 mg kg⁻¹ bodyweight i.v.; onset: after 10 to 20 s; offset: after 6 to 8 min</td>
<td>GABA receptor agonist</td>
<td>Respiratory depression, hypotension, histamine release</td>
<td>Dry substance, must be dissolved prior to application, NB: extravasation leads to necrosis</td>
</tr>
</tbody>
</table>

GABA, gamma-aminobutyric acid; i.v., intravenous (ly).

### Table 15  Fentanyl

<table>
<thead>
<tr>
<th>Dosage</th>
<th>Mechanism of action</th>
<th>Side-effects</th>
<th>Special characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anaesthesia induction: initially 2 μg kg⁻¹ bodyweight i.v., anaesthesia maintenance: 1 to 3 μg kg⁻¹ bodyweight i.v.; onset: after &lt;30 s; Offset (mean): after 0.3 to 0.5 h</td>
<td>Pure opiate receptor agonist with high affinity for µ-receptors and weak κ-receptor affinity</td>
<td>Respiratory depression, muscle rigidity, hypotension (especially in case of hypovolaemia), bradycardia</td>
<td>Antidote: naloxone Storage: protect from light</td>
</tr>
</tbody>
</table>

### Table 16  Sufentanil

<table>
<thead>
<tr>
<th>Dosage</th>
<th>Mechanism of action</th>
<th>Side-effects</th>
<th>Special characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initially 0.15 to 0.7 μg kg⁻¹ bodyweight i.v.; + 0.15 μg kg⁻¹ bodyweight i.v., repeated; onset: after &lt;2 to 3 min; Offset (mean): after 0.2 to 0.3 h</td>
<td>Pure opiate receptor agonist with high affinity for µ-receptors and weak κ-receptor affinity</td>
<td>Respiratory depression, muscle rigidity, hypotension (especially in case of hypovolaemia), bradycardia</td>
<td>Antidote: naloxone Storage: protect from light</td>
</tr>
</tbody>
</table>

i.v., intravenous (ly).
1.5.3 Muscle relaxants

Muscle relaxation is an integral part of rapid sequence induction and emergency anaesthesia management. Table 19 gives an overview of the advantages and disadvantages of muscle relaxation.

A short onset time is an important criterion when selecting a relaxant to induce anaesthesia. Rocuronium and succinylcholine are the only suitable drugs available. Succinylcholine is the most commonly used drug for this application (Table 20). However, hyperkalaemia (e.g. in patients who have been immobilised for more than 24 h and in patients with serious (burn) injuries) and malignant hyperthermia (e.g. predisposition) are relevant contraindications to the use of succinylcholine. The advantage of succinylcholine over rocuronium consists in its significantly shorter duration of effect and its lower price. As sugammadex has become available as an effective substance for blockade reversal, the prehospital use of rocuronium has been discussed in recent studies. So far, however, the data available is not sufficient to decide whether sugammadex must always be available when rocuronium is used in a prehospital setting. As rocuronium has a longer half-life, repeated prehospital administration is rarely required. The specific features of the other, nondepolarising muscle relaxants are summarised in Tables 21 and 22.

Table 17  ESKETAMINE

<table>
<thead>
<tr>
<th>Dosage</th>
<th>Mechanism of action</th>
<th>Side-effects</th>
<th>Special characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.25 to 0.5 mg kg⁻¹ bodyweight i.v. for analgesia with protective reflexes maintained; 0.5 to 1 mg kg⁻¹ bodyweight i.v. for anaesthesia induction or 1.5 to 5 mg kg⁻¹ bodyweight i.m.; onset (i.v.): after 30 s; Offset (i.v.): after 5 to 15 min</td>
<td>Noncompetitive antagonism at NMDA receptors; Agonist at opiate receptors; inhibition of peripheral catecholamine reuptake; Influence on central and peripheral monoaminergic and cholinergic transmission, causes dissociative anaesthesia</td>
<td>Sympathomimetic: increase in heart rate and blood pressure, respiratory depression, apnoea, increased reflexes in the pharyngeal and laryngeal areas (NB: laryngospasm when suctioning/ intubation is performed), anxiety, hallucinations</td>
<td>Esketamine decreases ICP and can be administered in case of TBI, careful use in cases of severe cardiac failure, storage below 0°C must be avoided because of the risk of container breakage</td>
</tr>
</tbody>
</table>

Table 18  KETAMINE

<table>
<thead>
<tr>
<th>Dosage</th>
<th>Mechanism of action</th>
<th>Side-effects</th>
<th>Special characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.5 to 1 mg kg⁻¹ bodyweight i.v. for analgesia with protective reflexes maintained; 1 to 2 mg kg⁻¹ bodyweight i.v. for anaesthesia induction or 4 to 10 mg kg⁻¹ bodyweight i.m.; onset (i.v.): after 30 s; Offset (i.v.): after 5 to 15 min</td>
<td>Noncompetitive antagonism at NMDA receptors, agonist at opiate receptors, inhibition of peripheral catecholamine reuptake, influence on central and peripheral monoaminergic and cholinergic transmission, causes dissociative anaesthesia</td>
<td>Sympathomimetic: increase in heart rate and blood pressure, respiratory depression, apnoea, increased reflexes in the pharyngeal and laryngeal areas (NB: laryngospasm during suctioning/intubation), anxiety, hallucinations</td>
<td>Ketamine decreases ICP and can be administered in case of TBI, careful use in cases of severe cardiac failure, bronchial inflammatory effect on asthmatic patients, storage below 0°C must be avoided because of risk of container breakage</td>
</tr>
</tbody>
</table>

Table 19  Pros and cons of muscle relaxation in emergency anaesthesia

<table>
<thead>
<tr>
<th>Pros</th>
<th>Cons</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improves conditions for laryngoscopy</td>
<td>In case of neuromuscular blockade, the patient stops breathing spontaneously – oesophageal intubations are always fatal [capnography]</td>
</tr>
<tr>
<td>Improves conditions for intubation</td>
<td>‘Cannot ventilate, cannot intubate’ situations may arise</td>
</tr>
<tr>
<td>Avoids high doses of hypnotics</td>
<td>Succinylcholine is associated with a risk in case of existing or developing hyperkalaemia</td>
</tr>
<tr>
<td>Avoids ICP peaks in cases of TBI</td>
<td>The success rate of endotracheal intubation is increased when muscle relaxants are administered by experienced staff</td>
</tr>
</tbody>
</table>

ICP, intracranial pressure; TBI, traumatic brain injury.


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