Acute incidents during anaesthesia

A small percentage of apparently routine anaesthetics will end in an anticipated or unforeseen acute incident.

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Anaesthesia is uneventful in the majority of cases but in a small percentage of routine and emergency cases there will be an anticipated or an unforeseen acute incident. These incidents need immediate theoretical knowledge and clinical skills to be managed effectively and to prevent further morbidity and mortality. Therefore all providers of anaesthesia, at different levels of experience, should be able to provide basic and advanced cardiopulmonary resuscitation (CPR).

The first death associated with an anaesthetic was reported in 1848 in the USA. Hannah Greener was due to have an ingrown toenail removed. She aspirated after induction of anaesthesia with chloroform by her surgeon and died.

Anticipation, prediction and recognition will all lead to the early and effective management of acute incidents and the prevention of adverse outcomes.

Although anaesthesia is a very well-controlled and governed discipline, acute incidents do occur. Incidents can occur during induction, maintenance and emergence from anaesthesia.

The following acute critical incidents are discussed in this article:
• Anaphylaxis
• Aspiration
• Laryngospasm
• High or total (complete) spinal blocks in obstetric anaesthesia.

Anaphylaxis

The prompt diagnosis and correct treatment of an anaphylatic reaction is paramount to avoid deleterious outcomes after these events. The estimated mortality once a reaction has started is approximately 5%.

Diagnosis of anaphylaxis is more challenging during general anaesthetic and a high index of suspicion is needed. The three most common initial presenting features during anaesthesia are:
• No pulse detected or hypotension (28%)
• Difficulty in inflating the lungs (26%)
• Flushing (21%).

Other presenting features are coughing, rash, desaturation, cyanosis, electrocardiograph (ECG) changes, wheezing, urticaria and angio-oedema.

Causes of anaphylactic reactions during anaesthesia include (the agents implicated are listed in order of most to least responsible):
• Neurumuscular blocking agents (70%)
• Latex (12.6%)
• Colloid solutions (4.7%)
• Induction agents (3.6%)
• Antibiotics (2.6%)
• Benzodiazepines (2%)
• Opioids (1.7%)
• Other agents (e.g. radio contrast media) (2.5%).

Treatment and management
• Stop administration of all suspected agents.
• Call for help.
• Airway must be secured and 100% oxygen given, and ensure adequate ventilation.
• Intravenous or intramuscular adrenaline must be administered, the dose depending on the cardiovascular status. CPR guidelines must be adhered to if cardiac arrest or near arrest is diagnosed.
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• Adequate amounts of the correct crystalloid solution should be given; in adult patients up to 2 - 4 litres might be needed (additionally both the legs can be raised to centrally pool the blood volume and to improve the preload of the heart).
• CPR should start when cardiac arrest is diagnosed (2010 South African guidelines).
• Additionally, antihistamines (H₁-receptor blocking agents, e.g. promethazine 25 mg intravenously (0.5 mg/kg in children), glucocorticosteroids (hydrocortisone 100 - 200 mg intravenously (4 - 5 mg/kg in children)) and bronchodilators must be administered if bronchospasm is severe or persistent.
• Catecholamine infusion (adrenaline 0.05 - 0.10 µg/kg/min) should be started, because cardiovascular instability may last for hours.
• Follow-up/step up in post-incident care is necessary, because a biphasic event may occur. The cause must be determined if possible, education given and the use of a MedicAlert disc advised.

Colloid solutions, and especially the gelatine solutions used for volume expansion post resuscitation of anaphylaxis, can cause anaphylactic reactions themselves. After the resuscitation and resolution of the event a causative agent should be identified and mast-cell tryptase (enzyme) should be measured at three intervals:
• Immediately after the reaction has been treated
• About 1 hour after the event
• 6 - 24 hours after the suspected anaphylaxis (tests not readily performed in South Africa) to confirm the diagnosis.

The samples should be separated and stored at 4°C if they can be analysed within 48 hours or at -20°C if sent for analysis later than 48 hours. The rise in tryptase is transient so timing of sample taking is important. Patients should be sent for further allergy testing and be informed about the anaphylaxis.

The prompt diagnosis and correct treatment of an anaphylactic reaction is paramount to avoid deleterious outcomes after these events.

Advises the patient to wear a form of identification to indicate the fact that he or she has had an anaphylactic response or previous anaphylaxis to a specific agent (e.g. anaesthetic hazard card or MedicAlert disc). Be aware of and take the necessary precautions when patients present with a history of previous allergic reactions – do not ignore the patient’s history.

Anaphylactoid reactions are indistinguishable from anaphylaxis and for all practical purposes are treated in a similar way. The most common cause of anaphylactoid reactions is exposure to contrast mediums.

Aspiration

Aspiration has a higher incidence in specific cases (upper and lower gastrointestinal pathology) but is unexpected in others. In Mendelson’s landmark paper, the aspiration of solid regurgitate had a much worse outcome than aspiration of fluids. The increased use of supraglottic airway devices (e.g. laryngeal mask airways (LMAs)) has increased the incidence of aspiration. Micro-aspiration will go unnoticed until it occurs in the debilitated patient with co-morbidities, and in this specific group of patients it will lead to morbidity and mortality.

The top three risk factors for aspiration are:
• Emergency surgery
• Light anaesthesia or unexpected response to stimulation
• Upper or lower gastrointestinal pathology.

Possible risk factors for regurgitation and pulmonary aspiration according to Engelhardt and Webster are shown in Table 1.

Table 1. Possible risk factors for regurgitation and pulmonary regurgitation

| Increased gastric content |
| Delayed gastric emptying |
| Gastric hypersecretion |
| Overfeeding |
| Lack of fasting |
| Increased tendency to regurgitate |
| Decreased lower oesophageal sphincter tone |
| Gastro-oesophageal reflux |
| Oesophageal strictures/carcinomas |
| Zenker's diverticulum |
| Achalasia |
| Extremes of age |
| Diabetic autonomic neuropathy |
| Laryngeal incompetence |
| General anaesthesia |
| Emergency surgery |
| Inexperienced anaesthetist |
| Night-time surgery |
| Head injury |
| Cerebral infarct/haemorrhage |
| Neuromuscular disorders |
| Multiple sclerosis |
| Parkinson's disease |
| Guillain-Barré syndrome |
| Muscular dystrophies |
| Cerebral palsy |
| Cranial neuropathies |
| Trauma/burns |
The following are good measures to prevent regurgitation and pulmonary aspiration:

- Keep to nil by mouth protocol and do not compromise.
- Keep a suctioning catheter/device at the head end of the operating theatre table during the peri-operative period.
- If in any doubt suction the stomach of the patient suspected of having a full stomach. Empty in three different positions before induction of anaesthesia.
- Contraindications to the use of LMAs should be adhered to.
- Patients in labour have delayed stomach emptying and the airway must be secured with an endotracheal tube.
- Pain and opioid analgesics lead to delayed gastric emptying.
- Err on the safe side and use muscle relaxants, rapid sequence induction and secure the airway with an endotracheal tube when in doubt.

Aspiration may be obvious macro-aspiration of regurgitated content or it may consist of micro-aspirates.

Diagnosis of macro-aspiration is usually obvious and the most important priority is early intervention to prevent further aspiration. Particulate matter can cause immediate airway obstruction and should be treated as such if it happens. Regurgitation of non-particulate matter/fluids is usually noticed once the airway device or oral cavity is soiled by them. The regurgitation and aspiration can go unnoticed and the first sign might be laryngospasm, airway obstruction, desaturation, bronchospasm, hypoventilation or even cardiac arrest.

Management

Management consists of:

- Early diagnosis – tell the rest of the theatre team and call for help.
- Position the patient head down in the left lateral decubitus position, if feasible.
- Open the airway and keep it patent.
- Apply cricoid pressure (relief of cricoid pressure if patient vomits).
- Clear the oro-, naso- and hypopharynx within direct vision from aspirate, if possible.
- Oxygenate and ventilate patient, use pulse oximetry to guide fractional inspired oxygen tension (FiO₂) delivery, and secure the airway by rapid sequence induction and endotracheal intubation.
- Suction the trachea and accessible bronchi through the endotracheal tube.
- Take a chest X-ray if aspiration suspected.
- Take a decision to continue or to postpone the case.
- Further management includes bronchoscopy (to clear aspirate from the lower airways) and – more controversially – steroids/antibiotics.
- Step up postoperative care and use intermittent positive pressure ventilation (IPPV) and bronchodilators if needed.
- Be vigilant and monitor the patient for any deterioration in his/her condition (especially during the first 2 hours post event) until the discharge criteria are met in a stable patient.

Criteria for monitored discharge of patients who have aspirated to a general ward are: 7

- Clinical stability
- Oxygen saturation (SpO₂) 95% on FiO₂ 50%
- Heart rate <100 beats/min
- Respiratory rate <20/min
- Temperature normal
- Minimal bronchodilation needed.

Laryngospasm

Laryngospasm is a protective reflex closure of the upper airway from spasm of the glottic musculature as the result of an abnormal stimulus.4 Laryngospasm is mediated by the superior laryngeal nerve.4 It occurs more commonly in paediatric anaesthesia and after certain procedures. Predisposing factors, including respiratory tract infections, must be elucidated in your pre-operative history taking and examination. Inexperienced anaesthetists will experience this complication more frequently than their more experienced colleagues.

Airway instrumentation or intravenous cannulation (any patient stimulation) should only be attempted after a sufficiently deep level of anaesthesia has been reached. Only sevoflurane and halothane must be used for inhalational induction. The use of muscle relaxant at the correct dose before attempting intubation will prevent laryngospasm. Extubating the patient fully awake or at a sufficiently deep level of anaesthesia is advised to prevent laryngospasm on extubation and emergence. Laryngospasm usually ceases spontaneously as hypoxia and hypercapnia develop, but it is a dangerous practice to wait for this to develop before you start to actively intervene.

Micro-aspiration will go unnoticed until it occurs in the debilitated patient with co-morbidities, and in this specific group of patients it will lead to morbidity and mortality.

The diagnosis of partial or complete laryngeal obstruction by looking and listening for signs and symptoms of laryngeal spasm are important. You should recognise suprasternal and supraclavicular retraction, a tracheal tug and paradoxical movement of the abdomen and chest during obstructive breathing. Inspiratory
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Fig. 1. Management of laryngospasm in paediatric patients. CPAP = continuous positive airway pressure; FiO₂ = fractional inspired oxygen tension; IV = intravenous; IM = intramuscular; PACU = post-anaesthesia care unit.

Diagnosis of laryngospasm

Identification and removal of the stimulus (secretions, blood, nociceptive stimulus)

Chin lift and jaw thrust
Oropharyngeal airway
CPAP and FiO₂ 100%

Assess air entry
Bag movement?

No

Complete laryngospasm

Call for help
Positive pressure ventilation with face mask

No improvement

IV access

IV suxamethonium
0.5 - 2 mg/kg after
IV atropine 0.02 mg/kg or
IV propofol 1 mg/kg

No IV access

IM (1.5 - 4 mg/kg)
or intraosseous
0.5 - 1 mg/kg
suxamethonium

Positive pressure ventilation with
FiO₂ 100%
Followed by tracheal intubation

No improvement

Cardiopulmonary resuscitation

Yes

Partial laryngospasm

Deepen anaesthesia with small doses of propofol or inhalational agent

Reassess air entry with CPAP

Improvement

Surgery or PACU

No improvement

Improvement
stridor with incomplete laryngospasm or no air movement with complete obstruction should be followed by prompt manoeuvres to treat the laryngospasm.

Bradycardia secondary to hypoxia should never be treated by atropine. Administration of suxamethonium may lead to severe bradycardia and even to cardiac arrest in a hypoxic patient. Therefore, giving intravenous atropine before an intravenous injection of suxamethonium to treat laryngospasm is mandatory.10

Treatment
The treatment of laryngospasm consists of:
• Removal (e.g. suctioning away of blood, secretions, surgical debris) of the offending trigger
• Administration of 100% oxygen by tight-fitting face mask, continuous positive airway pressure (CPAP) with gentle positive pressure ventilation with a chin lift and jaw thrust manoeuvre (painful stimuli in this position help to prevent and relieve laryngospasm)11
• If the abovementioned measures are ineffective, more aggressive pharmacological therapy is needed (succinylcholine, rocuronium – if there is a contraindication to succinylcholine’s use and no difficult airway is anticipated, use propofol etc.). Succinylcholine in a dose not less than 0.5 mg/kg should be used. If intravenous access is not secured at this stage, the best alternative option is to use the intramuscular route (intraosseous or intralingual are other alternatives) of administration at a dose of 1.5 mg/kg to 4 mg/kg. (Delayed onset of action is a real problem, but in this case there is no alternative available.)11

Fig. 1 details the management of laryngospasm in paediatric patients.10

General anaesthesia conducted with a facemask (and harness) is a technique now not frequently used by practitioners, but is less likely to cause laryngospasm than using either supraglottic airway devices (e.g. LMAs) or endotracheal intubation. However, this is also controversial.9

Table 2. Clinical manifestations of complete spinal block

<table>
<thead>
<tr>
<th>Cardiorespiratory</th>
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<tbody>
<tr>
<td>Hypotension*</td>
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<td>Bradycardia*</td>
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<td>Respiratory compromise*</td>
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<tr>
<td>Apnoea*</td>
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<tr>
<td>Reduced oxygen saturation</td>
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<tr>
<td>Difficulty speaking/coughing</td>
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<tr>
<td>Cardiac arrest (asystole)</td>
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<tr>
<td>Neurological</td>
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<tr>
<td>Nausea and anxiety*</td>
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<tr>
<td>Arm/hand dysesthesia or paralysis*</td>
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<tr>
<td>High sensory level block</td>
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<tr>
<td>Cranial nerve involvement</td>
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<tr>
<td>Loss of consciousness</td>
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* Commonly reported or ‘classic.’

The incidence of complications resulting from laryngospasm can vary as follows: *
• Cardiac arrest 0.5%
• Pulmonary aspiration 3%
• Obstructive negative pressure pulmonary oedema 4%
• Bradycardia 6%
• Oxygen desaturation 61%

According to Donlan et al., ‘In addition to hypoxemia some patients who are able to generate very large negative inspiratory pressures when attempting to breathe against the obstruction may succumb to “negative-pressure pulmonary edema”.’

High or total (complete) spinal blocks during caesarean section
Neuroaxial blockade by spinal anaesthesia is the choice of anaesthesia for caesarean section if no contraindications exist. A high or total (complete) spinal block is an anaesthetic emergency and prompt and definitive treatment and management should proceed before the airway protective reflexes are lost, ventilation becomes compromised or haemodynamic collapse happens (asystole or pulseless electrical activity).

Miller notes that ‘Pregnant patients develop more extensive spinal blocks than nonpregnant patients. This is related to increased sensitivity to local anaesthetics as well as the mechanical effects of epidural vein engorgement.’12

High spinal blocks (above T4) result because of excessively high spread of the local anaesthetic and can be diagnosed by the following signs and symptoms: nausea, vomiting, development of weakness of the upper extremities, breathing difficulty, feeling of impending doom and haemodynamic instability (bradycardia and hypotension). Haemodynamic instability is secondary to sympathetic blockade, blockade of the accelerator fibres to the heart (T1 - T4) or hypoperfusion of the vital centres in the brainstem.

Miller offers the following good advice. ‘Intraoperatively, during high spinal anaesthesia, patients occasionally complain excessively about dyspnea. This is not a result of significantly decreased inspiratory capacity but most often seems to be related to loss of chest wall sensation, which does not
allow patients to experience the reassurance of a deep breath. This impediment to patient acceptance can often be overcome by instructing the patient to raise a hand near the mouth and exhale forcefully. The tactile appreciation of the deep breath seems to provide reassurance.1

During spinal anaesthesia frequent monitoring (every minute) of the haemodynamic parameters should continue till the newborn has been delivered and the mother is haemodynamically stable, then blood pressure and other vital signs (heart rate) can be monitored and recorded less frequently.

A total (complete) spinal can be diagnosed if the patient loses consciousness not because of hypotension and hypoperfusion of the vital centres, but when the spread of local anaesthetic has been delivered and the mother is haemodynamically stable, then blood pressure and other vital signs (heart rate) can be monitored and recorded less frequently.

Immediate diagnosis of a total spinal block is essential, followed by prompt treatment. Protection of the airway (to prevent aspiration and asphyxia) and ventilation, together with haemodynamic support with adrenaline and intravenous fluid boluses, are the cornerstones of treatment. The patient must be placed in the left lateral tilt position during resuscitation, if not already in this position, to prevent aortocaval compression.

CPR must be started immediately when cardiac arrest is diagnosed. Cardiac compressions should be started at 100 compressions per minute. The immediate delivery of the newborn is part of the resuscitation attempt. Intravenous adrenaline at a dose of 1 mg of a 1:1 000 solution is indicated during a cardiac arrest.1

High and total spinals resolve within a period of time and the support for a good outcome needs to be instituted immediately and effectively and continued until the event resolves. Misdiagnosis or late diagnosis leads to morbidity and mortality.

Conclusion
Acute incidents happen during the perioperative care of patients. Paediatric, geriatric and pregnant patients are more prone to develop acute incidents and are more vulnerable to complications once these events have taken place. The prompt diagnosis and effective treatment and management of these incidents prevent morbidity and mortality. If an acute incident occurs, it must be documented meticulously. The event should be discussed with the patient and all other interested parties at a convenient time after the incident.

References available at www.cmej.org.za.