Complications

Intraoperative hypotension

Anaesthesia Related Mortality in Australia 1994-96

Postoperative confusion

Pulmonary aspiration in anaesthesia

Perioperative stroke

Respiratory depression under GA

Adverse effects of laryngoscopy

Complications of, benefits of, indications for IVs, CVCs, PA catheters, arterial lines

Perioperative myocardial ischaemia

Arrest protocol

Malignant hyperthermia

Post-operative nausea and vomiting

Crisis Management in Anaesthesia
Intraoperative hypotension

Differential diagnosis

Airway and breathing
   - Desaturation, hypoxia
   - Pneumothorax
   - High circuit pressure

Cardiac
   - Arrest
   - Arrhythmia with low output
   - Failure
   - Ischaemia or infarction
   - Vagally mediated bradycardia

Circulation
   - Hypovolaemia
   - Obstructed venous return
   - Sepsis with vasodilatation

Drugs
   - Indication agents, volatile agents, vasodilators
   - Incorrect dose or rate of infusion
   - Regional technique: sympathetic blockade, local anaesthetic toxicity
   - Anaphylaxis

Equipment
   - Drug administration equipment failure
   - Anaesthetic machine, syringe pumps
   - Monitoring equipment failure: artifact

Fluids, electrolytes, metabolism
   - Transfusion reaction, Haemaccel reaction
   - Hypoglycaemia, Addisonian crisis...
   - Hypothermia

Surgical
   - Blood loss
   - Embolism: air, fat, cement
   - Obstruction to venous return
   - Specific surgical complications
      - Clamp release, great vessel surgery, cardiac compression
Anaesthesia Related Mortality in Australia 1994-96

Classification

Deaths attributable wholly or partly to anaesthesia
   Under anaesthetist’s control
   Doubtful whether under anaesthetist’s control
   Combined anaesthetic and surgical factors

Deaths in which anaesthesia played no part
   Entirely due to surgical factors
   Inevitable deaths despite correct anaesthetic and surgical management
   Fortuitous deaths

Unassessable deaths
   Despite considerable data
   Due to inadequate data

1994-96

Deaths in Australia  1875  106.8 per million population
Related to anaesthesia  135  7.7 per million population
   7.2% of deaths considered
   15.9 per million procedures (1/63,000)

Major factors

   Anaesthetic technique
      Airway  16
      Ventilation  9
      Other  48
   Anaesthetic drugs
      Dosage  45
      Selection  18
      Inadequate reversal  3
      Adverse reaction  2
   Preoperative
      Assessment  40
      Management  13
   Anaesthetic management
      Crisis management  21
      Inadequate monitoring  12
      Equipment failure  3
      Inadequate supervision/assistance  19
      PA catheter  5

Gender
   Male 77, female 58

Age
   \( \leq 40 \text{ y} \)  6 (4.4%)
   \( >60 \text{ y} \)  117 (86.7%)

ASA status
   1  0
   2  13
   3  66
   4  45
   5  11

Complications

James Mitchell (December 24, 2003)
Postoperative confusion

Differential diagnosis
  Respiratory failure: hypoxia
  Cardiovascular
    Hypotension, arrhythmia, cardiac ischaemia, anaemia
  Neurological
    Cerebral bleed, embolic event, long-standing dementia or degenerative disease particularly in unfamiliar environment and in pain
  GIT
    Hepatic decompensation, gut ischaemia
  Renal
    Acute renal failure, uraemia
  Endocrine or metabolic
    Electrolyte disturbance, hypoglycaemia, Cushing’s syndrome, hypercalcaemia
  Septic
    Infection: respiratory or urinary most characteristic
  Drug-related
    Withdrawal of benzodiazepines, narcotics, alcohol, other sedatives
    Administration of sedatives, analgesics, antiemetics
    Adverse reaction to anaesthetic agents

History
Examination
  HR, BP, T, RR, SpO\textsubscript{2}
  General examination focussing on signs of sepsis or cardiorespiratory problems
  Neurological examination for focal signs and to assess degree of confusion

Investigation
  Directed by history and examination findings
  FBE, U&E, LFT, glucose, ABG, CXR, ECG, urine dipstick
  Consider CT head if no other cause found

Management
  Correction of cause
  Acutely: supportive care, reassurance, prevention of self-harm or wandering
Pulmonary aspiration of gastric contents in anaesthesia - T. Engelhardt and N.R. Webster
BJA 1999; 83: 453-60

Pathophysiology

Particle related
Acid related
Critical pH 2.5 and volume 0.4 ml/kg may be untrue
HCl LD$_{50}$ 1.0 ml/kg in monkeys

Process
- Burn within 5 s
- Neutralized by 15 s
- Desquamation with 6 h
- Alveolar type II cells most sensitive
- Second phase: inflammatory mediators
  - Local effect ARDS, systemic effects

Bacterial related
- Mixed aerobes and anaerobes
  - Klebsiella, P. aeruginosa, E. coli, S. aureus

Detection
- No specific tests if aspiration is not witnessed and no gastric contents from ETT
- Bronchoscopy, lavage, brushing may yield evidence
- CXR may show signs after hours
- $\dot{V}/Q$ scanning in children

Incidence
- 1 in 2000-3000
- Swedish study of 185385 GAs
  - Four fatalities
    - One ASA IV ICU patient, two failed intubation during resuscitation and one kyphoscoliosis
  - Non fatal: 47% pneumonitis, 17% ventilation
- Mayo Clinic study of 215488 GAs
  - Three fatalities in ASA III-IV
- Obstetric patients
  - Incidence around 1 in 900 for Caesarean section
  - Negligible mortality (denominator unknown)

Prevention
- No useful correlation with BMI, smoking, fasting, alcohol use, volume, pH
- Fasting
  - Intermediate markers used in studies (volume and pH)
- Drugs
  - Intermediate markers used
  - Cost-benefit analysis impossible
  - Likely minimal effect on morbidity or mortality
- Anaesthetic technique
  - Rapid sequence induction recommended
  - Quality of cricoid pressure is variable
  - Overuse of the LMA has potential to increase incidence of aspiration

Treatment
- Head-down, suctioning, intubation, tracheal suctioning
- Ventilation with 100% oxygen
- No proof for steroids or antibiotics

Conclusion
- Minimal demonstrated mortality or morbidity
- Changes in anaesthetic practice have probably contributed to this
- No proven benefit from prophylactic measures

Complications 3.A.4.5 James Mitchell (December 24, 2003)
Perioperative stroke


**Stroke**
- Rapidly developing episode of focal or global loss of cerebral function with symptoms lasting more than 24 hours or leading to death
- Less than 24 hours: TIA
- Peri-operative stroke commonly defined as occurring intra-operatively or within 3-30 days postoperatively

**Epidemiology**
- Third most common cause of death
- Annual incidence 1-2 per 1000 population
  - Rises with age: 3 per 1000 at 60 y, 10-25 per 1000 at 80 y
- Acute mortality 15-30%, 45% independent after 1 year

**Surgery**
- Incidence
  - General surgery 0.2-0.7% (six times background risk)
  - With previous stroke 2.9%
  - Peripheral vascular surgery 1-3%
  - Carotid surgery 3-5%
  - Usually between 2 and 10 days postop
- Natural history
  - Acute mortality 26%

**Aetiology**
- 42% emboli of cardiac origin (33% AF)
- Vascular emboli from plaque
- In situ thrombosis (hypercoagulable state)

**Risk factors**
- Hypertension, cardiac disease (AF), PVD, diabetes, age
- Carotid disease has not been established as a risk factor for periop stroke

**Prevention**
- Preoperative
  - Identify risk factors and modify if possible (e.g. revert AF, anticoagulate)
  - Consider heparinization of patients on warfarin
  - Delay surgery 4-6 weeks after a stroke
- Intraoperative
  - Maintain oxygen delivery
    - Normotension, maintain Hb, high PaO₂, normocapnia
    - Normal blood sugar
    - Avoid excessive neck rotation or extension
    - Anaesthetic technique has not been shown to cause a difference
- Postoperative
  - Avoid hypotension
  - Avoid dehydration and hypercoagulability
  - Control anticoagulation
Respiratory depression under GA

Definition
Inadequate ventilation caused by an abnormality in control of respiration
Manifest as rising PaCO\(_2\) or falling PaO\(_2\)

Respiratory control
Afferent
Peripheral chemoreceptors predominantly for PaO\(_2\)
   Carotid bodies via IX
   Aortic bodies via X
Central chemoreceptors for PaCO\(_2\) (via CSF pH)
Lung receptors
   Pulmonary stretch receptors, irritant receptors, J receptors
Other receptors
   Nose and upper airway
   Joint and muscle, \(\gamma\) afferents
   Arterial baroreceptors
   Pain and temperature sensation

Efferent
   Central integration: cortex, hypothalamus, pons, medulla
   Spinal cord: dorsolateral UMN system
   Anterior horn cells: \(\alpha\) and \(\gamma\) fibres
Muscles
   Diaphragm, intercostals, accessory muscles

Signs
Spontaneously ventilating anaesthetized patient
   ↑ ETCO\(_2\), ↓ SpO\(_2\), ABG findings
Ventilated patient (underventilation)
   Same gas changes
   Mild hypercapnia: sympathetic stimulation
   Profound hypoventilation
      Myocardial irritability and depression, cyanosis, circulatory collapse

Causes
Anaesthetic
Drugs
   Central
      Induction agents, volatiles, opioids
      All ↓ response to PaCO\(_2\)
Supplemental O\(_2\) in patients reliant on hypoxic drive
Spinal cord
   High block, total spinal
Peripheral nerve
   LA blockade of phrenic nerve (e.g. deep cervical block)
Neuromuscular
   Muscle relaxants, volatiles, Mg\(^{2+}\) etc
Physiological change
   Hypothermia, hypoglycaemia
   Hyperventilation

Surgical
   Interruption of any part of reflex control e.g. brainstem
   Mechanical disruption of thorax
   Airway obstruction, pulmonary blood flow obstruction

Patient
   OSA
   CVA
   Apnoea of newborn

Complications 3.A.4.7 James Mitchell (December 24, 2003)
Adverse effects of laryngoscopy

Mechanical
- Trauma to teeth, dental work, tongue, pharynx, epiglottis
- Compression of soft tissues: lip, gums
- Eye injury
- Cervical spine injury if preexisting instability

Physiological
- Airway
  - Coughing with inadequate anaesthesia
    - ↑ ICP, IOP
    - Damage if open eye injury or cerebral vascular anomaly
  - Laryngospasm
  - Vomiting, aspiration
- Neurological
  - ↑ CBF together with ↑ ICP
  - May cause vagal response
  - Commonly causes sympathetic response
- Cardiovascular
  - Usually tachycardia, hypertension
    - ↑ myocardial O₂ demand, risk of ischaemia
  - Bradycardia if vagal response, more common in children
- Pulmonary
- Bronchospasm

Drug-related
- Hypnotics and muscle relaxants

Failure to secure the airway
- Hypoxia, aspiration, death

Minimizing adverse effect
- Patient selection
  - Airway assessment
  - Alternative anaesthetic techniques if laryngoscopy is likely to be problematic
  - Removal of dentures
- Equipment
  - Suitable sized and well-maintained airway equipment
  - Gentle use of laryngoscope
- Drugs
  - Blunt airway response
    - Local anaesthetic to upper airway
    - Nerve blocks to IX, superior laryngeal nerve
    - Prophylactic IV lignocaine, opioid
  - Blunt haemodynamic response
    - β-blocker, clonidine, vasodilators
    - Reduce airway reactivity
      - β₂ agonists, anticholinergics, steroids
  - Induction agents
    - Suitable doses and adequate time for muscle relaxant to work
Complications of, benefits of, indications for IVs, CVCs, PA catheters, arterial lines.

IV

Complications

Cannula and insertion
- Pain of insertion
- Vessel damage, thrombosis, haematoma, haemorrhage, local irritation

Through the cannula
- Infection, septicaemia

Fluids
- Fluid overload, incorrect fluid: electrolyte disturbance, transfusion reaction, hypothermia

Drugs
- Incorrect drug or dose, incorrect route of administration, administration too rapidly
- Extravasation with vessel damage or cannula misplacement

Dressing
- Skin reactions, allergy

Benefits

Drug administration
- Rapid, 100% of drug delivered, more secure and reliable than oral or PR administration
- Suitable for emergency and resuscitation drugs

Fluids
- In fasting patients or patient with ileus, allows hydration and electrolyte supplementation, nutrition possible

Other uses
- May allow blood sampling with large cannula or in infants

Indications

Requirement for parenteral fluids or drugs where
- Rapid effect is required
- Volume is too large or agent unsuitable to give subcutaneously
- Possible requirement for resuscitation drugs e.g. during mask anaesthetic

CVC

As for IV, plus

Complications

Cannula and insertion
- Insertion technique
  - Arterial or other vessel damage
  - Damage to nerves or other viscera (e.g. femoral insertion)
  - Pneumothorax
  - Arrhythmia related to wire or cannula irritating endocardium
  - Loss of guidewire

Cannula
- Vessel wall damage: haemothorax, pericardial tamponade
- Misplacement into cerebral or other vessels

Benefits

Drug administration
- Highly secure access
- Suitable for irritant or hypertonic agents requiring rapid mixing

Fluids
- Suitable for TPN
Measurement of CVP provides information to guide fluid management

Other
- Suitable for venous blood sampling, normovolaemic haemodilution
- Useable for 7 days up to months depending on cannula type

Indications
- Secure IV access (e.g. TIVA)
- Prolonged access (e.g. chemotherapy)
- Need for CVP measurement (e.g. large fluid shifts with major laparotomy)
- Inadequate access elsewhere

PA catheter
- As for CVC, plus

Complications
- Cannula and insertion
  - Large sheath increases risk of vessel damage
  - Greater risk of arrhythmia
  - Potential for injury to right heart, PA, smaller pulmonary vessels
- Balloon
  - May injure surrounding vessel
  - Expands with N\textsubscript{2}O
  - Site of entry of gas into circulation
Through the cannula
- Cardiac output boluses risk bolus injection of other agents

Benefits
- Measurements
  - CVP, PAP, PAOP, CO, SmvO\textsubscript{2}, SVR, PVR, MRO\textsubscript{2}
  - May guide fluid and inotrope management
  - No proven benefit
- Fluids
  - Sheath allows rapid infusion

Indications
- Requirement for measuring PA pressures or cardiac output or SVR
  - e.g. pulmonary hypertension, septic shock
Perioperative myocardial ischaemia

Epidemiology
Most common cause of perioperative (and non-periop.) death
Occurs most frequently postoperatively (peak day 3)
Symptoms obscured by surgical pain or analgesia (silent)

Myocardial oxygen balance
Demand
- Heart rate
- Diastolic volume (preload)
- Contractility
- Blood pressure (afterload)

Supply
- Coronary blood flow
- Diastolic duration
- Coronary perfusion pressure
- Coronary vessel size and patency
- Oxygen content
  - Haematocrit
  - \( \text{PaO}_2 \)

Triggers
- Tachycardia
- Anaemia
  - Some evidence for maintaining Hb >90 g/l in CAD patients
  - Hb \( \geq 70 \) g/l well-tolerated in normal patients

Monitoring
ECG
- Subendocardial ischaemia causes ST elevation
- Transmural ischaemia causes ST elevation
- Criteria for ischaemia
  - Horizontal or downsloping ST depression \( \geq 1 \) mm, 60-80 ms after J point
  - Duration \( \geq 1 \) min
  - Separation from other episodes by \( \geq 1 \) min of normal baseline
- Sensitivity (intraoperative)
  - \( V_5 \): 75%
  - II, \( V_5 \): 80%
  - II, \( V_4, V_5 \): 96%
- Advantages
  - Least expensive, most automated
- Limitations
  - RBBB, LBBB, AF, LVH with strain interfere with interpretation

TOE
- Segmental wall motion abnormality with ischaemia
- Advantages
  - Most sensitive: earlier signs and more sensitive than ECG
  - Information about regional ischaemia, valve function, CO
- Limitations
  - Expensive equipment and experienced operator required
  - Not well-tolerated without sedation

PA catheter
- Rise in PAOP or change in waveform (e.g. mitral regurgitation) with ischaemia
- Advantages
  - Information about CO
- Limitations
  - Less sensitive than ECG or TOE
  - Expensive, invasive

Complications
3.A.4.11
James Mitchell (December 24, 2003)
Arrest protocol

Cardiac arrest

BLS algorithm
  Secure airway, ventilate with 100% O₂

Praecordial thump

Attach defibrillator/monitor, IV access

Assess rhythm and pulse
  VF or pulseless VT
  Defibrillate up to 3 times
    200 J, 300 J, 360 J first time
    360 J subsequent times (4 J/kg)
  CPR up to 1 min, then reassess rhythm and pulse
  non VF/VT
  CPR up to 3 min, then reassess rhythm and pulse

During CPR
  Verify electrode, paddle and ETT placement
  IV access if not present
  Adrenaline 1 mg every 3 min
  Consider atropine, K⁺, lignocaine, bicarbonate if indicated

Consider reversible causes
  Hypoxia, hypovolaemia, hypothermia, K⁺, Mg²⁺, Ca²⁺, tension pneumothorax,
  tamponade, drug toxicity, thromboembolism

Malignant Hyperthermia
Miller 5th Edition Chapter 27

**History**
1929 Ombrédanne’s syndrome: post-op hyperthermia and pallor
1960 Denborough & Lovell case report in Australia
1966 Stress-susceptible swine described
1975 Dantrolene use described in swine and trialled in humans

**Epidemiology**
1 in 62,000 anaesthetics with triggering agents

**Aetiology**
Normal excitation-contraction coupling
- ACh binds to nicotinic receptors and opens cation channels
- Na⁺ influx raises membrane potential
- Voltage-gated Na⁺ channels open: depolarization
- Voltage-gated Ca²⁺ channels in T tubules open (L-type channels or DHPR)
- Physical linkage to sarcoplasmic reticulum ryanodine receptor (Ca²⁺ channel)
- Ca²⁺ released from SR activates myofibril contraction
- Rapid reuptake of Ca²⁺ into SR and binding to calsequestrin
- Termination of contraction

MH defect
- Ry¹ coded on chromosome 19 in humans
  - Multiple mutations described covering fewer than 50% of MH families
- Defects also described on chromosome 17 (Na⁺ channel, L-type Ca²⁺ channel), chromosome 7 (L-type Ca²⁺ channel), chromosome 1 (DHPR)
- Functional abnormality is complex at a molecular level
  - Increased tendency for Ca²⁺ release from SR
  - Decreased inhibition by Mg²⁺ and Ca²⁺
- Sustained high sarcoplasmic Ca²⁺ level causes sustained contraction, aerobic and glycolytic metabolism and thus rigidity, acidosis, hyperkalemia...

**Risk factors**
- Family history of MH
- King-Denborough syndrome, central core disease

**Clinical Features**
- Triggered by volatile anaesthetics or suxamethonium, but not consistently
  - Rise in muscle intracellular Ca²⁺, rigidity
  - Venous ↓ pH, ↓ PO₂, ↑ PCO₂, ↑ lactate, ↑ [K⁺]
  - Subsequent ↑ HR, ↑ BP, ↑ T
  - Temperature rise up to 1°C per 5 min
- Secondary DIC, neurological dysfunction, renal and cardiac failure and arrest
- Clinical syndrome may be indistinguishable from other causes of hypermetabolism
- Masseter spasm
  - Caused by suxamethonium
  - Present to a variable extent in most patients
  - Due to slow tonic fibres in masseters and lateral pterygoids
  - Increased risk of MH

**Acute treatment**
- Institution protocol
- Call for assistance
- Clean anaesthetic machine, hyperventilate with 100% O₂
- Cold fluids and packs
- Curtail surgery
- Dantrolene
  - Lipid soluble hydantoin

**Complications**
James Mitchell (December 24, 2003)
Low water solubility
20% oral bioavailability
Vd, 0.5 l/kg
Clearance 0.6 ml/min/kg
t1/2, 12 h
Therapeutic concentration >3 µg/ml
Metabolized to 5-OH dantrolene (50% potency)

Pharmacodynamics
Molecular action uncertain
Inhibits Ca2+ release from sarcoplasmic reticulum without inhibiting uptake
Limits excitation-contraction coupling in skeletal muscle

Adverse effects
Muscle weakness
Negative inotrope
↑ [K-]
Electrolyte and volume disturbance due to water and mannitol load

Indications
Malignant hyperpyrexia
Also used in
Neuroleptic malignant syndrome
MDMA overdose, serotonin syndrome with hyperpyrexia
Muscle cramps

Clinical use
Ampoules of 20 mg with 3 g mannitol, pH 9.5
Dissolved in 60 ml water → 1 mg/3 ml
Dose 1 mg/kg up to 10 mg/kg
= up to 30 ml/kg free water, 1.5 g/kg mannitol

Post-operative ICU, supportive treatment of other abnormalities
MH-safe anaesthesia
Safe agents
Regional (amide LA almost certainly safe)
N2O, non-depolarizing relaxants, propofol, barbiturates, etomidate, ketamine, opioids, benzodiazepines

Follow up
Testing family members (through RMH)
Two protocols (North American and European) for muscle biopsies
Ry; testing not sufficient because of heterogeneity in humans
http://www.mhaus.org/
Postoperative nausea and vomiting (PONV)

Preoperative
Assessment
Detailed history of previous anaesthetic problems and nature of surgery
Severity of PONV, duration, delay in discharge
Examine previous anaesthetic charts if available
Particularly if there has been a nausea-free anaesthetic
History of drug sensitivity, particularly narcotic analgesics

Preparation
Minimal safe fasting time
Preoperative hydration
Reassurance

Premedication
Anxiolytic, antiemetic
Lorazepam, ranitidine, metoclopramide
Consider anticholinergic e.g. scopolamine (risk of dysphoria)

Transport
Gentle ride, sitting up
Consider walking to theatre

Intraoperative
Avoid GA if feasible: regional blockade
But also avoid hypotension
Avoid N₂O, opioids, volatile agents
Propofol TIVA may be best choice for GA
Avoid muscle relaxation if possible, in order to avoid neostigmine
If neostigmine unavoidable, give slowly
Avoid specific emetogenic drugs: e.g. ergometrine
Give prophylactic antiemetic
Ondansetron and dexamethasone
Give adequate hydration
Empty stomach and deflate insufflated gas prior to awakening

Postoperative
Regional blockade for analgesia
Otherwise aim for opioid-free analgesic regimen
Paracetamol, NSAID, ketamine
Regular antiemetic, rescue therapy available
Continue IV hydration
Crisis Management in Anaesthesia

COVER algorithm derived by Runciman from AIMS reports (http://www.apsf.net.au/)
- Based on incident reports collected since 1988
- Performs better than the anaesthetist in 20-30% of incidents
- Performs worse in 1%
- Not always the best strategy if the cause of a problem is obvious
- Must be considered at the same time as resuscitation/ABCD

C
Circulation
  - Pulse, BP, ECG
Colour
  - Saturation, skin colour

O
Oxygen flow
  - 100% O₂, increase flow
Oxygen concentration
  - Oxygen monitor, gas analyzer

V
Vaporizer
  - Turn off (also remember intravenous drug infusions)
Ventilation
  - Change to manual ventilation, feel compliance and flow

E
Endotracheal tube
  - Check position, cuff
Eliminate machine
  - Change to Laerdal bag

R
Recheck monitors
Review

Causes of incidents
- 30% equipment/human interface problems
- 14% contributed to by haste
- 8% drug problems
- 8% equipment failures

Vigilance mnemonic (SCARE)
  - Scan every five minutes
  - Check on the unexpected
  - Alert &
  - Ready if a problem is suspected
  - Emergency mode in a deteriorating situation