Anaesthesia for Plastic Surgeons

10

Daisy

The thin line between life and death

(Dr David Galler 2016)

The original version of this chapter was revised. A correction to this chapter can be found at https://doi.org/10.1007/978-981-13-0689-1_17

Drs. Sophie Klaassen and Katherine Lanigan contributed significantly to this chapter.

M. F. Klaassen and E. Brown, *An Examiner's Guide to Professional Plastic Surgery Exams*, https://doi.org/10.1007/978-981-13-0689-1_10

10.1 Evolution of Plastic Surgery and Anaesthesia

Ivan Whiteside Magill

It is significant and timely that the two specialties of anaesthesia and reconstructive plastic and maxillofacial surgery had their modern origins alongside each other at The Queens Hospital, Sidcup, Kent during the second half of the First World War (1916–1918). Major Harold Gillies with the support of the military hierarchy, a generous donation from Queen Mary and other private sponsorship established a purpose-built surgical hospital for facial and jaw injuries. His vision and goal was to bring all the selected facial injuries and the medical/nursing teams treating them to one specialist centre. British, Canadian, Australian and New Zealand divisions were working together, often with a friendly competitive edge and supported by multidisciplinary teams including anaesthetists, dental technicians, sculptors/artists and of course a dedicated nursing team. The anaesthetists were led by Captain R. Wade, who contributed to the 1920 book *Plastic Surgery of the Face* with six pages of remarks on anaesthesia. Later Captain Ivan Magill, an Ulsterman developed the first endotracheal tube at Sidcup to secure the airway of patients requiring complex facial reconstruction. The Magill forceps bear his name today.

10.2 Modern Surgeon/Anaesthetist Interactions

The skilful and expert delivery of peri-operative anaesthesia, monitoring of vital organs, fine-tuning of human physiology and pain management enable surgeons to achieve remarkable results without having to worry about the overall condition of the patient.

Successful surgery depends on a trilogy of anaesthetists (and their technicians), skilled operating room and ward nurses and surgeons (with their assistants).

The relationship between these health professionals is based on trust, respect, communication and teamwork.

The anaesthetic/surgeon/nursing team is a partnership and a professional working alliance that knows only too well, the sage words of Dr. David Galler (Senior Intensive Care Specialist, Middlemore)—*'the thin line between life and death'*.

I tell my patients that the anaesthetist caring for them and managing their anaesthetic during the operative procedure is the most important team member in the operating room (O.R.) … but collectively the medical team is all-important. The key factors for sophisticated and highly-developed anaesthetic surgical care are communication, situation awareness and clinical decision-making. The communication requires disclosure at a number of key periods in the surgical management:

1. Pre-operatively
2. Intra and peri-operatively
3. Post-operatively
4. Clinical audit and quality assurance

10.3 Pre-Operation

Diagnosis, considered options and definitive surgical plan must also address the co-morbidities, past medical history (including previous anaesthetic events, family history of drug allergies) and current medications which all contribute to the anaesthetic risk. The American Society of Anesthesiology (ASA) Physical Status (PS) Classification System developed by the American Society of Anesthesiologists (latest update, 2014) is summarised below.

ASA PS classification	Definition	Examples
ASA 1	A normal healthy patient	Healthy, non-smoking, no alcohol
ASA 2	Mild systemic disease	Mild disease without functional limitations (current smoker, social alcohol drinker, pregnancy, obesity (30 < BMI < 40), well-controlled diabetes mellitus/hypertension, mild lung disease)

(continued)

(continued)

ASA PS classification	Definition	Examples
ASA 3	Severe systemic disease	Substantive functional limitations—poorly controlled diabetes mellitus or hypertension, COPD, morbid obesity (BMI > 40), active hepatitis, alcohol dependence or abuse, implanted cardiac pacemaker, moderate reduction of ejection fraction with or without cardiac failure, End Stage Renal Disease (ESRD) undergoing regularly scheduled dialysis, premature infant <60 weeks, history (>3 months) of MI, CVA, TIA or CAD/stents
ASA 4	Severe systemic disease that is a threat to life	Recent (<3 months) MI, CVA, TIA or CAD/stents, ongoing cardiac ischaemia or severe valve dysfunction, severe reduction of ejection fraction, sepsis, DIC, ARD or ESRD not undergoing regularly scheduled dialysis
ASA 5	Moribund patient, not expected to survive without operation	Ruptured abdominal/thoracic aneurysm, massive trauma, intracranial bleed with mass effect, ischaemic bowel in the face of significant cardiac pathology or multiple organ/system dysfunction
ASA 6	Brain-dead patient whose organs are being removed for donor purposes	

The addition of 'E' denotes Emergency surgery.

e.g. ASA 2E.

In selected cases a pre-anaesthetic consult is required to address specific cardiovascular, respiratory and peri-anaesthetic risk factors. I generally send my anaesthetists a copy of my initial surgical report alerting them to a potential case and highlighting any obvious risk factors. This gives them time to arrange a workup specific to that individual patient. Recently we had a young woman with Ehlers-Danlos syndrome requiring major surgery and the workup required consultant anaesthetist and cardiologist involvement with echocardiography and other pre-surgery investigations. The early communication with your anaesthetic colleague is professionally appropriate, buys time and helps with the decisions about the most appropriate surgical facility for the case (outpatient, inpatient with or without ICU support).

10.4 Pre-Operative/Intra-Operative

The sequence of complex events, risk management and decision-making that occur during the surgical procedure are governed by all the modern strategies of communication skills, situation awareness, leadership, experience, clinical intuition and the sometimes urgent performance of practised emergency steps and manoeuvres. The realities of the very dangerous practice of surgery and anaesthesia require the full range of human factors and the familiarity of working within a team of known and

experience colleagues. The older I get as a surgeon, the more I value this environment of trust and shared history of working relationships, because it instils quiet confidence. This is the best environment for our patients as well. It is not critical, because trained professionals following common non-technical strategies in the theatre should be able to work with each other without familiarity. The ideal is not always logistically possible for team selection.

10.5 Shared Airway

In certain surgeries involving the aerodigestive tract and head and neck surgery, the shared airway concept is in play. This is particularly an intraoperative scenario with rhinoplasty, intraoral surgery, orthognathic surgery and craniofacial surgery. The anaesthetist may have limited access to the patient's airway during the surgical procedure. Where such a situation is envisaged, the anaesthetist needs to be forewarned, so that the most appropriate anaesthetic and flexible airway conduits (endotracheal tubes, flexible laryngeal mask airway, elective tracheostomy) can be chosen.

10.6 Post-Operative

Sir Harold Gillies stated that the aftercare is as important as the planning and the execution of the surgery itself. This rings true for the post-anaesthetic care as well, in terms of immediate recovery, step-down to later recovery and recovery over the succeeding days, weeks and months. Following surgery and a general anaesthetic, most patients have a feeling of tiredness and reduced intellectual function. This can vary between patients and procedures. As a generalisation, for every hour of anaesthesia, I tell my patients it will take at least a week to fully recover their energy and intellectual function. This is important for patients to know, so that they can plan their elective surgery around their physical and intellectual commitments, such as sporting events and student examinations.

10.7 Local Anaesthetic

Local anaesthetic drugs are either Amino Esters or Amino Amides. Their method of action is to *block* action potentials in the nerve cell membrane. The nerve impulses are not propagated along the membrane and depolarization and repolarisation cannot proceed. Skin anaesthesia results and sometimes the nearby motor nerves are also blocked, causing temporary muscle weakness, e.g. brow ptosis and facial asymmetry.

The Amino Esters include Procaine and Cocaine. The Amino Amides include Lidocaine (™Xylocaine), Bupivacaine (™Marcaine) and Ropivacaine (™Naropin).

10.8 Maximum Safe Doses

Drug	Plain	With adrenaline	Duration	Onset time
Lidocaine	3–4 mg/kg	5–7 mg/kg	Medium	Quick
Bupivacaine	2.5 mg/kg	2.5 mg/kg	Long	Moderate
Ropivacaine	3–4 mg/kg	3-–4 mg/kg	Long	Moderate

Lidocaine has a rapid onset of anaesthesia whilst Ropivacaine is characterised by slow onset of action and longevity. Lignocaine with Adrenaline and Ropivacaine can be combined to gain the benefits of both rapid onset and longevity of action. Remember that the collective dose is combined so safest to use 50% of the safe dose of the first local anaesthetic and no more than 50% of the second local anaesthetic.

Lidocaine has a rapid onset of anaesthesia whilst Ropivacaine is characterised by slow onset and prolonged duration of action.

10.9 Toxicity

Monitoring during local anaesthetic procedures is important for patient safety.

We suggest routine use of pulse meter or pulse oximeter.

Marcaine is the most toxic local anaesthetic potentially and has the highest lipid solubility. Cardiotoxicity from Marcaine overdose is almost always fatal and intravenous Intralipid 20% in the dose of 1.5 mL/kg stat over 60 seconds by infusion at 0.25 mL/kg/min is recommended as CPR continues. Naropin is the safest local anaesthetic in my experience and has a bonus vasoconstrictor affect, independent of adrenaline.

The earliest signs of local anaesthetic toxicity are CNS signs with cerebral excitation, restlessness, tinnitus, perioral tingling and light headedness followed progressively by seizures, loss of consciousness and eventually death, if not treated with emergency resuscitation. The ABCD of cardiac arrest management is initiated with Oxygen, airway and circulation support. Cardiovascular toxicity includes: hypotension, conduction blockade and cardiac arrest. Marcaine is associated with the highest risk of severe cardiac dysrhythmias and irreversible cardiovascular collapse.

10.10 ABCDE Management

*A*irway.
*B*reathing.
*C*irculation.
*D*isability (GCS: Glasgow Coma Score).
*E*xposure.

10.11 Cardiac Arrest During a Local Anaesthetic Procedure

When cardiac arrest is suspected, immediate cardiopulmonary resuscitation is critical to maintain oxygenation of vital organs. A vaso-vagal event can lead to pulselessness for a short period of time [1].

Send for urgent help.

Commence ABCDE with a cycle of 30 chest compressions, followed by two rescue breaths and then further compressions.

The first 2 min of proper CPR will give your patient 8 min more chance of survival.

Establishment of an airway with an endotracheal tube and administration of Oxygen.

Should circulation NOT be established, defibrillation should be initiated.

10.12 Safety Guidelines for Management of Severe LA Toxicity

1. *Recognition*
   Signs: sudden alteration in mental status, severe agitation or loss of consciousness with or without tonic-clonic convulsion. Cardiovascular collapse: sinus bradycardia, conduction blocks, asystole and ventricular tachyarrhythmias.
2. *Immediate management*
   Stop injecting. Call for help. Maintain airway, secure with an endotracheal tube. Give 100% oxygen and ensure adequate lung ventilation. Establish intravenous access. Control seizures with a benzodiazepam, thiopental or propofol in small incremental doses. Assess cardiovascular status throughout.
3. *Treatment*
   In Circulatory Arrest: start CPR, manage arrhythmias. Give Intravenous Lipid Emulsion (20% Lipid emulsion bolus 1.5 mL/kg over 1 min, start an IV infusion of 20% lipid emulsion at 15 mL/kg/h). After 5 min can give a maximum of two repeat boluses (same dose), 5 min between boluses. Continue infusion at same rate but Double rate to 30 mL/kg/h after 5 min.

   Without Circulatory Arrest: treat hypotension, bradycardia and tachyarrhythmias. Consider intravenous lipid emulsion.
4. *Follow-up*
   Arrange safe transfer to a clinical area with appropriate equipment for recovery of patient Post Anaesthesia Care Unit (PACU). Exclude pancreatitis by regular clinical review, including daily Amylase or Lipase assays for 2 days. Report case to national safety agency.

10.13 Different Types of Anaesthesia

General anaesthesia.
Local anaesthesia.
Regional anaesthesia.

Procedural sedation.
Conscious sedation.
Analgesia.

10.14 Guidelines for the Management of Postoperative Nausea and Vomiting (PONV)

(endorsed by Australian and New Zealand College of Anaesthetists)

Incidence of nausea is 50% and of vomiting is about 30%. Postoperative nausea and vomiting prophylaxis is therefore important. There is a subset of high risk patients. Risk factors include: females, <50 years, history of PONV, opioid use in PACU and nausea in PACU. The most likely causes of PONV are volatile anaesthetics, nitrous oxide and postoperative opioids. Strategies to reduce the risk of PONV include: avoidance of GA by using Regional Anaesthetic (in adults and children), use of propofol for induction and maintenance of anaesthesia, avoidance of nitrous oxide and volatile anaesthetics, minimization of intraoperative and postoperative opioids and adequate hydration.

Prophylaxis for PONV: this is reserved for patients who are at high risk of PONV and the evidence from multiple literature studies is that combination therapy (multimodal approach) is best.

For Adults pharmacologic combinations include:
Droperidol + dexamethasone.
5-HT3 receptor antagonist + dexamethasone.
5-HT3 receptor antagonist + droperidol.
5-HT3 receptor antagonist + dexamethasone + droperidol.
Ondansetron + casopitant.
For Children pharmacologic combinations include:
Ondansetron 0.05 mg/kg + dexamethasone 0.015 mg/kg.
Ondansetron 0.1 mg/kg + droperidol 0.015 mg/kg.
Tropisetron 0.1 mg/kg + dexamethasone 0.5 mg/kg.
All of the above drugs have potential side effects.

When nausea and vomiting occur postoperatively, treatment should be administered with an antiemetic from a pharmacologic class that is different from the prophylactic drug initially given [2].

10.15 Anaesthetic Emergencies/Critical Events

1. Can't intubate can't oxygenate (CICO)
2. Emergency tracheostomy
3. Anaphylaxis in the operating theatre
4. Malignant hyperthermia in the operating theatre
5. Fire in the operating theatre

10.16 Can't Intubate Can't Oxygenate (CICO)

CICO arises when attempts to manage the airway by tracheal intubation, face-mask ventilation, or placement of a supraglottic airway device have all failed. Hypoxic brain damage and death will result unless there is a rapid resolution. This is an emergency. Rehearsed protocols should be initiated early and surgical airway access achieved with either a mini-tracheotomy or an emergency tracheostomy [3].

10.17 Emergency Tracheostomy

Midline incision inferior to the cricoid cartilage (4–5 cm long), dissect between and retract the strap muscles laterally. Identify the tracheal rings and make either a cruciate incision or Bjork cartilage flap and introduce the tracheostomy tube for connection to the Oxygen supply and ventilation system. When the first sign of CI (Can't Intubate) is defined, call for tracheostomy set.

10.18 Anaphylaxis

Anaphylaxis during anaesthesia can present as cardiovascular collapse, airway obstruction, and/or skin manifestations.

Usual suspects include: neuromuscular blocking agents, natural rubber latex, antibiotics, plasma volume expanders, IV anaesthetic drugs and Betadine.

10.19 Management of Patient with Suspected Anaphylaxis During Anaesthesia:

1. Stop administration of all agents likely to have caused the anaphylaxis.
2. Call for help.
3. Maintain airway, give 100% oxygen and lie patient flat with legs elevated.
4. Give epinephrine (adrenaline). This may be given intramuscularly in a dose of 0.5–1 mg (0.5–1 mL of 1:1000) and may be repeated every 10 min according to the arterial pressure and pulse until improvement occurs. Alternatively, 50–100 μg intravenously (0.5–1 mL of 1:10,000) over 1 min has been recommended for hypotension with titration of further doses as required.

Secondary therapy

1. Give antihistamines (chlorpheniramine 10–20 mg by slow intravenous infusion).
2. Give corticosteroids (100–500 mg hydrocortisone slowly iv).
3. Bronchodilators may be required for persistent bronchospasm.

10.20 Malignant Hyperthermia (MH)

This is a rare pharmacogenetic disorder. It is fatal if prompt treatment is not instituted. Multiple high priority tasks must be attended to simultaneously and Resource Kits are available in all theatres and a laminated MH double-sided A4 sized *MH Crisis Initial Management Card* should be attached to each anaesthetic machine.

The most senior anaesthetist should co-ordinate crisis management;

1. Declare Emergency
2. Call for HELP
3. Send for MH box and supplies
4. Turn off volatile agent and remove vaporises from anaesthetic machine
5. Hyperventilate with 100% oxygen at >15 L/min
6. Commence IV anaesthesia maintenance (e.g. Propofol infusion)
7. *Dantrolene administration is the priority*

Signs and symptoms of MH.
Masseter spasm after Suxamethonium.
Tachypnoea and raised end tidal carbon dioxide.
Tachycardia.
Cardiac arrhythmias.
Rapid rise in temperature.
Respiratory and metabolic acidosis.
Hyperkalaemia.
Profuse sweating.
Cardiovascular instability.
Decreased Sp02 (peripheral capillary oxygen saturation) or mottling of skin.
Generalised muscular rigidity.
Myoglobinuria (dark-coloured urine).
Generalised muscle ache (awake patient).
Grossly raised serum CK.
Coagulopathy.
Cardiac Arrest.

10.21 Fires in the Operating Room

Involving patient.
As part of a building fire.
Chemical burns.

These can be either airway or non-airway fires. Fire is always a risk in the operating theatre when there exists the 'fire triad' of oxidizer, ignition and fuel. The emergency management of an airway fire includes: stopping the procedure, removing the tracheal tube, stopping the flow of all airway gases and saline irrigation of the airway. Once fire is extinguished the ventilation of the patient is re-established

with the circuit or a self-inflating resuscitation bag. Ventilate with room air. Consider bronchoscopy and airway examination to remove tracheal fragments and other debris. Continue an ongoing management plan for the patient's damaged airway.

References

1. Thim T, Krarup NHV, Grove EL, Rohde CV, Løfgren B. Initial assessment and treatment with the Airway, Breathing, Circulation, Disability, Exposure (ABCDE) approach. Int J Gen Med. 2012;5:117–21. Published online 2012 Jan 31. https://doi.org/10.2147/IJGM.S28478.
2. Gan TJ, et al. Consensus guidelines for the management of postoperative nausea and vomiting. Anesth Analg. 2014;118(1):85–113.
3. Pracy JP, Brennan L, Cook TM, Hartle AJ, Marks RJ, McGrath BA, Narula A, Patel A. Surgical intervention during a Can't intubate Can't oxygenate (CICO) event: Emergency Front-of-neck Airway (FONA)? Br J Anaesth. 2016;117(4):426–8. https://doi.org/10.1093/bja/aew221. Published:19 September 2016.