Making Sense of the 2-Phase Examination System

What is tested?
Why?
How to approach each phase?

Laetitia Jaillardon and Niki Skeldon

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Making Sense of the 2-Phase Exam System



1. Why a 2-phase exam?
2. Phase 1 or phase 2 topic?
3. Do I have the right methods for learning?
4. Top tips!

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Ethos behind 2-phase testing

- To ensure a **strong foundation in pathophysiology** and mechanisms of disease to underpin the practical skills
- To encourage **application of Quality principles** to every aspect of clinical pathology right from the start
- To encourage **study of core texts early** in the residency
- To **facilitate the examination experience** for residents
- To bring the examination **process in line with other European colleges**

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Phase 1 v. Phase 2

Phase 1

- Lab management and Quality
- Mechanisms of disease
- Pathophysiology

Phase 2

- Applied knowledge
- Analytical concepts and methodologies
- Interpretation of clinical pathology data
- Diagnostic reasoning
- Practical skills

Phase 1 v. Phase 2

Phase 1

- Causes of test inaccuracies
  (relates to Quality)
- Mechanism of renal biomarker increases
  (relates to renal pathophysiology)
- Coagulation cascade details

Phase 2

- Test analytic concepts and interpretation
- Interpretation of a renal panel of results
- Interpretation of a TEG graph

Pop Quiz



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Quality control procedures are primarily designed to detect failure in which phase of the laboratory testing process?



A. Analytical phase
B. Preanalytical phase
C. Postanalytical phase
D. Clinical interpretation phase

Ref: Arnold, J. E. et al. (2019). ASVCP Guidelines: Principles of Quality Assurance and Standards for Veterinary Clinical Pathology (version 3.0): Developed by the American Society for Veterinary Clinical Pathology's (ASVCP) Quality Assurance and Laboratory Standards (QALS) Committee. *Veterinary Clinical Pathology*, *48*(4), 542–618. https://doi.org/10.1111/vcp.12810

LAB MANAGEMENT AND QUALITY

Proteinuria associated with chronic glomerular disease results from disruption of which component?

A. Renal medullary interstitial urea concentration gradient
B. Glomerular basement membrane filtration barrier integrity
C. Renal tubular epithelial apical brush border transport proteins
D. Juxtaglomerular apparatus renin secretion signalling pathways

Ref: Stockham & Scott, 3rd ed., Chapter 8: Urinary System



PATHOPHYSIOLOGY

Normal synovial fluid viscosity is primarily maintained by which molecular component?



A. Albumin
B. Fibrinogen
C. Hyaluronan
D. Immunoglobulin G

Ref: Stockham & Scott, 3rd ed., Chapter 20: Synovial Fluid

(PATHO)PHYSIOLOGY

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A blood gas analysis in a dog yields the following results. Which acid–base disturbance is most consistent with these findings?

pH: 7.24 (RI: 7.35–7.45)
HCO_3^-: 14 mmol/L (RI: 20–24 mmol/L)
$PaCO_2$: 28 mmHg (RI: 35–45 mmHg)



A. Uncompensated metabolic acidosis
B. Fully compensated respiratory alkalosis
C. Partially compensated metabolic acidosis
D. Mixed metabolic acidosis and respiratory acidosis

Ref: Stockham & Scott, 3rd ed., Chapter 10: Blood gases, Blood pH and Strong-Ion Difference

BIOCHEM – DIAGNOSTIC INTEPRETATION

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A 7-year-old Labrador Retriever presents with these findings. Which factor most likely contributes to the decreased tT4 concentration?

- Lethargy
- Weight gain
- Exercise intolerance
- Serum tT4 decreased
- Serum TSH increased



A. Increased clearance
B. Underactive pituitary gland
C. Altered thyroglobulin conformation
D. Autoimmune destruction of the thyroid gland

Ref: Stockham & Scott, 3rd ed., Chapter 17: Thyroid function

PATHOPHYSIOLOGY

A dog has clinical signs compatible with hypothyroidism. Serum tT4 is decreased, and serum TSH is increased. Which diagnosis is most strongly supported?



A. Euthyroidism
B. Hyperthyroidism
C. Primary hypothyroidism
D. Secondary hypothyroidism

Ref: Stockham & Scott, 3rd ed., Chapter 17: Thyroid function

BIOCHEM – DIAGNOSTIC INTERPRETATION

A dog with suspected hypothyroidism has a decreased tT4 concentration. Which additional test is most useful for evaluating the concentration of biologically available thyroxine?



A. Free T4
B. Total T3
C. Serum TSH
D. Thyroglobulin antibody

Ref: Stockham & Scott, 3rd ed., Chapter 17: Thyroid function

BIOCHEM – DIAGNOSTIC PATHWAY

A dog has severe bacterial peritonitis. What is the best explanation for the leukogram findings?

WBC: 7.2 × 10^9/L
Segmented neutrophils: 2.1 × 10^9/L
Bands: 3.4 × 10^9/L
Metamyelocytes: 1.1 × 10^9/L
Smear examination: toxic changes



A. Marrow exhaustion
B. Storage-pool depletion
C. Increased granulopoiesis
D. Neutrophil production exceeding demand

Ref: Stockham & Scott, 3rd ed., Chapter 2: Leukocytes

BONE MARROW PATHOPHYSIOLOGY AND LEUKOCYTE KINETICS

A greyhound repeatedly has a neutrophil count slightly below the reference interval established for the general canine population. Which interpretation is most appropriate?



A. Bone marrow pathology
B. Breed-associated variation
C. Overwhelming inflammation
D. Immune mediated neutropenia

Ref: Stockham & Scott, 3rd ed., Chapter 2: Leukocytes

HAEMATOLOGY – BREED SPECIFIC INTERPRETATION

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Which principle is most important for obtaining an accurate cell concentration from a Neubauer counting chamber?



A. Count cells in one area
B. Apply a consistent dilution
C. Use identical chamber volumes
D. Relate cells counted to the defined volume

Ref: Stockham & Scott, 3rd ed., Chapter 2: Leukocytes

HAEMATOLOGY – ANALYTICAL PRINCIPLES AND METHODS

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Persistent fasting hyperlipemia most strongly suggests abnormality of which process?



A. Intestinal lipid absorption
B. Clearance of circulating lipoproteins
C. Hepatic synthesis of bile acid molecules
D. Renal excretion of non-esterified fatty acids

Ref: Stockham & Scott, 3rd ed., Chapter 16: Lipids

PATHOPHYSIOLOGY

Lipemia may interfere with photometric laboratory methods primarily through which analytical mechanism?

A. Altered enzymatic substrate binding
B. Increased light scattering by lipid particles
C. Increased light absorption absorption within the sample
D. Reduced analyte diffusion through reagent reaction mixtures



Ref: Stockham & Scott, 3rd ed., Chapter 1: Analytical and Diagnostic Concepts: Getting it Right

ANALYTICAL ERRORS

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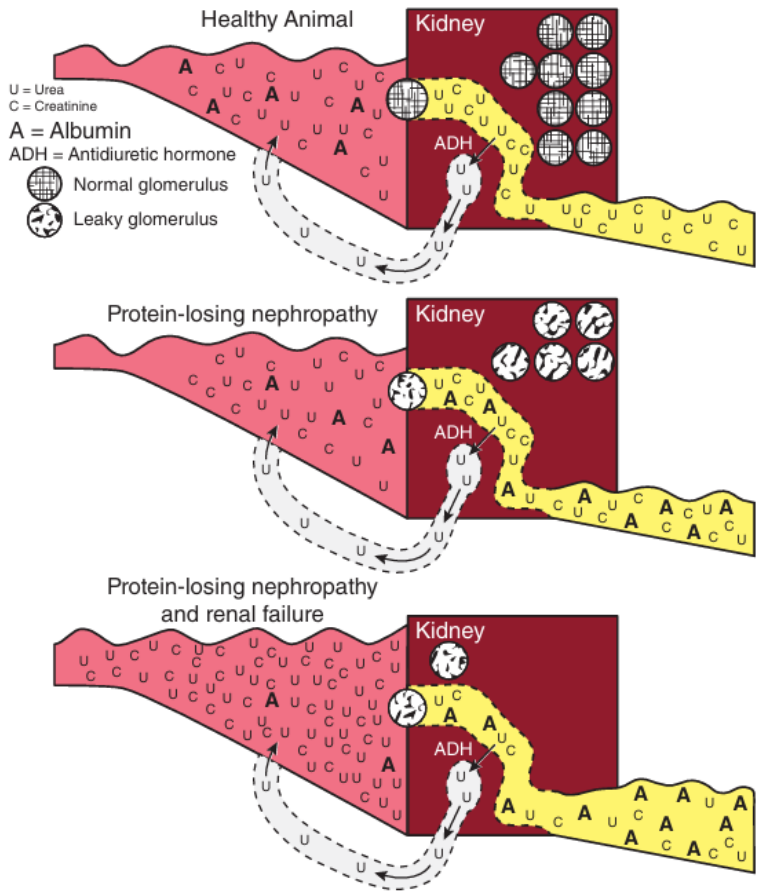


Figure 8.13. Protein-losing nephropathy and renal failure. Illustrations depict a body's extracellular fluid as a lake, kidneys as a dam, and the urine as the river below the dam. Urea and Crt molecules are small fish in the lake, and albumin molecules are big fish. The nephron consists of the sieve or filter at the lake outlet and the tubes that run through the dam. Some H_2O and urea are reclaimed to maintain extracellular fluid H_2O and urea content. Only 1 nephron per dam is shown as a filter with a connected tubular system, but each would have one.

- In healthy animals, urea and Crt pass freely through 10 functional filters, into the dam's tubes, and into the river. Some urea (and H_2O) is reclaimed from the dam's tubes in a process enhanced by ADH. Albumin

PHASE 1 – RENAL PATHOPHYSIOLOGY

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Table 2.9. Disorders and conditions that cause neutropenia
Inflammation
Overwhelming bacterial infections and endotoxemia* (e.g., horses with equine salmonellosis or Potomac horse fever)
Some viral infections causing secondary bacterial sepsis/endotoxemia:* canine and feline parvovirus (myelotoxicity also contributes), equine influenza
Variety of acute inflammatory states in cattle:* mastitis, pneumonia
Mature-neutrophil destruction
Idiopathic IMN
Alloimmune neonatal neutropenia
Hemophagocytic syndrome
Bone marrow lymphocytosis of granular lymphocytes (neoplastic or nonneoplastic)
Granulocytic or generalized marrow hypoplasia (decreased production neutropenia)
Infectious agents:* parvovirus (dogs and cats), FeLV, *Toxoplasma*, *Ehrlichia*
Neoplasms: primary or metastatic
Toxicants (see Chapter 6)
Predictable:* cytotoxic chemotherapeutics, azathioprine, chloramphenicol (cats), endogenous or exogenous estrogen (dogs), trichothecene mycotoxins, bracken fern, colchicine
Idiosyncratic: albendazole, fenbendazole, griseofulvin, phenylbutazone, trimethoprim-sulfadiazine, D-penicillamine
Whole body irradiation
Myelophthisis
Immune-mediated aplastic anemia (including bovine neonatal pancytopenia)
Gelatinous transformation of bone marrow (severe malnutrition)
Anti–G-CSF antibodies caused by recombinant human G-CSF (dogs)
Ineffective production
Precursor-targeted IMN
Phenobarbital-induced cytopenias
Severe cobalamin deficiency
G-CSF deficiency (possibly)
Myelodysplastic neoplasms
Other, unknown, or multiple mechanisms
Trapped-neutrophil syndrome (Border collies)
Toxicants: zonisamide (suspected), lithium carbonate (cats), *Cuscuta capestris* (horses)
[illegible]yclic neutropenia) (mostly grey collies or their crosses)
[illegible]d with FeLV
G-CSF administration (acute and transient)

PHASE 2 (HAEM) – DDx NEUTROPENIA

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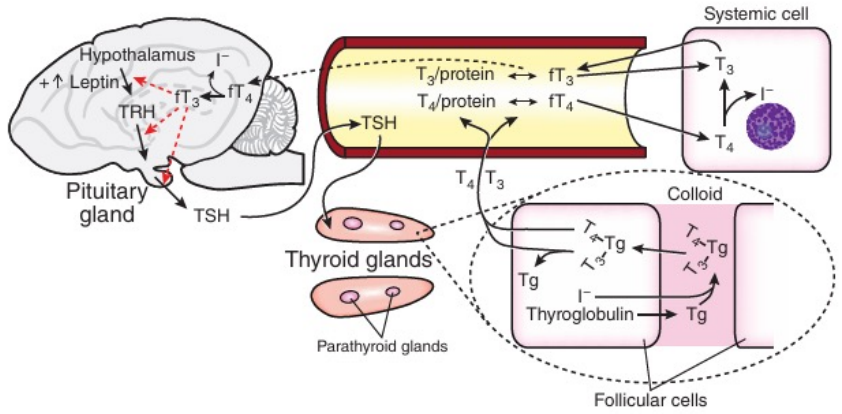


Figure 17.1. Hormonal control of thyroid glands.

- TRH released from the hypothalamus stimulates the release of TSH from the pituitary gland.
- TSH stimulates the production and release of T_4 and T_3 in pathways involving thyroglobulin in the thyroid glands.
  - Thyroglobulin is synthesized in follicular cells and is released into the luminal colloid by exocytosis.
  - Iodide (I^-) is transported from blood into the follicular cells via a Na^+/I^- symporter, and then into the colloid where it is oxidized to reactive iodine species by hydrogen peroxide and thyroid peroxidase on apical membranes of follicular cells. The reactive iodine species then rapidly bind to tyrosyl residues of thyroglobulin, a process called *organification* of thyroglobulin. Iodinated tyrosyl groups join together to form about 30 T_4 and few T_3 units within each thyroglobulin molecule. Thyroglobulin is stored within thyroid follicles, providing enough hormone for weeks to months.
  - The iodinated thyroglobulin enters follicular cells by pinocytosis of colloid, and the pinocytic vesicles fuse with lysosomes containing proteases that free T_4 and T_3 molecules from some of the thyroglobulin molecules.
- T_4 and T_3 are released from follicular cells and diffuse to blood via a rich network of fenestrated capillaries. Iodine within thyroglobulin tyrosyl groups that do not form T_4 and T_3 is recycled by a deiodinase enzyme. In blood, most T_4 and T_3 molecules are protein bound, but fT_4 and fT_3 can enter systemic cells where some of the T_4 is converted to T_3. T_3 has more activity than T_4. In the negative-feedback system (*red dashed lines*), fT_3 produced locally from fT_4 inhibits the secretion of TSH and TRH. Leptin stimulates the release of TRH; conditions that lower leptin concentrations inhibit TRH secretion.

3. Physiologic variations in [tT_4] have been reported. Age-related differences are addressed along with causes of hyper- and hypothyroxinemia (see Thyroxine (T_4) Concentration,

Chapter 17

PHASE 1 – THYROID (PATHO)PHYSIOLOGY

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B. Most capillary walls have minimal permeability to plasma proteins, but some proteins do enter the interstitium and the resultant lymph. The proteins in the interstitial fluid create an extravascular oncotic pressure (colloid osmotic pressure) (see Chapter 7, Colloid Osmotic Pressure).
   1. Oncotic pressure in the interstitium of skeletal muscle is about 30 % of the plasma oncotic pressure in health.[26]
   2. Oncotic pressure in the hepatic interstitium is nearly equal to plasma oncotic pressure because the walls of hepatic sinusoids are fenestrated and permeable to most plasma proteins.[26]
   3. Oncotic pressure in the pulmonary interstitium is about 70 % of plasma oncotic pressure. Alveolar capillaries are less permeable to proteins than are the hepatic sinusoids but more than are the skeletal muscle capillaries.[26]

C. The permeability of capillaries is expressed as a reflection coefficient (Eq. 19.1), which ranges from 0 (completely permeable to proteins) to 1 (impermeable to proteins). The reflection coefficient approaches 1 in most capillary beds but is smaller in hepatic and pulmonary capillary beds.

D. For most tissues, the extravascular fluid has a protein concentration much less than a plasma protein concentration, but the concentrations of other solutes (electrolytes, urea, Crt, glucose, and most small molecules) are nearly the same as those found in plasma. Minor differences in electrolyte concentrations are created by the Gibbs–Donnan effect (see Chapter 7, Colloid Osmotic Pressure, I.C.2).

E. There are "safety factors" that reduce or prevent the accumulation of extravascular fluid when there are changes in either hydraulic pressures or oncotic pressures.[26]
   1. Drainage by lymphatic vessels increases.
   2. When more protein-poor fluid enters the extravascular space because of increased hydraulic pressure, that fluid dilutes the extravascular protein concentration and thus increases the oncotic pressure gradient, promoting less fluid extravasation. Addition of extravascular fluid also increases extravascular hydraulic pressure and thus lowers the hydraulic pressure gradient.
   3. A reduction of plasma oncotic pressure (due to hypoproteinemia) may, if not too severe, be matched in time by a lowering of the extravascular protein concentration such that the oncotic pressure gradient does not change.

F. Extracellular fluid (ECF) spaces: When an animal has a cavitary effusion, it has an increased amount of extracellular fluid. It is useful to remember the three "spaces" of ECF.
   1. First space: intravascular fluid (plasma)
   2. Second space: extravascular interstitial fluid (extravasation of plasma fluid to cause interstitial edema is a second-space loss of fluid)

PHASE 1 – PATHOPHYSIOLOGY (EFFUSION FORMATION)

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adult cat intervals were about 1.8–6.4 mg/dL. A corresponding age-related difference is that these young animals have greater serum alkaline phosphatase activity than mature animals (usually < 3 times the URL for adults).

II. Analytical concepts of [Pi]
  A. Sample
    1. Serum or heparinized plasma may be used.
    2. In vitro hemolysis or delayed removal of serum from a blood clot may increase serum or plasma [Pi] because PO_4 is liberated from RBCs.[29]
  B. Assays
    1. In most common assays, PO_4 reacts with ammonium molybdate to form a colored complex that is measured by photometry.
    2. In some systems, conjugated bilirubin may interfere with the assay to produce falsely decreased results (*pseudohypophosphatemia*),[228] whereas, in other assay systems, bilirubin has no effect or may produce falsely increased values (*pseudohyperphosphatemia*).
    3. In spectrophotometric assays that use the strongly acidic ammonium molybdate reagent, the reagent can precipitate certain immunoglobulins to produce a turbidity that results in a falsely increased [Pi] by 1–2 mmol/L (≅ 3–6 mg/dL).[229,230] This positive interference has been seen in samples with monoclonal gammopathies despite assay modifications such as the addition of detergent to prevent protein precipitation.
  C. Methods of expressing concentrations
    1. [Pi] is often referred to as the phosphorus concentration. More accurately, [Pi] is the serum PO_4 reported as phosphorus in mg/dL. It is not reported as a PO_4 concentration in mg/dL because the amount of each form of PO_4 changes with pH, and each form has a slightly different relative molecular mass. This problem is overcome by reporting PO_4 in SI units of mmol/L (the number of molecules per volume rather than mass of molecules per volume).
    2. Relationship between [PO_4] and [Pi]
      a. 1 mmol of $H_2PO_4^-$ weighs 97 mg. In 1 mmol of $H_2PO_4^-$ there are 31 mg of P. Therefore, a solution of 97 mg/dL of $H_2PO_4^-$ contains 31 mg/dL of P.
      b. 1 mmol of HPO_4^{2-} weighs 96 mg. In 1 mmol of HPO_4^{2-} there are 31 mg of P. Therefore, a solution of 96 mg/dL of HPO_4^{2-} contains 31 mg/dL of P.
      c. Because of the equilibrium among the different PO_4 molecules at a pH of 7.4, 1 mmol of PO_4 averages about 96.2 mg/dL.
  D. Unit conversion for [Pi]: mg/dL × 0.3229 = mmol/L (SI unit)[31]

Chapter 11

PHASE 2 (BIOCHEM) – ANALYTICAL CONCEPTS

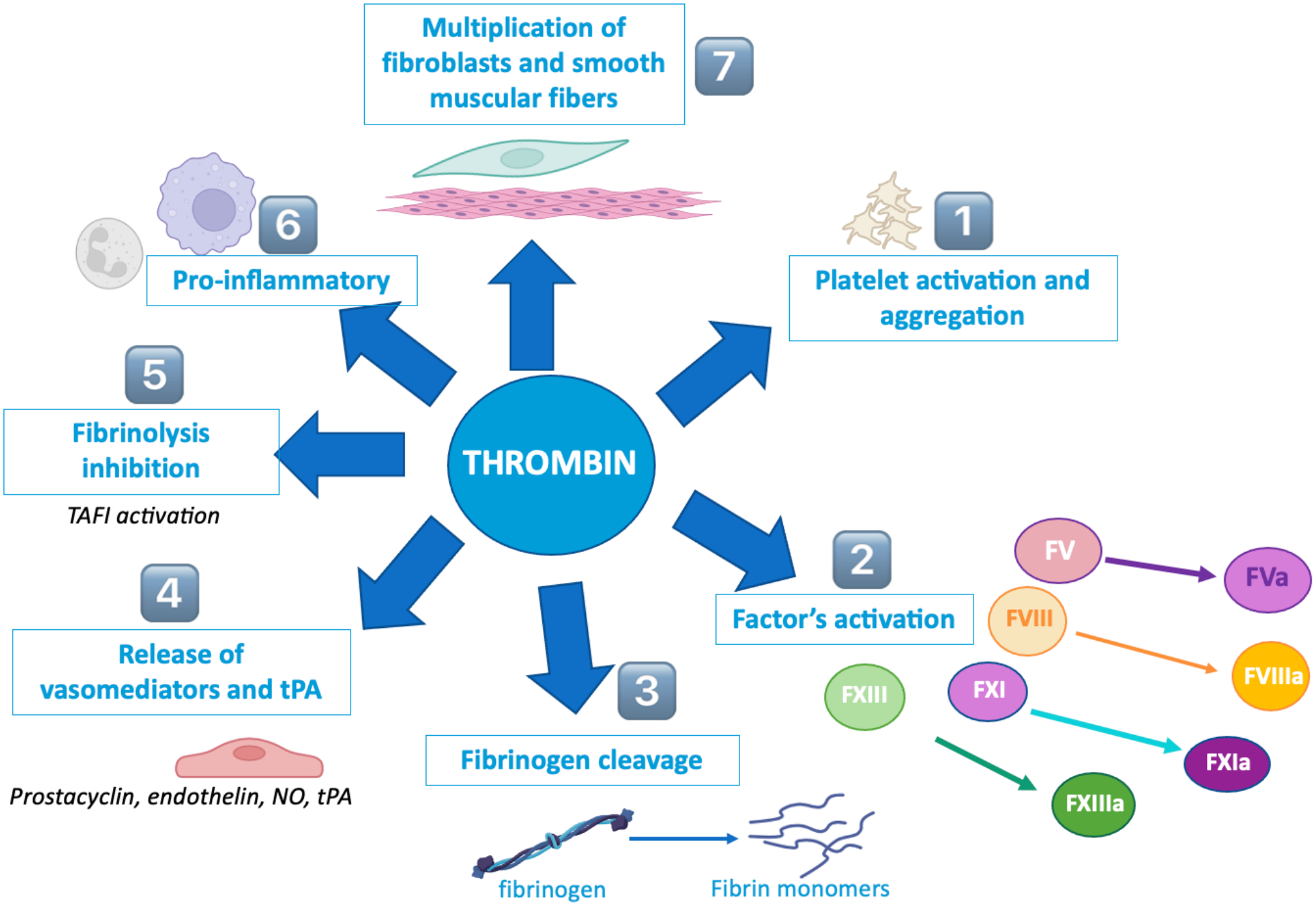
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Learning how to learn

Do I really know myself?



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Multiplication of fibroblasts and smooth muscular fibers
7
6
Pro-inflammatory
1
Platelet activation and aggregation
5
Fibrinolysis inhibition
TAFI activation
THROMBIN
2
Factor's activation
FV
FVa
FVIII
FVIIIa
FXIII
FXI
FXIa
FXIIIa
4
Release of vasomediators and tPA
Prostacyclin, endothelin, NO, tPA
3
Fibrinogen cleavage
fibrinogen
Fibrin monomers

WHAT YOU'VE JUST EXPERIENCED HAS A NAME:

Metacognition



Knowledge component

- Oneself as a learner
- How the brain works

Effective learning strategies

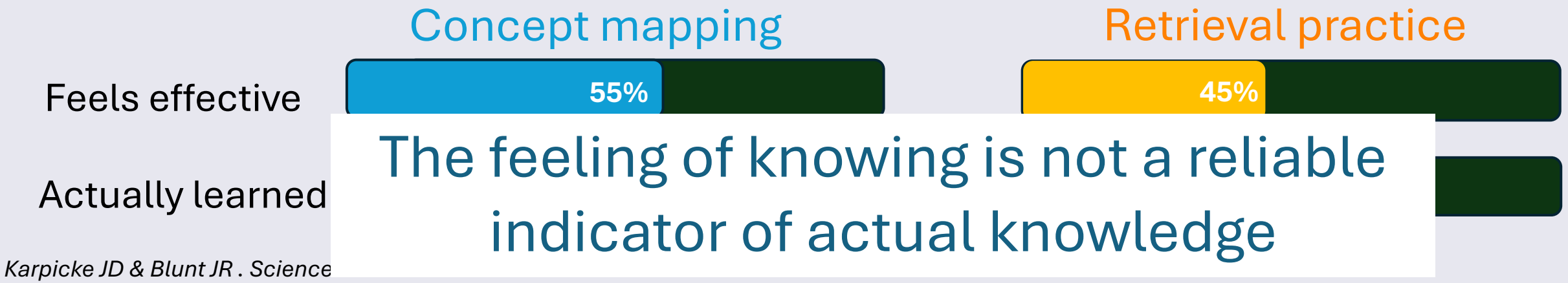
Regulatory component

- Planification (before)
- Monitoring (during)
- Evaluation (after)

Improvement of metacognitive skills <=> academic success

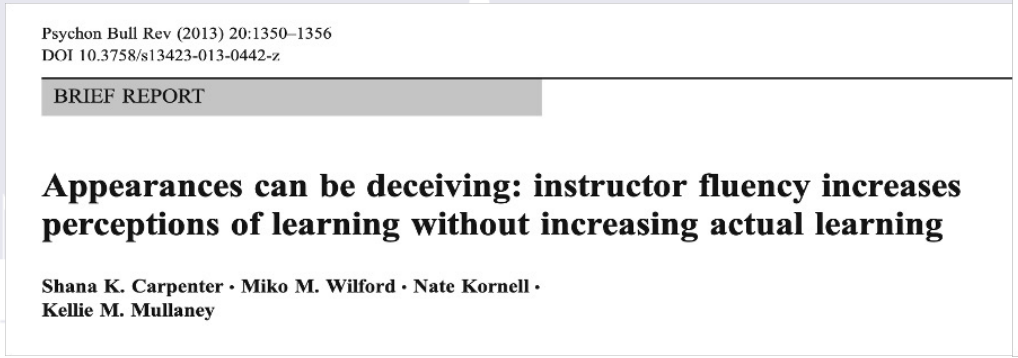
WHY WERE SOME OF YOU CONFIDENT (OR NOT)…AND WRONG?

The illusion of learning



The feeling of knowing is not a reliable indicator of actual knowledge

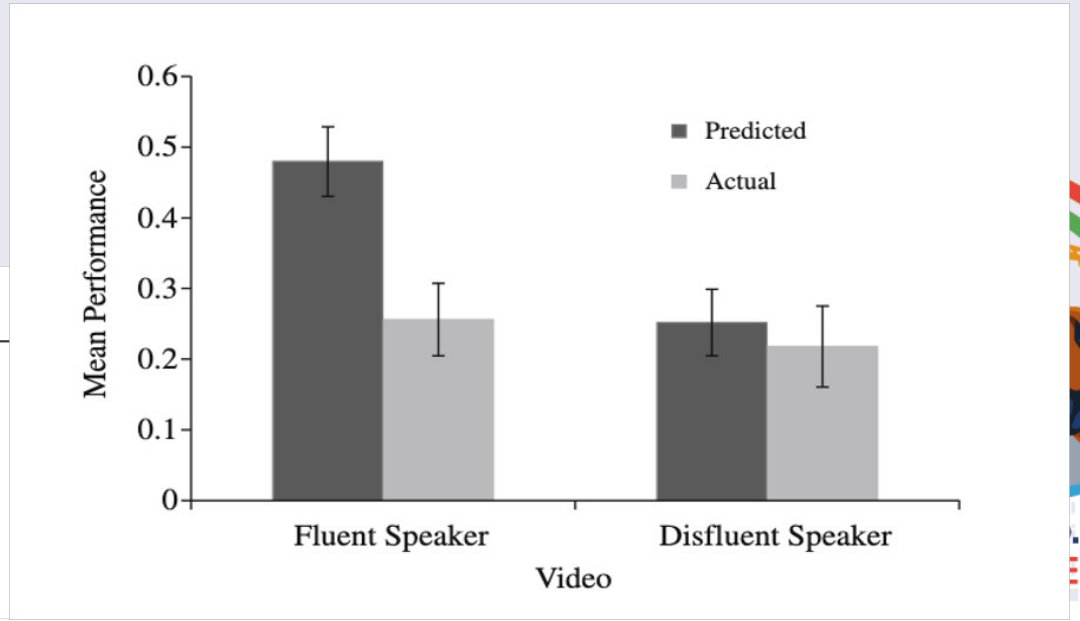
Karpicke JD & Blunt JR . Science


Psychon Bull Rev (2013) 20:1350–1356
DOI 10.3758/s13423-013-0442-z

BRIEF REPORT

Appearances can be deceiving: instructor fluency increases perceptions of learning without increasing actual learning

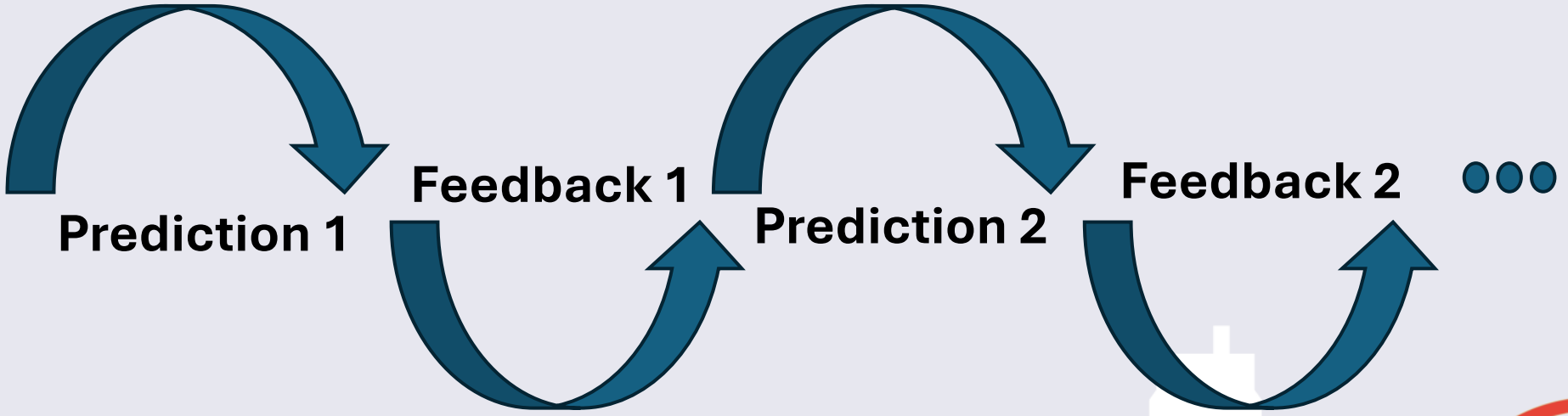
Shana K. Carpenter · Miko M. Wilford · Nate Kornell · Kellie M. Mullaney



THE MECHANISM BEHIND THE ERROR

We learn ONLY if we make mistakes

Your brain constantly learns by predicting and correcting



Making mistakes while practising is the mechanism, not an accident.

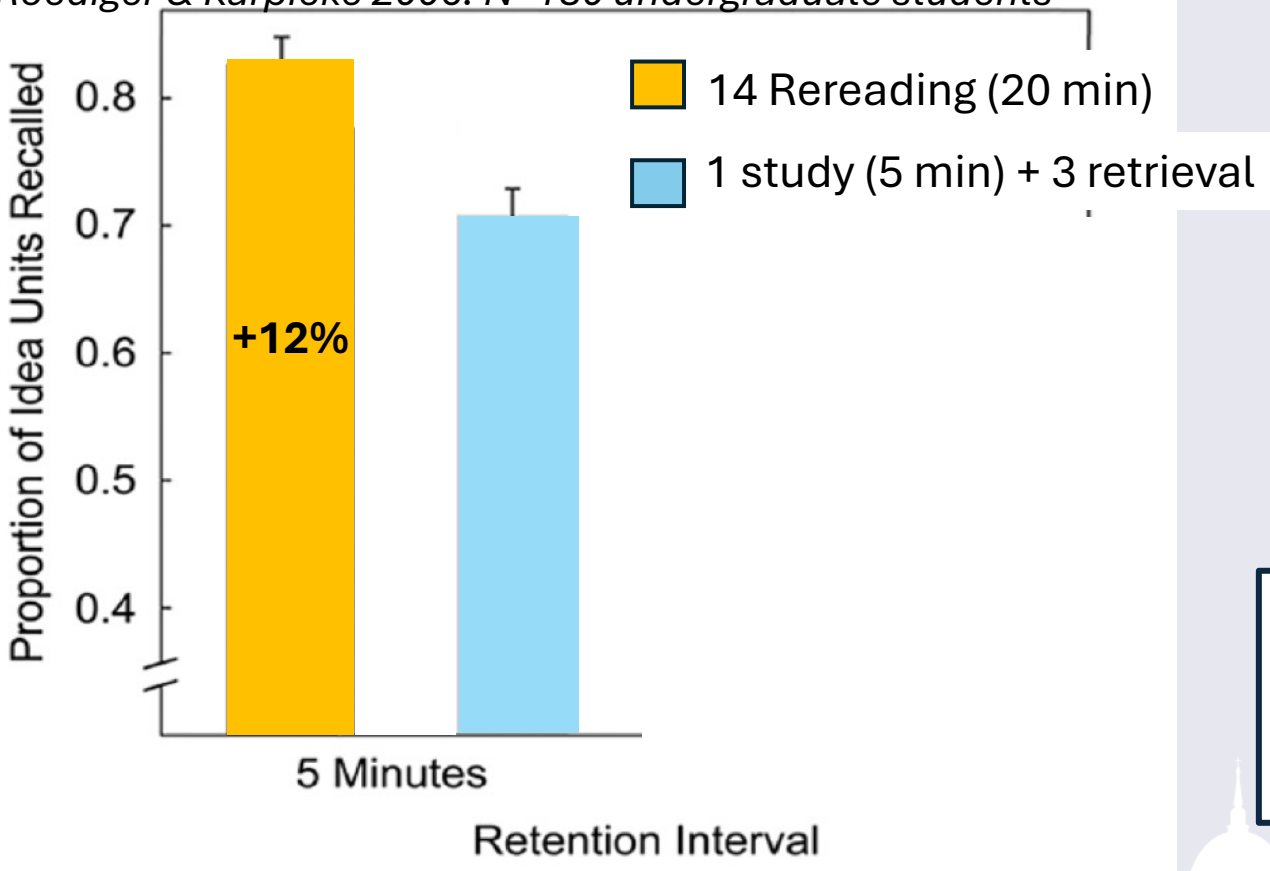
Stahl AE, Feigenson L. Science. 2015.
Clarck A. Behav Brain Sci. 2013
Dehaene S. 2018

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ACTIVE RETRIEVAL >> PASSIVE REREADING

Retrieval beats rereading, even when it feels worse

Roediger & Karpicke 2006. N=180 undergraduate students



Passive rereading	Active retrieval
Feels easy	Feels difficult
Creates familiarity	Reveals what you can retrieve
Can create false confidence	Strenghens long-term retention

Before rereading a topic studied weeks ago, try to recall it first. It may feel difficult but that difficulty is not failure!

Bjork & Bjork, 2011

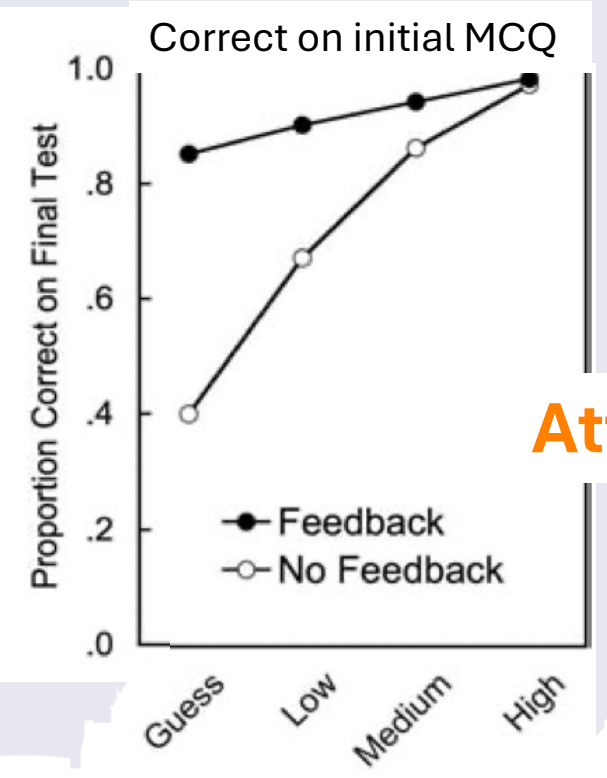
Rereading can improve immediate performance but repeated retrieval produces more durable learning.

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FEEDBACK INCREASES LEARNING RETENTION

Feedback closes the loop

Self-testing creates a prediction → **Feedback reveals the gap and turns it into learning** → **Retrieval after feedback makes the correction durable**



Butler AC, Karpicke JD, Roediger HL. 2008. J Exp Psychol. N=30 students

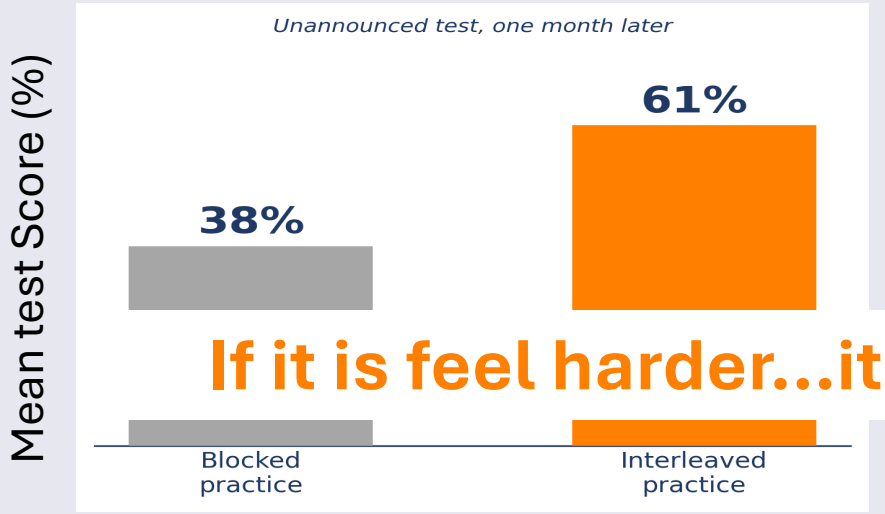
Feedback is not only for wrong answers.
It stabilizes correct answers that learners are unsure about.

Attempt first, check feedback, and retrieve again later

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MIXING SUBJECT EVEN WHEN IT FEELS HARDER

Distributed and mixed practice > massed practice



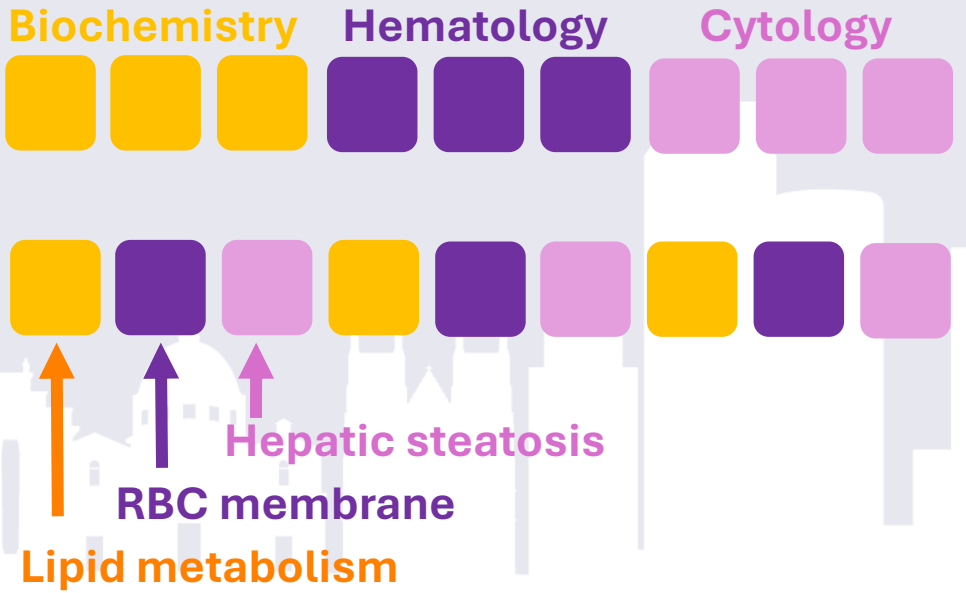
- N=787 7th grade children
- 4 mathematics problems studied for 4 months
- Either by blocked either by interleaved practice

Rohrer D et al. 2019. J Educ Psy.

If it is feel harder...it's probably working better

Massed/blocked practice

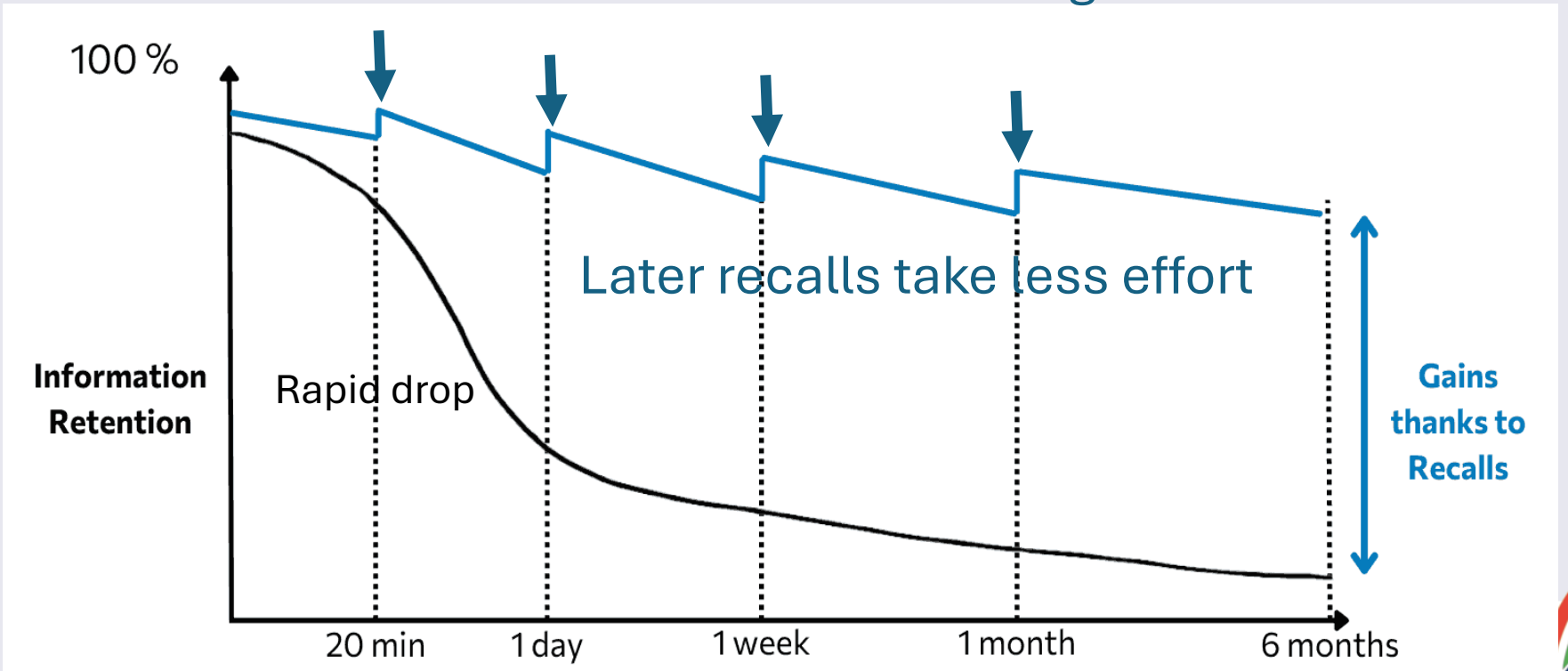
Distributed/Interleaved practice



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The forgetting curve and the spacing effect

Each recall resets the curve to a higher level



Spacing beats cramming right before the exam

Making sense of a 2-phase exam

Ebbinghaus 1885

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Can I improve my memory?

First, let's retire a myth: your memory isn't "visual" or "auditory."
The VAK/VARK learning-styles model is a neuromyth, not supported by evidence.



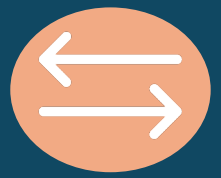
Mnemonics / Acronyms

Turn a dry list into a memorable phrase or word



Method of loci / Memory palace

Associate each piece of information with a real or imagined place



Elaborative encoding

Link new information to an image or something you already know

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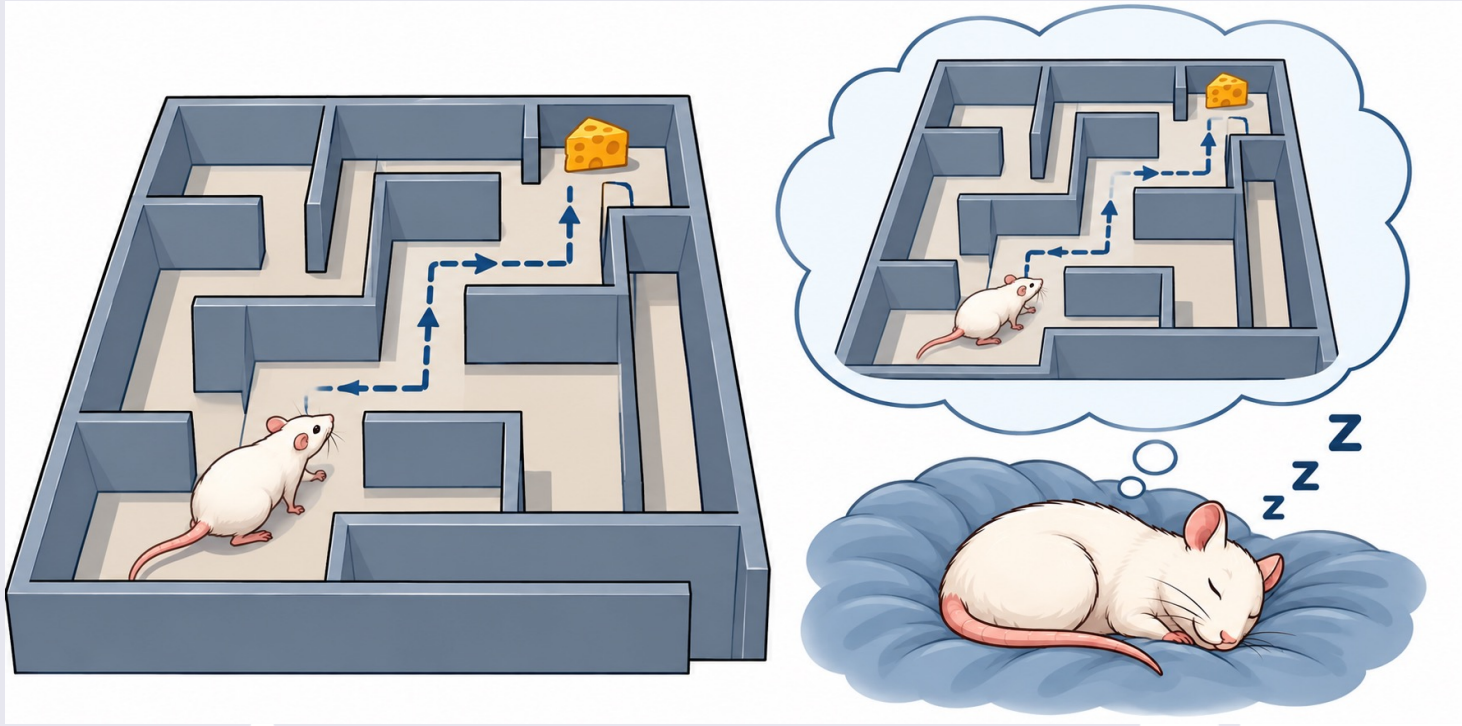
Example of a memory palace:



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SLEEP HELPS CONSOLIDATE WHAT YOU PRACTISED

The last but not least: SLEEP!!!



Sleep is an active phase of consolidation

Skaggs & McNaughton. 1996. Science

Exam blueprint

SOURCE	% OF EXAM
Textbook	65-75
Journal	25-35

SPECIES	% OF PHASE 2 EXAM
Non-species specific	20-25
Canine	25-30
Feline	20-25
Equine	10-15
Farm animal	5-10
Exotics	5-10
Laboratory animal	5-10

GCP TOPIC	% OF PHASE 1 EXAM
Lab management and Quality	25-30
Mechanisms of disease	35-40
Pathophysiology	35-40

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Vital Resources

- ECVCP 2026 Reading List
https://www.ecvcp.org/sites/www.ecvdi.org/files/medias/documents/ECVCP/2026%20ECVCP%20Board%20Examination%20Reading%20List.pdf

- ECVCP Information Brochure, Section 5.3
https://www.ecvcp.org/sites/www.ecvdi.org/files/medias/documents/ECVCP/ECVCP%20Information%20Brochure%20March%202024.pdf



European College of Veterinary Clinical Pathology

5.3 The Format of the Examination

The Examination is designed to test Candidates'.

(a) Understanding of the broad principles of Clinical Pathology;

(b) Ability to competently practice Veterinary Clinical Pathology; and

(c) Ability to synthesise data and observations in problem solving.

The Examination is divided into two phases:

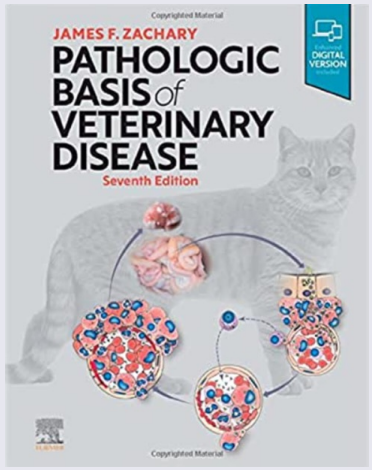
Phase 1: Examination in General Clinical Pathology **(Duration: 2 hours)**

General Clinical Pathology: This section examination tests the Candidate's understanding of the pathophysiological bases of clinical pathology changes and general principles of laboratory management. It will consist of **100** multiple choice questions (MCQ), comprising a mixture of those testing knowledge, and those testing interpretative ability and understanding. Some questions may include short data sets, graphs, diagrams or tables. Questions are focused on:

(a) Pathophysiological mechanisms responsible for haematological, biochemical or cytological changes; and

(b) Laboratory management, including, for example, method and technology validation, good laboratory practice, quality control, laboratory statistics and reference intervals, basic epidemiology, evaluation of diagnostic efficacy and efficiency.

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Top tips for tackling Zachary

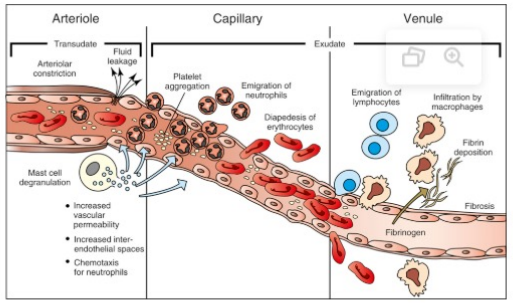


FIGURE 3.2 The Primary Vascular and Cellular Responses during Acute Inflammation. The majority of these responses occur in capillaries and postcapillary venules.

ESSENTIAL CONCEPT 3.1 Acute Inflammation

Acute inflammation is a vascular response to cell and tissue injury triggered by numerous physical and biologic stimuli. In affected tissue, its objectives are (1) to kill and/or eliminate the cause of injury (e.g., microbes, foreign material, thermal, radiation), (2) to phagocytose and remove debris, and (3) to repair the damage, thereby returning the tissue to normal structure and function. Acute inflammation progresses chronologically in sequence through three phases: fluidic, cellular, and reparative. The purpose of the fluidic phase is to dilute, surround (i.e., isolate), and contain (i.e., trap) the stimulus and the damage, thereby limiting the extent of involvement of adjacent normal cells and tissue. It is caused by direct or indirect injury of microvessels

- Don’t lose sight of the titles and subtitles: provide the overview
- Focus on the “Essential Concepts” boxes and Figures
- Always consider clinical implications to determine which details are relevant
- MCQs WON’T be taken from E-tables/supplements
- Repetition vital

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A dog with acute bacterial pneumonia has neutrophils accumulating within pulmonary microvessels. Molecular analysis shows markedly impaired neutrophil integrin activation.
Which step of leukocyte recruitment is most directly impaired?

A. Rolling
B. Margination
C. Firm adhesion
D. Transendothelial migration

Ref: Zachary, 7th ed., Chapter 3: Inflammation and Healing

Classic FACT RECALL-type question

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Two strains of the same bacterium are administered experimentally to identical groups of dogs.
Which conclusion is best supported by these data?

Finding	Strain A	Strain B
Infectious dose required	Low	High
Tissue invasion	Extensive	Minimal
Mortality	80%	5%
Toxin production	High	Low

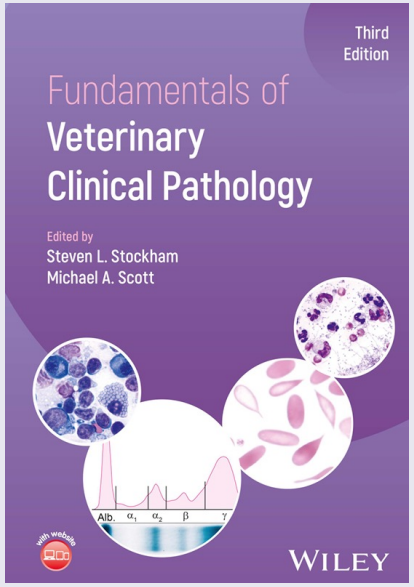
A. Strain A has greater virulence
B. Strain B has greater infectivity
C. Strain A produces less tissue injury
D. Strain B produces greater host susceptibility

Ref: Zachary, 7th ed., Chapter 3: Inflammation and Healing

INTERPRETATIVE-type question

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Top tips for tackling Stockham



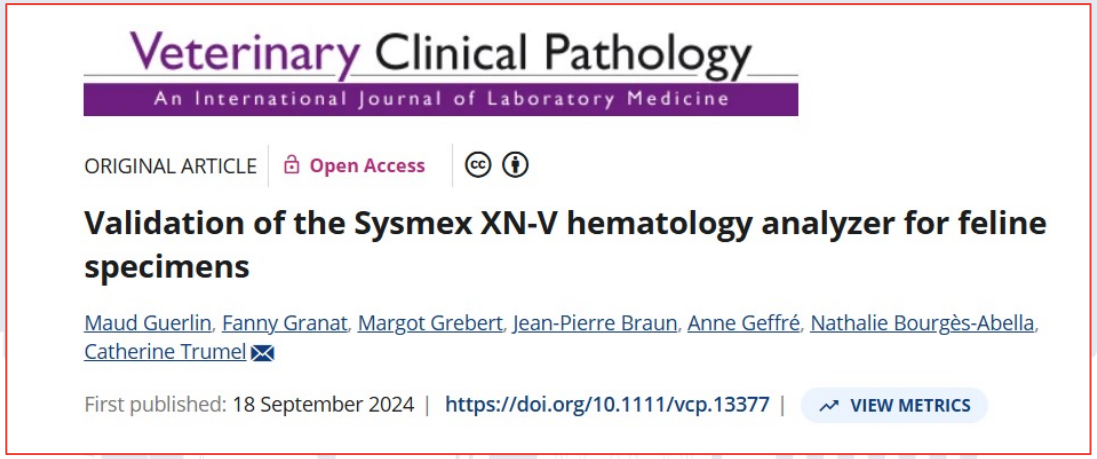
- Intimate familiarity required
- Constantly consider whether the information describes the causes of a change in a test result, or the interpretation of that change
- For Phase 1, focus on early parts of each chapter (in general!)
  - i.e. Physiologic concepts
- For Phase 2, learn the hypo/hyper tables

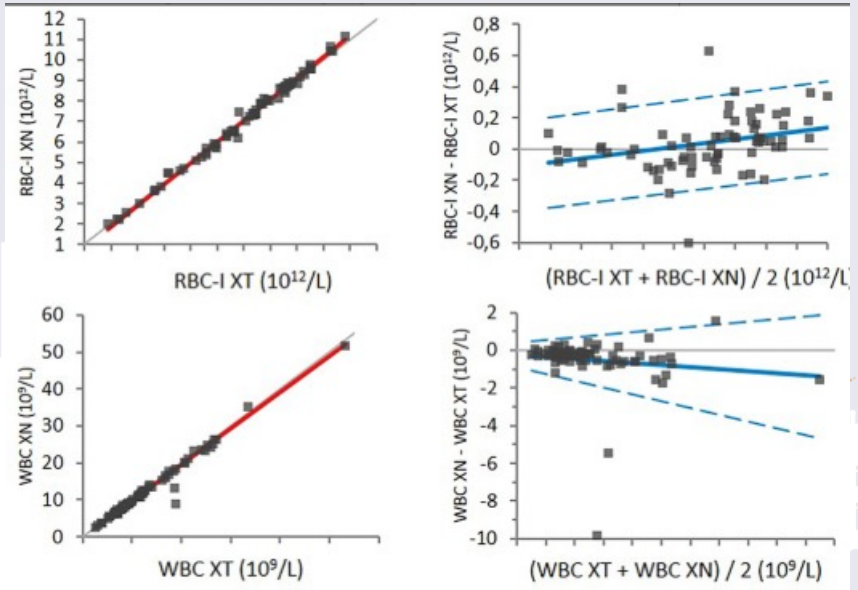
Causes/Mechanisms = Phase 1
Interpretation = Phase 2

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Top tips for tackling journals

- >90% questions taking from core reading list: VCP!
- Avoid single articles demonstrating an esoteric new finding that's not part of a wider trend of research
- Pay attention to reviews
- Focus on the key "take-home" message
- Consider whether an article may invoke Quality questions





Top tips for Phase 2 – all sections

- Attend **Summer School** mock exam (every 2 years – next one 2027)
- Practice at **speed**, including "marathon sessions"
- Unfamiliar topics – exotics, tox path etc.: **apply first principles**
- **Breed specifics**
- Get advice from **recent candidates**

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Top tips - Haematology



Rigid adherence to **systematic** evaluation of **blood films and bone marrows**

Don’t forget **quantitative estimations** of any change

- Estimated differential count
- ‘Mild/moderate/marked’

Focus on **less familiar species** (Schalm species-specific chapters)

Practice **dot plot analysis** for the principal analysers

Practice **TEG interpretation**

Papers: species-specific publications and method validation studies

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In cats, Heinz body haemolytic anaemia can cause which error when the CBC is obtained with the ADVIA 2120?

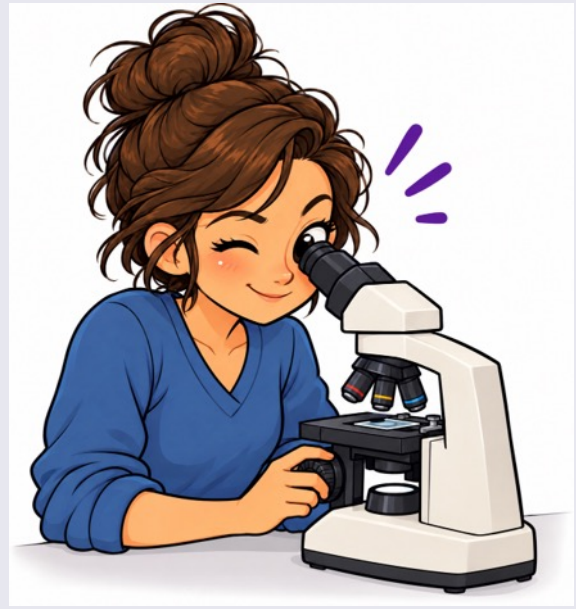
A. Decreased MCHC
B. Increased haematocrit
C. A marked spurious leukopenia
D. An erroneous BASO WBC count

Reference: Spurious, marked leukocytosis in 2 cats with Heinz body hemolytic anemia Courtney E. Johnson, Davis M. Seelig, Frances M. Moore, Tammy J. Ruska, Daniel A.Heinrich. Pages: 232-239. First Published: 26 May 2020, https://doi.org/10.1111/vcp.12860

Classic FACT RECALL-type question

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Top tips - Cytology



Don’t neglect technical aspects:

- **Special stains** and ICC antibodies
- Microscope set-up: **Raskin final chapters**
- Principles of flow cytometry, ICC, PARR, cell block techniques

Don’t forget unfamiliar tissues – **Raskin chapters on:**

- Reproductive
- Neuroendocrine
- CNS and eye

What level of anatomic histo is relevant?

- Consider what is useful for a cytologist to know
- What cells where? What tumours where?

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A tongue FNA from a dog reveals the features below. What is the most likely interpretation?

A. Liposarcoma
B. Rhabdomyoma
C. Clear cell carcinoma
D. Granular cell tumour

Cytology	• Polyhedral cells, individualised or in groups • Abundant cytoplasm with clear vacuoles • Moderate anisocytosis and anisokaryosis, multinucleation
Special staining	PAS positive, Oil red O negative
Immunohistochemistry	Demin positive, Cytokeratin negative

Reference:Sebastian, K. N., Magestro, L., Kiupel, M., & Radin, M. J. (2021). What is your diagnosis? Lingual mass aspirate from a dog. *Veterinary Clinical Pathology*, *50*(4), 615–617. https://doi.org/10.1111/vcp.12985

INTERPRETATIVE-type question

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Top tips – Glass slides

Practice low power scanning to gain speed and return to lower power before leaving a slide

"What have I missed?" "What else could it be?"



Practice at speed, including "marathon sessions"

Paste template in Examsoft (don't waste time formatting)

If you're struggling with a case: Description!

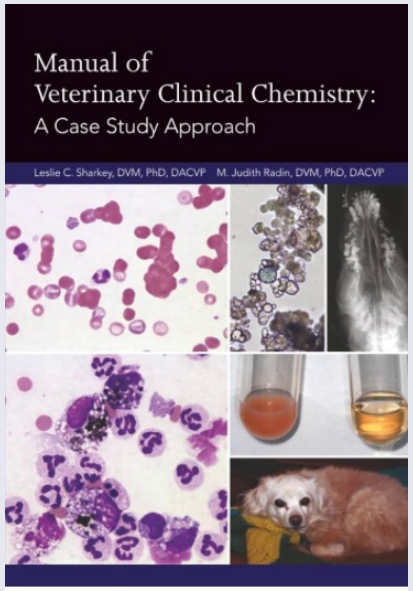
Haem: Scan whole slide – microfilaria, platelet clumps, agglutination etc.

Cyto: Don't forget:

- Background
- Cellularity and **preservation**
- Nucleoli – more marks than you might expect
- Organisation – 1 pt for quality of answer

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Top tips - Biochemistry



Practise case write-ups at speed: Sharkey LC, Radin J (2010). Manual of Veterinary Clinical Chemistry: A Case Study Approach. Teton Newmedia.

For cases, consider **your target audience: clinicians**

When listing DDx for a change, must **highlight the most important**

Summarise the main findings in your interpretation

Don't panic if not all aspects of a case fit perfectly

- Real cases
- Discuss comorbidities and unexpected results

Familiarise yourself with **toxpath terminology** ("reduced RBC mass", not "anaemia")

For tox path and exotics: **first principles**

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Signalment: 9-year-old female entire cocker spaniel.
Presenting complaints: vomiting, diarrhoea, weight loss, and icterus.
What is the most likely cause of these laboratory findings?

Analyte	Value	Reference Ranges
Albumin (g/dL)	3	2.5-4.5
Globulin (g/dL)	**4.2**	1.5-3.5
Total Bil (mg/dL)	**6**	0-0.3
ALP (IU/L)	**880**	10-130
ALT (IU/L)	**346**	15-45
AST (IU/L)	**302**	30-260
Cholesterol (mg/dL)	**601**	130-350

A. Hepatic insufficiency
B. Hyperadrenocorticism
C. Cholestatic hepatopathy
D. Acute hepatocellular necrosis

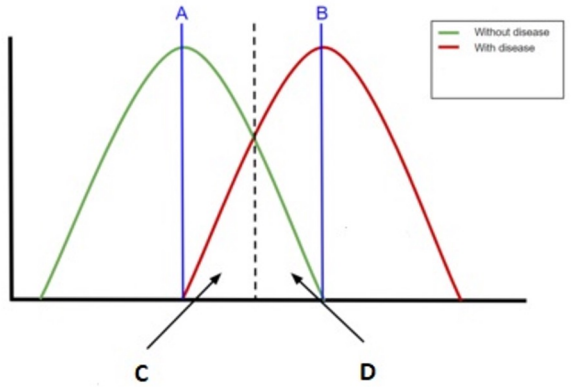
Reference: Stockham SL, Scott MA. Enzymes In: "*Fundamentals of Veterinary Clinical Pathology*". Ames, IA: Iowa State Press; 2008: 650-658, 690-695 and 770.

INTERPRETATIVE-type question

Multi-stage interpretative

17. This histogram illustrates distribution of test results for populations with and without a disease. A and B are proposed cutoff values for disease diagnosis. What do A, B, C and D represent?
X axis = concentration of measurand. Y axis = frequency of results.

A. A = 100% sensitivity, B = 100% specificity, C = false negatives, D = false positives
B. A = 100% specificity, B = 100% sensitivity, C = false negatives, D = false positives
C. A = 100% sensitivity, B = 100% specificity, C = false negatives, D = false positives
D. A = 100% specificity, B = 100% sensitivity, C = false positives, D = false negatives



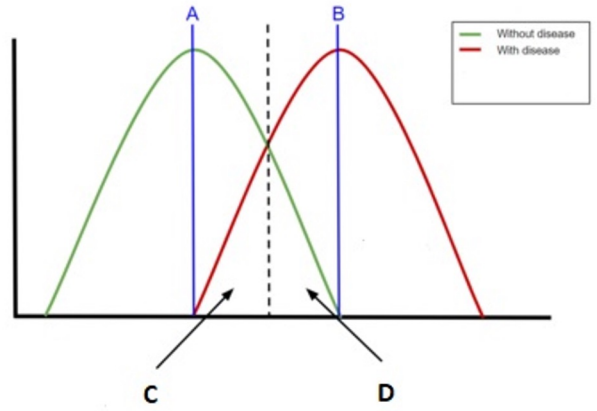
Multi-stage INTERPRETATIVE-type question

18. The vertical dashed line is the test cut off point (results to the right of this are deemed positive). If this is moved to the RIGHT, what effect would this have on sensitivity, specificity, and the number of false positive test results?

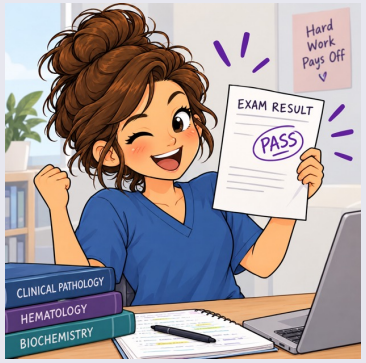
A. Specificity increases, sensitivity decreases, and false positives increase
B. Specificity decreases, sensitivity increases, and false positives increase
C. Specificity increases, sensitivity decreases, and false positives decrease
D. Specificity decreases, sensitivity increases, and false positives decrease

19. In which scenario would it be appropriate to move the test cut off point to the RIGHT?

A. The prevalence of the disease is high
B. The treatment has significant side effects
C. The treatment is cheap and relatively harmless
D. The consequences of missing a diagnosis are severe



Reference: Bellamy JEC, Olexson DW (2000). Quality Assurance Handbook for Veterinary Laboratories, 1st ed. Wiley-Blackwell. p. 71-3



Conclusions

2 phase-exam will help you to get (even) better. And you are now ready to tackle it!

Phase 1: Lab management and Quality, Pathophysiology and Mechanisms of Diseases

Phase 2: Applied knowledge, analytical methodology, test interpretation, diagnostic reasoning and practical skill

Practice and learn regularly by actively retrieving. And sleep!

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THANK YOU!

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Any questions?



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