Passing the Final FFICM

High-Yield Facts for the MCQ & OSCE Exams

Muzzammil Ali

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Passing the Final FFICM

This innovative resource serves as a fusion of an MCQ guide and a textbook, providing essential content for postgraduate trainees gearing up for the FFICM Final exam, a vital part of the CCT in Intensive Care Medicine.

Crafted to meet the unique requirements of the MCQ while also addressing the necessity for concise, well-structured responses in the OSCE, this book prioritises quick topic transitions, delivering focused, streamlined learning across a range of ICM topics. Its uniqueness lies in its unorthodox structure; rather than having a conventional table of contents, it gives you the freedom to start your learning adventure from any page.

Each of the 1,400 facts is structured as a question, reflecting the format of both exams. Answers are conveniently positioned just below, eliminating the need for constant page-flipping. The material is carefully curated to cover the breadth of the ICM curriculum, weaving in valuable insights from the author's and colleagues' exam experiences. Random questions replicate the unpredictability of both exams and enhance the reader's capacity to swiftly switch between topics during self-assessment. This balances knowledge application and active recall while using memory-enhancing methods like self-quizzing, chunking, and spaced repetition.

Muzzammil Ali, a graduate of the University of Birmingham, UK, currently holds the position of Senior Registrar in Intensive Care and Acute Internal Medicine in the West Midlands. His dedication extends to leadership roles at Queen Elizabeth Hospital Birmingham, an honorary Clinical Lecturer position at the University of Birmingham, and representing the West Midlands as the trainee representative for Intensive Care Medicine.

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Dedication

To my beloved family,
Amina, Nizam, and Nura,
for your unwavering belief in me.

And to my dearest, Joanna,
for your enduring support, endless inspiration,
and reliable companionship throughout this journey.

Also, to my esteemed colleagues,
for your invaluable mentorship, constant encouragement,
and for fostering an environment that has nurtured my growth.

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Contents

This book does not contain separate chapters but comprises a compilation of bite-sized question-and-answer segments, designed to emulate the unpredictable and erratic nature of both exam components.

With a collection of 1,400 essential facts, each fact functions as a multi-purpose instrument for self-review and study, applicable to a diverse range of postgraduate Intensive Care exams.

How to Use This Book

The key is simple: Cover up!

To get the most out of this book, active mental engagement is essential. Simply skimming through the answers without first attempting the questions hinders the learning process.

I advise using a bookmark, a piece of paper, or even your hand to cover the answers while you tackle each question. Don't be afraid of making mistakes; view them as invaluable opportunities for learning. Keep in mind that making errors while studying is completely acceptable.

I also recommend keeping a notebook close by to jot down any challenging facts or topics that you encounter. This practice will assist you in recognising and subsequently addressing specific knowledge gaps.

You'll notice that some text is intentionally bolded to create acronyms, which are designed to aid in memory retention. I've included this feature because I found it to be particularly helpful during my own review process.

When to Use This Book?

Crafted for quick, digestible learning, this book offers you the flexibility to dive into any section that piques your interest. I highly recommend taking advantage of 'hidden moments' throughout your day – be it while waiting for a train, picking up your kids from school, or queuing for your morning brew. Whether you're studying alone or with peers, these brief moments can be converted into productive revision opportunities. A mere five-minute commitment can deliver tangible progress. This adaptable format is especially useful for making your study routine less daunting, particularly after exhausting on-call shifts.

To drive the point home, consider this succinct example:

Question:

*Which substances primarily cause crystal nephropathy in **tumour lysis syndrome (TLS)**?*

Answer:

Uric acid and calcium phosphate

It's a quick read, but it offers crucial knowledge: in TLS, acute kidney injury can arise from crystal nephropathy, primarily driven by uric acid and calcium phosphate. Consequently, should you face an OSCE question like, 'Why may acute kidney injury occur in TLS?', you are well-prepared to offer an insightful answer.

But this book is more than an exam-prep tool; it acts as a catalyst for lifelong learning. Consistent interaction with the content, even after your exams, ensures that the knowledge remains fresh in your memory. Avoid the trap of letting your acquired knowledge diminish by incorporating this resource into the 'hidden moments' of your daily life.

Disclaimer

- This book serves as a fusion of an MCQ guide and a textbook, primarily intended as a supplementary study resource rather than a comprehensive reference. I highly suggest pairing this book with specialised MCQ and OSCE question banks for a well-rounded study approach. If you discover particular areas where your knowledge is lacking while using this book, I recommend seeking out additional resources that concentrate on those topics to enhance your understanding.
- It's important to underscore that this book includes a limited number of images, which may not fully cover certain image-intensive aspects of the OSCE exam, where you may encounter numerous ECGs and radiological images.
- The frequent use of the phrase 'most likely' aligns with the style of exam questions, indicating that while other diagnoses are possible, the provided answer is the most probable one.
- The information presented is up-to-date as of the book's publication, including details such as the new global definition of ARDS, the revised definition of pulmonary artery hypertension, and the Sepsis-3 definition of sepsis. However, it's important to be aware that information may change or become outdated over time.
- For the sake of brevity, standard medical acronyms like MRI, JVP, CXR, and ECG are used without further elaboration.

Preface

Tackling the FFICM exam was a rigorous endeavour that pushed the boundaries of my medical expertise and adaptability. As I prepared, I noticed an intriguing parallel between the MCQ and OSCE. Despite their differences, both components shared elements of unpredictability, and the occasional inclusion of niche subjects. Achieving success required more than just a fundamental understanding of common topics; it necessitated a distinctive perspective and approach.

This book was specifically crafted to meet the unique requirements of the MCQ, while also addressing the necessity for concise, well-structured responses in the OSCE. It prioritises quick topic transitions, delivering focused, streamlined learning across a range of ICM topics. Its uniqueness lies in its unorthodox structure; rather than having a conventional table of contents, it gives you the freedom to start your learning adventure from any page.

Each of the 1,400 facts is structured as a question, reflecting the format of both exams. Answers are conveniently positioned just below, eliminating the need for constant page-flipping. The material is carefully curated to cover the breadth of the ICM curriculum, incorporating valuable insights from my own exam experiences and those of my colleagues. It truly offers a little bit of everything. Random questions replicate the unpredictability of both exams and enhance your capacity to swiftly switch between topics during self-assessment. My objective is to balance the application of knowledge with active recall, while incorporating memory-boosting techniques such as self-quizzing, chunking and spaced repetition.

When composing this book, I drew upon a wide range of resources to ensure its relevance in the constantly evolving field of medicine. Depending on your clinical experience, you may find some facts either elementary or complex. Yet, virtually any topic, including esoteric ones like 'fire safety', can appear in the exam. Getting acquainted with these topics, even without memorising them, will enhance your likelihood of success. This book also covers specialised topics such as mechanical cardiovascular support and rare conditions like HLH. While these topics may not regularly appear in your day-to-day clinical practice, they are crucial for exam preparation. That said, the emphasis on basic sciences has been scaled back, in alignment with the FICM's assumption of your proficiency through your primary base-specialty exams.

As you work through this resource, you'll find that some of the longer facts can serve as valuable material for the Short Oral Examination (SOE). Consequently, comprehensive preparation for the MCQ and OSCE will undeniably elevate your performance in the SOE. It's worth noting, however, that this book lacks visual aids like ECGs or radiological scans, crucial for OSCE readiness. For this purpose, I recommend using a separate image repository.

My dedication to you goes beyond merely helping you pass the FFICM exam; consider this book a resource for continuous learning. Whether you need a knowledge refresher or a confidence boost, a quick glance at any section of the book should suffice. It also serves as a handy guide for ward rounds, enabling you to deepen your understanding and engage in informed discussions with colleagues.

I extend my warmest wishes to you on your journey.

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About the Author

Muzzammil Ali, a University of Birmingham UK graduate, currently holds the position of Senior Registrar in Intensive Care and Acute Internal Medicine within the West Midlands. He is a fervent advocate for medical education, committed to supporting trainees in achieving point-of-care ultrasound accreditation and excelling in their medical exams.

Renowned as the author of the medical textbook *MRCP Facts* and the creator of @ mrcpfacts, the largest verified Instagram page dedicated to MRCP-related content, Muzzammil's dedication to education extends to leadership roles at the Queen Elizabeth Hospital Birmingham. He also holds an honorary position as a Clinical Lecturer at the University of Birmingham and represents the West Midlands as the trainee representative for Intensive Care Medicine.

Drawing upon his expertise in postgraduate exams, including FFICM, MRCP, and the Acute Medicine SCE, Dr Ali has written this book with the sincere aspiration of guiding others toward success.

Resources

Websites

- BMJ OnExamination: https://www.onexamination.com/
- Brain Trauma Foundation (BTF): https://braintrauma.org/
- British National Formulary (BNF): https://bnf.nice.org.uk/
- British Society of Echocardiography: https://www.bsecho.org/
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- Deranged Physiology: https://derangedphysiology.com/
- EMCrit Project: https://emcrit.org/
- European Society of Cardiology (ESC): https://www.escardio.org/
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- Fire Service: https://www.fireservice.co.uk/
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Fact 1:

What causes a high and low **mixed venous oxygen saturation (S_vO_2)**?

S_vO_2 is obtained from a pulmonary artery catheter. It measures the end result of O_2 consumption and delivery, and contains blood from both the SVC and IVC. The normal range is approximately 65–70%.

↑ S_vO_2	↓ S_vO_2
• ↑ O_2 delivery, e.g. ↑ FiO_2, hyperbaric O_2 • ↓ O_2 extraction, e.g. hypothermia, general anaesthetic, neuromuscular blockade • ↑ Flow states, e.g. sepsis, thyrotoxicosis, severe liver disease	• ↓ O_2 delivery, e.g. shock states, hypoxemia, anaemia • ↑ O_2 extraction, e.g. hyperthermia, shivering, pain, seizures

$S_{cv}O_2$, on the other hand, measures oxygen saturation in the SVC, taken from an internal jugular, subclavian or axillary vein catheter and is sometimes used as a surrogate for S_vO_2.

Typically, in healthy individuals, $S_vO_2 > S_{cv}O_2$ because the brain (SVC-drained) has a higher oxygen demand compared to organs like the kidneys (IVC-drained) with lower oxygen demands.

$S_{cv}O_2$ can surpass S_vO_2 in cases where the brain's metabolic requirement decreases, such as during anaesthesia, in TBI, or in shock, when body oxygen extraction increases, which leads to reduced oxygen saturation in the IVC.

Fact 2:

What factors affect **functional residual capacity (FRC)**?

- FRC = expiratory reserve volume + residual volume.
- It is the volume of air in the lungs after normal expiration, measured by either gas dilution or body plethysmography.

↑ FRC	↓ FRC
• Standing position • Asthma/COPD • PEEP/CPAP	• Supine position • Obesity • Pregnancy • Restrictive lung disorders • General anaesthesia

Fact 3:

Which should be the normal **cuff pressure** of a tracheostomy?

20–30 cm H_2O – should be checked every 8–12 hours, or more frequently depending on the clinical picture

Higher cuff pressures may compress tracheal capillaries, limit blood flow, and predispose to tracheal necrosis (ischaemic damage).

Fact 4:

What is the difference between **cardiac output** and **cardiac index**?

- Cardiac Output = Heart Rate × Stroke Volume
  Normal range ~ 4–8 L/min
- Cardiac Index = Cardiac Output/Body Surface Area
  Normal range ~ 2.5–4 L/min

Fact 5:

What is the physiological role of **C-reactive protein**?

- A pentraxin protein synthesised in the liver
- ↑ In response to inflammation
- Binds to phosphocholine on the surface of dead/dying cells, which activates the complement system

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Fact 6:

Which cardiac structural abnormality may the presence of a **right bundle branch block** in a young adult indicate?

Atrial septal defect

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Fact 7:

What are some of the causes of a **raised MCV**?

D	**D**rugs, e.g. anticonvulsants, antimicrobials, chemotherapy
R	**R**eticulocytosis
A	**A**lcohol abuse
M	**M**egaloblastic anaemia, e.g. pernicious anaemia, B_{12}/folate deficiency
A	**A**rtefact, e.g. aplasia, myelofibrosis, hyperglycaemia, cold agglutinins
T	**T**hyroid (hypothyroidism)
I	**I**mmature bone marrow cells, e.g. myelodysplastic syndrome
C	**C**hronic liver disease

Fact 8:

What is the dose of IV salbutamol in treating **life-threatening asthma**, and what are some side effects?

- Dose: 3–20 mcg/min
- Side effects: tachycardia, arrhythmias, tremors, hyperglycaemia, hypokalaemia, and type B lactic acidosis

Fact 9:

What are the mechanisms of **drug-induced hyperkalaemia**?

K^+ supplements	• Sando-K • IV fluids with K^+
Drugs that impair K^+ distribution	• **B**eta blockers • **A**rginine • **D**igoxin • **S**uxamethonium
Drugs that ↓ renal K^+ excretion	• Calcineurin inhibitors, e.g. tacrolimus and ciclosporin • Potassium-sparing diuretics, e.g. spironolactone, eplerenone • Some antibiotics, e.g. trimethoprim
Drugs that impact on the RAAS	• NSAIDs • ACE inhibitors, ARBs • Heparin

Fact 10:

When do you control hypertension in the first 24 hours after an **acute ischaemic stroke** according to NICE?

NICE advises against actively managing hypertension during this period, except in the following situations:

- To facilitate thrombolysis–target BP < 185/110.
- In cases of pre-eclampsia, aortic dissection, or hypertensive encephalopathy/nephropathy/cardiac failure.

Fact 11:

What percentage TBSA **burn** would meet the criteria for referral to a burns centre on area alone?

>40% Total Body Surface Area (TBSA)

Fact 12:

Where is **propofol** predominantly metabolised?

Hepatic metabolism, primarily via glucuronidation and sulfation pathways.

Fact 13:

What is the dose of IV magnesium in the management of **acute asthma**, and how does it work as a bronchodilator?

Dose: 1.2–2 g IV over 20 minutes

Mechanism as a bronchodilator:

1. Calcium blocker in bronchial smooth muscle
2. ↓ Ach release at the NMJ
3. ↑ Sensitivity of β-receptors to catecholamines

Fact 14:

What is the **Parkland formula** for IV fluid replacement after a burn, and does it take into account pre-hospital fluid administration?

Volume of IV fluids =
4 mL/kg / %TBSA over 24 hours

Half of total is given in the first 8 hours after the injury.

This formula takes into account pre-hospital fluid administration. Therefore, any prehospital fluid is subtracted from total.

When calculating TBSA, erythematous regions are omitted unless there is additional blistering or underlying evidence of a partial-thickness burn.

Fact 15:

What did the **PROPPR trial (2015)** demonstrate for blood product administration in a 1:1:1 ratio compared to a 1:1:2 plasma:platelet:red cell ratio in patients with severe trauma and major bleeding?

- No difference in all-cause 24-hour or 90-day mortality
- Post-hoc analysis found a significant reduction in death by exsanguination within the first 24 hours and a higher rate of achieving haemostasis in the 1:1:1 group compared to the 1:1:2 group.

Fact 16:

What are the 12 physiological variables of the **APACHE II score**?

CNS	CVS	RESP	RENAL	MICRO/HAEM
GCS	MAP HR	RR PaO_2	Arterial pH Na^+ K^+ Creatinine	Temperature WCC Hct
The worst of these variables within the first 24 hours of critical care admission is used				

Effects of age and chronic health are incorporated to give a single score with a maximum of 71. A score of >25 represents a predicted mortality of >50%.

Fact 17:

What dose of adrenaline do you give in adult **anaphylaxis**?

0.5–1 mL of 1:1,000 IM (0.5–1 mg)

OR

0.5–1 mL of 1:10,000 IV (50–100 mcg)

Fact 18:

What is the difference between **intra-abdominal hypertension (IAH)** and **abdominal compartment syndrome (ACS)**, and how do you measure intra-abdominal pressure (IAP)?

- IAH: sustained or repeated pathological elevation of IAP ≥ 12 mmHg
- ACS: sustained IAP > 20 mmHg + new organ dysfunction/failure +/– abdominal perfusion pressure (APP) < 60 mmHg

IAP is measured:

- Direct: puncture of the abdominal cavity
- Indirect: via a urinary catheter in the bladder or a balloon-tipped catheter inserted into the stomach. Correlates well with direct measurements but can be inaccurate when there are adhesions, pelvic fractures, and abdominal packs.

Fact 19:

Where in adults does the **trachea** start and divide anatomically?

- Starts at C6
- Extends to T4 where it bifurcates
- It is approximately 10–12 cm long
- The right main bronchus separates at a 25° angle and the left main bronchus separates at a 45° angle.

Fact 20:

What are the differences between a **Minnesota tube (MT)** and **Sengstaken–Blakemore tube (SBT)**?

Both are used for bleeding UGI varices resistant to medical and/or endoscopic treatment.

- SBT has three ports – oesophageal balloon, gastric balloon, and gastric aspiration port. MT has an additional port for oesophageal suction to ↓ the risk of aspiration.
- The MT has a higher-volume gastric balloon (450–500 mL vs. 250–300 mL).

Fact 21:

What is the evidence for a **decompressive hemicraniectomy (DH)** in malignant middle cerebral artery syndrome according to the DECIMAL (2007), HAMLET (2009) and DESTINY II (2011) trials?

- **Mortality**: Decompressive hemicraniectomy significantly reduces mortality compared to conservative treatment in all three studies. This benefit appears to be particularly strong when surgery is performed early (within 48 hours) after stroke onset. This was observed in younger patients (18–55 years in DECIMAL) and older patients (≥61 years in DESTINY II), as well as in the varied population of HAMLET.
- **Neurological Disability**: The findings on functional outcome are more complex and are potentially dependent on factors like age, stroke severity, and time to surgery:
    - Decompressive hemicraniectomy did not significantly improve the proportion of patients achieving a 'good' functional outcome (mRS ≤ 3) at 6 or 12 months in DECIMAL and HAMLET.
    - However, it significantly increased the proportion of patients achieving a 'moderate' functional outcome (mRS ≤ 4) at six months in DECIMAL and DESTINY II.
    - Notably, no surviving patients in DESTINY II achieved the best possible functional outcome (mRS 0–2).
- **Overall**: Decompressive hemicraniectomy offers a clear and substantial mortality benefit. While it may not guarantee good recovery and return to pre-stroke levels of function, it can increase the chances of achieving moderate disability as opposed to severe disability or death. This decision requires individualised assessment and MDT involvement.

Fact 22:

How do you distinguish between moderate, severe and life-threatening **acute asthma**?

<table>
<tr><th>Moderate</th><th>Severe</th></tr>
<tr><td>PEFR > 50%</td><td>PEFR 33–50%</td></tr>
<tr><td rowspan="2">No features of severe asthma</td><td>RR ≥ 25

HR ≥ 110

Inability to complete sentences in one breath</td></tr>
<tr><td>Life-threatening</td></tr>
<tr><td colspan="2">Features of severe asthma + at least one of:
• PEFR < 33%
• SpO_2 < 92%
• Normal or ↓ PCO_2: implies poor ventilation
• <u>C</u>yanosis, confusion or coma
• <u>H</u>ypotension or ↓ HR
• <u>E</u>xhaustion or poor respiratory effort
• <u>S</u>ilent chest
• <u>T</u>achy(arrhythmia)</td></tr>
</table>

Fact 23:

When is it safe to use **suxamethonium** after a significant burn injury?

- Within the first 24 hours after the burn
- One year after the burn

Fact 24:

How do you calculate the internal diameter of an **endotracheal tube** in the paediatric population?

Cuffed : [age / 4] + 3.5

Uncuffed : [age / 4] + 3.5 + 0.5

Fact 25:

What is the recommended therapeutic management for a **variceal haemorrhage** that continues to bleed despite pharmacological intervention, endoscopic banding and balloon tamponade?

Transjugular Intrahepatic Portosystemic Shunt (TIPS)

Fact 26:

When is **damage control surgery** more preferable than definite surgery in trauma?

When there is severe haemorrhagic shock and/or ongoing bleeding. This is particularly necessary if the **lethal diamond** is present (hypothermia, acidosis, coagulopathy and hypocalcaemia) and in patients who have inaccessible major venous injuries or those who require time-consuming procedures.

Fact 27:

What risk is associated with the administration of **suxamethonium** after a spinal cord injury, and how soon after the injury does this risk occur?

- Life-threatening hyperkalaemia
- 72 hours after spinal cord injury

Fact 28:

How is the **rapid shallow breathing index (RSBI)** useful as a weaning predictor?

RSBI = RR / Tv

- RSBI < 105: 80% chance of successful extubation
- RSBI >105: strongly suggests weaning failure

Fact 29:

What are the approximate proportions of Na^+ and K^+ in some commonly used **crystalloid solutions**?

Fluid	Na^+ (mmol/L)	K^+ (mmol/L)
0.9% N. saline	154	0
Hartmann's	131	5
5% Dextrose	0	0
0.18% N. saline + 4% dextrose	30	0
0.45% N. saline + 5% dextrose	77	0

Fact 30:

What happens to pulmonary artery pressure after a **cardiac arrest**?

↑ Multifactorial reasons why this may occur include:

- Post-ROSC
  - ↑ PVR secondary to hypoxia/acidosis
  - ↑ PAP secondary to cardiac dysfunction as part of post-cardiac arrest syndrome
- Precipitant of cardiac arrest
  - ↑ PAP secondary to cardiac dysfunction (e.g. STEMI) or pulmonary embolism

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Fact 31:

What **adrenaline** dose would you give to a three-year-old in cardiac arrest?

10 mcg / kg = 0.1 mL / kg of 1 in 10,000 solution

Formula for weight	Calculation	Estimated weight (kg)
3 (age) + 7	3(3) + 7	16
2 (age + 4)	2(3 + 4)	14

Using the average weight of 15 kg, this is 150 mcg, which is **1.5 mL of 1:10,000 of adrenaline solution**.

Fact 32:

What is meant by **intention to treat** analysis in a randomised controlled trial?

- All participants are analysed based on their originally assigned treatment groups, regardless of whether they completed or received the intended treatment.
- This approach helps maintain the randomisation and avoids biases caused by crossover or dropout.

Fact 33:

What are the differences in **relative humidity** in a ventilator circuit when using an ultrasonic nebuliser as opposed to a heat and moisture exchanger (HME)?

- Ultrasonic nebuliser: exceeds 100% relative humidity
- HME: achieves approximately 70% relative humidity

Fact 34:

Which clinical features in someone with a **burn** may indicate the need for early intubation?

- GCS < 8
- Respiratory distress or failure
- Noticeable swelling or blistering in the lips, tongue or oropharynx
- Voice changes, e.g. hoarseness or stridor
- Singed nasal hair
- Carbonaceous sputum
- Extensive burns of the face or neck, including circumferential burns

Fact 35:

How does **digoxin** work in treating atrial fibrillation with a fast ventricular rate?

Direct	Indirect
Inhibits cardiac Na^+/K^+-ATPase ▼ ↑ Intracellular Na^+ ▼ Exchange of Na^+ for Ca^{2+} via the Na^+/Ca^{2+} pump ▼ ↑ Intracellular Ca^{2+} ↓ Intracellular Na^+	↑ Acetylcholine at cardiac muscarinic receptors
↑ Intracellular Ca^{2+} causes ↑ cardiac contraction **↓ Intracellular Na^+ prolongs refractory time of the bundle of His**	**Results in prolongation of the refractory period at the AV node and bundle of His**

Fact 36:

Which alternative drug can be used in the management of AVNRT if **adenosine** is contraindicated?

Verapamil 2.5–5 mg IV

Fact 37:

Why may someone with **primary hyperaldosteronism (Conn's syndrome)** develop muscle weakness and tetany?

Due to hypokalaemic metabolic alkalosis

Conn's syndrome causes a low renin hypertension.

It is diagnosed by a ↑ aldosterone:renin ratio.

Fact 38:

What modifications have been implemented in advanced life support algorithms for resuscitating individuals with **hypothermia**?

- Refrain from administering adrenaline or any other drugs until the temperature is >30°C.
- When the temperature ranges from 30°C to 35°C, double the dose intervals for ALS drugs.
- In cases of VF, consider delivering up to three shocks if needed, but hold off on further shocks until the temperature is >30°C.

Fact 39:

What is the most likely diagnosis if someone develops **hypocalcaemia and seizures** two days after starting chemotherapy?

Tumour lysis syndrome (TLS) – electrolyte abnormalities can precipitate neurological dysfunction.

Common abnormalities include:

- ↓ Calcium
- ↑ Phosphate, potassium, urate, LDH, lactate

TLS is due to the sudden and large-scale death of cells following the initiation of chemotherapy. It is often associated with acute leukaemias and high-grade lymphomas, e.g. Burkitt's.

Fact 40:

What are the most likely causes for developing drowsiness one week after undergoing endovascular coiling for a **subarachnoid haemorrhage**, when a CT scan indicates no rebleeding, infarction or hydrocephalus?

- Delayed cerebral ischaemia (DCI)
  - Cerebral vasospasm
  - Local hypoperfusion or disordered autoregulation
- Non-convulsive seizures

Fact 41:

What's the rationale for including **clindamycin or linezolid** alongside broad-spectrum antibiotics in the treatment of necrotising fasciitis?

For the termination of toxin production

Fact 42:

Besides **hypothermia**, what are some alternative reasons for the presence of J-waves on an ECG?

Normal variant (early repolarisation)

Hypercalcaemia
Angina – vasospastic
Brain injury including a subarachnoid haemorrhage
Idiopathic ventricular fibrillation
Type 1 Brugada syndrome

Fact 43:

When would you consider placing an inferior vena cava (IVC) filter after a **pulmonary embolism (PE)**?

- If anticoagulation is contraindicated, e.g. the bleeding risk is very high
- If thrombosis has recurred despite adequate anticoagulation
- If temporary cessation of anticoagulation within one month is anticipated, e.g. pregnant patients within one month of the expected date of delivery

IVC filters have no long-term mortality benefit. As foreign material, they are thrombogenic (↑ incidence of DVT).

Fact 44:

What is the utility of the **LRINEC (Laboratory Risk Indicator for Necrotising Fasciitis)** score?

- The LRINEC score distinguishes necrotising fasciitis from other soft tissue infections, e.g. cellulitis.
- The score incorporates CRP, WCC, Hb, sodium, creatinine and glucose.
- A LRINEC score of ≥6 could be used as a potential tool to rule in necrotising fasciitis, but a score <6 should not be used to rule out the diagnosis.
- A score ≥8 has a positive predictive value >90%.

Fact 45:

Is it necessary to check digoxin levels during and after administering **digoxin-specific antibody fragments** for digoxin toxicity?

No – the assay measures both digoxin bound to antibody fragments and free digoxin. This overestimates free levels.

Fact 46:

What fibrinogen level may warrant administering **cryoprecipitate** in a trauma patient?

Fibrinogen < 1.5–2 g/L

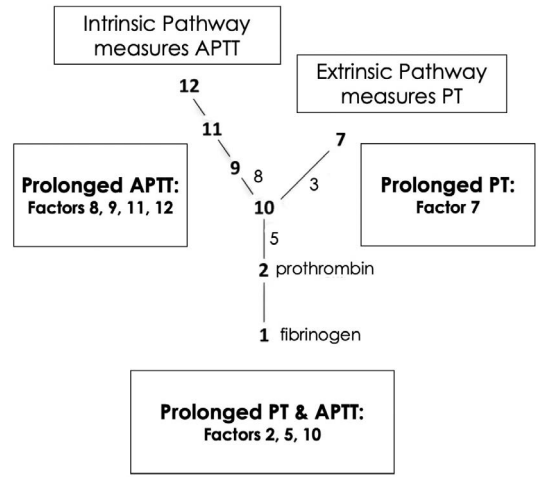
Fact 47:

What is the normal axis for left ventricular depolarisation in an adult, and what are the Sokolow-Lyon criteria for **left ventricular hypertrophy (LVH)**?

- Normal axis: –30 to +90 degrees
- LVH: If the height of the R wave in $V_{5/6}$ + the depth of the S wave in $V_{1/2}$ is ≥35 mm
- The most common cause of LVH is hypertension

Fact 48:

What are the two pathways of **coagulation**?



Fact 49:

Which substances are primarily responsible for crystal nephropathy in **tumour lysis syndrome**?

Uric acid and calcium phosphate

↑ Cell turnover → ↑ purine metabolism → ↑ serum urate

↑ Cell lysis → ↑ serum phosphate which binds to calcium

Uric acid precipitates readily in the presence of calcium phosphate, and calcium phosphate precipitates readily in the presence of uric acid.

Fact 50:

According to the BTS guidelines, when do you insert a chest drain for a **spontaneous pneumothorax (PTX)**?

1. Primary PTX when aspiration fails. Aspiration is indicated for a primary PTX when breathless and/or size is >2 cm.
2. Secondary PTX when aspiration fails. Aspiration is indicated for a secondary PTX when the size is 1–2 cm.
3. Secondary PTX when the patient is breathless and/or the size of the pneumothorax is >2 cm.

Fact 51:

What clinical feature would indicate a **haemodynamically unstable pulmonary embolism** (previously called a massive or high-risk PE)?

Significant hypotension:

Blood pressure of <90 mmHg OR a drop of >40 mmHg from baseline, which is not explained by something else, e.g. arrhythmia, hypovolaemia or sepsis.

Fact 52:

What makes **ketamine** effective in enhancing lung function and alleviating bronchospasm in asthma?

It is a bronchodilator.

It is a phencyclidine derivative. It has little effect on the laryngeal reflexes, and a patent airway can often be maintained. However, it can induce ↑ secretion production, potentially leading to laryngospasm due to these retained reflexes.

Fact 53:

What CT features are associated with an increased severity of **acute pancreatitis**?

- Extensive fat stranding
- Peri-pancreatitis fluid collections
- Pancreatic necrosis

Fact 54:

On which side of the body do traumatic **diaphragmatic injuries** tend to occur?

Left side

Fact 55:

What are the principles of ventilating someone with **life-threatening asthma** and which strategies can achieve this?

Principles	Strategies
Lung protective ventilation: • Limit peak and mean airway pressures • Allow for a prolonged expiratory time • Maintain adequate oxygenation	• ↓ Tidal volume • ↓ RR • ↓ Or removal of extrinsic PEEP • ↓ Inspiratory time or ↑ expiratory time • Permissive hypercapnia • Intermittent disconnection from ventilator and manual chest decompression

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Fact 56:

Why should care be taken when giving **flumazenil**?

It reverses the effects of benzodiazepines.

It has a relatively short half-life when compared to benzodiazepines and has a risk of provoking seizures when administered.

Fact 57:

What do the **Berlin criteria** for ARDS entail, and what are the four key recommendations of the **New Global Definition of ARDS** that expand upon these criteria?

Berlin criteria:

Timing	Within one week of a known clinical insult or new/worsening respiratory symptoms
Oxygenation (P/F ratios)	PaO_2/FiO_2 < 40 kPa (≤300 mmHg) with a minimum PEEP of 5 cm H_2O: • Mild ≤ 39.9 kPa • Moderate ≤ 26.6 kPa • Severe ≤ 13.3 kPa
CXR or CT	Bilateral opacities not explained by effusions/collapse/nodules
Origin of oedema	Respiratory failure NOT fully explained by cardiac failure or fluid overload

Four key recommendations:

- Intubation is not required for making a diagnosis. This includes patients on high-flow nasal oxygen (HFNO) ≥ 30 L/min or NIV/CPAP with end-expiratory pressure ≥ 5 cmH_2O.
- Uses SpO_2/FiO_2 as an alternative to P/F to assess oxygenation. A SpO_2/FiO_2 ≤ 315 + SpO_2 ≤ 97% is indicative of compromised oxygenation.
- The requirement of bilateral lung opacities as an imaging criterion remains, but now includes using ultrasound, provided it is performed by a well-trained operator.
- In settings with limited resources, the following are not essential for diagnosis: PEEP, specific oxygen flow rates, or particular types of respiratory support devices.

Fact 58:

Which ion has the most significant impact on the **resting potential** of neural tissue?

Potassium – This has a large concentration gradient across the cell membrane *and* the greatest permeability at rest.

Every cell membrane has a transmembrane potential difference of –70 mV. This difference is dependent on two factors: The transmembrane concentration gradient *and* the permeability of the membrane to the ions.

Fact 59:

What are some general respiratory changes that occur by the third trimester of **pregnancy**?

Tidal volume	↑	Chest wall compliance	↓
Respiratory rate		Airway resistance	
pH		FRC	
PaO_2		HCO_{3^-}	
Maternal 2,3-DPG		$paCO_2$	

Lung compliance remains largely the same.

Fact 60:

What is the pathogenesis of **hepatic encephalopathy (HE)** in chronic liver disease and what are some treatment options?

- **Pathogenesis**: HE occurs when the liver cannot remove ammonia from enteric sources. This ammonia enters the systemic circulation. It goes to the brain to cause neurotoxicity and cerebral oedema.
- **Management**:
  - Treat any precipitating factor, e.g. SBP, UGIB, electrolyte disturbances. May also need organ support on ICU, e.g. for ↓ GCS in Grade 4 HE
  - Lactulose & Rifaximin: ↓ Ammonia levels in the gut by ↑ transit time and ↓ bacterial numbers
  - LOLA: Removes ammonia from the blood through extrahepatic metabolism of ammonia to glutamine. This treatment is not widely available and has little evidence of benefit.

Fact 61:

Which parameters on a **blood gas** are directly and indirectly measured?

- Direct: pH, PaO_2, $PaCO_2$
- Indirect: Standard bicarbonate, base excess, SaO_2

Fact 62:

How do you treat **prilocaine** toxicity?

Methylthioninium chloride (methylene blue) 1 mg/kg IV

- Prilocaine is metabolised in the liver to O-toluidine.
- O-toluidine oxidises haemoglobin into methaemoglobin.
- Methaemoglobin has ↓ oxygen-carrying capacity resulting in cyanosis.
- Methylene blue accelerates the reduction of methaemoglobin.

Fact 63:

How do you distinguish between **hyperacute, acute and subacute liver failure** using the O'Grady classification?

Acute liver failure is triad of jaundice, coagulopathy and encephalopathy. It is classified according to the time from the onset of jaundice to the development of encephalopathy:

- Hyperacute disease: <7 days
- Acute disease: 1–4 weeks
- Subacute disease: 4–12 weeks

Fact 64:

What is meant by **pulsus paradoxus** and in which clinical contexts may it be present?

An amplification of the typical drop in systolic BP during inhalation by >10 mmHg. When severe, the radial pulse vanishes during inspiration. The 'paradox' is that the pulse disappears despite cardiac contraction. It is often due to pericardial disease, particularly cardiac tamponade, but can occur in many contexts including ('PRACTICE'):

- **P**ulmonary embolism
- **R**V infarction and **R**estrictive cardiomyopathy
- **A**sthma and COPD (severe)
- **C**ardiac tamponade and **C**onstrictive pericarditis
- **T**ension pneumothorax
- **I**atrogenic during surgery
- **C**ompression (obesity, pectus excavatum)
- **E**ffusions (large bilateral pleural effusions)

Fact 65:

What ECG pattern has developed if a person who took a large quantity of **cocaine** now has ST elevation in $V_{1–3}$ and T-wave inversion in $V_{1–2}$?

Drug-induced Brugada

Fact 66:

What type of lung injury does a titrated PEEP strategy aim to decrease when treating **ARDS**?

↓ Atelectotrauma

Fact 67:

What are the King's criteria for **non-paracetamol-induced acute liver failure**?

INR > 6.5 (PT > 100 seconds)	OR	Any three of the following: • Aetiology: Non-A, non-B hepatitis or idiosyncratic drug reaction • **B**ilirubin > 300 µmol/L • **A**ge < 11 or > 40 years • **I**NR > 3.5 (PT > 50 seconds) • **T**ime from onset of jaundice to encephalopathy > 7 days

Fact 68:

Which three clinical signs make up Beck's triad in acute **pericardial tamponade**?

- ↓ BP
- ↓ Heart sounds (quiet/muffled)
- ↑ CVP – distended neck veins

Fact 69:

How does **mannitol** reduce intracranial pressure (ICP)?

Mannitol is a diuretic and 'free radical scavenger'. It is freely filtered through the glomerulus and is not re-absorbed. It decreases ICP in two main ways:

Immediate effect (Plasma expansion)	**Slightly delayed effect (Osmotic gradient)**
Plasma expansion results in: • ↓ Blood viscosity • ↑ Intravascular volume • ↑ Cardiac output Overall, these effects result in ↑ regional cerebral blood flow and compensatory cerebral vasoconstriction in brain regions where autoregulation is intact → ↓ ICP	↑ Plasma osmolality → shifts water from cerebral extracellular space into plasma → ↓ cerebral oedema (if the BBB is intact)

Fact 70:

Which arrhythmias can cause **cannon-A** waves in the JVP waveform?

- Complete heart block
- Ventricular tachycardia
- Junctional rhythms including AVNRT

Fact 71:

Which complication of **heparin** therapy carries the highest mortality?

Type 2 HIT (heparin-induced thrombocytopaenia) due to arterial/venous thrombotic complications

	Type 1 HIT	Type 2 HIT
Onset (days)	1–4	5–14
Nadir platelet count	>100,000	50–100,000
Immune complexes	No	Yes (IgG-Heparin-platelet factor 4)
Thrombosis	No	Arterial and venous
Treatment	Observation	Stop heparin Change to an alternative anticoagulant to prevent thromboses, e.g. direct thrombin inhibitors (lepirudin, argatroban, bivalirudin) or anti-thrombin-dependent factor Xa inhibitors (fondaparinux, danaparoid)

Fact 72:

How may **somatosensory evoked potentials (SSEPs)** suggest poor neurological outcome post-cardiac arrest?

They test the somatosensory pathway integrity.

Stimulating a peripheral nerve, e.g. median nerve can be detected at the cortical level. The bilateral absence of the N20 spike on SSEP testing can be used as part of neuro-prognostication in predicting poor neurological outcome in comatosed patients following cardiac arrest.

Fact 73:

What is the classical CSF finding in **Guillain–Barré syndrome (GBS)**?

Albuminocytological dissociation

(↑ CSF protein, normal glucose and no pleocytosis)

GBS is a post-infectious immune-mediated acute inflammatory demyelinating polyneuropathy (AIDP). The majority of cases occur within one month of a respiratory or GI infection, e.g. *Campylobacter jejuni*, *Mycoplasma pneumoniae*, CMV or EBV. The pathogenesis is a cross reaction of antibodies with gangliosides in the peripheral nervous system. Anti-ganglioside antibodies (e.g. anti-GM1) are present in 25% of patients. The majority of cases exhibit Landry's ascending paralysis, areflexia and autonomic dysfunction. Sensory symptoms tend to be mild/absent.

Fact 74:

Which reversal agent can you use to manage major bleeding in someone who takes **dabigatran**?

Idarucizumab–5 mg IV bolus

This is humanised monoclonal antibody fragment that binds to dabigatran and its metabolites.

HD or HF can also remove dabigatran.

Fact 75:

What are the **RIFLE criteria** for acute kidney injury?

	Creatinine	GFR	Urine output
Risk	↑ Cr × 1.5	>25% ↓	<0.5 mL/kg/hr for 6 hrs
Injury	↑ Cr × 2	>50% ↓	<0.5 mL/kg/hr for 12 hrs
Failure	↑ Cr × 3		<0.3 mL/kg/hr for 24 hrs OR Anuria for 12 hrs
Loss	Loss of renal function > Four weeks		
End stage	Loss of renal function > Three months		

Fact 76:

What is the **GCS** if someone with a head injury is opening his eyes to painful stimuli, is saying occasional inappropriate words and is extending his limbs to pain?

GCS = 7

Score	Eye opening	Verbal response	Motor response
6	-	-	Obeys commands
5	-	Alert and orientated	Localises to pain
4	Spontaneous	Confused	Withdrawal from pain
3	On speech	**Inappropriate words**	Flexion to pain
2	**On pain**	Incomprehensible sounds	**Extension to pain**
1	None	None	None

Fact 77:

Why is **atropine** ineffective for treating bradycardia in a heart transplant?

The heart is completely denervated. Therefore, the lack of a vagal input would prevent any anticholinergic effects of atropine. Sympathomimetic agents are used instead for chronotropy or inotropy.

Fact 78:

Which organism is most likely to cause right-sided **infective endocarditis** in an intravenous drug user?

Staphylococcus aureus

Fact 79:

How may hyperoxia cause harm in an **acute myocardial infarction**?

- Through free radical production
- Through coronary vasoconstriction

Fact 80:

What are the four mechanisms of AKI in **rhabdomyolysis**?

1. Myoglobin combines with Tamm–Horsfall protein to form insoluble casts and tubular obstruction.
2. Hyperuricaemia worsens this tubular obstruction.
3. The haem moiety is directly nephrotoxic.
4. There is inappropriate renal vasoconstriction because of hypovolaemia and third-spacing.

Fact 81:

What is the $PaCO_2$ level in **life-threatening asthma**?

Normal or ↓ $PaCO_2$

Fact 82:

Why may peripheral oedema develop in **cor pulmonale** if there is preserved ventricular function?

Chronic hypoxia causes sympathetic stimulation. This activates the renin–angiotensin–aldosterone system (RAAS) which results in fluid retention.

Fact 83:

What is meant by **FVC, FEV_1/FVC ratio, TLC and DLCO**?

- FVC: Volume from maximal inspiration to maximal expiration
- FEV_1/FVC ratio: The portion of FVC exhaled in the first second
- TLC: The total volume of gas in the lungs at maximal inspiration
- DLCO: Lung diffusion capacity for carbon monoxide, approximating oxygen transfer from alveoli to red blood cells

Fact 84:

Which **laxative** class is least likely to cause diarrhoea?

Bulk-forming laxatives, e.g. psyllium, methylcellulose

These laxatives work by absorbing water in the intestine, which increases stool bulk and stimulates peristalsis.

Fact 85:

What is meant by **pre-excitation** in electrophysiology?

There is an accessory pathway between the atria and ventricles. This prematurely activates the ventricles.

Fact 86:

What is the **static compliance** if the expired tidal volume is 800 mL, plateau pressure is 50 cmH_2O and PEEP is 10 cm H_2O?

$$C_{stat} = V_t / (P_{plateau} - PEEP)$$

$$C_{stat} = 800 / 50 - 10 = 20 \text{ mL} / cmH_2O$$

Static compliance represents pulmonary compliance during periods without gas flow, e.g. inspiratory hold

Fact 87:

What are the two types of **amiodarone-induced thyrotoxicosis (AIT)**?

	Type 1 AIT	**Type 2 AIT**
Pre-existing thyroid disease	Often	No
Pathology	Excessive hormone synthesis due to excess iodine found in amiodarone	Excessive release of preformed hormones due to thyroiditis
Goitre	Mostly multinodular	Absent or small
Radioiodine isotope uptake	Normal	↓/ absent
Thyroid ABS	Present	Absent
IL-6	Normal/mildly raised	Very high
Treatment options	• Stop amiodarone • Antithyroid drugs • K^+ perchlorate • Thyroidectomy	• Stop amiodarone • Anti-inflammatory drugs, e.g. prednisolone