## Contents

Preface ................................................................................................................................. 2  
Interpreting Pathflow ........................................................................................................... 3  
Abbreviations ....................................................................................................................... 4  
Abdominal Aortic Aneurysm (AAA) ............................................................................. 5  
Anaphylaxis ......................................................................................................................... 6  
Acute Pulmonary Oedema (Secondary to Cardiac Failure) ........................................... 7  
Asthma .................................................................................................................................... 10  
Atheroma (Acute Coronary Syndromes) ...................................................................... 12  
Autonomic Dysreflexia ..................................................................................................... 15  
The Coagulation Cascade ................................................................................................ 16  
Coeliac Disease .................................................................................................................. 18  
Croup ....................................................................................................................................... 20  
Hypovolaemic Shock ........................................................................................................... 21  
Meningococcal Septicaemia ................................................................................................. 24  
Obstructive Sleep Apnoea ............................................................................................... 25  
Organophosphate Poisoning ............................................................................................ 28  
Pulmonary Embolism (PE) ............................................................................................. 30  
Renin-Angiotensin-Aldosterone System ........................................................................ 31  
Seizure ..................................................................................................................................... 33  
Stroke (CVA) ..................................................................................................................... 36  
Tension Pneumothorax ....................................................................................................... 39  
References .......................................................................................................................... 41  
References by Condition ..................................................................................................... 43
Pathoflow was developed to provide a concise and structured resource representing common medical conditions. Its design will provide a starting point for health care students to progress to more advanced condition investigation, and encourage experienced health care professionals to revise.

The genesis for this product was the need for an accessible resource that reinforced essential medical information, and structured in a way to maximize learning and understanding.

The flow chart format eliminates extraneous information common in most textbooks, delivering a clear and accurate summary of each disease process. Each Pathoflow adheres to a template that includes:

- A practical definition of the condition.
- A flow chart of the pathophysiological process.
- Common clinical signs and symptoms.

Treatment regimes are not provided to avoid inconsistencies between health providers and conflicts in current research evidence.

Pathoflow will consolidate your existing knowledge, and complement the use of other texts, journals & clinical guidelines.

Special thanks go to Rory Flynn from Clever Digital for his expertise in project design and format development.

Feedback or suggestion is appreciated, email to: info@pathoflow.com.au.

Colin Knight
Geelong - Australia

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Direct all requests and enquiries regarding use and availability to:
Website: www.pathoflow.com.au
Email: info@pathoflow.com.au

Written by: Colin Knight
Designed by: Clever Digital
Website: cleverdigital.com.au

To access the app, download a pdf sample or order a hardcopy go to: www.pathoflow.com.au

“He who studies medicine without books sails an uncharted sea, but he who studies medicine without patients does not go to sea at all.”

William Osler
Interpreting Pathoflow

Pathoflow was designed to be as succinct as possible. Many terms have been abbreviated, and a list of these has been provided for easy referral.

Each page contains different coloured boxes and symbols that represent different information of significance, designed to provide a consistent, easy-to-read, free-flowing concept.

A typical Pathoflow example may include:

- **Condition**

- **A clear and accurate definition, identifying what it is; where it occurs in the body; what it is characterized by and what it leads to**

- **General pathophysiology**

- **Sub-headings within the process, other significant information & points of interest**

- **Important information that may not directly be part of the flow chart, however is relevant or a vital part of the process**

- **Signs & symptoms within the pathophysiologic process that are a consequence of the condition we see or measure**

After each Pathoflow, the following is provided, encouraging further research:

- Web site links.
- Journal articles.
- Texts.
- References for each condition.

A full list of references is provided at the rear of the document.
Abbreviations

AAA  Abdominal Aortic Aneurism
ACE  Angiotensin Converting Enzyme
AF   Atrial Fibrillation
ACH  Acetylcholine
ACS  Altered Conscious State
ADH  Antidiuretic Hormone
ADP  Adenosine Diphosphate
AMI  Acute Myocardial Infarction
ANP  Atrial Natriuretic Peptide
AP   Action Potential
APO  Acute Pulmonary Oedema
ATP  Adenosine Triphosphate
BP   Blood Pressure
Ca+  Calcium
Ca2+ Calcium
CO   Cardiac Output
CO2  Carbon Dioxide
CNS  Central Nervous System
COPD Chronic Obstructive Pulmonary Disease
CPP Cerebral Perfusion Pressure
CSF  Cerebro-Spinal Fluid
CVA  Cerebrovascular Accident
DIC  Disseminated Intravascular Coagulation
DKA  Diabetic Keto-Acidosis
dVT  Deep Venous Thrombosis
ECM  Extracellular Matrix
EPO  Erythropoietin
FHx  Family History
GABA Gamma-Aminobutyric Acid
GFR  Glomerular Filtration Rate
GIT  Gastro-Intestinal Tract
GORD Gastric Oesophageal Reflux Disease
H2O  Water
HDL  High Density Lipoprotein
HIV  Human Immunodeficiency Virus
HLA  Human Leukocyte Antigen
HMW  High Molecular Weight
HR   Heart Rate
HSP  Hydrostatic Pressure
HT   Hypertension
IgE  Immunoglobulin E
IC   Intra Cellular
ICP  Intra Cranial Pressure
IDDM Insulin Dependant Diabetes Mellitus
IHD  Ischaemic Heart Disease
IL   Interleukin
IV   Intravascular
JVP Jugular Venous Pressure
K+   Potassium
LAP Left Atrial Pressure
LDL Low Density Lipoprotein
LV   Left Ventricle
LVEDP Left Venticular End Diastolic Pressure
LVEDV Left Venticular End Diastolic Volume
LVH Left Ventricular Hypertrophy
LVF Left Ventricular Failure
MAP Mean Arterial Pressure
NDMA N-Methyl-D-Aspartate
NIDDM Non Insulin Dependant Diabetes Mellitus
Na+ Sodium
Na+K+ Sodium Potassium
NaCl Sodium Chloride
O2 Oxygen
OSA Obstructive Sleep Apnoea
PaCO2 Arterial CO2
PaO2 Arterial Oxygen
PE  Pulmonary Embolus
PEA Pulseless Electrical Activity
pH  Acidity
PNS  Peripheral Nervous System
PTH  Parathyroid Hormone
PVR  Peripheral Vascular Resistance
RA  Right Atrium
RBC  Red Blood Cell
REM Rapid Eye Movement
RR  Respiration Rate
RV  Right Ventricle
RVH Right Ventricular Hypertrophy
SCI  Spinal Cord Injury
SOB  Short Of Breath
SpO2 Oxygen Saturation
SV  Stroke Volume
T6  Thoracic Vertebrae No.6
Tb  Tuberculosis
TIA Transient Ischaemic Attack
tTG Tissue Transglutaminase
TxA2 Thromboxane A2
URT Upper Respiratory Tract
URTI  Upper Respiratory Tract Infection
UTI  Urinary Tract Infection
V/Q Ventilation/Perfusion
VR  Venous Return
vWF Von Willebrand Factor
An episodic, reversible inflammatory disease of the small airways mediated by multiple trigger factors and characterised by mucosal oedema, bronchospasm and mucosal plugging, potentially leading to airway obstruction, respiratory failure and cardiac arrest.

Trigger/Stimuli exposure: pollution, URTI, allergen, cold air, drugs, exercise, stress/emotion, foods (dairy, eggs, nuts, seafood, wheat, soy, berries)

Reaction with IgE antibody causes mast cell degranulation

Inflammatory & chemotactic mediator release (histamine, prostaglandins, neutrophils, eosinophils, leukotrienes, lymphocytes, macrophages)

Increased vascular permeability & vasodilation

Bronchospasm + Mucosal plugging + Mucosal oedema = Increase bronchial hyper-responsiveness to stimuli

Increased airflow resistance & pressure on bronchiole walls

Small/lower airways obstructed

Natural increased intrathoracic pressure during expiration further increases airflow resistance & bronchiole wall pressure

Expiratory wheeze

Acute changes

Decreased expiratory flow while maintaining inspiratory respiration volume

Distal airway hyperinflation & “gas trapping”

Increased intrathoracic & alveoli gas pressures & decreased ventilation

Chronic changes

Chronic inflammatory activation

Eosinophil recruitment

Airway smooth muscle hypertrophy & hyperplasia, sub-epithelial collagen deposition and thickening, mucous secreting cell hypertrophy and hyperplasia, neo-vascularisation

Permanent airway remodelling

Decreased lung function

Collapse of low pressure venae cavae

Decreased VR & CO

PEA &/or other heart arrythmias

Decreased alveoli perfusion

Hypoxaemia stimulates brain respiratory centre to increase R/R

Decreased cerebral perfusion

ACS

(cont.)
Asthma (CONT.)

- Decreased PaCO2, increase pH (respiratory alkalosis)
- Persistent decreased alveoli ventilation & perfusion
- Increased breathing workrate (respiratory distress)
- CO2 retention
- Decreased pH, increased PaCO2 (respiratory acidosis)
- Respiratory failure

- Increased catecholamine release
- Increased heart rate & contractility
- Increased workload of heart
A life-threatening, uncontrolled sympathetic reflex discharge in patients with spinal cord injury at T6 or above in response to external or visceral stimulus below the lesion causing parasympathetic/sympathetic CNS imbalances, resulting in conditions associated with severe hypertension.

**Autonomic Dysreflexia**

- SCI at T6 or above
- Complete loss of reflex function below SCI (Spinal Shock)
- Complete loss of bladder & bowel, autonomic & temperature control
- Days/weeks/months post SCI
- Spinal shock resolves; reflex activity returns

Strong sensory stimulus below level of SCI: **Common:** Kinked or irritating catheter, distended bladder or bowel  
**Other:** UTI, burns, tight clothing, haemorrhoids, fracture, DVT, pressure ulcer, pregnancy/labour, sunburn

Sensory neurons transmit stimulus to intact spinal cord and it ascends to level of SCI

Massive sympathetic reflex response cause noradrenaline & dopamine release

- Severe vasoconstriction below SCI
  - Pallor, piloerection
- Increased BP
- Normal inhibitory response from vasomotor centre in medulla oblongata blocked at SCI
- Uncontrolled sympathetic hyperreflexia **below** SCI
- Persistent vasoconstriction typically **BELOW** level T6 causes splanchnic blood vessel bed vasoconstriction shunting large blood volume into systemic circulation

HT detected at carotid & aortic arch baroreceptors

- Increased parasympathetic response to excessive sympathetic NS activity **above** SCI via vagus nerve
- Vasodilation, skin flushing, diaphoresis, severe headache, bradycardia

Sympathetic response **BELOW** SCI overwhelms parasympathetic response **ABOVE** SCI

- Uncontrolled increased BP
- Increased SOB/anxiety
- Cardiac arrhythmias, cerebral haemorrhage, seizure

Remove stimulus. Sympathetic reflex ceases; symptoms resolve
Laryngeal infection (usually viral) causing acute inflammation and oedema at the narrow glottic and subglottic region of the upper airway, characterised by inspiratory stridor, leading to airway obstruction and associated respiratory distress and hypoxia.

NB. Common in children 6 months- 5 years; peak at 2 years. Rare in patients over 5 years as larger airway diameter allows adequate airflow amid upper airway inflammation and oedema associated with URTI.

- URT viral infection; 1-5 days prodrome is common
- Moves into glottic & subglottic areas of larynx
- Fever
- Loosely attached subglottic membranes cause oedema accumulation
- Inflammation
- Barking cough/ hoarse voice
- Decreased lumen size around narrowest area of cricoid cartilage
- Increased extrathoracic (above trachea) airway resistance/occlusion on inspiration
- Decreased intrathoracic (below trachea) airway pressure on inspiration
- Partial airway collapse on inspiration at area of occlusion
- Increased anxiety
- Decreased ventilation
- Inspiratory stridor
- Increased catecholamine release
- Tachycardia
- Increase work of breathing to supplement increase O2 requirement
- Increased hypoxia
- Lethargy, restlessness, agitation
- Rapid fatigue
- Decreased respiratory effort/increased hypoxia
- Decreased stridor
- Bradypnoea & Bradycardia
- Chest wall retraction (children have increased compliance), accessory and/or intercostal muscle use, nasal flaring, tachypnoea
- Respiratory arrest
- ACS
- Decrease SPO2

Children have higher oxygen consumption requirements & metabolic rate than adults, with smaller muscle glycogen reserves. As oxygen availability decreases & work of breathing increases, the endurance of accessory breathing muscles is limited so they fatigue very quickly.