EJA

Eur J Anaesthesiol 2016; **33:**881–897

SPECIAL ARTICLE

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

Bjoern Hossfeld, Bertold Bein, Bernd W. Boettiger, Andreas Bohn, Matthias Fischer, Jan-Thorsten Graesner, Jochen Hinkelbein, Clemens Kill, Carsten Lott, Erik Popp, Markus Roessler, Alin Schaumberg, Volker Wenzel and Michael Bernhard

Published online 14 September 2016

Table of content

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

From the Department of Anaesthesiology and Intensive Care Medicine, Section Emergency Medicine, Armed Forces Hospital, Ulm (BH); Department of Anaesthesiology, Intensive Care Medicine, Emergency Medicine, Pain Service, ASKLEPIOS Hospital St. Georg, Hamburg (BB); Department of Anaesthesiology and Intensive Care Medicine, University Hospital of Cologne, Cologne (BWB, JH); City of Münster Fire Department, Münster (AB); Department of Anaesthesiology, Intensive Care Medicine, Emergency Medicine, Pain Service, Hospital Am Eichert, Goeppingen (MF); Department of Emergency Medicine, University of Schleswig-Holstein, Kiel (J-TG); Department of Emergency Medicine, University of Giessen and Marburg, Campus Marburg (CK); Department of Anesthesiology, University Medical Center, Johannes Gutenberg-University of Mainz, Mainz (CL); Department of Anesthesiology, University of Heidelberg, Heidelberg (EP); Department of Anesthesiology, University Medical Center Göttingen, Göttingen (MR); Department of Anaesthesiologie and Intensive Care Medicine University of Giessen and Marburg, Campus Giessen, Germany (AS); Department of Anesthesiology and Critical Care Medicine, Innsbruck Medical University, Innsbruck, Austria (VW); Emergency Department, University of Leipzig, Leipzig, Germany (MB)

Correspondence to Bjoern Hossfeld, LtCol MD, Department of Anaesthesiology and Intensive Care Medicine, Section Emergency Medicine, Armed Forces Hospital Ulm, Oberer Eselsberg 40, 89081 Ulm, Germany
Tel: +49 731 1710 26501; e-mail: bjoern.hossfeld@extern.uni-ulm.de

DOI:10.1097/EJA.0000000000000533

DGAI German Society of Anaesthesiology and Intensive Care Medicine

DIVI German Interdisciplinary Association for Intensive Care and Emergency Medicine

$etCO_2$ End-tidal carbon dioxide

GCS Glasgow Coma Score

ICP Intracranial pressure

ICU Intensive Care Unit

LIKS AirRescue Information and Communication Systems

MIND German minimal emergency data record

NIBP Noninvasive Blood Pressure

NIV Noninvasive Ventilation

BP Blood Pressure (according to Riva Rocci)

SOP Standard operating procedure

SpO_2 Oxygen saturation

TBI Traumatic Brain Injury

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. Rapid sequence induction of emergency anaesthesia includes standard monitoring, preoxygenation, standardised preparation of emergency anaesthesia, drug administration, manual in-line stabilisation during intubation (if necessary), airway management and checking of correct tube placement. Spontaneously breathing casualties should receive preoxygenation for at least 3 to 4 min with a tight-fitting facemask with reservoir using 12 to 15 l min_1 of oxygen or with a demand valve providing 100% oxygen. As an alternative, preoxygenation may be performed as noninvasive ventilation with 100% oxygen. Standardised anaesthesia preparation comprises filling drugs into syringes and labelling them, checking ventilation equipment, preparing endotracheal tube and syringe for inflating the cuff and the introducer, stethoscope and fixation material, preparing alternative instruments for airway management as well as checking suction, ventilation and standard monitoring devices, including capnography. Standard monitoring for out-of-hospital emergency anaesthesia comprises ECG, blood pressure measurement and pulse oximetry. Continuous capnography is always and exclusively performed to check the placement of airway devices, as well as to indirectly monitor haemodynamics.

1.1 Rationale, frequency and indication

1.1.1 Rationale

Emergency anaesthesia, airway management and ventilation are important therapeutic measures in emergency medicine.[1,2] 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.[3] 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.[4,5]

The following recommendations of the German Society of Anaesthesiology and Intensive Care Medicine (DGAI) have been prepared for EMS-physicians

Table 1 Overview of the recommendations for out-of-hospital emergency anaesthesia

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, standardised 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)

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.

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 agreement of 12 out of 14 participants (> 85%) could be reached.

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.[7] Out-of-hospital anaesthesia is induced in approximate 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, GSC <9), uncooperative, severely injured or critically ill patients with a full stomach and unstable cardiopulmonary conditions.[13] 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.[14] 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.[15,16] 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.[17] Tables 2 and 3 provide an overview of the indications for and objectives of out-of-hospital emergency anaesthesia.

If emergency anaesthesia is required, personnel must take into account the guideline on out-of-hospital airway management[18] by the DGAI as well as the information on emergency anaesthesia, airway management and ventilation in the German S3 guideline on treatment of major

Table 2 Indications for out-of-hospital emergency anaesthesia

Acute respiratory insufficiency (hypoxia and/or respiratory rate[a] <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 SpO_2 < 90% despite oxygenation or
craniocerebral trauma with GCS < 9

[a] If causes are not rapidly reversible.

Table 3 Objectives of out-of-hospital emergency anaesthesia

Amnesia
Anxiolysis
Stress reduction
Hypnosis
Pain management through comprehensive and sufficient analgesia
Rapid and effective airway management (with oxygenation and ventilation as well as protection against pulmonary aspiration through endotracheal intubation)
Reduction of oxygen consumption
Protection of vital organs and avoidance of secondary myocardial and cerebral damage

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.[19] 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-hospital anaesthesia '... should have the same level of training and competence that would allow them to perform unsupervised ...' emergency intubation '... in the emergency department'.[7,20,21]

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.[1,4,22] According to Timmermann *et al.*[18], 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.[23]

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).[24] If peripheral venous cannulation is difficult, anaesthetics may also be administered through intraosseous access.[25,26] All drugs mentioned below may be administered through intraosseous access using the same dose.

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 (SpO_2) of less than 70% after only 1 to 2 min of apnoea despite preoxygenation.[27] 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.[28,29]

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.[13] 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.[30] Airway management equipment must be prepared and checked for functionality (Table 4).

Table 4 Standardised preparation of emergency anaesthesia equipment

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

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.

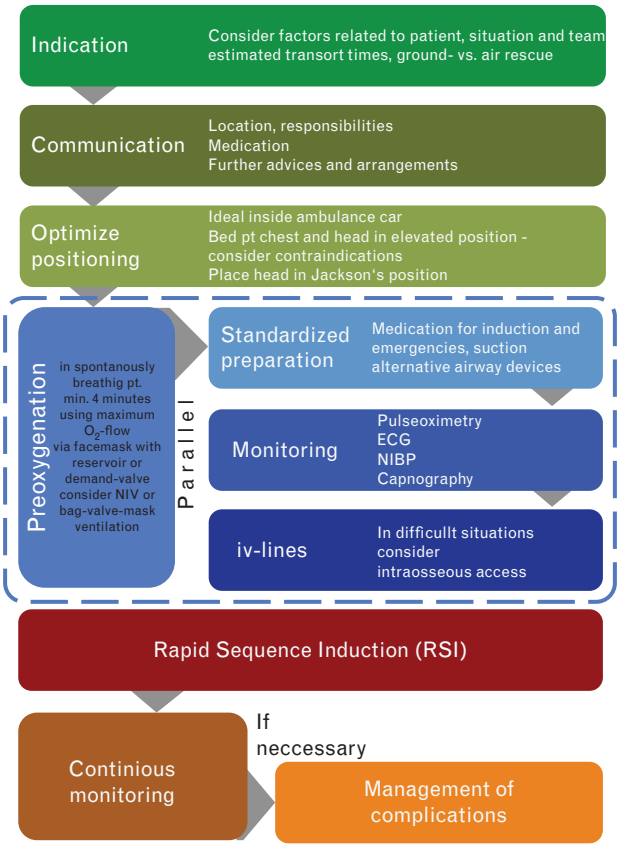
After paramedics prepare the drugs and the equipment for airway management and ventilation as instructed by the EMS-physician, preoxygenation is initiated as soon as the EMS-physician decides to induce emergency anaesthesia (Fig. 1). To prevent desaturation during anaesthesia induction and airway management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

Table 5 Standardised performance of out-of-hospital anaesthesia

Critically evaluate indications for emergency anaesthesia
Inform all team members about indications for emergency anaesthesia
Optimise out-of-hospital conditions (e.g. transport of patient into ambulance, head position)
Immediately begin preoxygenation of spontaneously breathing patients
Prepare drugs and airway management equipment (Table 2)
Monitor patient (place ECG electrodes, SpO_2, automatic NIBP, have capnograph ready)
Place and fix two peripheral venous catheters with continuous infusions (if possible)
Perform rapid sequence induction
If necessary, remove cervical collar and start manual in-line stabilisation
Call out drugs, indicating the active agent and dose; consecutive administration
Wait for loss of consciousness and muscle relaxation
Airway management without intermittent ventilation in normoxic patients
Confirm tube placement (capnography, auscultation, insertion depth)
If necessary, stop manual in-line stabilisation and close cervical collar
Perform continuous monitoring, including continuous capnography and adjust ventilation device
Maintain and monitor anaesthesia
Recognise and treat problems regarding vital signs
Manage complications if necessary

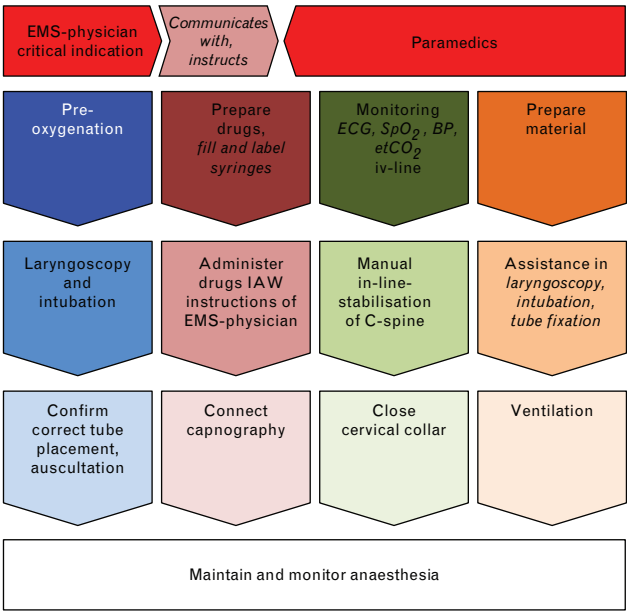
NIBP, noninvasive blood pressure.

Fig. 1



Schematic diagram of the performance and procedure of out-of-hospital emergency anaesthesia.

Fig. 2



Phase model of the performance and procedure of out-of-hospital emergency anaesthesia.

patients are given oxygen for 3 to 4 min, whenever possible.[31,32] 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.[32] 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 SpO_2). 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 http://www.divi.de/images/Dokumente/Empfehlungen/Spritzenetiketten/DIVI-Etiketten-Empfehlung_2012_07_02.pdf, Assessed 16 May 2016)

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).[32,33] 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.[33–35] 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[36]: 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).[37]

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.[38,39] Continuous automated oscillometric BP measurement is, therefore, vital. Patients with acute hypovolaemia have an increased risk of hypotension. Treat hypotension with fluids, cafedrine–theodrenaline, 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, H_1/H_2 antagonists, fluids and adrenaline (intravenously).

Bleeding in oral, nasal and pharyngeal cavities and aspiration: Out-of-hospital anaesthesia involves a 14 to 20% [12] higher risk of bleeding/secretions 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.[38–40] Even short-term hypoxia increases mortality by a factor of 2.6 in patients with traumatic brain injury (TBI).[41] In many cases, hypoxia persists over a longer period and incidence increases especially during rapid sequence induction.[32] To ensure ideal conditions, the patient should be properly preoxygenated.[40] Hypoxia may occur especially on account of failed or prolonged airway management.[40]

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).[18,42]

Difficult airway management: Please refer to the DGAI recommendations for out-of-hospital airway management and other literature.[18,42] In the operating theatre, the incidence of a life-threatening 'cannot ventilate, cannot intubate' situation is approximately 0.4%.[43] This figure is much higher in out-of-hospital situations.[5] These rare complications may lead to the death of a patient in a very short time.[36] 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.[44] 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.[45] This option remains theoretical only, however, and is not considered in the algorithms for out-of-hospital airway management and anaesthesia ('forward strategy').

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.[18] 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.[36] A subjective inspection of patient physical characteristics helps experienced anaesthesiologists to assess a difficult airway.[46] 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.[39] 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.[36] 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.[47] 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.[48] 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.

Table 6 Indicators of difficult airways

Diseases/injuries: major trauma, (mid)facial trauma, sleep apnoea syndrome
Patient characteristics: beards, short/strong neck, reduced mobility in the cervical spine area, pregnancy, obesity (BMI $> 30\,kg\,m^{-2}$), Mallampati score III and IV, reduced thyromental distance, men, previous irradiation/surgery in the neck area

Reproduced with permission from.[42]

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[18,36]:

(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.[49]
(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,[28,42] 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.[50,51]
(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.[1]
(6) All personnel performing these procedures must have sufficient practice, training and experience in the use of (airway) devices and their application.[52]

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

Hypovolaemia/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. β-blockers in elderly patients).[53] As many anaesthetics have the side-effect

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.[54]

Reduced oxygenation: Trauma patients often exhibit haemorrhaging 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 cmH_2O (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.

Table 7 Emergency anaesthesia for seriously injured patients

Example: Road accident, 26-year-old man, SBP 100 mmHg, HR 110/min, S_pO_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
Analgosedation for technical rescue with maintained spontaneous breathing:
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
Anaesthesia preparation and preoxygenation
Anaesthesia induction[a]
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.
Airway management[a]
if necessary, enhancement of anaesthesia with 3 to 5 mg midazolam i.v.
Anaesthesia maintenance[a]
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)

i.v., intravenous (ly). [a]Cardiovascular support with noradrenaline, 10 µg bolus dose administered according to target SBP or by a syringe pump.

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 8 Emergency anaesthesia for patients with neurotrauma/stroke/intracranial bleeding

Example: 65-year-old woman, SBP 160 mmHg, HR 70/min, S_pO_2 92%, weight approx. 70 kg, her husband had found her lying in bed, the blanket is soiled by vomit, unequal pupil sizes (right > left), GCS 6	
Anaesthesia preparation and preoxygenation	
Anaesthesia induction[a]	300 mg thiopental[b] or 140 mg propofol i.v.
	+ 0.2 mg fentanyl or 20 µg sufentanil or 100 mg esketamine i.v.
	+ 70 to 100 mg rocuronium or 70 mg succinylcholine i.v.
Airway management[a]	if necessary, enhancement of anaesthesia with 3 to 5 mg midazolam i.v.
Anaesthesia maintenance[a]	3 to 5 mg midazolam i.v. (repeated approximately every 20 min)
	+ 0.15 mg fentanyl i.v. (repeated approximately every 20 min)

i.v., intravenous (ly). [a]Cardiovascular support with noradrenaline, 10 µg bolus doses administered according to target SBP or by a syringe pump. [b]Barbiturates are traditional anaesthetics for lowering intracranial pressure and should be given preference by the emergency physician, whenever possible.

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.[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.[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.[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. [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.[32] 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).[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

Example: 76-year-old man, known three-vessel coronary disease, weight: approximately 70 kg	
Variation 1: hypertensive crisis and consecutive pulmonary oedema, SBP 190 mmHg, HR 110/min, S_pO_2 84%, failure of noninvasive ventilation	
Variation 2: cardiogenic shock, SBP 80 mmHg, HR 150/min, S_pO_2 83%	
Variation 1[a]:	
Anaesthesia preparation and preoxygenation	
Anaesthesia induction	0.2 mg fentanyl or 20 μg sufentanil i.v.
	+ 20 mg etomidate i.v.
	+ 70 to 100 mg rocuronium or 70 mg succinylcholine i.v.
Airway management	if necessary, enhancement of anaesthesia with 3 to 5 mg midazolam i.v.
Anaesthesia maintenance	0.1 mg fentanyl i.v. (repeated approximately every 20 min)
	+ 3 to 5 mg midazolam i.v. (repeated approximately every 20 min)
Variation 2[a]:	
Anaesthesia preparation during preoxygenation with 100% O_2 through tightly fitting face mask with oxygen reservoir for 3 to 4 min or continuation of noninvasive ventilation	
Anaesthesia induction	0.2 mg fentanyl or 20 μg sufentanil i.v.
	+ 7 mg midazolam i.v.
	+ 70 to 100 mg rocuronium or 70 mg succinylcholine i.v.
Airway management	If necessary, enhancement of anaesthesia with 3 to 5 mg midazolam i.v.
Anaesthesia maintenance	0.1 mg fentanyl i.v. (repeated approximately every 20 min)
	+ 3 to 5 mg midazolam i.v. (repeated approximately every 20 min)

i.v., intravenous (ly). [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

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	
Anaesthesia option 1[a]:	
Anaesthesia preparation and preoxygenation	
Anaesthesia induction	0.2 mg fentanyl or 20 μg sufentanil i.v.
	+ 110 to 160 mg propofol or 20 mg etomidate i.v.
	+ 70 to 100 mg rocuronium or 100 mg succinylcholine i.v.
Airway management	If necessary, enhancement of anaesthesia with 3 to 5 mg midazolam i.v.
Anaesthesia maintenance	0.15 mg fentanyl i.v. (repeated approximately every 20 min)
	+ 3 to 5 mg midazolam i.v. (repeated approximately every 20 min)
Anaesthesia option 2[a]:	
Anaesthesia preparation, including preoxygenation with 100% O_2 through tightly fitting face mask with oxygen reservoir for 3 to 4 min or continuation of noninvasive ventilation	
Anaesthesia induction	+ 35 to 100 mg esketamine i.v.
	+ 7 mg midazolam i.v.
	+ 70 to 100 mg rocuronium or 100 mg succinylcholine i.v.
Airway management	if necessary, enhancement of anaesthesia with 3 to 5 mg midazolam i.v.
Anaesthesia maintenance	20 mg esketamine i.v. (repeated approximately every 20 min)
	+ 3 to 5 mg midazolam i.v. (repeated approximately every 20 min)

i.v., intravenous (ly). [a] 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.[65–67] 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).[69–71] These undesired effects are increased in hypovolaemic patients. Particular care should thus be taken when treating patients with cardiovascular insufficiency and/or hypovolaemia.[68,72] 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

Table 11 Propofol

Dosage	Mechanism of action	Side-effects	Special characteristics
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/h 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	GABA receptor agonist	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	Minor bronchodilatory effect, favourable in case of TBI and increased ICP, Store at room temperature (below 25°C), protect from light

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

equivalent to etomidate in terms of intubation success and cardiovascular stability.[74,75] Etomidate may cause both myoclonus and dyskinesia (NB: mask ventilation may be complicated; Table 12). Prior application of a benzodiazepine prevents myoclonus.

The significance of cortisol synthesis inhibition in the adrenal cortex and the associated increase in possible complications (e.g. acute respiratory distress syndrome, multiple organ failure, longer hospital stay, increase in ventilation days, longer ICU stays, higher mortality) is the subject of controversial discussion.[76] The S3 guideline on treatment of major trauma/severe injuries and the revision of the S2k guideline on sepsis recommend that the use of etomidate should be carefully considered.[77] In contrast, according to the American Eastern Association for the Surgery of Trauma guidelines, there is no evidence against the use of etomidate as an hypnotic induction agent.[78] In light of unclear evidence and the principle of 'primum non nocere', one review does not recommend the use of etomidate, especially in septic patients.[79]

Given the side-effects described and their relevant impact on morbidity and mortality, which has not yet been conclusively determined, the authors believe that the use of etomidate can be completely abandoned in favour of other anaesthetic agents.

Midazolam: As a fast-acting benzodiazepine with a short duration of effect, midazolam possesses a wide therapeutic range in the treatment of anxiety, arousal and stress. Several studies showed no significant differences between midazolam and etomidate regarding intubation conditions and drops in BP during rapid sequence induction.[80,81] 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.[13] Table 13 gives an overview of the most important characteristics of midazolam.

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

Table 12 Etomidate

Dosage	Mechanism of action	Side-effects	Special characteristics
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	Not entirely clear, hypnotic effect partly mediated through GABAergic mechanism	Nausea and vomiting, mild respiratory depression, localised pain on injection, myoclonus	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

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

Table 13 Midazolam

Dosage	Mechanism of action	Side-effects	Special characteristics
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	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	Paradoxical arousal; NB: combination with alcohol (increased effect of alcohol), respiratory failure when combined with opioids	NB: dosage errors because of confusion when 5 mg/5 ml (=1 mg/ml) ampoules and 15 mg/3 ml (=5 mg/ml) ampules are stored together, storage: protect from light

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

Table 14 Thiopental

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

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

Table 15 Fentanyl

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

i.v., intravenous (ly). Reproduced with permission from.[13]

emergency anaesthesia, there are no absolute contraindications. 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 respiratory 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 dissociative 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 16 Sufentanil

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

i.v., intravenous (ly).

Table 17 Esketamine

Dosage	Mechanism of action	Side-effects	Special characteristics
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.): 5 to 15 min	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	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	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

ICP, intracranial pressure; i.v., intravenous (ly); NMDA, *N*-methyl-D-aspartate; TBI, traumatic brain injury. Reproduced with permission from.[13]

Table 18 Ketamine

Dosage	Mechanism of action	Side-effects	Special characteristics
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	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	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	ketamine decreases ICP and can be administered in case of TBI, careful use in cases of severe cardiac failure, bronchodilatory effect on asthmatic patients, storage below 0°C must be avoided because of risk of container breakage

ICP, intracranial pressure; i.v., intravenous (ly); NMDA, *N*-methyl-D-aspartate; TBI, traumatic brain injury. Reproduced with permission from[13].

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.[33] 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.[29,61,82,83] 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.

1.5.4 Storage instructions for emergency drugs

The need to refrigerate various drugs used for emergency anaesthesia is a particularly important aspect of storage precautions. Such drugs are labelled 'Store in refrigerator'. This means storage at +2 to +8°C (refrigerator temperature). Drugs requiring cool storage can also be temporarily stored and transported at normal room temperature but their quality will deteriorate. Many manufacturers have conducted stability tests and provide recommendations on shelf life if continuous cool storage cannot be ensured. The following recommendations (Table 23) assume a normal room temperature of 25°C.

Table 19 Pros and cons of muscle relaxation in emergency anaesthesia

Pros	Cons
Improves conditions for laryngoscopy[28]	In case of neuromuscular blockade, the patient stops breathing spontaneously – oesophageal intubations are always fatal (capnography)[60]
Improves conditions for intubation[60]	'Cannot ventilate, cannot intubate' situations may arise[28]
Avoids high doses of hypnotics[28]	Succinylcholine is associated with a risk in case of existing or developing hyperkalaemia[81]
Avoids ICP peaks in cases of TBI[28]	
The success rate of endotracheal intubation is increased when muscle relaxants are administered by experienced staff[20]	

ICP, intracranial pressure; TBI, traumatic brain injury.

Table 20 Succinylcholine

Dosage	Mechanism of action	Side-effects	Special characteristics
Single dose: 1.0 to 1.5 mg kg^{-1} bodyweight i.v. across all age groups; onset: after 60 to 90 s; offset: 3 to 6 min	The only depolarising muscle relaxant, effect at the nicotinic acetylcholine receptor on the motor end plate	Arrhythmias, tachycardia, bradycardia, potassium release up to asystole, abnormal blood pressure, muscle pains following fasciculation, allergic reactions, increase in intraocular pressure (NB: penetrating injuries), increase in intragastric pressure, hypersalivation, jaw muscle rigidity (up to 60 s), malignant hyperthermia	Increased sensitivity in patients with neuromuscular diseases (reduce dose if required), precurarisation with nondepolarising muscle relaxants reduces side effects, noticeable rigidity of the masseter muscle is considered a warning sign of rhabdomyolysis or malignant hyperthermia, if cholinesterase activity is reduced, the duration of effect is extended, for storage see Chapter 1.5.4

i.v., intravenous (ly). Reproduced with permission from.[13,32]

Table 21 Rocuronium

Dosage	Mechanism of action	Side-effects	Special characteristics
For rapid sequence induction: 1.0 to 1.2 mg kg^{-1} bodyweight i.v.; dose for geriatric patients: 0.6 mg kg^{-1} bodyweight i.v. (duration of effect may be prolonged); onset: after 60 to 120 s; offset: after 30 to 67 min	Nondepolarising neuromuscular blockade with medium duration of action, competitive effect at the nicotinic acetylcholine receptor on the motor end plate	Tachycardia, pain on injection, allergic reaction	Reversible with sugammadex, physically incompatible with: dexamethasone, diazepam, furosemide, hydrocortisone sodium succinate, insulin, intralipid, methylprednisolone, prednisolone sodium succinate, thiopental, for storage see Chapter 1.5.4

i.v., intravenous (ly). Reproduced with permission from.[13,32]

Table 22 Vecuronium

Dosage	Mechanism of action	Side-effects	Special characteristics
Relaxation after intubation with succinylcholine: 0.03 to 0.05 mg kg^{-1} bodyweight i.v.; onset: after 2 to 4 min; offset: after 20 to 60 min	Competitive binding at the motor end plate	Anaphylactic reactions (rare)	Reversible with sugammadex, increased potency in patients with hypokalaemia, hypermagnesaemia, hypocalcaemia, dehydration, acidosis, hypercapnia, cachexia, store below 25°C and protect from light

i.v., intravenous (ly). Reproduced with permission from.[13]

Table 23 Drugs requiring refrigeration

Substance	Shelf life at 25 to 30°C
Rocuronium	up to 3 months* at +8 to +30°C, do not refrigerate again once removed from refrigerator
Succinylcholine	up to 1 month* if a storage temperature of 25°C is not exceeded, up to 7 days to ensure safety at higher temperatures from +25°C to +30°C; according to the manufacturer, higher temperatures must be avoided
Noradrenaline	up to 6 months* once removed from refrigerator
Adrenaline	up to 6 months* once removed from refrigerator

*Within the indicated shelf life.

Acknowledgements related to this article

Assistance with the guidelines: these guidelines was approved by the presidium of the German Society of Anaesthesiology and Intensive Care Medicine (DGAI) and acknowledged by the German Interdisciplinary Society of Intensive Care and Emergency Medicine (DIVI).

Financial support and sponsorship: none.

Conflicts of interest: BB is a member of the MSD Advisory Board. Travel allowance were granted by Weinmann EMT (to JTG, BH, CK), Storz (to BH), Verathon (to CK), O-Two Medical Technologies (to VW) and Ambu (to EP).

Presentation: these guidelines were previously published in German in the Journal *Anästh Intensivmed* 2015; 56: 2–19.

Note/Disclaimer: the authors have taken the utmost care to present the drugs and provide dosage recommendations. Manufacturers' recommendations (technical information) on dosages, adverse effects and contraindications are nevertheless decisive.

Comment from the Editor: the editors of the *European Journal of Anaesthesiology* were informed by the authors that the guidelines had been published previously in German. The guidelines were checked and accepted by the editors of the *European Journal of Anaesthesiology* but did not undergo external peer review. The European Society of Anaesthesiology (ESA) was not involved in the development of these guidelines and did not endorse them. BWB is an associate editor of the *European Journal of Anaesthesiology*.

References

1 Helm M, Gries A, Mutzbauer T. Surgical approach in difficult airway management. *Best Practice Res Clin Anaesthesiol* 2005; **19**:623–640.
2 Schlechtriemen T, Reeb R, Ensle G, *et al.* Überprüfung der korrekten Tubuslage in der Notfallmedizin. *Notfall Rettungsmed* 2004; **7**:231–236.
3 Sefrin P, Kuhnigk H, Wurmb T. Narkose im Rettungsdienst. *Notarzt* 2014; **30**:73–84.
4 Cook T, Behringer EC, Benger J. Airway management outside the operating room: hazardous and incompletely studied. *Curr Opin Anesthesiol* 2012; **25**:461–469.
5 Rognås L, Hansen TM, Kirkegaard H, *et al.* Anaesthesiologist-provided prehospital airway management in patients with traumatic brain injury: an observational study. *Eur J Emerg Med* 2014; **21**:418–423.
6 Williams PL, Webb C. The Delphi technique: a methodological discussion. *J Adv Nurs* 1994; **19**:180–186.
7 Gries A, Zink W, Bernhard M, *et al.* Realistic assessment of the physician-staffed emergency services in Germany. *Anaesthesist* 2006; **55**:1080–1086.
8 Bernhard M, Helm M, Luiz T, *et al.* Pädiatrische Notfälle in der prähospitalen Notfallmedizin. *Notfall Rettungsmed* 2011; **14**:554–566.
9 Bernhard M, Hilger T, Sikinger M, *et al.* Patientenspektrum im Notarztdienst. *Anaesthesist* 2006; **33**:1157–1165.
10 Eich C, Roessler M, Nemeth M, *et al.* Characteristics and outcome of prehospital pediatric tracheal intubation attended by anaesthesia-training emergency physicians. *Resuscitation* 2009; **80**:1371–1377.
11 Helm M, Biehn G, Lampl L, *et al.* Pädiatrischer Notfallpatient im Luftrettungsdienst. *Anaesthesist* 2010; **59**:896–903.
12 Helm M, Hossfeld B, Schäfer S, *et al.* Factors influencing emergency intubation in the out-of-hospital setting: a multicentre study in the German Helicopter Emergency Medical Service. *Br J Anaesth* 2006; **96**:67–71.
13 Stollings JL, Diedrich DA, Oyen LJ, *et al.* Rapid-sequence intubation: a review of the process and considerations when choosing medications. *Ann Pharmacolther* 2014; **48**:62–76.
14 Thierbach A, Piepho T, Wolcke B, *et al.* Präklinische Sicherung der Atemwege: Erfolgsraten und Komplikationen. *Anaesthesist* 2004; **53**:543–550.
15 Christ M, Popp S, Erbguth E. Algorithmen zur Abklärung von Bewusstseinsstörungen in der Notaufnahme. *Intensivmed* 2010; **47**: 83–93.
16 Edlow JA, Robinstein A, Traub SJ, *et al.* Diagnosis of reversible causes of coma. *Lancet* 2014; **384**:2064–2076.
17 Moulton C, Pennycook AG. Relation between Glasgow coma score and cough reflex. *Lancet* 1994; **343**:1261–1262.
18 Timmermann A, Byhahn C, Wenzel V, *et al.* Handlungsempfehlungen für das präklinische Atemwegsmanagement. *Anaesth Intensivmed* 2012; **53**:294–308.
19 Von Goedecke A, Herff A, Paal P, *et al.* Field airway management disasters. *Anesth Analg* 2007; **104**:481–483.
20 Herff H, Wenzel V, Lockey D. Prehospital intubation: the right tools in the right hands at the right time. *Anesth Analg* 2009; **109**:303–305.
21 Lockey D, Carter J, Nolan J et al. 2009 AAGBI Safety Guideline. Out-of-hospital anaesthesia, www.aagbi.org/publications/guidelines/docs/prehospital_glossy09.pdf. [Accessed 26 May 2015]
22 Heuer JF, Barwing TA, Barwing J, *et al.* Incidence of difficult intubation in intensive care patients: analysis of contributing factors. *Anaesth Intensive Care* 2012; **40**:120–127.
23 Schmidt J, Strauß JM, Becke K, *et al.* Handlungsempfehlung zur Rapid-Sequence-Induction im Kindesalter. *Anästh Intensivmed* 2007; **48**: S88–S93.
24 Gräsner JT, Heller G, Dörges V, *et al.* Narkose im Rettungsdienst. *Notfallmedizin up2date* 2007; **2**:197–202.
25 Barnard EB, Moy RJ, Kehoe AD, *et al.* Rapid sequence induction of anaesthesia via the intraosseous route: a prospective observational study. *Emerg Med J* 2015; **32**:449–452.
26 Bernhard M, Gräsner JT, Gries A, *et al.* Die intraossäre Infusion in der Notfallmedizin. Erste deutsche Empfehlung der DGAI-Arbeitskreise. *Anästh Intensivmed* 2010; **51 (Suppl)**:S615–S620.
27 Pehböck D, Wenzel V, Voelckel W, *et al.* Effects of preoxygenation on desaturation time during hemorrhagic shock in pigs. *Anesthesiology* 2010; **113**:593–599.
28 Bretschneider I, Hossfeld B, Helm M, *et al.* Präklinisches Atemwegsmanagement beim Erwachsenen. *Notarzt* 2011; **27**:270–286.
29 Hossfeld B, Maier B, Kuhnigk H, *et al.* Narkose im Notarztdienst. *Notfall Rettungsmed* 2011; **14**:15–20.
30 Neumayr A, Ganster A, Schinnerl A, *et al.* Unterschätzte Gefahr am Notfallort. Plädoyer zur Einführung von Risikomanagement. *Notfall Rettungsmed* 2014; **17**:25–31.
31 Paal P, Herff H, Mitterlechner T, *et al.* Anaesthesia in prehospital emergencies and in the emergency room. *Resuscitation* 2010; **81**:148–154.
32 Weingart SD, Levitan RM. Preoxygenation and prevention of desaturation during emergency airway management. *Ann Emerg Med* 2012; **59**:165–175.
33 El-Orbany M, Connolly LA. Rapid sequence induction and intubation: current controvery. *Anesth Analg* 2010; **110**:1318–1325.
34 Steinmann D, Priebe HJ. Krikoiddruck. *Anaesthesist* 2009; **58**:695–707.
35 Timmermann A, Byhahn C. Krikoiddruck. Schützender Handgriff oder etablierter Unfug. *Anaesthesist* 2009; **58**:663–664.
36 Henderson JJ, Popat MT, Latto IP, *et al.* Difficult Airway Society guidelines for management of the unanticipated difficult intubation. *Anaesthesia* 2004; **59**:675–694.
37 Sitzwohl C, Langheinrich A, Schober A, *et al.* Endobronchial intubation detected by insertion depth of endotracheal tube, bilateral auscultation, or observation of chest movements: randomised trial. *BMJ* 2010; **341**:c5943.
38 Newton A, Ratchford A, Khan I. Incidence of adverse events during prehospital rapid sequence intubation: a review of one year on the London helicopter emergency medical service. *J Trauma* 2008; **64**:487–492.
39 Rognås L, Hansen TM, Kirkegaard H, *et al.* Out-of-hospital advanced airway management by experienced anaesthesiologists: a prospective descriptive study. *Scand J Trauma Resusc Emerg Med* 2013; **21**:58.
40 Helm M, Kremers G, Lampl L, *et al.* Incidence of transient hypoxia during out-of-hospital rapid sequence intubation by anaesthesiologists. *Acta Anaesthesiol Scand* 2013; **57**:199–205.
41 Chi JH, Knudson MM, Vassar MJ, *et al.* Prehospital hypoxia affects outcome in patients with traumatic brain injury: a prospective multicenter study. *J Trauma* 2006; **61**:1134–1141.
42 Hossfeld B, Lampl L, Helm M. Notwendigkeit eines Algorithmus für den ?schwierigen Atemweg' in der Präklinik. *Notfall Rettungsmed* 2011; **14**:10–14.
43 Kheterpal S, Healy D, Aziz MF, *et al.* Incidence, predictors, and outcome of difficult mask ventilation combined with difficult laryngoscopy: a report from the multicenter perioperative outcomes group. *Anesthesiology* 2013; **119**:1360–1369.
44 Benumof JL, Dagg R, Benumof R. Critical hemoglobin desaturation will occur before return to an unparalayzed state following 1 mg/kg intravenous succinylcholine. *Anesthesiology* 1997; **87**:979–982.
45 Sørensen MK, Bretlau C, Gätke MR, *et al.* Rapid sequence induction and intubation with rocuronium-sugammadex compared with succinylcholine: a randomized trial. *Br J Anaesth* 2012; **108**:682–689.
46 Connor CW, Segal S. The importance of subjective facial appearance on the ability of anesthesiologists to predict difficult intubations. *Anesth Analg* 2014; **118**:419–427.
47 Breckwoldt J, Kleimstein S, Brunne B, *et al.* Expertise in prehospital endotracheal intubation by emergency physicians. Comparing 'proficient performer' and 'experts'. *Resuscitation* 2012; **83**:434–439.
48 Sakles JC, Chiu S, Mosier J, *et al.* The importance of first pass success when performing orotracheal intubation in the emergency department. *Acad Emerg Med* 2013; **20**:71–78.
49 Niven AS, Doerschug KC. Techniques for the difficult airway. *Curr Opin Crit Care* 2013; **19**:9–15.
50 Wetsch WA, Carlitscheck M, Spelten O, *et al.* Success rates and tube insertion times of experienced emergency physicians using different video laryngoscopes: a study in simulated entrapped car accident victims. *Eur J Anaesthesiol* 2011; **28**:849–858.
51 Wetsch WA, Hellmich M, Spelten O, *et al.* Endotracheal intubation in ice-pick position with video laryngoscopes: a randomised controlled trial in a manikin. *Eur J Anaesthesiol* 2013; **30**:537–543.
52 Bernhard M, Beres W, Timmermann A, *et al.* Prehospital airway management using the laryngeal tube. An emergency department point of view. *Anaesthesist* 2014; **63**:589–596.
53 Voelckel WG, von Goedecke A, Fries D, *et al.* Die Behandlung des hämorrhagischen Schocks. Neue Therapieoptionen. *Anaesthesist* 2004; **53**:1151–1167.
54 Hasler RM, Nuesch E, Jüni P, *et al.* Systolic blood pressure below 110 mmHg is associated with increased mortality in blunt major trauma patients: multicenter cohort study. *Resuscitation* 2012; **83**:476–481.

55 Brain Trauma Foundation. Guidelines for the management of severe traumatic brain injury. 1. Blood Pressure and Oxygenation. *J Neurotrauma* 2007; **24 (Suppl 1)**:S7–S13.
56 Innerklinische Akutversorgung des Patienten mit Schädel-Hirn-Trauma. Aktualisierte Empfehlungen des Wissenschaftlichen Arbeitskreis Neuroanästhesie der DGAI. *Anästh Intensivmed* 2009; **50**:S489–S501.
57 Adams HP, Adams RJ, Brott T, *et al.* Guidelines for the early management of patients with ischemic stroke: a scientific statement from the stroke council of the American stroke association. *Stroke* 2003; **34**:1056–1083.
58 European Stroke Organisation (ESO) Executive Committee, ESO Writing Committee (2008). Guidelines for management of ischaemic stroke and transient ischaemic attack 2008. *Cerebrovasc Dis* 2008; **25**:457–507.
59 Werdan K, Ruß M, Buerke M, *et al.* Deutsch-österreichische S3-Leitlinie Infarktbedingter kardiogener Schock: Diagnose, Monitoring und Therapie. *Kardiologe* 2011; **5**:1666–2224.
60 Zollinger A, Hofer CK, Kuhnle G, *et al.* Anästhesie bei Patienten mit Lungenerkrankungen. In: Rossaint R, Werner C, Zwißler B, editors. *Die Anästhesiologie*. Springer; 2012.
61 Braun P, Wenzel V, Paal P. Anaesthesia in prehospital emergencies and in the emergency department. *Curr Opin Anesthesiol* 2010; **23**:500–506.
62 Hedenstierna G, Edmark L. The effects of anesthesia and muscle paralysis on the respiratory system. *Intensive Care Med* 2005; **31**:1327–1335.
63 Liccardia G, Salzilloa A, De Blasiob F, *et al.* Control of asthma for reducing the risk of bronchospasm in asthmatics undergoing general anesthesia and/or intravascular administration of radiographic contrast media. *Curr Med Res Opin* 2009; **25**:1621–1630.
64 Van der Walt J. Anaesthesia in children with viral respiratory tract infections. *Paediatr Anaesth* 1995; **5**:257–262.
65 Cowan GM, Burton F, Newton A. Prehospital anaesthesia: a survery of the current practice in the UK. *Emerg Med J* 2012; **29**:136–140.
66 Mrugalla HR, Samberger M, Schuhmann W, *et al.* Übergabemanagement für Beatmungspatienten im Luftrettungsdienst. Ergebnisse einer bundesweiten Umfrage. *Notfall Rettungsmed* 2003; **6**:233–241.
67 Rortgen D, Schaumberg A, Skorning M, *et al.* Stocked medications in emergency physician-based medical services in Germany. Reality and requirements according to guidelines. *Anaesthesist* 2011; **60**:312–324.
68 Easby J, Dodds C. Emergency induction of anaesthesia in the prehospital setting: a review of the anaesthetic induction agents. *J Trauma* 2004; **6**:217–224.
69 El-Beheiry H, Kim J, Milne B. Prophylaxis against the systemic hypotension induced by propofol during rapid-sequence intubation. *Can J Anaesth* 1995; **42**:875–878.
70 Hugg CJ, McLeskey C, Nahrwold M. Haemodynamic effects of propofol: data from over 25.000 patientes. *Anesth Analg* 1993; **77 (Suppl)**:21–29.
71 Koenig SJ, Lakticova V, Narasimhan M, *et al.* Safety of propofol as an induction agent for urgent endotracheal intubation in the medical intensive care unit. *J Intensive Care Med* 2015; **30**:499–504.
72 Wilbur K, Zed PJ. Is propofol an optimal agent for procedural sedation and rapid sequence intubation in the emergency department? *CJEM* 2001; **3**:302–310.
73 Weller D, Domke A, Gahn G. Management des refraktären Status epilepticus mit Propofol. *AktNeurologie* 2006; **33**:532.
74 Jabre P, Combes X, Lapostolle F, *et al.* Etomidate versus ketamine for rapid sequence intubation in acutely ill patients: a multicentre randomised controlled trial. *Lancet* 2009; **374**:293–300.
75 Patanwala AE, McKinney CB, Erstad BL<ET-AL. Retrospective analysis of etomidate versus ketamine for first-pass-success in an academic emergency department. *Acad Emerg Med* 2014; **21**:87–91.
76 Cotton B, Guillamondegui O, Fleming S. Increases risk of adrenal insufficiency following etomidate exposure in critcally injured patients. *Arch Surg* 2008; **143**:62–67.
77 Reinhart K, Brunkhorst FM, Bone HG, *et al.* Prävention, Diagnose, Therapie und Nachsorge der Sepsis. Erste Revision der S2k-Leitlinie der Deutschen Sepsis-Gesellschaft e.V. (DSG) und der Deutschen Interdisziplinären Vereinigung für Intensivmedizin und Notfallmedizin (DIVI). *Anaesthesist* 2010; **59**:347–370.
78 Mayglothling J, Duane TM, Gibbs M, *et al.* Emergency tracheal intubation immediately following traumatic injury: an eastern association for the surgery of trauma practice management guideline. *J Trauma Acute Care Surg* 2012; **73**:S333–S340.
79 Van den Heuvel I, Wurmb TE, Böttiger BW<ET-AL. Pros and Con of etomidate: more discussion than evidence. *Curr Opin Anesthesiol* 2013; **26**:404–408.
80 Jacoby J, Heller M, Nicholas J, *et al.* Etomidate versus midazolam for out-of-hospital intubation: aprospective, randomized trial. *Ann Emerg Med* 2006; **47**:525–530.
81 Swanson E, Fosnocht D, Jensen S. Comparison of etomidate and midazolam for prehospital rapid-sequence intubation. *Prehosp Emerg Care* 2004; **8**:273–279.
82 Luxen J, Trentzsch H, Urban B. Rocuronium und Sugammadex in der Notfallmedizin. *Anaesthesist* 2014; **63**:331–337.
83 Perry J, Lee J, Sillberg V, *et al.* Rocuronium versus succinylcholine for rapid sequence induction intubation (Review). *Cochrane Database Syst Rev* 2008;CD002788.