Anaesthesia, Intensive Care and Perioperative Medicine A-Z

An Encyclopaedia of Principles and Practice

Sixth Edition

Steve M. Yentis
Nicholas P. Hirsch
James K. Ip

Original contributions by
Gary B Smith

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To
Gill, Emma and Abigail

to
Alison, Jonathan, Sophie and Alexander

and to
Pat, Ethan and Molly

Anaesthesia, Intensive Care and Perioperative Medicine A-Z

An Encyclopaedia of Principles and Practice

Sixth Edition

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Preface

In the 25 years since the publication of the first edition of our textbook, we have been delighted to find that 'the *A–Z*' has been adopted by both trainees and established practitioners alike. Whilst our original idea was to produce a readily accessible source of information for those sitting the Royal College of Anaesthetists' Fellowship examinations, it soon became obvious that the book appeals to a far wider readership. We hope that the *A–Z* will continue to be useful to all staff who help us care for patients on a daily basis, as well as to anaesthetists and intensivists of all grades.

As with previous new editions, each entry has been reviewed and, where appropriate, revised, and new ones inserted. We have also developed further the structured 'revision checklist' of entries, introduced in the 5th edition, that we hope will be useful to those preparing for examinations.

The difference between the list of entries in the first edition and those in the current one continues to increase, with a huge expansion of new entries and revision of existing ones. This change acknowledges the enormous breadth of information needed to satisfy the vast range of activities performed by our anaesthetic, intensive care, nursing and other colleagues, and also reflects the ever-changing field in which we work. With current consolidation of the role of anaesthetists as 'perioperative physicians', we have also developed and/or introduced entries that are particularly relevant to this aspect of our work, and emphasise this in the altered title of the book.

The publication of a textbook requires the support of a multitude of people. We are indebted to our colleagues, both junior and senior, who have gently criticised previous editions; their suggestions have been invaluable and have directly resulted in changes found in each new edition over the years. We are particularly grateful to Drs Helen Laycock and Harriet Wordsworth, Chelsea and Westminster Hospital/Imperial College, for advising on entries related to pain and pain management for this edition. We also thank the staff of Elsevier and their predecessors for their support during the life of this project. Finally, this is the second edition of the *A–Z* on which two of the original authors, SMY and NPH, have worked without the third, Gary Smith, though his contributions exist throughout the book in both the format and content of entries from previous editions. We are both delighted to continue to work with James Ip on this edition, having successfully done so on the previous one.

SMY
NPH
JKI

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Explanatory notes

Arrangement of text

Entries are arranged alphabetically, with some related subjects grouped together to make coverage of one subject easier. For example, entries relating to tracheal intubation may be found under **I** as in **Intubation, awake**; **Intubation, blind nasal**, etc.

Cross-referencing

Bold type indicates a cross-reference. An abbreviation highlighted in bold type refers to an entry in its fully spelled form. For example, '**ALI** may occur …' refers to the entry **Acute lung injury**. Further instructions appear in italics.

References

Reference to a suitable article is provided at the foot of the entry where appropriate.

Proper names

Where possible, a short biographical note is provided at the foot of the entry when a person is mentioned. Dates of birth and death are given, or the date of description if these dates are unknown. No dates are given for contemporary names. Where more than one eponymous entry occurs, e.g., **Haldane apparatus** and **Haldane effect**, details are given under the first entry. The term 'anaesthetist' is used in the English sense, i.e., a medical practitioner who practises anaesthesia; the terms 'anesthesiologist' and 'anaesthesiologist' are not used.

Drugs

Individual drugs have entries where they have especial relevance to, or may by given by, the anaesthetist or intensivist. Where many different drugs exist within the same group, for example, β-adrenergic receptor antagonists, those which may be given intravenously have their own entry, whilst the others are described under the group description. The reader is referred back to entries describing drug groups and classes where appropriate.

Recommended International Nonproprietary Names (rINNs) and chemical names

Following work undertaken by the World Health Organization, European law requires the replacement of existing national drug nomenclature with rINNs. For most drugs, rINNs are identical to the British Approved Name (BAN). The Medicines Control Agency (UK) has proposed a two-stage process for the introduction of rINNs. For substances where the change is substantial, both names will appear on manufacturers' labels and leaflets for a number of years, with the rINN preceding the BAN on the drug label. For drugs where the change presents little hazard, the change will be immediate. For some drugs which do not appear in either of the above two categories, the British (or USP) name may still be used.

There are over 200 affected drugs, many of them no longer available. Affected drugs that are mentioned in this book (though not all of them have their own entries) are listed below—though please note that, in common with the British Pharmacopoeia, the terms 'adrenaline' and 'noradrenaline' will be used throughout the text in preference to 'epinephrine' and 'norepinephrine', respectively, because of their status as natural hormones. Thus (except for adrenaline and noradrenaline), the format for affected drugs is rINN (BAN), e.g., **Tetracaine hydrochloride** (Amethocaine). Non-BAN, non-rINN names are also provided for certain other drugs (for example, **Isoproterenol**, *see Isoprenaline*) to help direct non-UK readers or those unfamiliar with UK terminology.

Similarly, the International Union of Pure and Applied Chemistry (IUPAC) nomenclature of inorganic chemistry is used for consistency, even though some of the spelling (outside of drug names) is not widely used in UK English, e.g., sulfur/sulfate instead of sulphur/sulphate.

Examination revision checklist

At the front of the book is a checklist based on entries of particular relevance to examination candidates, which have been classified and listed alphabetically in order to support systematic study of examination topics according to the subject area.

continued over page

BAN	*rINN*
Adrenaline	Epinephrine
Amethocaine	Tetracaine
Amoxycillin	Amoxicillin
Amphetamine	Amfetamine
Amylobarbitone	Amobarbital
Beclomethasone	Beclometasone
Benzhexol	Trihexyphenidyl
Benztropine	Benzatropine
Busulphan	Busulfan
Cephazolin	Cefazolin
Cephradine	Cefradine
Cephramandole	Ceframandole
Chlormethiazole	Clomethiazole
Chlorpheniramine	Chlorphenamine
Corticotrophin	Corticotropin
Cyclosporin	Ciclosporin
Dicyclomine	Dicycloverine
Dothiepin	Dosulepin
Ethacrynic acid	Etacrynic acid
Ethamsylate	Etamsylate
Frusemide	Furosemide
Indomethacin	Indometacin
Lignocaine	Lidocaine
Methohexitone	Methohexital
Methylene blue	Methylthioninium chloride
Noradrenaline	Norepinephrine
Oxpentifylline	Pentoxifylline
Phenobarbitone	Phenobarbital
Sodium cromoglycate	Sodium cromoglicate
Sulphadiazine	Sulfadiazine
Sulphasalazine	Sulfasalazine
Tetracosactrin	Tetracosactide
Thiopentone	Thiopental
Tribavarin	Ribavarin
Trimeprazine	Alimemazine

Abbreviations

ACE inhibitors angiotensin converting enzyme inhibitors
ACTH adrenocorticotrophic hormone
ADP adenosine diphosphate
AF atrial fibrillation
AIDS acquired immune deficiency syndrome
ALI acute lung injury
APACHE acute physiology and chronic health evaluation
ASA American Society of Anesthesiologists
ASD atrial septal defect
ATP adenosine triphosphate
AV atrioventricular
bd twice daily
BP blood pressure
cAMP cyclic adenosine monophosphate
$CMRO_2$ cerebral metabolic rate for oxygen
CNS central nervous system
CO_2 carbon dioxide
COPD chronic obstructive pulmonary disease
CPAP continuous positive airway pressure
CPR cardiopulmonary resuscitation
CSE combined spinal–extradural
CSF cerebrospinal fluid
CT computed tomography
CVP central venous pressure
CVS cardiovascular system
CXR chest x-ray
DIC disseminated intravascular coagulation
DNA deoxyribonucleic acid
2,3-DPG 2,3-diphosphoglycerate
DVT deep vein thrombosis
ECF extracellular fluid
ECG electrocardiography
EDTA ethylenediaminetetraacetate
EEG electroencephalography
EMG electromyography
ENT ear, nose and throat
FEV_1 forced expiratory volume in 1 s
F_IO_2 fractional inspired concentration of oxygen
FRC functional residual capacity
FVC forced vital capacity
G gauge
GABA γ-aminobutyric acid
GFR glomerular filtration rate
GIT gastrointestinal tract
GTN glyceryl trinitrate
HCO_3^- bicarbonate
HDU high dependency unit
HIV human immunodeficiency virus
HLA human leucocyte antigen
5-HT 5-hydroxytryptamine
ICP intracranial pressure
ICU intensive care unit
IgA, IgG, etc. immunoglobulin A, G, etc.
im intramuscular
IMV intermittent mandatory ventilation
IPPV intermittent positive pressure ventilation
iv intravenous
IVRA intravenous regional anaesthesia
JVP jugular venous pressure
LM laryngeal mask
MAC minimal alveolar concentration
MAP mean arterial pressure
MH malignant hyperthermia
MI myocardial infarction
MODS multiple organ dysfunction syndrome
MRI magnetic resonance imaging
mw molecular weight
NAD(P) nicotinamide adenine dinucleotide (phosphate)
NHS National Health Service
NICE National Institute for Health and Care Excellence
NMDA *N*-methyl-D-aspartate
N_2O nitrous oxide
NSAID non-steroidal anti-inflammatory drug
O_2 oxygen
od once daily
ODA/P operating department assistant/practitioner
P_{CO_2} partial pressure of carbon dioxide
PE pulmonary embolus
PEEP positive end-expiratory pressure
P_{O_2} partial pressure of oxygen
PONV postoperative nausea and vomiting
pr per rectum
qds four times daily
RNA ribonucleic acid
RS respiratory system
SAD supraglottic airway device
sc subcutaneous
SIRS systemic inflammatory response syndrome
SLE systemic lupus erythematosus
SVP saturated vapour pressure
SVR systemic vascular resistance
SVT supraventricular tachycardia
TB tuberculosis
TBI traumatic brain injury
tds three times daily
TENS transcutaneous electrical nerve stimulation
THRIVE transnasal humidified rapid-insufflation ventilatory exchange
TIVA total intravenous anaesthesia
TPN total parenteral nutrition
TURP transurethral resection of prostate
UK United Kingdom
US(A) United States (of America)
VF ventricular fibrillation
$\dot{V}/\dot{Q}$ ventilation/perfusion
VSD ventricular septal defect
VT ventricular tachycardia

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Examination revision checklist

This checklist has been compiled from entries of particular relevance to examination candidates, classified and listed alphabetically in order to support systematic study of examination topics. This list is not exhaustive, and, for clarity, entries that summarise a topic and incorporate multiple cross-references (e.g. Opioid analgesic drugs) have been included in preference to listing every relevant entry, e.g. Alfentanil, Fentanyl, Remifentanil, etc. The latter should still be referred to where appropriate, to gain relevant detail.

Checklist index:

PHYSIOLOGY

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- Atrial natriuretic peptide.
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- Cardiac output.
- Cardioinhibitory centre.
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GASTROINTESTINAL

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- Alkalosis, respiratory.
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CLINICAL ANATOMY

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MUSCULOSKELETAL SYSTEM

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- Skull.
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- Vertebrae.

NERVOUS SYSTEM

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- Brain.
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- Opioid receptors.

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CARDIOVASCULAR

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- α-Adrenergic receptor antagonists.
- β-Adrenergic receptor agonists.
- β-Adrenergic receptor antagonists.
- Antiarrhythmic drugs.
- Anticholinergic drugs.
- Antihypertensive drugs.
- Calcium channel blocking drugs.
- Calcium sensitisers.
- Cardiac glycosides.
- Diuretics.
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GASTROINTESTINAL

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PHYSICS AND MEASUREMENT

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- Work.

CLINICAL MEASUREMENT

- Amplifiers.
- Arterial blood pressure measurement.
- Arterial cannulation.
- Arterial waveform.
- Becquerel.
- Bispectral index monitor.
- Body mass index (BMI).
- Calibration.
- Capnography.
- Carbon dioxide measurement.
- Cardiac output measurement.
- Cerebral function monitor.
- cgs system of units.
- Damping.
- Dilution techniques.
- End-tidal gas sampling.
- Fade.
- Fick principle.
- Flame ionisation detector.
- Flowmeters.
- Flow–volume loops.
- Gain, electrical.
- Gas analysis.
- Gas chromatography.
- Haldane apparatus.
- Hygrometer.
- Hysteresis.
- Impedance plethysmography.
- Isosbestic point.
- Korotkoff sounds.
- LiMON.
- Mass spectrometer.
- Monitoring.
- Neuromuscular blockade monitoring.
- Oscillotonometer.
- Oximetry.
- Oxygen measurement.
- Peak flowmeters.
- pH measurement.
- Phase shift.
- Plethysmography.
- Pneumotachograph.
- Pressure measurement.
- Pulse oximeter.
- Respirometer.
- Rotameter.
- Spectroscopy.
- Spirometer.
- Thromboelastography (TEG).
- Transducers.

ELECTRICITY

- Antistatic precautions.
- Capacitance.
- Conductance.
- Coulomb.
- Current.
- Current density.
- Defibrillation.
- Electrical symbols.
- Electrocution and electrical burns.
- Impedance, electrical.
- Inductance.
- Resistance.
- Volt.

STATISTICS

- Absolute risk reduction.
- Confidence intervals.
- Data.
- Degrees of freedom.
- Errors, statistical.
- Likelihood ratio.
- Mean.
- Median.
- Meta-analysis (Systematic review).
- Mode.
- Null hypothesis.
- Number needed to treat (NNT).
- Odds ratio.
- Percentile.
- Populations.
- Power (in Statistics).
- Predictive value.
- Probability (*P*).
- Randomisation.
- Receiver operating characteristic curves.
- Relative risk reduction.
- Samples, statistical.
- Sensitivity.
- Specificity.
- Standard deviation.
- Standard error of the mean.
- Statistical frequency distributions.
- Statistical significance.
- Statistical tests.
- Variance.

CLINICAL ANAESTHESIA

GENERAL TOPICS

- Altitude, high.
- Altitude, low.
- Anaesthesia, depth of.
- Anaesthesia, stages of.
- Anaesthetic morbidity and mortality.
- Anaesthetists' non-technical skills.

- Anaphylaxis.
- ASA physical status.
- Aspiration of gastric contents.
- Awareness.
- Bariatric surgery.
- Blood loss, perioperative.
- Bronchospasm.
- Carbon dioxide narcosis.
- Cardiac risk indices.
- Cardiopulmonary exercise testing.
- Cell salvage.
- Central venous cannulation.
- Confusion, postoperative.
- Consent for anaesthesia.
- Cricoid pressure (Sellick's manoeuvre).
- Cricothyroidotomy.
- Day-case surgery.
- Elderly, anaesthesia for.
- Electroconvulsive therapy.
- Emergence phenomena.
- Emergency surgery.
- Environmental impact of anaesthesia.
- Environmental safety of anaesthetists.
- Explosions and fires.
- Extubation, tracheal.
- Eye care.
- Fluid balance.
- Gas embolism.
- Heat loss, during anaesthesia.
- Hypotensive anaesthesia.
- Hypothermia.
- Hypoventilation.
- Hypovolaemia.
- Induction of anaesthesia.
- Induction, rapid sequence.
- Intubation, awake.
- Intubation, blind nasal.
- Intubation, complications of.
- Intubation, difficult.
- Intubation, failed.
- Intubation, fibreoptic.
- Intubation, oesophageal.
- Intubation, tracheal.
- Investigations, preoperative.
- Jehovah's Witnesses.
- Laparoscopy.
- Laryngoscopy.
- Laryngospasm.
- Liver transplantation.
- Malignant hyperthermia.
- Medicolegal aspects of anaesthesia.
- Nerve injury during anaesthesia.
- Obesity.
- Plastic surgery.
- Positioning of the patient.
- Postoperative analgesia.
- Postoperative cognitive dysfunction.
- Postoperative nausea and vomiting.
- Premedication.
- Preoperative assessment.
- Preoperative optimisation.
- Preoxygenation.
- Radiology, anaesthesia for.
- Recovery, enhanced.
- Recovery from anaesthesia.
- Regurgitation.
- Sedation.
- Seldinger technique.
- Shivering, postoperative.
- Smoking.
- Sore throat, postoperative.
- Stress response to surgery.
- Substance abuse.
- Teeth.
- Temperature regulation.
- Total intravenous anaesthesia (TIVA).
- Tourniquets.
- Transnasal humidified rapid-insufflation ventilator exchange (THRIVE).

CARDIOTHORACIC

- Cardiac surgery.
- Cardiopulmonary bypass.
- Heart transplantation.
- Lung transplantation.
- One-lung anaesthesia.
- Thoracic surgery.

ENT/MAXILLOFACIAL

- Airway obstruction.
- Bronchoscopy.
- Dental surgery.
- Ear, nose and throat surgery.
- Epistaxis.
- Facial trauma.
- Foreign body, inhaled.
- Injector techniques.
- Insufflation techniques.
- Ludwig's angina.
- Maxillofacial surgery.
- Stridor.
- Tonsil, bleeding.
- Trismus.

NEUROANAESTHESIA

- Head injury.
- Neurosurgery.
- Spinal surgery.

OBSTETRICS

- Amniotic fluid embolism.
- Antepartum haemorrhage.
- Aortocaval compression.
- Caesarean section.
- Confidential Enquiries into Maternal Deaths.
- Fetal monitoring.
- Fetus, effects of anaesthetic drugs on.
- HELLP syndrome.
- Obstetric analgesia and anaesthesia.
- Placenta praevia.
- Placental abruption.
- Postpartum haemorrhage.
- Pre-eclampsia.

OPHTHALMIC

- Eye, penetrating injury.
- Intraocular pressure.
- Oculocardiac reflex.
- Ophthalmic surgery.

ORTHOPAEDICS

- Bone cement implantation syndrome.
- Bone marrow harvest.
- Fat embolism.
- Fractured neck of femur.
- Kyphoscoliosis.
- Orthopaedic surgery.

PAEDIATRICS

- Apgar scoring system.
- Choanal atresia.
- Croup.
- Diaphragmatic herniae.
- Epiglottitis.
- Facial deformities, congenital.
- Gastroschisis and exomphalos.
- Necrotising enterocolitis.
- Neonate.
- Paediatric anaesthesia.
- Pyloric stenosis.
- Tracheo-oesophageal fistula.
- Transposition of the great arteries.

PAIN

- Acupuncture.
- Central pain.
- Coeliac plexus block.
- Complex regional pain syndrome.
- Gasserian ganglion block.
- Pain.
- Pain evaluation.
- Pain management.
- Pain, neuropathic.
- Patient-controlled analgesia.
- Phantom limb.
- Spinal cord stimulation.
- Stellate ganglion block.
- Sympathetic nerve blocks.
- Transcutaneous electrical nerve stimulation.
- Trigger points.

REGIONAL

- Adductor canal block.
- Ankle, nerve blocks.
- Blood patch, epidural.
- Brachial plexus block.
- Caudal analgesia.
- Cervical plexus block.
- Combined spinal–epidural anaesthesia.
- Dural tap.
- Epidural anaesthesia.
- Fascia iliaca compartment block.
- Femoral nerve block.
- Inguinal hernia field block.
- Intercostal nerve block.
- Interpleural analgesia.
- Intravenous regional anaesthesia.
- Knee, nerve blocks.
- Paravertebral block.
- Pecs block.
- Penile block.
- Peribulbar block.
- Post-dural puncture headache.
- Psoas compartment block.
- Rectus sheath block.
- Regional anaesthesia.
- Retrobulbar block.
- Sciatic nerve block.
- Serratus anterior plane block.
- Spinal anaesthesia.
- Sub-Tenon's block.
- Transversus abdominis plane block.
- Wrist, nerve blocks.

UROLOGY

- Extracorporeal shock wave lithotripsy.
- Renal transplantation.
- Transurethral resection of the prostate.
- TURP syndrome.
- Urinary retention.

VASCULAR

- Aortic aneurysm, abdominal.
- Aortic aneurysm, thoracic.
- Aortic dissection.
- Carotid endarterectomy.

CRITICAL CARE

GENERAL TOPICS

- Critical care.
- Imaging in intensive care.
- Lactic acidosis.
- Multiple organ dysfunction syndrome (MODS).
- Organ donation.
- Paediatric intensive care.
- Targeted temperature management.
- Transportation of critically ill patients.
- Withdrawal of treatment in ICU.

CARDIOVASCULAR

- Cardiogenic shock.
- Heparin-induced thrombocytopenia.
- Pulmonary artery catheterisation.
- Pulmonary capillary wedge pressure.
- Septic shock.
- Shock.

GASTROINTESTINAL

- Abdominal compartment syndrome.
- Glycaemic control in the ICU.
- Nutrition, enteral.
- Nutrition, total parenteral (TPN).
- Pancreatitis.

- Refeeding syndrome.
- Selective decontamination of the digestive tract.
- Stress ulcers.

NEUROLOGICAL

- Brainstem death.
- Cerebral hypoxic ischaemic injury.
- Cerebral protection/resuscitation.
- Coma.
- Confusion in the intensive care unit.
- Coning.
- Critical illness polyneuropathy.
- Guillain–Barré syndrome.
- Intracranial pressure monitoring.
- Sedation scoring systems.
- Spinal cord injury.
- Status epilepticus.
- Subarachnoid haemorrhage.
- Vegetative state.

ORGANISATIONAL

- APACHE scoring system.
- Care bundles.
- Intensive care follow-up.
- Intensive care unit.
- Mortality/survival prediction on intensive care unit.

RESPIRATORY

- Acute lung injury.
- Alveolar recruitment manoeuvre.
- Assisted ventilation.
- Barotrauma.
- Continuous positive airway pressure.
- Dynamic hyperinflation.
- Extracorporeal membrane oxygenation.
- High-frequency ventilation.
- Hypercapnia.
- Hypoxaemia.
- Inspiratory: expiratory ratio (I:E ratio).
- Intermittent positive pressure ventilation.
- Lung protection strategies.
- Non-invasive positive pressure ventilation.
- Pleural effusion.
- Pneumothorax.
- Respiratory failure.
- Respiratory muscle fatigue.
- Tracheostomy.
- Transfusion-related acute lung injury (TRALI)
- Ventilator-associated lung injury.
- Ventilator-associated pneumonia.
- Ventilators.
- Weaning from ventilators.

RESUSCITATION

- Advanced life support, adult.
- Basic life support, adult.
- Cardiac arrest.
- Cardiopulmonary resuscitation (CPR).
- Cardiopulmonary resuscitation, neonatal.
- Cardiopulmonary resuscitation, paediatric.
- Choking.
- Intraosseous fluid administration.
- Near-drowning.

TRAUMA

- Abdominal trauma.
- Burns.
- Chest trauma.
- Compartment syndromes.
- ‘Golden hour’.
- Pelvic trauma.
- Peritoneal lavage.
- Rib fractures.
- Trauma.
- Traumatic brain injury.

EQUIPMENT

AIRWAY

- Airway exchange catheter.
- Airways.
- Cuffs, of tracheal tubes.
- Endobronchial tubes.
- Facemasks.
- Fibreoptic instruments.
- Intubation aids.
- Laryngeal mask (LM).
- Laryngoscope.
- Laryngoscope blades.
- Minitracheotomy.
- Oesophageal obturators and airways.
- Supraglottic airway device (SAD).
- Tracheal tubes.

BREATHING SYSTEMS

- Adjustable pressure-limiting valves.
- Anaesthetic breathing systems.
- Carbon dioxide absorption, in anaesthetic breathing systems.
- Circle systems.
- Coaxial anaesthetic breathing systems.
- Demand valves.
- Filters, breathing system.
- Heat–moisture exchanger (HME).
- Humidification.
- Nebulisers.
- Non-rebreathing valves.
- Reservoir bag.
- Scavenging.
- Self-inflating bags.
- Soda lime.
- Triservice apparatus.

GAS SUPPLY

- Air.
- Bodok seal.
- Cylinders.
- Filling ratio.
- Oxygen.
- Oxygen concentrator.

- Oxygen failure warning device.
- Pin index system.
- Piped gas supply.
- Pressure regulators.
- Vacuum insulated evaporator (VIE).

OTHER

- Anaesthetic machines.
- Blood filters.
- Checking of anaesthetic equipment.
- Contamination of anaesthetic equipment.
- Diathermy.
- Gauge.
- Luer connectors.
- Needles.
- Suction equipment.
- Syringes.
- Vaporisers.

MEDICINE

CARDIOLOGY

- Acute coronary syndromes.
- Aortic regurgitation.
- Aortic stenosis.
- Arrhythmias.
- Bundle branch block.
- Cardiac catheterisation.
- Cardiac enzymes.
- Cardiac failure.
- Cardiac pacing.
- Cardiac tamponade.
- Cardiomyopathy.
- Cardioversion, electrical.
- Congenital heart disease.
- Cor pulmonale.
- Defibrillators, implantable cardioverter.
- Echocardiography.
- Electrocardiography (ECG).
- Endocarditis, infective.
- Heart block.
- Hypertension.
- Ischaemic heart disease.
- Mitral regurgitation.
- Mitral stenosis.
- Myocardial ischaemia.
- Myocarditis.
- Percutaneous coronary intervention (PCI).
- Pericarditis.
- Prolonged Q–T syndromes.
- Pulmonary hypertension.
- Pulmonary oedema.
- Pulmonary valve lesions.
- Stokes–Adams attack.
- Torsades de pointes.
- Transoesophageal echocardiography.
- Tricuspid valve lesions.

DERMATOLOGY/MUSCULOSKELETAL/RHEUMATOLOGY

- Ankylosing spondylitis.
- Connective tissue diseases.
- Marfan's syndrome.
- Muscular dystrophies.
- Myotonic syndromes.
- Rheumatoid arthritis.
- Sarcoidosis.
- Stevens–Johnson syndrome.
- Systemic lupus erythematosus.
- Vasculitides.

ENDOCRINOLOGY

- Acromegaly.
- Adrenocortical insufficiency.
- Cushing's syndrome.
- Diabetes mellitus.
- Diabetic coma.
- Hyperaldosteronism.
- Hyperthyroidism
- Hypopituitarism.
- Hypothyroidism.
- Phaeochromocytoma.
- Sick euthyroid syndrome.
- Thyroid crisis.

GASTROENTEROLOGY

- Ascites.
- Carcinoid syndrome.
- Diarrhoea.
- Gastrointestinal haemorrhage.
- Gastro-oesophageal reflux.
- Hepatic failure.
- Hepatitis.
- Hiatus hernia.
- Liver function tests.

GENERAL

- Alcoholism.
- Anaemia.
- Decompression sickness.
- Deep vein thrombosis (DVT).
- Dehydration.
- Down's syndrome.
- Hypercalcaemia.
- Hyperglycaemia.
- Hyperkalaemia.
- Hypernatraemia.
- Hyperthermia.
- Hypocalcaemia.
- Hypoglycaemia.
- Hypokalaemia.
- Hypomagnesaemia.
- Hyponatraemia.
- Hypophosphataemia.
- Inborn errors of metabolism.
- Malignancy.
- Malnutrition.
- Porphyria.
- Pyrexia.
- Syndrome of inappropriate antidiuretic hormone secretion (SIADH).
- Systemic inflammatory response syndrome.
- Vasovagal syncope.

HAEMATOLOGY/IMMUNOLOGY

- Autoimmune disease.
- Blood compatibility testing.
- Blood products.
- Blood storage.
- Blood transfusion.
- Bone marrow transplantation.
- Coagulation disorders.
- Coagulation studies.
- Disseminated intravascular coagulation.
- Glucose-6-phosphate dehydrogenase deficiency.
- Haemoglobinopathies.
- Haemolysis.
- Haemophilia.
- Immunodeficiency.
- Latex allergy.
- Methaemoglobinaemia.
- Rhesus blood groups.
- Thrombocytopenia.
- Thrombotic thrombocytopenic purpura.
- Tumour lysis syndrome.
- von Willebrand's disease.

MICROBIOLOGY AND INFECTIOUS DISEASES

- Bacteria.
- Blood cultures.
- Catheter-related sepsis.
- Cellulitis.
- Clostridial infections.
- Human immunodeficiency viral (HIV) infection.
- Infection control.
- Influenza.
- Meningococcal disease.
- Necrotising fasciitis.
- Nosocomial infection.
- Notifiable diseases.
- Pseudomonas infections.
- Sepsis.
- Staphylococcal infections.
- Streptococcal infections.
- Tropical diseases.
- Tuberculosis (TB).

NEUROLOGY/PSYCHIATRY

- Amnesia.
- Anorexia nervosa.
- Anterior spinal artery syndrome.
- Autonomic hyperreflexia.
- Autonomic neuropathy.
- Cauda equina syndrome.
- Central pontine myelinolysis.
- Cerebral abscess.
- Cerebral ischaemia.
- Cerebral oedema.
- Cholinergic crisis.
- Coma scales.
- Convulsions.
- Demyelinating diseases.
- Electroencephalography (EEG).
- Electromyography (EMG).
- Encephalopathy.
- Epilepsy.
- Extradural (epidural) haemorrhage.
- Horner's syndrome.
- Hydrocephalus.
- Lumbar puncture.
- Meningitis.
- Migraine.
- Motor neurone disease.
- Motor neurone, lower.
- Motor neurone, upper.
- Myasthenia gravis.
- Neurofibromatosis.
- Neuroleptic malignant syndrome.
- Paralysis, acute.
- Parkinson's disease.
- Peripheral neuropathy.
- Post-traumatic stress disorder.
- Stroke.
- Subdural haemorrhage.
- Trigeminal neuralgia.

POISONING

- β-Adrenergic receptor antagonist poisoning.
- Alcohol poisoning.
- Barbiturate poisoning.
- Benzodiazepine poisoning.
- Carbon monoxide poisoning.
- Charcoal, activated.
- Chelating agents.
- Cocaine poisoning.
- Cyanide poisoning.
- Heavy metal poisoning.
- Iron poisoning.
- Opioid poisoning.
- Organophosphorus poisoning.
- Paracetamol poisoning.
- Paraquat poisoning.
- Poisoning and overdoses.
- Salicylate poisoning.
- Serotonin syndrome.
- Tricyclic antidepressant drug poisoning.

RENAL

- Acute kidney injury (AKI).
- Crush syndrome.
- Diabetes insipidus.
- Dialysis.
- Glomerulonephritis.
- Hepatorenal syndrome.
- Myoglobinuria.
- Oliguria.
- Renal failure.
- RIFLE criteria.

RESPIRATORY

- Aspiration pneumonitis.
- Asthma.
- Atelectasis.
- Bronchial carcinoma.
- Bronchiectasis.
- Bronchoalveolar lavage.

- Chest drainage.
- Chest infection.
- Chronic obstructive pulmonary disease.
- Cystic fibrosis.
- Dyspnoea.
- Forced expiration.
- Fowler's method.
- Helium.
- Hypocapnia.
- Lung function tests.
- Obstructive sleep apnoea.
- Oxygen, hyperbaric.
- Oxygen therapy.
- Oxygen toxicity.
- Pulmonary embolism (PE).
- Pulmonary fibrosis.

ORGANISATIONAL

- Advance decision.
- Clinical governance.
- Clinical trials.
- Coroner.
- COSHH regulations (Control of Substances Hazardous to Health).
- Critical incidents.
- Do not attempt resuscitation orders.
- Ethics.
- Incident, major.
- Mental Capacity Act 2005.
- National audit projects.
- Never events.
- Organ donation.
- Pollution.
- Recovery room.

RADIOLOGY

- Chest x-ray.
- Computed (axial) tomography (CT).
- Magnetic resonance imaging (MRI).
- Positron emission tomography (PET).
- Radioisotope scanning.
- Radiological contrast media.
- Ultrasound.

A

A severity characterisation of trauma (ASCOT). **Trauma scale** derived from the **Glasgow coma scale**, systolic BP, **revised trauma score**, **abbreviated injury scale** and age. A logistic regression equation provides a probability of mortality. Excludes patients with very poor or very good prognoses. Has been claimed to be superior to the **trauma revised injury severity score** system, although is more complex.
Champion HR, Copes WS, Sacco WJ, et al (1996). J Trauma; 40: 42–8

A–adO_2, *see Alveolar–arterial oxygen difference*

ABA, *see American Board of Anesthesiology*

Abbott, Edward Gilbert, *see Morton, William*

Abbreviated injury scale (AIS). **Trauma scale** first described in 1971 and updated many times since. Comprises a classification of injuries with each given a six-digit code (the last indicating severity, with 1 = minor and 6 = fatal). The codes are linked to International Classification of Diseases codes, thus aiding standardisation of records. The anatomical profile is a refinement in which the locations of injuries are divided into four categories; the AIS scores are added and the square root taken to minimise the contribution of less severe injuries.
Gennarelli TA, Wodzin E (2006). Injury; 37: 1083–91

Abciximab. Monoclonal antibody used as an **antiplatelet drug** and adjunct to **aspirin** and **heparin** in high-risk patients undergoing **percutaneous coronary intervention**. Consists of Fab fragments of **immunoglobulin** directed against the glycoprotein IIb/IIIa receptor on the **platelet** surface. Inhibits platelet aggregation and thrombus formation; effects last 24–48 h after infusion. Careful consideration of risks and benefits should precede use because risk of bleeding is increased. Licensed for single use only.

- Dosage: initial loading of 250 µg/kg over 1 min iv, followed by iv infusion of 125 ng/kg/min (max 10 µg/min) 10–60 min (up to 24 h in unstable angina) before angioplasty with 125 µg/kg/min (up to 10 µg/min for 12 h afterwards).
- Side effects: bleeding, hypotension, nausea, bradycardia. Thrombocytopenia occurs rarely.

Abdominal compartment syndrome. Combination of increased **intra-abdominal pressure** (>12 mmHg [16 cmH_2O]) and organ dysfunction (e.g. following **abdominal trauma** or extensive surgery) resulting from haemorrhage or expansion of the **third space** fluid compartment. May also follow **liver transplantation**, **sepsis**, **burns** and acute **pancreatitis**. Intra-abdominal pressures above 15–18 mmHg (20–25 cmH_2O) may impair ventilation and be associated with reduced venous return, cardiac output, renal blood flow and urine output. Increased CVP may lead to raised ICP. Diagnosed by clinical features and intra-abdominal pressure measurement (performed via a bladder catheter or nasogastric tube, in combination with a water column manometer).

Management includes laparotomy ± Silastic material to cover the abdominal contents. **Paracentesis** may be effective if raised intra-abdominal pressure is due to accumulation of fluid, e.g. ascites. Full resuscitation must be performed before decompression as rapid release of pressure may result in sudden washout of inflammatory mediators from ischaemic tissues, causing acidosis and hypotension. Mortality of the syndrome is 25%–70%.
Kirkpatrick AW, Roberts DJ, De Waele J, et al (2013). Int Care Med; 39: 1190–206
See also, Compartment syndromes

Abdominal field block. Technique using 100–200 ml **local anaesthetic agent**, involving infiltration of the skin, subcutaneous tissues, abdominal muscles and fascia. Provides analgesia of the abdominal wall and anterior peritoneum, but not of the viscera. Now rarely used. **Rectus sheath block**, **transversus abdominis plane block**, **iliac crest block** and **inguinal hernia field block** are more specific blocks.

Abdominal sepsis, *see Intra-abdominal sepsis*

Abdominal trauma. May be blunt (e.g. road traffic accidents) or penetrating (e.g. stabbing, bullet wounds). Often carries a high morbidity and mortality because injuries may go undetected. Massive intra-abdominal blood loss or **abdominal compartment syndrome** may follow. The abdomen can be divided into three areas:

- intrathoracic: protected by the bony thoracic cage. Contains the spleen, liver, stomach and **diaphragm**. Injury may be associated with **rib fractures**. The diaphragm may also be injured by blows to the lower abdomen (which impart pressure waves to the diaphragm) or by penetrating injuries of the chest.
- true abdomen: contains the small and large bowel, bladder and, in the female, uterus, fallopian tubes and ovaries.
- retroperitoneal: contains the kidneys, ureters, pancreas and duodenum. May result in massive blood loss from retroperitoneal venous injury.

- Management:
  - basic resuscitation as for **trauma** generally.
  - initial assessment: examination of the anterior abdominal wall, both flanks, back, buttocks, perineum (and in men, the urethral meatus) for bruises, lacerations, entry and exit wounds. Signs may be masked by unconsciousness, **spinal cord injury** or the effects of **alcohol** or drugs. Abdominal swelling usually indicates intra-abdominal haemorrhage; abdominal guarding or rigidity usually indicates visceral injury. Absence of bowel sounds may indicate intraperitoneal haemorrhage or peritoneal soiling with bowel contents. Colonic or rectal injuries may cause blood pr. A high

Table 1 Antigens and antibodies in ABO blood groups

Group	Incidence in UK (%)	Red cell antigen	Plasma antibody
A	42	A	Anti-B
B	8	B	Anti-A
AB	3	A and B	None
O	47	None	Anti-A and anti-B

index of suspicion is required for retroperitoneal injuries because examination is difficult.
- imaging: abdominal x-ray may reveal free gas under the diaphragm (erect or semi-erect; may also be visible on CXR) or laterally (lateral decubitus x-ray); other investigations include pelvic x-ray and urological radiology if indicated (e.g. iv urogram), **CT** and **MRI scanning** and **ultrasound**.
- **peritoneal lavage** is indicated in blunt abdominal trauma associated with:
  - altered pain response (**TBI**, spinal cord injury, drugs, etc.).
  - unexplained **hypovolaemia** following multiple trauma.
  - equivocal diagnostic findings.
- insertion of a nasogastric tube and urinary catheter (provided no urethral injury; a suprapubic catheter may be necessary).
- indications for laparotomy include penetrating injuries, obvious intra-abdominal haemorrhage, signs of bowel perforation or a positive peritoneal lavage.

Al-Mudhaffar M, Hormbrey P (2014). Br Med J; 348: g1140
See also, Pelvic trauma

ABO blood groups. Discovered in 1900 by Landsteiner in Vienna. Antigens may be present on red blood cells, with antibodies in the plasma (Table 1). The antibodies, mostly type-M **immunoglobulins**, develop within the first few months of life, presumably in response to naturally occurring antigens of similar structure to the blood antigens. Infusion of blood containing an ABO antigen into a patient who already has the corresponding antibody may lead to an adverse reaction; hence the description of group O individuals as universal donors, and of group AB individuals as universal recipients.
[Karl Landsteiner (1868–1943), Austrian-born US pathologist]
See also, Blood compatibility testing; Blood groups; Blood transfusion

ABPI, *see Ankle–Brachial Pressure Index*

Abruption, *see Antepartum haemorrhage*

Absolute risk reduction. Indicator of treatment effect in clinical trials, representing the decrease in risk of a given treatment compared with a control treatment, i.e., the inverse of the **number needed to treat**. For a reduction in incidence of events from a% to b%, it equals $(a - b)$%.
See also, Meta-analysis; Odds ratio; Relative risk reduction

Abuse of anaesthetic agents. May occur because of easy access to potent drugs by operating theatre or ICU staff. Anaesthetists are 2.5 times as likely to abuse agents than other physicians. **Opioid analgesic drugs** (especially **fentanyl**) are the most commonly abused agents, but others include **benzodiazepines**, **propofol** and **inhalational anaesthetic agents**. Abuse may be suggested by behavioural or mood changes or excessive and inappropriate requests for opioids. Main considerations include the safety of patients, counselling and psychiatric therapy for the abuser and legal aspects of drug abuse. May be associated with **alcoholism**.
Bryson EO, Silverstein JH (2008). Anesthesiology; 109: 905–17
See also, Misuse of Drugs Act; Sick doctor scheme; Substance abuse

Acarbose. Inhibitor of intestinal alpha glucosidases and pancreatic amylase; used in the treatment of **diabetes mellitus**, usually in combination with a **biguanide** or **sulfonylurea**. Delays digestion and absorption of starch and sucrose and has a small blood glucose-lowering effect.
See also, Meglitinides; Thiazolidinediones

Accessory nerve block. Performed for spasm of trapezius and sternomastoid muscles (there is no sensory component to the nerve). 5–10 ml **local anaesthetic agent** is injected 2 cm below the mastoid process into the sternomastoid muscle, through which the nerve runs.

Accident, major, *see Incident, major*

ACD, Acid–citrate–dextrose solution, *see Blood storage*

ACD-CPR, Active compression decompression CPR, *see Cardiac massage; Cardiopulmonary resuscitation*

ACE, Angiotensin converting enzyme, *see Renin/angiotensin system*

ACE anaesthetic mixture. Mixture of alcohol, chloroform and diethyl ether, in a ratio of 1:2:3 parts, suggested in 1860 as an alternative to chloroform alone. Popular into the 1900s as a means of reducing total dose and side effects of any one of the three drugs.

ACE inhibitors, *see Angiotensin converting enzyme inhibitors*

Acetaminophen, *see Paracetamol*

Acetazolamide. **Carbonic anhydrase** inhibitor. Reduces renal **bicarbonate** formation and hydrogen ion excretion at the proximal convoluted tubule, thereby inducing a metabolic **acidosis**. A weak **diuretic**, but rarely used as such. Also used to treat **glaucoma**, metabolic alkalosis, altitude sickness and childhood epilepsy. Useful in the treatment of severe **hyperphosphataemia** as it promotes urinary excretion of phosphate. May be used to lower **ICP** (e.g. in benign intracranial hypertension) by reducing **CSF** production. Has been used to alkalinise the urine in **tumour lysis syndrome** and to enhance excretion in drug intoxications, e.g. with salicylates.
- Dosage: 0.25–0.5 g orally/iv od/bd.

Acetylcholine (ACh). **Neurotransmitter**, the acetyl ester of choline (Fig. 1). Synthesised from acetylcoenzyme A and choline in **nerve** ending cytoplasm; the reaction is catalysed by choline acetyltransferase. Choline is actively

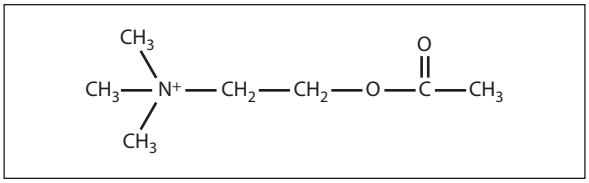

Fig. 1 Structure of acetylcholine

transported into the nerve and acetylcoenzyme A is formed in mitochondria. ACh is stored in vesicles.

- ACh is the transmitter at:
  - autonomic ganglia.
  - parasympathetic postganglionic nerve endings.
  - sympathetic postganglionic nerve endings at sweat glands and some muscle blood vessels.
  - the **neuromuscular junction**.
  - many parts of the CNS, where it has a prominent role in CNS plasticity (and therefore learning), attention and memory. Dysfunction of the CNS cholinergic system contributes to the memory disorder in Alzheimer's disease.

Actions may be broadly divided into either muscarinic or nicotinic, depending on the **acetylcholine receptors** involved. ACh is hydrolysed to choline and acetate by **acetylcholinesterase** on the postsynaptic membrane. Other esterases also exist, e.g. plasma **cholinesterase**.

[Alois Alzheimer (1864–1915), German neurologist and pathologist]

See also, Acetylcholine receptors; Neuromuscular transmission; Parasympathetic nervous system; Sympathetic nervous system; Synaptic transmission

Acetylcholine receptors. Transmembrane receptors activated by **acetylcholine** (ACh). Classified according to their relative sensitivity to **nicotine** or **muscarine** (Fig. 2a).

- Nicotinic receptors: ligand-gated ion channels present at numerous sites within the nervous system; notable examples include the **neuromuscular junction** (NMJ) and autonomic ganglia. Each receptor consists of five glycosylated protein subunits that project into the synaptic cleft. The adult receptor consists of 2 α, β, δ and ε units. The ε subunit is replaced by a γ subunit in the neonate. The subunits span the postsynaptic **membrane**, forming a cylinder around a central ion channel (Fig. 2b). The two α subunits of each receptor carry the binding sites for ACh. Occupation of these sites opens the ion channel, allowing cations (mainly sodium, potassium and calcium) to flow into the cell down their concentration gradients; this produces an excitatory postsynaptic potential. If these summate and exceed the threshold potential, an **action potential** is generated. Non-depolarising **neuromuscular blocking drugs** are reversible competitive antagonists of these receptors at the NMJ.
- Muscarinic receptors: **G protein-coupled receptors**, largely coupled to either **adenylate cyclase** or phospholipase C, via Gi and Gq proteins, respectively. Mediate postganglionic neurotransmission via parasympathetic neurones, as well as sympathetic outflow to sweat glands (Fig. 2a). Classified according to structural subtype, distribution and function:
  - M1: Gq-coupled; stomach (stimulates acid secretion) and brain (memory formation).
  - M2: Gi-coupled; heart; decreases heart rate, contractility and atrioventricular nodal conduction.

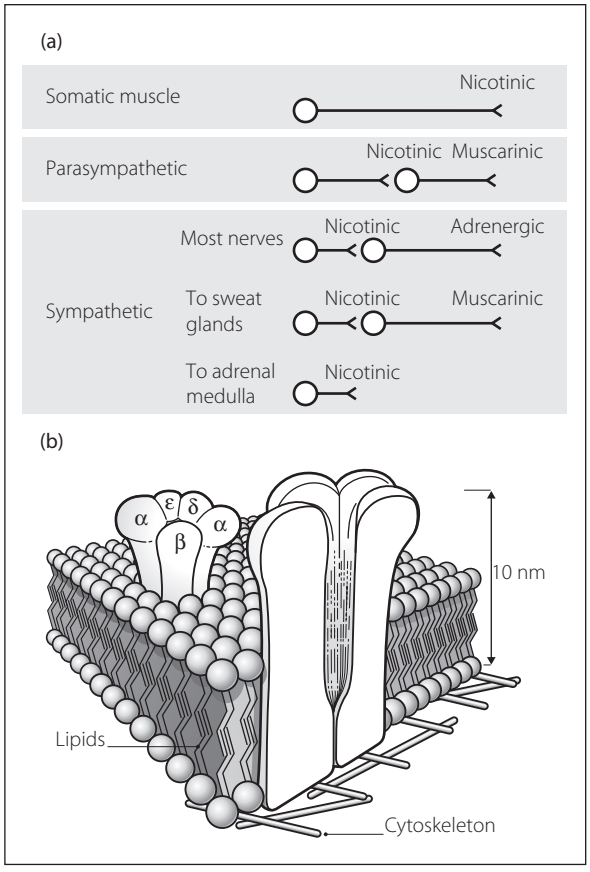

Fig. 2 (a) Types of acetylcholine receptors. (b) Structure of nicotinic acetylcholine receptor

  - M3: Gq-coupled; smooth muscle (increased tone, e.g. bronchiolar, intestinal), exocrine glands (stimulatory), brain (stimulatory at vomiting centre).
  - M4/5: brain and adrenal medulla.

Muscarinic receptor agonists include bethanechol, carbachol and pilocarpine (in the eye); antagonists include **hyoscine**, **atropine** and **ipratropium bromide**.

The activation threshold of muscarinic receptors is lower than that of nicotinic receptors. Injection of ACh or poisoning with **anticholinesterases** thus causes parasympathetic stimulation and sweating at lower doses, before having effects at autonomic ganglia and the NMJ at higher doses.

See also, Neuromuscular transmission; Parasympathetic nervous system; Sympathetic nervous system; Synaptic transmission

Acetylcholinesterase. **Enzyme** present at the synaptic membranes of cholinergic **synapses** and **neuromuscular junctions**. Also found in red blood cells and the placenta. Metabolises **acetylcholine** (ACh) to acetate and choline, thus terminating its action. Has a high catalytic activity, each molecule of acetylcholinesterase catalysing 25 000 molecules of ACh per second. The $N(CH_3)_3^+$ moeity of ACh binds to the anionic site of the enzyme, and the acetate end of ACh forms an intermediate bond at the esteratic site. Choline is liberated, and the intermediate substrate/enzyme complex is then hydrolysed to release acetate (Fig. 3).

See also, Acetylcholinesterase inhibitors; Neuromuscular transmission; Synaptic transmission

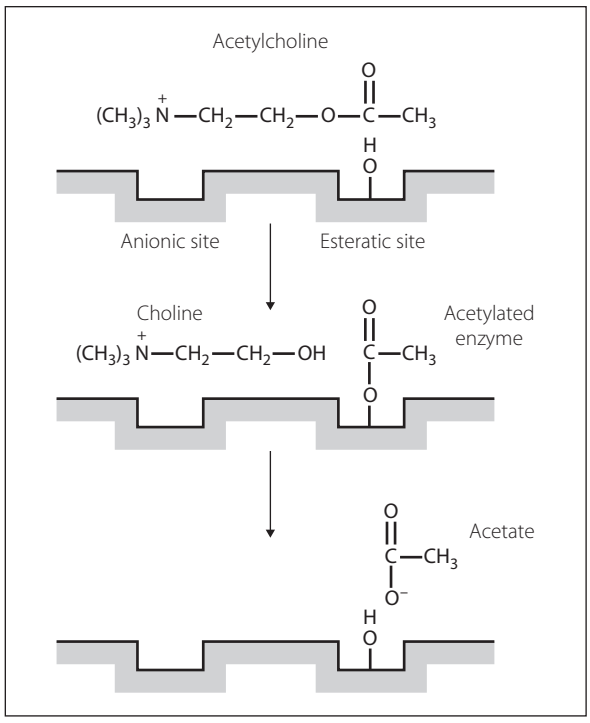

Fig. 3 Action of acetylcholinesterase

Acetylcholinesterase inhibitors. Substances that increase **acetylcholine** (ACh) concentrations by inhibiting **acetylcholinesterase** (AChE). Used clinically for their action at the **neuromuscular junction** in **myasthenia gravis** and in the reversal of **non-depolarising neuromuscular blockade**. Concurrent administration of an antimuscarinic agent, e.g. **atropine** or **glycopyrronium**, reduces unwanted effects of increased ACh concentrations at muscarinic receptors. Effects at ganglia are minimal at normal doses. Central effects may occur if the drug readily crosses the **blood–brain barrier**, e.g. **physostigmine** (used to treat the **central anticholinergic syndrome**).

Have also been used to treat tachyarrhythmias.

- Classified according to mechanism of action:
  - reversible competitive inhibitors: competitive inhibition at the anionic site of AChE prevents binding of ACh, e.g. **edrophonium**, **tetrahydroaminacrine**.
  - oxydiaphoretic (or 'acid-transferring') inhibitors: act as an alternative substrate for AChE, producing a more stable carbamylated enzyme complex. Subsequent hydrolysis of the complex and thus reactivation of the enzyme is slow. Examples:
    - **neostigmine**, physostigmine (few hours).
    - **pyridostigmine** (several hours).
    - **distigmine** (up to a day).
  - organophosphorus compounds: act by irreversibly phosphorylating the esteratic site of AChE; inhibition can last for weeks until new enzyme is synthesised. Examples include: **ecothiopate** (used for the treatment of glaucoma); parathion (an insecticide); sarin nerve gas (a chemical weapon).

Acetylcholinesterase inhibitors augment **depolarising neuromuscular blockade** and may cause depolarising blockade in overdose. They may also cause bradycardia, hypotension, agitation, miosis, increased GIT activity, sweating and salivation.

Centrally acting acetylcholinesterase inhibitors (e.g. donepezil, rivastigmine, galantamine) are used for symptomatic treatment of Alzheimer's dementia. Of anaesthetic relevance because of their side effects (including nausea, vomiting, fatigue, muscle cramps, increased creatine kinase, convulsions, bradycardia, confusion), enhancement of the actions of **suxamethonium**, and possible antagonism of non-depolarising **neuromuscular blocking drugs**.
[Alois Alzheimer (1864–1915), German neurologist and pathologist]
See also, Neuromuscular transmission; Organophosphorus poisoning

***N*-Acetylcysteine**. Derivative of the naturally occurring **amino acid**, L-cysteine. A **free radical** scavenger, licensed as an antidote to **paracetamol poisoning**. Acts by restoring depleted hepatic stores of glutathione and providing an alternative substrate for a toxic metabolite of paracetamol. Also used as an ocular lubricant and to prevent nephropathy due to **radiological contrast media** in patients with reduced renal function.

Has been investigated for the treatment of fulminant **hepatic failure**, **MODS**, **ALI** and neuropsychiatric complications of **carbon monoxide poisoning**, as well as a possible role in protection against myocardial **reperfusion injury**. Also used as a mucolytic because of its ability to split disulfide bonds in mucus glycoprotein.

- Dosage:
  - paracetamol poisoning: 150 mg/kg (to a maximum of 12 g) in 200 ml 5% dextrose iv over 1 h, followed by 50 mg/kg in 500 ml dextrose over 4 h, then 100 mg/kg in 1 l dextrose over 16 h (maximum of 110 kg body weight used for obese patients).
  - to reduce viscosity of airway secretions: 200 mg 8 hourly, orally. May be delivered by nebuliser.
- Side effects: rashes, anaphylaxis. Has been associated with bronchospasm in asthmatics.

Achalasia. Disorder of oesophageal motility caused by idiopathic degeneration of nerve cells in the myenteric plexus or vagal nuclei. Results in dysphagia and oesophageal dilatation. A similar condition may result from American trypanosomal infection (Chagas' disease). **Aspiration pneumonitis** or repeated chest infections may occur. Treated by mechanical distension of the lower **oesophagus** or by surgery. Heller's cardiomyotomy (longitudinal myotomy leaving the mucosa intact) may be undertaken via abdominal or thoracic approaches. Preoperative respiratory assessment is essential. Patients are at high risk of aspirating oesophageal contents, and rapid sequence induction is indicated.
[Carlos Chagas (1879–1934), Brazilian physician; Ernst Heller (1877–1964), German surgeon]
See also, Aspiration of gastric contents; Induction, rapid sequence

Achondroplasia. Skeletal disorder, inherited as an autosomal dominant gene, although most cases arise by spontaneous mutation. Results in dwarfism, with a normal size trunk and shortened limbs. Flat face, bulging skull vault and spinal deformity may make tracheal intubation difficult, and the larynx may be smaller than normal. **Obstructive sleep apnoea** may occur. Foramen magnum and spinal canal stenoses may be present, the former resulting in cord compression on neck extension, the latter making neuraxial blockade difficult and reducing volume requirements for **epidural anaesthesia**.

Aciclovir. **Antiviral drug**; an analogue of nucleoside 2′-deoxyguanosine. Inhibits viral DNA polymerase; active against herpes viruses and used in the treatment of **encephalitis**, varicella zoster (chickenpox/shingles) and **postherpetic neuralgia**, and for prophylaxis and treatment of herpes infections in immunocompromised patients. Treatment should start at onset of infection; the drug does not eradicate the virus but may markedly attenuate the clinical infection.

- Dosage:
  - as topical cream, 5 times daily.
  - 200–800 mg orally, 2–5 times daily in adults.
  - 5–10 mg/kg iv tds, infused over 1 h.
- Side effects: rashes, GIT disturbances, hepatic and renal impairment, blood dyscrasias, headache, dizziness, severe local inflammation after iv use, confusion, convulsions, coma.

Acid. Species that acts as a proton (H^+) donor when in solution (Brønsted–Lowry definition).
[Johannes N Brønsted (1879–1947), Danish chemist; Thomas M Lowry (1874–1936), English chemist]
See also, Acid–base balance; Acidosis

Acidaemia. Arterial **pH** <7.35 or **hydrogen ion** concentration >45 nmol/l.
See also, Acid–base balance; Acidosis

Acid–base balance. Maintenance of stable **pH** in body fluids is necessary for normal **enzyme** activity, ion distribution and protein structure. Blood pH is normally maintained at 7.35–7.45 (**hydrogen ion** [H^+] concentration 35–45 nmol/l); intracellular pH changes with extracellular pH. During normal metabolism of neutral substances, organic acids are produced that generate hydrogen ions.

- Maintenance of pH depends on:
  - **buffers** in tissues and blood, which minimise changes in H^+ concentration.
  - regulation by kidneys and lungs; the kidneys excrete about 60–80 mmol and the lungs about 15 000–20 000 mmol H^+ per day.

Because of the relationship between CO_2, carbonic acid, **bicarbonate** (HCO_3^-) and H^+, and the ability to excrete CO_2 rapidly from the lungs, respiratory function is important in acid–base balance:

$$H_2O + CO_2 \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$$

Thus hyper- and hypoventilation cause **alkalosis** and **acidosis**, respectively. Similarly, hyper- or hypoventilation may compensate for non-respiratory acidosis or alkalosis, respectively, by returning pH towards normal.

Sources of H^+ excreted via the kidneys include lactic acid from blood cells, muscle and brain, sulfuric acid from metabolism of sulfur-containing proteins, and acetoacetic acid from fatty acid metabolism.

- The kidney can compensate for acid–base disturbances in three ways:
  - by regulating the reabsorption of filtered HCO_3^- at the proximal convoluted tubule (normally 80%–90%):
    - filtered Na^+ is exchanged for H^+ across the tubule cell membrane.
    - filtered HCO_3^- and excreted H^+ form carbonic acid.
    - carbonic acid is converted to CO_2 and water by **carbonic anhydrase** on the cell membrane.
    - CO_2 and water diffuse into the cell and reform carbonic acid (catalysed again by carbonic anhydrase).
    - carbonic acid dissociates into HCO_3^- and H^+.
    - HCO_3^- passes into the blood; H^+ is exchanged for Na^+, etc.
  - by forming dihydrogen phosphate from monohydrogen phosphate in the distal tubule ($HPO_4^- + H^+ \rightarrow H_2PO_4^-$). The H^+ is supplied from carbonic acid, leaving HCO_3^-, which passes into the blood.
  - by combination of ammonia, passing out of the cells, with H^+, supplied as mentioned earlier. Resultant ammonium ions cannot pass back into the cells and are excreted.

In acid–base disorders, the primary change determines whether a disturbance is respiratory or metabolic. The direction of change in H^+ concentration determines acidosis or alkalosis. Renal and respiratory compensation act to restore normal pH, not reverse the primary change. For example, in the **Henderson–Hasselbalch equation**:

$$pH = pK_a + \log\frac{[HCO_3^-]}{[CO_2]}$$

adjustment of the HCO_3^-/CO_2 concentration ratio restores pH towards its normal value, e.g.:

- primary change: increased CO_2; leads to decreased pH (respiratory acidosis).
- compensation: HCO_3^- retention by kidneys; increased ammonium secretion, etc.

An alternative approach, suggested by Stewart in 1983, focuses on the **strong ion difference** to explain the underlying processes rather than the above 'traditional approach', which concentrates more on interpretation of measurements. It is based on the degree of dissociation of ions in solution, in particular the effects of strong ions and weak acids, and the role of bicarbonate as a marker of acid–base imbalance rather than a cause.
[Peter Stewart (1921–1993), Canadian physiologist]
See also, Acid; Base; Blood gas interpretation; Breathing, control of; Davenport diagram; Siggaard-Andersen nomogram

Acid–citrate–dextrose solution, *see Blood storage*

Acidosis. A process in which arterial **pH** <7.35 (or **hydrogen ion** >45 mmol/l), or would be <7.35 if there were no compensatory mechanisms of **acid–base balance**.
See also, Acidosis, metabolic; Acidosis, respiratory

Acidosis, metabolic. **Acidosis** due to metabolic causes, resulting in an inappropriately low **pH** for the measured arterial P_{CO_2}.

- Caused by:
  - increased acid production:
    - **ketone bodies**, e.g. in **diabetes mellitus**.
    - **lactate**, e.g. in **shock**, **exercise**.
  - acid ingestion: e.g. **salicylate poisoning**.
  - failure to excrete **hydrogen ions** (H^+):
    - **renal failure**.
    - distal **renal tubular acidosis**.
    - **carbonic anhydrase** inhibitors.
  - excessive loss of **bicarbonate**:
    - diarrhoea.
    - gastrointestinal fistulae.
    - proximal renal tubular acidosis.
    - ureteroenterostomy.

- May be differentiated by the presence or absence of an **anion gap**:
  - anion gap metabolic acidosis occurs in renal failure, lactic acidosis, ketoacidosis, rhabdomyolysis and following ingestion of certain toxins (e.g. salicylates, methanol, ethylene glycol).
  - non-anion gap (hyperchloraemic) metabolic acidosis is caused by the administration of chloride-containing solutions (e.g. saline) in large volumes, amino acid solutions, diarrhoea, pancreatic fistulae, ileal loop procedures, after rapid correction of a chronically compensated respiratory alkalosis or renal tubular acidosis.
- Primary change: increased H^+/decreased bicarbonate.
- Compensation:
  - **hyperventilation**: plasma bicarbonate falls by about 1.3 mmol/l for every 1 kPa acute decrease in arterial P_{CO_2}, which usually does not fall below 1.3–1.9 kPa (10–15 mmHg).
  - increased renal H^+ secretion.
- Effects:
  - hyperventilation (**Kussmaul breathing**).
  - confusion, weakness, coma.
  - cardiac depression.
  - **hyperkalaemia**.
- Treatment:
  - of underlying cause.
  - bicarbonate therapy is reserved for treatment of severe acidaemia (e.g. pH under 7.1) because of problems associated with its use. If bicarbonate is required, a formula for iv infusion is:

$$\frac{\text{base excess} \times \text{body weight (kg)}}{3} \text{ mmol}$$

    Half this amount is given initially.
  - other rarely used agents include **sodium dichloroacetate**, **Carbicarb** (sodium bicarbonate and carbonate in equimolar concentrations) and THAM (**2-amino-2-hydroxymethyl-1,3-propanediol**).

Morris CG, Low J (2008). Anaesthesia; 63: 294–301 and 396–411

See also, Acidaemia; Acid–base balance

Acidosis, respiratory. Acidosis due to increased arterial P_{CO_2}. Caused by alveolar **hypoventilation**.

- Primary change: increased arterial P_{CO_2}.
- Compensation:
  - initial rise in plasma bicarbonate due to increased carbonic acid formation and dissociation.
  - increased acid secretion/bicarbonate retention by the kidneys. In acute hypercapnia, bicarbonate concentration increases by about 0.7 mmol/l per 1 kPa rise in arterial P_{CO_2}. In chronic hypercapnia it increases by 2.6 mmol/l per 1 kPa.
- Effects: those of hypercapnia.
- Treatment: of underlying cause.

See also, Acidaemia; Acid–base balance

ACLS, *see Advanced Cardiac Life Support*

Acquired immune deficiency syndrome (AIDS), *see Human immunodeficiency viral infection*

Acromegaly. Disease caused by excessive **growth hormone** secretion after puberty; usually caused by a **pituitary** adenoma but ectopic secretion may also occur. Incidence is 6–8 per million population.

- Features:
  - enlarged jaw, tongue and larynx; widespread increase in soft tissue mass; enlarged feet and hands. Nerve entrapment may occur, e.g. carpal tunnel syndrome.
  - respiratory obstruction, including **obstructive sleep apnoea**.
  - tendency towards **diabetes mellitus**, **hypertension** and **cardiac failure** (may be due to **cardiomyopathy**). Thyroid and adrenal impairment may occur. Colorectal malignancy is more common.

Apart from the above diseases, acromegaly may present difficulties with tracheal intubation and maintenance of the airway.

Treatment is primarily pituitary surgery with or without subsequent radiotherapy. Some patients respond to bromocriptine or **somatostatin** analogues.

Nemergut EC, Dumont AS, Barry UT, Laws ER (2007). Anesth Analg; 101: 1170–81

ACS, *see Acute coronary syndromes*

ACT, Activated clotting time, *see Coagulation studies*

Acta Anaesthesiologica Scandinavica. Official journal of the Scandinavian Society of Anaesthesiology and Intensive Care Medicine, first published in 1957.

ACTH, *see Adrenocorticotrophic hormone*

Actin. One of the protein components of **muscle** (mw 43 000). In muscle, arranged into a double strand of thin filaments (F-actin) with globular 'beads' (G-actin), to which myosin binds, along their length. Present in all cells as microfilaments.

See also, Muscle contraction

Action potential. Sequential changes in **membrane potential** that result in the propagation of electrical impulses in excitable cells. Neuronal, myocardial and cardiac nodal action potentials have distinct characteristics, determined by their underlying ionic fluxes (Fig. 4).

- Neuronal action potential (Fig. 4a):
  - A: depolarisation of the membrane by 15 mV (threshold level).
  - B: rapid depolarisation to +40 mV.
  - C: repolarisation, rapid at first then slow.
  - D: hyperpolarisation.
  - E: return to the resting membrane potential.

Slow initial depolarisation causes opening of voltage-gated sodium channels (VGSCs) and influx of Na^+ into the cell, which causes further rapid depolarisation. Na^+ conductance then falls as the VGSCs enter an inactivated state. K^+ efflux via voltage-gated potassium channels occurs more slowly and helps bring about repolarisation. Normal ion distribution (and hence the resting membrane potential) is restored by the action of the **sodium/potassium pump**. The action potential is followed by a **refractory period**.

- Myocardial action potential (Fig. 4b):
  - phase 0: fast depolarisation and Na^+ influx via VGSCs.
  - phase 1: onset of repolarisation due to sodium channel closure.
  - phase 2: plateau due to Ca^{2+} influx via voltage-gated calcium channels (VGCCs).
  - phase 3: repolarisation and K^+ efflux.
  - phase 4: resting membrane potential.

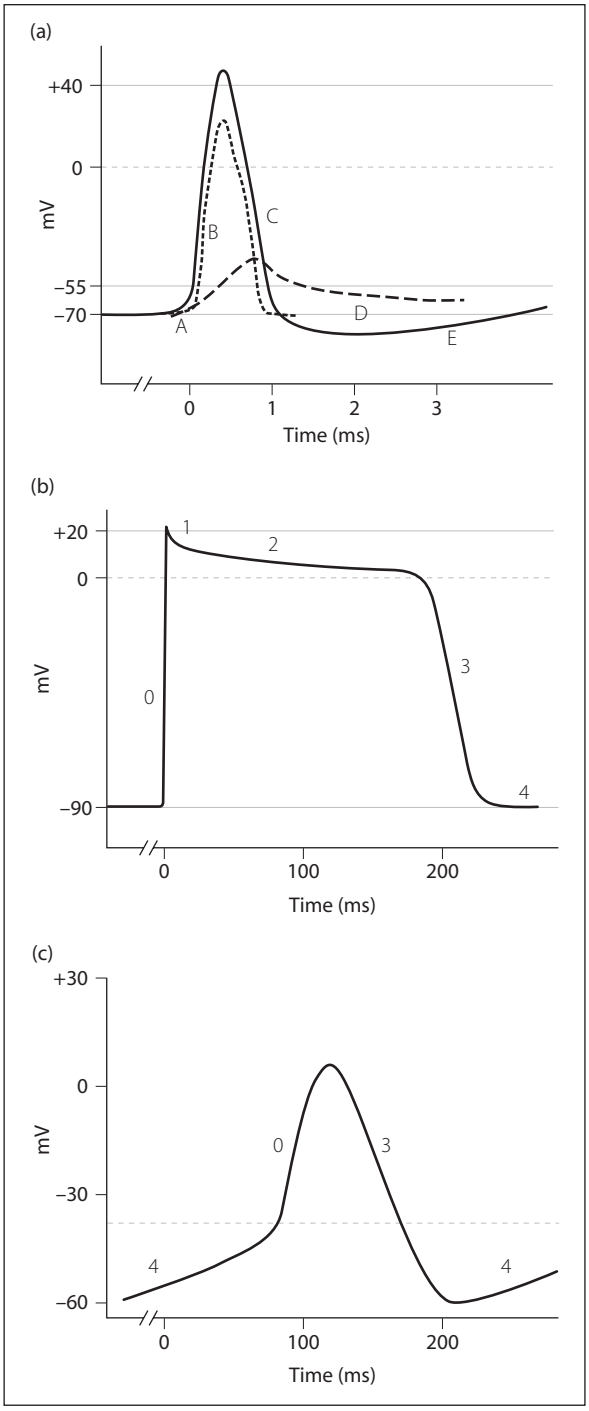

Fig. 4 (a) Nerve action potential *(solid)* showing changes in sodium *(dotted)* and potassium *(dashed)* conductance. (b) Cardiac action potential (see *text*). (c) Sinoatrial nodal action potential

The long plateau of phase 2 prolongs the refractory period, preventing tetanisation.

- Cardiac nodal action potential (Fig. 4c):
  - phase 4: slow spontaneous depolarisation (pacemaker potential) caused by a fall in K^+ efflux and slow Ca^{2+} influx via T-type VGCCs.
  - phase 0: depolarisation caused by opening of L-type VGCCs and Ca^{2+} influx.
  - phase 3: repolarisation caused by K^+ efflux.

Notably, there is no contribution by Na^+ flux to the action potential in **pacemaker cells**.

Cardiac excitability is modulated by autonomic inputs and antiarrythmic drugs via their effects on Na^+, K^+ and Ca^{2+} conductance.

See also, Nernst equation; Nerve conduction

Activated charcoal, *see Charcoal, activated*

Activated clotting time, *see Coagulation studies*

Activated protein C, *see Protein C*

Activation energy. Energy required to initiate a chemical reaction. For ignition of explosive mixtures of anaesthetic agents the energy may be provided by sparks, e.g. from electrical equipment or build-up of static electricity. Combustion of **cyclopropane** requires less activation energy than that of diethyl **ether**. Activation energy is less for mixtures with O_2 than with air, and least for **stoichiometric mixtures** of reactants.

See also, Explosions and fires

Active compression/decompression cardiopulmonary resuscitation, *see Cardiac massage; Cardiopulmonary resuscitation*

Active transport. Energy-requiring transport of particles across cell **membranes**. Protein 'pumps' within the membranes utilise energy (which is usually supplied by **ATP** metabolism) to move ions and molecules, often against concentration gradients. A typical example is the **sodium/potassium pump**.

Acupuncture. Use of fine **needles** (usually 30–33 G) to produce healing and pain relief. Originated in China thousands of years ago, and closely linked with the philosophy and practice of traditional Chinese medicine. Thus abnormalities in the flow of Qi (Chi: the life energy that circulates around the body along meridians, nourishing the internal organs) result in imbalance between Yin and Yang, the two polar opposites present in all aspects of the universe. Internal abnormalities may be diagnosed by pulse diagnosis (palpation of the radial arteries at different positions and depths). The appropriate organ is then treated by acupuncture at specific points on the skin, often along the meridian named after, and related to, that organ. Yin and Yang, and flow of Qi, are thus restored.

Modern Western acupuncture involves needle insertion at sites chosen for more 'scientific' reasons; e.g. around an affected area, at **trigger points** found nearby, or more proximally but within the appropriate **dermatome**. These may be combined with distant or local traditional points, although conclusive evidence for the existence of acupuncture points and meridians has never been shown. The needles may be left inserted and stimulated manually, electrically or thermally to increase intensity of stimulation. Pressure at acupuncture points (acupressure) may produce similar but less intense stimulation.

- Possible mechanisms:
  - local reflex pathways at spinal level.
  - closure of the 'gate' in the **gate control theory of pain**.
  - central release of **endorphins**/**enkephalins**, and possibly involvement of other **neurotransmitters**.
  - modulation of the 'memory' of pain.

Still used widely in China. Increasingly used in the West for chronic pain, musculoskeletal disorders, headache and migraine, and other disorders in which modern Western medicine has had little success. Claims that acupuncture may be employed alone to provide analgesia for surgery are now viewed with scepticism, although it has been used to provide analgesia and reduce **PONV**, e.g. 5 min stimulation at the point P6 (pericardium 6: 1–2 inches [2.5–5 cm] proximal to the distal wrist crease, between flexor carpi radialis and palmaris longus tendons).
Ernst E, Lee MS, Choi TY (2011). Pain; 152: 755–64

Acute coronary syndromes (ACS). Group of clinical conditions characterised by acute **myocardial ischaemia** and including unstable angina and **MI**; usually caused by acute thrombus formation within a coronary artery upon the exposed surface of a ruptured or eroded atheromatous plaque. May also occur due to coronary artery spasm, arteritis or sudden severe hypo- or hypertension.

- Classification:
  - **S–T segment** elevation MI (STEMI): ACS with S–T segment elevation on 12-lead **ECG**. Accounts for 40% of MI. Suggestive of total coronary artery occlusion. Consistently associated with elevated plasma biomarkers of myocardial damage. Immediate reperfusion therapy (*see later*) significantly improves outcomes. ACS with new-onset left **bundle branch block** (LBBB) or evidence of posterior infarction is included in this category for treatment purposes.
  - non-S–T segment elevation acute coronary syndromes (NSTEACS): suggestive of subtotal arterial occlusion. Immediate reperfusion therapy is not indicated, although early (within 24 h) **percutaneous coronary intervention** (PCI) may be considered in high-risk patients. Further subdivided into:
    - non-S–T segment elevation MI (NSTEMI); normal or non-specific changes on ECG, with elevated cardiac biomarkers.
    - unstable angina: ACS without elevated cardiac biomarkers.
- Clinical features:
  - pain as for **myocardial ischaemia**.
  - **arrhythmias**; **cardiac arrest** may occur.
  - anxiety, sweating, pallor, dyspnoea.
  - **hypertension** or **hypotension**.
  - **cardiac failure** and **cardiogenic shock**.

Severe infarction is usually associated with more severe symptoms and signs than unstable angina, although painless/silent infarction is also common.

- Differential diagnosis: pain and ECG changes may occur with lesions of:
  - heart/great vessels, e.g. **aortic dissection**, **pericarditis**.
  - lung, e.g. **PE**, **chest infection**.
  - oesophagus, e.g. spasm, inflammation, rupture.
  - abdominal organs, e.g. **peptic ulcer disease**, **pancreatitis**, **cholecystitis**.
- Investigations:
  - 12-lead ECG (*see Myocardial infarction for characteristic features of STEMI and their localisation by ECG pattern*). Bundle branch block may be evident. NSTEACS may coexist with a normal ECG or: S–T segment depression; S–T segment elevation insufficient to meet reperfusion therapy criteria (*see later*); T wave flattening or inversion; or biphasic T waves.
  - cardiac biomarkers:
    - **cardiac enzymes**: largely replaced by troponins.
    - cardiac troponins:
      - regulatory proteins involved in cardiac and skeletal **muscle contraction**.
      - composed of three subunits: C, T and I; plasma levels of the latter two are both specific and sensitive markers for myocardial damage.
      - levels may not rise until 6–8 h after onset of symptoms although more recent high-sensitivity assays of cardiac troponins may allow accurate diagnosis at 3 h.
      - levels peak at around 24 h, correlating with the extent of infarction, and may remain elevated for 7–10 days.
      - may be elevated in myocardial damage due to other causes, e.g. **myocarditis**, contusion and also other non-cardiac critical illness (e.g. **sepsis**, **renal failure**, PE), probably reflecting myocardial injury but in most cases not related to coronary artery disease. Likely benefit of NSTEMI treatment in these cases should be assessed on an individual patient basis.
  - **echocardiography**: may be used to assess regional and global ventricular function; regional wall motion abnormalities and loss of thickness suggest acute infarction. Also useful in diagnosing complications of MI (e.g. ventricular aneurysm, mitral regurgitation, mural thrombus).
- Immediate management of suspected ACS:
  - O_2 via facemask (only if evidence of **hypoxia** [S_pO_2 >94%], **pulmonary oedema** or ongoing ischaemia), cardiac monitoring, 12-lead ECG, iv access.
  - **aspirin** 300 mg orally.
  - analgesia (e.g. iv **morphine** in 2 mg increments).
  - sublingual **GTN**.
  - associated pulmonary oedema and arrhythmias should be treated in the usual way.
  - consideration for immediate reperfusion therapy if:
    - presentation <12 h after symptom onset, unrelieved by GTN.
    - S–T segment elevation >0.1 mV in two or more contiguous chest leads or two adjacent limb leads.
    - new-onset LBBB.
    - posterior infarction (dominant R wave and S–T depression in V_1–V_2 chest leads).
- Reperfusion strategies include:
  - pharmacological thrombolytic therapy: agents include **streptokinase**, **alteplase**, **tenecteplase** and **reteplase**. Survival benefit is reduced with increasing delay, and is negligible from 12 h after onset of symptoms. Administration of thrombolysis within 1 h of the patient calling for professional help is a national **audit** standard. Contraindications include active bleeding, recent trauma (including surgery and **CPR**), previous haemorrhagic **stroke**, uncontrolled hypertension and pregnancy.
  - primary PCI.
  - emergency **coronary artery bypass surgery**.

Thrombolysis is generally only preferred if primary PCI is unavailable or there would be delay of >90 min in delivering it and the presentation is within 3 h of symptom onset. Primary PCI is particularly superior if: there is **cardiogenic shock**; there are contraindications to thrombolysis; or the patient is at high risk of death (e.g. age >75, previous MI, extensive anterior infarct). Emergency surgery

is generally reserved for those known to have disease uncorrectable by PCI or in whom primary PCI fails.

Patients not meeting criteria for immediate reperfusion (i.e., those with NSTEACS) are managed either invasively (PCI within 24 h plus **abciximab** iv) or conservatively (pharmacological management only). High-risk patients are most likely to benefit from invasive therapy.

- Pharmacological adjuncts include:
  - **clopidogrel** and low-molecular weight **heparin** (e.g. **enoxaparin**): should be given to all patients (in the absence of contraindications) with definite or strongly suspected ACS, in addition to aspirin. **Prasugrel** and **ticagrelor** are newer alternatives to clopidogrel, but the former is associated with increased risk of life-threatening bleeding.
  - GTN sublingually or by iv infusion if pain persists.
  - glycoprotein IIB/IIIa inhibitors (e.g. abciximab and **tirofiban**): beneficial in patients undergoing PCI, those at high risk of death, or both.
  - **β-adrenergic receptor antagonists**: reduce the rate of reinfarction and **VF** and should be commenced within 24 h if there are no contraindications, e.g. **heart block**, pulmonary oedema, hypotension. Those unable to receive β-blockers should receive one of the non-dihydropyridine **calcium channel blocking drugs** (e.g. **verapamil**).
  - **ACE inhibitors**: improve long-term survival after MI and should be commenced within 24 h, assuming no contraindications.
  - **magnesium** and **potassium** supplementation to maintain normal levels reduces the incidence of **arrhythmias**. Prophylactic administration of **anti-arrhythmic drugs** is no longer recommended.
  - implantable cardioverter defibrillators should be considered in patients following MI who have an ejection fraction <35%, and those with persisting ventricular arrhythmias.

Timms A (2015). Br Med J; 351: h5153

See also, Defibrillators, implantable cardioverter

Acute cortical necrosis, *see Renal failure*

Acute crisis resource management, *see Crisis resource management*

Acute demyelinating encephalomyelopathy, *see Demyelinating diseases*

Acute kidney injury (AKI). Previously referred to as acute **renal failure**, describing a rapid deterioration (within 48 h) from baseline renal function; classified according to severity by the **RIFLE criteria**. An independent risk factor for in-hospital morbidity and mortality and a major cause of death in ICU, especially as part of **MODS**. May develop with or without pre-existing renal impairment. May follow any severe acute illness, dehydration, **trauma** or major surgery (especially involving the heart and great vessels), **hepatic failure**, obstetric emergencies, and any condition involving sustained hypotension. It is estimated that 20% of AKI is avoidable with good management.

- May be classified as:
  - prerenal: caused by renal hypoperfusion, e.g. **shock**, **hypovolaemia**, **cardiac failure**, renal artery stenosis.
  - renal: caused by renal disease:
    - glomerular, e.g.:
      - **glomerulonephritis**.
      - **diabetes mellitus**.
      - amyloid.
    - tubulointerstitial, e.g.:
      - acute tubular necrosis (ATN): accounts for 75% of hospital AKI. Caused by renal hypoperfusion or ischaemia and/or chemical toxicity, trauma or sepsis. Nephrotoxins include analgesics (e.g. chronic **aspirin** and **paracetamol** therapy), **NSAIDs**, **aminoglycosides**, **immunosuppressive drugs**, metformin, **radiological contrast media** and heavy metals. Usually (but not always) associated with **oliguria** (caused by tubular cell necrosis, tubular obstruction and cortical arteriolar vasoconstriction).
      - acute cortical necrosis: typically associated with **placental abruption**, **pre-eclampsia** and septic abortion, but also with factors causing ATN. Confirmed by renal biopsy. Usually irreversible.
      - tubulointerstitial nephritis/pyelonephritis.
      - polycystic renal disease.
      - tubular obstruction, e.g. in myeloma, **myoglobinuria**.
    - vascular, e.g. hypertension, connective tissue disease.
  - postrenal: caused by obstruction in the urinary tract, e.g. bladder tumour, prostatic hypertrophy.

Distinction between renal and pre- or postrenal failure is important because diagnosis guides treatment, and early intervention can prevent irreversible injury.

- Features:
  - oliguria/anuria.
  - **uraemia** and accumulation of other substances (e.g. drugs): nausea, vomiting, malaise, increased bleeding and susceptibility to infection, decreased healing.
  - reduced sodium and water excretion and **oedema**, **hypertension**, **hyperkalaemia**, **acidosis**.
- The following may aid diagnosis:
  - analysis of **urine**: e.g. tubular casts may be seen in ATN, myoglobinuria may be present.
  - plasma and urine indices (Table 2).
  - flushing of the urinary catheter using aseptic technique.
  - assessment of cardiac and volume status to exclude hypovolaemia.
  - a fluid challenge of, e.g. 200–300 ml increased urine output, may occur in incipient prerenal failure.

Table 2 Investigations used to differentiate between prerenal oliguria and acute kidney injury

Investigation	*Prerenal oliguria*	*Renal failure*
Specific gravity	>1.020	<1.010
Urine osmolality (mosmol/kg)	>500	<350
Urine sodium (mmol/l)	<20	>40
Urine/plasma osmolality ratio	>2	<1.1
Urine/plasma urea ratio	>20	<10
Urine/plasma creatinine ratio	>40	<20
Fractional sodium excretion (%)	<1	>1
Renal failure index	<1	>1

$$\text{Fractional sodium excretion} = \frac{\text{urine/plasma sodium ratio}}{\text{urea/plasma creatinine ratio}} \times 100\%$$

$$\text{and renal failure index} = \frac{\text{urine sodium}}{\text{urine/plasma creatinine ratio}}$$

- diuretic administration, e.g. **furosemide** or **mannitol**: increased urine output may occur in incipient ATN but there is no evidence of a prophylactic or therapeutic effect; however, reduction in renal O_2 demand (furosemide) and scavenging of **free radicals** (mannitol) have been suggested as being theoretically beneficial.
- renal **ultrasound** or biopsy.

- Management:
  - directed at the primary cause with optimisation of **renal blood flow**.
  - monitoring of weight, cardiovascular status, including JVP/CVP/pulmonary capillary wedge pressure as appropriate, urea and electrolytes, and acid–base status. Accurate recording of fluid balance is vital.
  - fluid restriction if appropriate, e.g. previous hour's urine output + 30 ml/h while oliguric.
  - **H_2 receptor antagonists** are commonly administered to reduce GIT haemorrhage.
  - treatment of hyperkalaemia.
  - monitoring of drug levels, as **clearance** may be reduced considerably.
  - various **dialysis** therapies.
  - adequate **nutrition**.

Goren O, Matot I (2015). Br J Anaesth; 115 (Suppl 2): ii3–14

Acute life-threatening events—recognition and treatment (ALERT). Multiprofessional course aimed at reducing the incidence of potentially avoidable **cardiac arrests** and admissions to **ICU**. Targeted especially at junior doctors and ward nurses. Sharing principles of many life-support training programmes (e.g. **ALS**, **ATLS**, **APLS**, **CCrISP**), its development embraces both **clinical governance** and multiprofessional education. Uses a structured and prioritised system of patient assessment and management to recognise and treat seriously ill patients or those at risk of deterioration.

Smith GB, Osgood VM, Crane S (2002). Resuscitation; 52: 281–6

See also, Early warning scores; Medical emergency team; Outreach team

Acute lung injury (ALI). Syndrome of pulmonary inflammation and increased pulmonary capillary permeability associated with a variety of clinical, radiological and physiological abnormalities that cannot be explained by, but may coexist with, left atrial or pulmonary capillary hypertension. Associated with **sepsis**, major **trauma**, **aspiration pneumonitis**, **blood transfusion**, **pancreatitis**, **cardiopulmonary bypass** and **fat embolism**. Onset is usually within 2–3 days of the precipitating illness or injury, although direct lung insults usually have a shorter latency. Acute respiratory distress syndrome (ARDS) is now regarded to be the most severe form of ALI, with a mortality of 30%–45%. Mortality is dependent on age (higher in the elderly), racial origin (higher in non-Caucasians) and aetiology (highest in sepsis-related ALI). The definitions of ALI and ARDS are increasingly being challenged and diagnostic criteria vary, but the 2012 Berlin definition has largely superseded that of the 1994 American-European Consensus Conference (AECC):

- Major criterion: acute-onset arterial **hypoxaemia** resistant to **oxygen therapy** alone (P_aO_2/F_IO_2 <39.9 kPa [300 mmHg] for definition of ALI; <26.6 kPa [200 mmHg] for definition of moderate ARDS; <13.3 kPa [100 mmHg] for definition of severe ARDS), regardless of the level of ventilatory support.
- Other features:
  - bilateral diffuse infiltrates seen on the CXR although it is often difficult to differentiate between ARDS and cardiogenic **pulmonary oedema**.
  - reduced respiratory **compliance** (≤40 ml/cm), lung volumes and increased **work of breathing**.
  - $\dot{V}/\dot{Q}$ **mismatch** with increased **shunt**.
  - increased **pulmonary vascular resistance** occurs in 25% of patients with severe ALI and is a risk factor for increased mortality.
  - **MODS** may occur and is a common cause of death.

Measurement of **pulmonary wedge pressure** has been removed from the criteria.

- Pathophysiology: ALI results from damage to either the lung epithelium or endothelium. Two pathways of injury exist:
  - direct insult to lung, e.g. aspiration, **smoke inhalation**.
  - indirect result of an acute systemic inflammatory response involving both humoral (activation of **complement**, **coagulation** and **kinin** systems; release of mediators including **cytokines**, oxidants, **nitric oxide**) and cellular (neutrophils, macrophages and lymphocytes) components. Pulmonary infiltration by neutrophils leads to interstitial fibrosis, possibly because of damage caused by **free radicals**. Examples include sepsis, pancreatitis and fat embolism.

Histopathological findings can be divided into three phases: exudative (oedema and haemorrhage); proliferative (organisation and repair); and fibrotic.

- Management involves prompt treatment of the underlying cause and supportive therapy:
  - general support: **nutrition**, **DVT** prophylaxis, prevention of nosocomial infection.
  - O_2 therapy, accepting S_pO_2 >90%. **CPAP** is often helpful as it improves **FRC**.
  - ventilatory support: **IPPV** may be necessary if CPAP is ineffective. **Lung protection strategies** improve survival in ARDS and consist of using low tidal volumes/inspiratory pressures (e.g. 4–6 ml/kg and $P_{Plateau}$ <30 cmH_2O) with moderate PEEP, tolerating a degree of respiratory acidosis (**permissive hypercapnia**). High-pressure recruitment manoeuvres and high PEEP are often beneficial in life-threatening hypoxaemia, but increase the risk of **barotrauma** and impaired cardiac output.

    Additional ventilatory strategies include **inverse ratio ventilation** (at **I:E ratios** of up to 4:1), **airway pressure release ventilation** and **high-frequency ventilation**. **Extracorporeal membrane oxygenation** has been used with varying success. **Extracorporeal CO_2 removal** may be useful in life-threatening hypercapnia.
  - **prone ventilation** improves oxygenation and outcome in patients with severe hypoxaemia due to ARDS, especially in obese patients.
  - short-term use of **neuromuscular blocking drugs** may improve outcome.
  - diuresis and fluid restriction are often instituted to reduce lung water, although avoidance of initial fluid overload is thought to be more important.
  - **corticosteroid** therapy is controversial. There appears to be no benefit to its prophylactic administration, or in high-dose, short-term therapy at the onset of ALI/ARDS. However, corticosteroids may have a role in refractory ARDS.

- anti-inflammatory agents including nitric oxide, **prostaglandins**, **prostacyclin**, **surfactant** and **N-acetylcysteine** have not been shown to improve outcome.

MacSweeney R, McAuley DF (2016). Lancet; 388: 2416–30

Acute-phase response, A reaction of the haemopoietic and hepatic systems to inflammation or tissue injury, assumed to be of benefit to the host. There is a rise in the number/activity of certain cells (neutrophils, platelets) and plasma proteins (e.g. fibrinogen, **complement**, **C-reactive protein**, plasminogen, **haptoglobin**) involved in host defence, while there is a reduction in proteins with transport and binding functions (e.g. albumin, haemoglobin, transferrin). Initiated by actions of **cytokine** mediators such as interleukins (IL-1α, IL-1β, IL-6 and IL-11), tumour necrosis factors (α and β) and leukaemia inhibitory factor.

Serum levels of acute-phase proteins (e.g. C-reactive protein) can be helpful in diagnosis, monitoring and prognosis of certain diseases. The rise in fibrinogen levels causes an elevation in **ESR**. The fall in albumin is due to redistribution and decreased hepatic synthesis.

Acute physiology, age, chronic health evaluation, *see APACHE III scoring system*

Acute physiology and chronic health evaluation, *see APACHE scoring system; APACHE II scoring system*

Acute physiology score (APS). Physiological component of severity of illness scoring systems, such as **APACHE** II/III and Simplified APS. Weighted values (e.g. 0–4 in APACHE II) are assigned to each of a range of physiological variables (e.g. temperature, mean arterial blood pressure, serum creatinine) based on its derangement from an established 'normal' range, as measured either upon ICU admission or within 24 h of entry. The sum of all assigned weighted values for the physiological variables that comprise a given scoring system constitutes the acute physiological score. The higher the acute physiology score, the sicker the patient.

See also, Mortality/survival prediction on intensive care unit; Simplified acute physiology score

Acute respiratory distress syndrome (ARDS), *see Acute lung injury*

Acute tubular necrosis, *see Renal failure*

Acyclovir, *see Aciclovir*

Addiction, *see Alcoholism; Substance abuse*

Addison's disease, *see Adrenocortical insufficiency*

Adductor canal block. Distal approach to **femoral nerve block**, below the level at which the quadriceps motor innervation has left the **femoral nerve**. Thus provides analgesia to the anteromedial knee and lower leg while quadriceps strength is relatively preserved, aiding early postoperative mobilisation.

The adductor canal extends from the apex of the **femoral triangle** to the adductor hiatus. It contains the femoral vessels, saphenous nerve, obturator nerve (posterior division) and nerve to vastus medialis. With the patient supine and the leg to be blocked slightly flexed at the knee and externally rotated, 10–15 ml **local anaesthetic agent** is placed medial and 10–15 ml lateral to the **femoral artery** via a needle inserted midway down the medial aspect of the thigh, under **ultrasound** guidance.

Mariano ER, Perlas A (2014). Anesthesiology; 120: 530–2

ADEM, Acute demyelinating encephalomyelopathy, *see Demyelinating diseases*

Adenosine. Nucleoside, of importance in energy homeostasis at the cellular level. Reduces O_2 consumption, increases coronary blood flow, causes vasodilatation and slows atrioventricular conduction (possibly via increased potassium and reduced calcium **conductance**). Also an inhibitory CNS **neurotransmitter**.

The drug of choice for treatment of **SVT** (including that associated with **Wolff–Parkinson–White syndrome**), and diagnosis of other tachyarrhythmias by slowing atrioventricular conduction. Its short half-life (8–10 s) and lack of negative inotropism make it an attractive alternative to verapamil. Not included in the Vaughan Williams classification of **antiarrhythmic drugs**.

Has also been used as a directly acting **vasodilator drug** in **hypotensive anaesthesia**. Increases cardiac output, with stable heart rate. Its effects are rapidly reversible on stopping the infusion.

- Dosage:
  - SVT: 6 mg by rapid iv injection into a central or large peripheral vein; if unsuccessful after 1–2 min, may be followed by up to two further boluses of 12 mg.
  - hypotensive anaesthesia: 50–300 μg/kg/min. **ATP** has also been used.
- Side effects are usually mild and include flushing, dyspnoea and nausea. Bronchoconstriction may occur in asthmatics. Bradycardia is resistant to atropine. Adenosine's action is prolonged in **dipyridamole** therapy (because uptake of adenosine is inhibited) and reduced by **theophylline** and other **xanthines** (because of competitive antagonism). Transplanted hearts are particularly sensitive to adenosine's effects.

[EM Vaughan Williams (1918–2016), English pharmacologist]

Adenosine monophosphate, cyclic (cAMP). Cyclic adenosine 3′,5′-monophosphate, formed from **ATP** by the enzyme adenylate cyclase. Activation of surface receptors may cause a guanine nucleotide regulatory protein (**G protein**) to interact with adenylate cyclase with resultant changes in intracellular cAMP levels (Fig. 5). Many substances act on surface receptors in this way, including **catecholamines**, **vasopressin**, **ACTH**, **histamine**, **glucagon**, parathyroid hormone and **calcitonin**.

Some substances inhibit adenylate cyclase via an inhibitory regulatory protein, e.g. **noradrenaline** at α_2-**adrenergic receptors** (Fig. 5).

cAMP is termed a 'second messenger' as it causes phosphorylation of proteins, particularly enzymes, by activating protein kinases. Phosphorylation changes enzyme and thus cellular activity. cAMP is inactivated by phosphodiesterase to 5′-AMP. **Phosphodiesterase inhibitors**, e.g. **aminophylline** and **enoximone**, increase cAMP levels.

Adenosine triphosphate and diphosphate (ATP and ADP). ATP is the most important high-energy phosphate compound. When hydrolysed to form ADP, it releases **energy** that may be utilised in many cellular processes,