Complete Cardiology Course Handbook - All 12 Lessons

Course 03 · Cardiovascular, Vascular and Hematology · October 2026

Contents: 12 core lessons · clinical cases · questions to consider · recommended guideline links. 120 original MCQs are available in the linked website question bank.

Lesson 01 | Cardiovascular Assessment, ECG and Diagnostic Reasoning

Focused cardiac history, examination, 12-lead ECG patterns, biomarkers and investigation selection.

Focused history and differential diagnosis

• Define the presenting syndrome: chest discomfort, dyspnea, palpitations, syncope, edema, fatigue or reduced exercise tolerance; establish onset, duration, triggers and associated symptoms.

• Assess immediate instability: airway, breathing, circulation, mental status, vital signs, pulse oximetry and signs of poor tissue perfusion.

• Clarify cardiac risk: prior CAD, hypertension, diabetes, dyslipidemia, smoking, kidney disease, family history, stimulant exposure, pregnancy status and current medication use.

• Separate ischemic, arrhythmic, structural, pulmonary, infectious, gastrointestinal and musculoskeletal causes; normal examination does not exclude life-threatening illness.

Cardiac examination and red flags

• Record bilateral pulses when indicated, jugular venous pressure, cardiac murmurs, gallops, peripheral temperature and edema; listen for lung crackles.

• New severe pressure-like chest pain, syncope during exertion, shock, new focal deficit or acute severe breathlessness warrants immediate emergency assessment.

• A new murmur with fever, embolic features or recent invasive procedures raises concern for infective endocarditis.

• Orthopnea, elevated JVP, crackles and edema support congestion; unilateral leg swelling with sudden dyspnea raises pulmonary embolism concern.

12-lead ECG fundamentals

• Check calibration and acquisition, rate, regularity, P waves, PR interval, QRS duration, axis, QT/QTc and ST-T changes.

• Identify atrial fibrillation by an irregularly irregular rhythm without consistent discrete P waves; consider artifact and other irregular rhythms.

• Recognize STEMI-equivalent and ischemic patterns in the clinical context; a normal or non-diagnostic first ECG does not rule out ACS.

• A wide-complex tachycardia in an ill patient should be treated as ventricular tachycardia until proven otherwise by a trained emergency team.

• Long QT, high-grade AV block, pre-excitation, hyperkalemia changes and Brugada-pattern findings require appropriate urgent evaluation.

Testing and interpretation

• Use serial high-sensitivity troponin with a validated local protocol for suspected ACS; troponin elevation signals myocardial injury, not automatically type 1 infarction.

• BNP/NT-proBNP supports heart failure assessment but may rise with age, renal dysfunction or atrial fibrillation; low values can help make HF less likely.

• Echocardiography evaluates chamber size, ejection fraction, valve disease, wall motion and pericardial effusion; choose it for suspected structural disease.

• Ambulatory ECG monitoring can capture intermittent palpitations or syncope when a resting ECG is unrevealing; choose monitoring duration based on symptom frequency.

• Use clinical pretest probability to select stress testing, coronary CT angiography or specialist referral; avoid indiscriminate testing.

Decision documentation

• Record differential diagnoses and why major emergencies were considered or excluded, including time course and examination findings.

• Use shared decision-making to discuss benefits, limitations, false positives, downstream procedures and urgent return precautions.

• Do not defer emergency referral while waiting for outpatient blood tests or imaging if a patient has concerning symptoms.

Applied clinical case

A 59-year-old with diabetes reports 40 minutes of chest pressure, diaphoresis and nausea. BP 102/70, pulse 108; first ECG is non-diagnostic.

Model reasoning and disposition

High-risk possible ACS remains the leading concern. Activate emergency transfer and obtain time-sensitive ECG/serial troponin in a monitored setting; do not reassure based on the first ECG.

Self-check prompts

• Which conditions must be excluded first?

• Why is one normal ECG insufficient?

• What changes make an outpatient plan unsafe?

Selected guidelines (open online)

AHA/ACC — 2025 Acute Coronary Syndromes Guideline

Canadian Cardiovascular Society — Guidelines Library

Lesson 02 | Hypertension, Dyslipidemia and Cardiovascular Prevention

Evidence-based blood-pressure diagnosis, vascular risk modification and primary/secondary prevention.

Measurement and diagnosis

• Use an appropriately sized cuff and validated automated device, seated rest, supported arm and consistent standardized technique.

• Hypertension Canada 2025 recommends a standardized BP threshold of at least 130/80 mm Hg; confirm with out-of-office readings to detect white-coat or masked hypertension.

• Differentiate diagnosis threshold, medication initiation threshold and individualized treatment target; these are not identical.

• Consider secondary causes when onset is young, severe or resistant: renal disease, sleep apnea, primary aldosteronism, medications and endocrine causes.

Treatment and target

• Promote sodium reduction, regular activity, weight optimization, sleep health, smoking cessation and moderation of alcohol.

• Guideline pharmacotherapy is generally initiated at BP at least 140/90 mm Hg or at SBP 130–139 in high cardiovascular-risk adults, after clinical assessment.

• Aim for a tolerated systolic BP below 130 mm Hg as recommended in the 2025 primary-care guideline, with individual adjustments for frailty, orthostasis and adverse effects.

• Common first-line drug classes include thiazide/thiazide-like diuretics, ACE inhibitors/ARBs and dihydropyridine calcium-channel blockers; monitor electrolytes and renal function when relevant.

• Hypertensive emergency requires elevated BP with acute target-organ damage; severe BP alone without injury does not automatically define an emergency.

Lipids and risk assessment

• Review lipids, diabetes, smoking, renal function, family history, BP and existing atherosclerotic cardiovascular disease to estimate absolute risk.

• Document indications for statin therapy in established ASCVD and high-risk primary prevention; intensification depends on response and guideline thresholds.

• Discuss myalgias, adherence, pregnancy considerations, potential drug interactions and follow-up lipid measurements.

• Coronary artery calcium may refine risk in selected uncertain primary-prevention decisions; a zero score does not eliminate risk in all people.

Secondary prevention

• After atherosclerotic events prioritize evidence-based statins, smoking cessation, exercise/cardiac rehab, glucose/BP optimization and appropriate antithrombotic plans.

• Recognize that aspirin for primary prevention is not routinely indicated and can cause bleeding; review individualized benefits and harms.

• Assess drug interactions and possible blood-pressure or arrhythmia effects of stimulants, NSAIDs, decongestants, licorice and other complementary products.

Communication and follow-up

• Use home BP logs with date/time readings and a jointly agreed reassessment schedule; verify adherence before labeling resistant hypertension.

• Explain absolute risk reduction and the difference between treating a risk factor and treating a symptom.

• Build a realistic plan incorporating cost, access, culture, health literacy and personal preferences.

Applied clinical case

A 55-year-old with repeated clinic BP 151/92 mm Hg has no symptoms. Home monitoring averages 148/90; history includes smoking and diabetes.

Model reasoning and disposition

This is confirmed hypertension needing timely clinician-directed treatment and global CVD risk reduction; screen for target-organ damage, discuss medication plus lifestyle, and follow BP/labs.

Self-check prompts

• Is this a hypertensive emergency?

• How should home readings affect diagnosis?

• Which other risk factors should be treated?

Selected guidelines (open online)

Hypertension Canada — 2025 recommendations

Canadian Cardiovascular Society — Guidelines Library

Lesson 03 | Coronary Artery Disease, Acute Coronary Syndromes and Rehabilitation

Stable angina, ACS presentations, reperfusion principles and follow-up risk reduction.

Ischemic syndromes

• Classical angina is often exertional pressure relieved by rest; dyspnea, epigastric discomfort or fatigue may be ischemic equivalents.

• ACS includes unstable angina, NSTEMI and STEMI; new rest pain, crescendo symptoms or prolonged discomfort require emergency assessment.

• Differentiate acute coronary occlusion from pulmonary embolism, aortic dissection, pericarditis, pneumothorax and esophageal causes.

• Assess symptom onset, prior coronary interventions, antiplatelet use, anticoagulants and bleeding risk.

Initial emergency workup

• Prompt 12-lead ECG and serial assessment are essential; ST elevation or occlusive patterns mandate immediate reperfusion-pathway activation.

• Use serial high-sensitivity troponin in a validated algorithm; distinguish acute myocardial injury from type 1 MI using clinical and ECG evidence.

• Do not use a single normal troponin at symptom onset to rule out infarction.

• High-risk features include hemodynamic instability, ventricular arrhythmia, ongoing pain, acute HF and dynamic ischemic ECG changes.

Evidence-based treatment principles

• Emergency teams decide on reperfusion, antithrombotics, hemodynamic support and invasive strategy based on ECG, clinical instability and local protocols.

• Antiplatelet therapy, lipid lowering and risk-factor management are central after ACS; choice and duration of dual antiplatelet therapy depend on bleeding/ischemic risk.

• Check contraindications and bleeding risk before antithrombotics; do not administer unreviewed medication in undifferentiated chest pain with possible dissection.

• Avoid routine oxygen for normoxemic ACS patients; provide oxygen according to clinical need and current resuscitation standards.

Stable CAD and rehabilitation

• For stable symptoms use symptom assessment, probability-based investigation, antianginal management and referral when symptoms or findings are high risk.

• Cardiac rehabilitation includes progressive activity, medication education, tobacco cessation, nutrition support and psychosocial assessment.

• Recurrent chest pain after a recent MI, stent placement or bypass graft requires urgent reassessment; adherence to antiplatelets matters.

Communication

• Explain why time matters, what ECG/troponin can and cannot tell us, and why emergency transport is preferable to self-driving.

• Review safety-net symptoms after discharge: new prolonged chest pain, fainting, rapidly progressive dyspnea or severe palpitations.

• Use shared decisions and document bleeding-risk discussions around antiplatelet treatment.

Applied clinical case

A 67-year-old has 50 minutes of central chest pressure radiating to the left arm. ECG shows new ST elevation in contiguous leads.

Model reasoning and disposition

Presume STEMI until evaluated by the emergency team; activate the local reperfusion pathway immediately and avoid delays for nonessential investigations.

Self-check prompts

• Why does the ECG require urgent action?

• Which mimics alter immediate safety?

• What is the role of secondary prevention?

Selected guidelines (open online)

AHA/ACC — 2025 Acute Coronary Syndromes Guideline

Canadian Cardiovascular Society — Guidelines Library

Lesson 04 | Heart Failure, Cardiomyopathy, Myocarditis and Pericardial Syndromes

Congestive states, EF phenotypes, cardiac muscle disease, inflammatory injury and decompensation.

Diagnosis and phenotypes

• Heart failure is a clinical syndrome of congestion and/or inadequate cardiac output due to cardiac dysfunction; assess dyspnea, orthopnea, weight gain, edema and fatigue.

• Examine JVP, pulses, edema, lung findings and perfusion; obtain ECG, labs, natriuretic peptide where appropriate and echocardiography.

• Differentiate reduced EF from mildly reduced, preserved and improved EF; management options vary with EF and phenotype.

• Consider mimics and contributors: COPD, anemia, renal failure, obesity, thyroid disease, valvular disease and medications.

HFrEF evidence-based care

• The central disease-modifying drug classes for symptomatic HFrEF are an ARNI/ACEi/ARB pathway, evidence-based beta blocker, mineralocorticoid receptor antagonist and SGLT2 inhibitor when eligible.

• Diuretics relieve volume overload but do not replace the disease-modifying foundation; monitor renal function, potassium, BP and congestion.

• Assess need for specialist involvement, device therapy and cardiac rehabilitation; medication sequencing is individualized.

• Avoid drugs that may aggravate HF and monitor for adverse effects, including nonprescribed products that alter fluid balance or BP.

HFpEF and HF with EF above 40%

• Confirm objective evidence of congestion or elevated filling pressures; dyspnea with a normal EF does not by itself prove HF.

• Treat contributory hypertension, diabetes, sleep apnea, AF, ischemia and obesity where applicable.

• SGLT2 inhibitors are important evidence-based options in appropriate symptomatic HF with non-reduced EF under current Canadian guidance.

• Recognize frequent overlap of HF with CKD, pulmonary disease and frailty.

Cardiomyopathy and myocarditis

• Dilated cardiomyopathy may follow ischemic, genetic, toxic, metabolic or inflammatory injury; family history and echocardiography inform assessment.

• Hypertrophic cardiomyopathy may present with exertional syncope, dyspnea or family history of sudden death; avoid dismissing these as benign.

• Myocarditis may occur after viral illness with chest pain, dyspnea, palpitations and troponin elevation; urgent evaluation is needed for possible ventricular dysfunction or arrhythmia.

• Peripartum cardiomyopathy should be considered with new HF late in pregnancy or postpartum.

Pericardial syndromes and decompensation

• Pericarditis may cause sharp positional pleuritic pain and diffuse ST elevation; exclude ACS and evaluate for effusion.

• Tamponade may cause hypotension, distended neck veins, tachycardia and obstructive shock; urgent emergency management is necessary.

• Acute pulmonary edema, hypoxemia, altered mentation, hypotension or cool extremities signals possible decompensated HF/shock.

• Provide clear return precautions and arrange appropriate rapid reassessment following a change in symptoms or diuretic regimen.

Applied clinical case

A 46-year-old develops dyspnea, palpitations and chest discomfort 10 days after a febrile illness. Troponin is elevated and echo shows a new depressed EF.

Model reasoning and disposition

Possible myocarditis with acute ventricular dysfunction requires urgent cardiology/emergency assessment, rhythm monitoring and evaluation of alternative causes including ACS.

Self-check prompts

• How do injury and infarction differ?

• What findings suggest decompensated HF?

• Which drugs modify HFrEF outcomes?

Selected guidelines (open online)

Canadian Cardiovascular Society — 2025 Heart Failure Guideline

Canadian Cardiovascular Society — Guidelines Library

AHA/ACC — 2025 Acute Coronary Syndromes Guideline

Lesson 05 | Arrhythmias, Conduction Disease and Syncope

ECG-driven rhythm differential diagnosis, AF stroke prevention and unstable rhythm escalation.

Rhythm approach

• Determine stability first: altered mental status, hypotension, ischemic chest pain, shock and acute HF indicate need for immediate resuscitation-level care.

• Read rate, regularity, P-QRS relation and QRS width; use 12-lead ECG when possible without delaying resuscitation.

• Sinus tachycardia is usually a response to an underlying cause such as fever, pain, hypovolemia, anemia or PE rather than a primary arrhythmia.

• Wide-complex tachycardia in a sick patient should be assumed ventricular until specialist assessment.

Atrial fibrillation and flutter

• Atrial fibrillation shows an irregularly irregular rhythm and lacks consistent discrete P waves; AF may be paroxysmal and absent on one ECG.

• Assess thromboembolic risk with a validated approach, bleeding modifiers, renal function and contraindications.

• DOACs are generally preferred to warfarin in eligible nonvalvular AF; mechanical valves and moderate/severe rheumatic mitral stenosis are important exceptions.

• Aspirin alone is not an adequate substitute for indicated anticoagulation for AF stroke prevention.

SVT, bradycardia and blocks

• Regular narrow-complex tachycardia suggests SVT; assess stability and look for accessory pathways before selecting medication strategies.

• Symptomatic severe bradycardia or high-grade AV block can cause syncope and low output and may require urgent pacing.

• Long QT is associated with torsades risk; review electrolyte abnormalities, congenital causes and QT-prolonging medications.

• Palpitations with a family history of sudden cardiac death, exertional syncope or structural heart disease deserve prompt specialist evaluation.

Syncope differentiation

• Distinguish vasovagal and orthostatic causes from cardiac syncope; ask posture, triggers, exertion, palpitations and recovery.

• High-risk clues include syncope during exertion, no prodrome, abnormal ECG, family history of sudden death, heart disease, hypotension and associated chest pain.

• Measure orthostatic vitals when safe; consider targeted rhythm monitoring and echo based on risk rather than ordering a battery of indiscriminate tests.

• Document driving and activity restrictions according to applicable local standards after concerning syncope.

Safety, medication review, communication

• Check reversible arrhythmia contributors: potassium, magnesium, thyroid status, ischemia, stimulants, alcohol and medications.

• Explain when to seek emergency help for fainting, persistent severe palpitations, chest pain or shortness of breath.

• Use informed discussions around anticoagulant bleeding risks, adherence, procedures and drug interactions.

Applied clinical case

A 74-year-old with hypertension and prior TIA has new irregularly irregular pulse and ECG-confirmed AF; vital signs are stable.

Model reasoning and disposition

Assess stroke risk and bleeding modifiers and arrange guideline-directed anticoagulation evaluation; do not rely on aspirin monotherapy as AF stroke protection.

Self-check prompts

- What factors determine stroke prevention?
- Which AF phenotypes require different anticoagulants?
- What features would make this unstable?

Selected guidelines (open online)

AHA/ACC — Atrial Fibrillation Clinical Practice Guideline

AHA — 2025 Adult Advanced Life Support

Canadian Cardiovascular Society — Guidelines Library

Lesson 06 | Valvular Disease, Endocarditis, Rheumatic Disease and Congenital Conditions

Murmurs, valve lesions, infective endocarditis and structural cardiac disorders.

Murmur and valve assessment

• Use symptom history and focused auscultation to assess murmurs: onset, timing, radiation, positional changes, dyspnea, exertional syncope and HF signs.

• Symptomatic aortic stenosis may present with exertional angina, dyspnea and syncope; assess urgently and obtain echocardiography.

• Mitral regurgitation can cause an apical pansystolic murmur and pulmonary congestion; echo defines severity and ventricular effects.

• Acute severe valve regurgitation may cause flash pulmonary edema and shock even without a loud chronic murmur.

Endocarditis

• Fever with new murmur, embolic signs, positive blood cultures or a prosthetic valve raises concern for infective endocarditis.

• When stable, obtain appropriate blood cultures before antibiotics as directed by the treating emergency/inpatient team; do not delay antibiotics in sepsis.

• Echocardiography and specialist-directed microbiology assess vegetations and complications.

• Prompt referral is needed for suspected endocarditis, especially with HF, neurological deficit or sepsis.

Rheumatic heart disease

• Prior rheumatic fever can lead to chronic mitral stenosis/regurgitation and AF; a low-pitched diastolic rumble suggests mitral stenosis.

• Acute rheumatic fever follows group A streptococcal infection and is diagnosed using clinical criteria with evidence of preceding infection.

• Rheumatic mitral stenosis has important implications for anticoagulation selection when AF is present.

• Appropriate prevention and long-term specialist follow-up reduce recurrent rheumatic injury.

Congenital and inherited disease

• A bicuspid aortic valve is associated with premature valve stenosis and possible aortopathy requiring imaging follow-up.

• Adult congenital heart disease may present with cyanosis, murmurs, arrhythmia or exercise limitation; refer complex lesions to specialty services.

• Family history of inherited cardiomyopathy, unexplained sudden death or thoracic aneurysm can warrant cardiogenetic evaluation.

• Pregnancy modifies hemodynamics and the risk profile of severe valve disease or aortopathy.

When to escalate

• Urgent red flags: new murmur plus fever/sepsis, syncope with severe stenosis, sudden pulmonary edema or embolic neurological deficits.

• Do not use exercise stress testing for symptomatic severe aortic stenosis without specialist oversight.

• Explain echocardiographic findings in patient-centred terms and coordinate with cardiology for surveillance/intervention decisions.

Applied clinical case

A 70-year-old with exertional syncope has a harsh systolic ejection murmur at the right upper sternal edge radiating to the carotids.

Model reasoning and disposition

Suspect symptomatic aortic stenosis; arrange urgent echocardiography and cardiology evaluation, and avoid treating this as uncomplicated vasovagal syncope.

Self-check prompts

• Which findings support aortic stenosis?

• Which symptoms indicate high risk?

• How does rheumatic mitral stenosis change AF care?

Selected guidelines (open online)

Canadian Cardiovascular Society — Guidelines Library

AHA/ACC — Atrial Fibrillation Clinical Practice Guideline

Lesson 07 | Cardiac Emergencies, Shock, Arrest and Acute Resuscitation

Identify unstable patients, differentiate shock states and recognize arrest pathways.

Recognize shock

• Shock is inadequate tissue perfusion; assess hypotension together with mental status, skin temperature, capillary refill, urine output and lactate trends.

• Distinguish cardiogenic, obstructive, distributive and hypovolemic shock; some patients present before BP falls.

• Cardiogenic shock may follow extensive MI, acute severe valvular dysfunction, arrhythmia or myocarditis.

• Obstructive causes include massive PE, tamponade and tension pneumothorax; each needs immediate cause-specific action.

Initial stabilization

• Activate emergency response, provide high-quality CPR if pulseless, secure airway/oxygenation as indicated and arrange appropriate monitoring.

• Obtain rhythm assessment promptly, but do not delay chest compressions for testing in confirmed arrest.

• Defibrillation is indicated for ventricular fibrillation/pulseless VT; asystole and PEA are nonshockable rhythms.

• Reversible arrest causes include hypoxia, hypovolemia, acidosis, potassium disorders, hypothermia, tamponade, tension pneumothorax, thrombosis and toxins.

Post-arrest and acute instability

• After return of spontaneous circulation, optimize oxygenation, ventilation, BP and identify the underlying cause, including ACS or PE.

• Treatment of unstable tachyarrhythmia versus stable arrhythmia requires trained emergency care and guideline-directed synchronized cardioversion or other measures.

• New chest pain with pulse deficit and abrupt tearing back pain suggests aortic dissection; do not delay transfer or prescribe reflexive anticoagulation.

• Pregnancy, pediatric age and major comorbidities may require distinct algorithms beyond this adult overview.

Anaphylaxis and mimics

• Anaphylaxis with hypotension and airway symptoms is distributive shock; rapid intramuscular epinephrine and emergency activation are first-line.

• Vasovagal faint may cause bradycardia with rapid recovery when supine; persistent dyspnea, wheeze, swelling or hypotension suggests an alternative emergency.

• Obtain help rather than attempting complex procedural management outside scope or available equipment.

Communication and handover

• Use a structured handover: symptoms/onset, vitals, ECG/rhythm, suspected etiology, interventions, response and known medications/allergies.

• Clarify roles for CPR, AED and calling emergency services; document precise time-sensitive events.

• Discuss prognosis and consent with patients/families when appropriate without delaying life-saving interventions.

Applied clinical case

A 61-year-old collapses and is unresponsive, apneic and pulseless; the AED identifies a shockable rhythm.

Model reasoning and disposition

Start high-quality CPR, deliver an AED shock promptly as instructed and resume CPR according to the 2025 adult resuscitation algorithms while emergency help arrives.

Self-check prompts

• Which arrest rhythms are shockable?

• Name reversible causes of PEA.

• Why is early defibrillation critical?

Selected guidelines (open online)

AHA — 2025 Adult Advanced Life Support

AHA/ACC — 2025 Acute Coronary Syndromes Guideline

Lesson 08 | Peripheral and Cerebrovascular Disease, Aortic and Venous Conditions

PAD, acute limb ischemia, stroke/TIA, aortic emergencies and chronic venous disease.

Peripheral arterial disease

• Claudication is reproducible exertional muscle discomfort relieved by rest; pulses, skin changes and ankle-brachial index guide assessment.

• ABI at or below 0.90 supports PAD; ABI above 1.40 suggests noncompressible vessels and may require toe-brachial index evaluation.

• Manage global cardiovascular risk with smoking cessation, statins, antithrombotic strategies when appropriate, diabetes/BP care and structured exercise.

• Rest pain, nonhealing ulcer or gangrene suggests chronic limb-threatening ischemia and warrants expedited vascular referral.

Acute limb ischemia and aortic disease

• Acute limb ischemia presents with pain, pallor, pulse deficit, paresthesia, paralysis and poikilothermia; urgent vascular emergency care is essential.

• Sudden tearing chest/back pain, pulse or BP asymmetry, neuro deficits or shock raises suspicion for aortic dissection; emergency imaging and transfer are needed.

• Abdominal aortic aneurysm risk increases with age and smoking; rupture may cause abdominal/back pain, hypotension or collapse.

• A known aneurysm with new pain warrants immediate emergency assessment.

TIA and acute stroke

• Sudden facial asymmetry, arm weakness, speech change or visual loss requires emergency stroke activation, even if symptoms resolve.

• Establish last-known-well time, glucose, anticoagulant exposure and focal deficits; imaging is required to distinguish ischemia from hemorrhage.

• Thrombolysis eligibility and mechanical thrombectomy windows are determined by trained stroke teams and imaging; do not delay transport.

• Transient symptoms still require urgent evaluation and secondary prevention; do not assume a TIA is harmless.

Venous disease and edema

• Chronic venous insufficiency causes dependent edema, varicosities, hyperpigmentation and sometimes ulceration.

• Differentiate chronic bilateral edema from new unilateral swelling suggestive of DVT, and evaluate for systemic HF, renal or hepatic disease.

• Compression may benefit venous disease but requires appropriate arterial assessment when PAD is suspected.

• Consider lymphedema, cellulitis and medication-related edema in the differential.

Long-term prevention

• Atherosclerotic PAD increases the risk of MI, stroke and limb events; address the entire cardiovascular risk profile.

• Foot inspection, ulcer care, exercise adaptation and smoking cessation are key long-term components.

• Discuss urgent signs: acute neurological deficit, cold painful limb, sudden tearing chest/back pain or syncope.

Applied clinical case

A 66-year-old smoker has calf pain after 100 metres relieved by rest. ABI 0.72 and pedal pulses are diminished.

Model reasoning and disposition

Findings support PAD: initiate comprehensive cardiovascular risk assessment and guideline-based prevention, structured exercise and vascular follow-up as indicated.

Self-check prompts

• Which ABI values suggest PAD?

• What would indicate acute limb ischemia?

• Why must transient speech loss prompt emergency care?

Selected guidelines (open online)

AHA/ACC — 2024 Peripheral Artery Disease Guideline

Canadian Stroke Best Practices — Acute Ischemic Stroke Treatment

Canadian Cardiovascular Society — Guidelines Library

Lesson 09 | Venous Thromboembolism, Anticoagulants and Clinical Safety

Suspected DVT/PE pathways, anticoagulant selection, complications and monitoring.

VTE risk and presentation

• DVT may present with unilateral swelling, tenderness and asymmetry; PE may cause pleuritic chest pain, acute dyspnea, tachycardia, syncope or hypoxemia.

• Recognize precipitating risk: surgery, immobility, cancer, estrogen exposure, pregnancy/postpartum and prior VTE.

• A normal oxygen saturation does not rule out pulmonary embolism, and not every calf pain is DVT.

• Hemodynamic instability with suspected PE requires emergency care and rapid specialist evaluation.

Probability-led diagnosis

• Use a validated pretest probability tool (e.g., Wells criteria) in appropriate stable patients before choosing D-dimer or imaging.

• D-dimer can help rule out VTE in appropriately low-probability situations, but a positive value is nonspecific.

• Compression venous ultrasonography evaluates suspected lower-limb DVT; CT pulmonary angiography or alternative imaging evaluates PE when indicated.

• Do not delay emergency assessment to arrange outpatient D-dimer testing in a hemodynamically unstable patient.

Anticoagulant principles

• Anticoagulation prevents thrombus extension and recurrence; agent choice is affected by renal function, pregnancy, active malignancy, bleeding risk, weight and drug interactions.

• Oral factor Xa inhibitors and direct thrombin inhibitors are used in many VTE and AF settings, but selection, dosing and duration are indication-specific.

• Warfarin requires INR monitoring and interaction review; mechanical heart valves are a prominent indication for vitamin K antagonist rather than DOAC therapy.

• Distinguish anticoagulants from antiplatelets: they target different clotting mechanisms and cannot be freely substituted.

Bleeding safety

• Major bleeding warning signs include hematemesis, melena, intracranial symptoms, unexplained hypotension and severe bruising.

• Ask about NSAIDs, OTC aspirin, herbal supplements and alcohol when reviewing bleeding risk.

• Before procedures or medication changes, review renal function and consult the treating anticoagulation team; do not stop medication independently.

• Reversal decisions depend on agent and urgency and belong to hospital protocols.

Follow-up and patient education

• Explain adherence, missed-dose instructions specific to the prescribed agent, and when to seek emergency help.

• Consider provoking factors, persistent risks and patient preferences when selecting VTE treatment duration.

• Document a clear follow-up plan and monitoring for recurrent symptoms, bleeding and medication complications.

Applied clinical case

A 42-year-old on combined oral contraceptives has a new swollen painful calf after prolonged travel, but normal BP and oxygen saturation.

Model reasoning and disposition

Assess clinical probability, choose compression ultrasound and D-dimer only as appropriate to the probability pathway, and review alternative diagnoses and bleeding risk.

Self-check prompts

• When is D-dimer helpful?

• What makes suspected PE an immediate emergency?

• How do DOACs differ from warfarin?

Selected guidelines (open online)

AHA/ACC — Atrial Fibrillation Clinical Practice Guideline

Canadian Cardiovascular Society — Guidelines Library

Lesson 10 | Anemia, Red Cell Disease and Hematologic Diagnostic Reasoning

Structured CBC interpretation, anemia mechanisms, red cell emergencies and referral.

CBC and reticulocyte approach

• Classify anemia by MCV: microcytic, normocytic and macrocytic; confirm clinical severity and symptoms.

• Use reticulocyte response to differentiate underproduction from destruction or blood loss, alongside smear and relevant clinical information.

• Consider iron status, B12, folate, renal function, inflammation, hemolysis indices and bleeding history as directed by the differential.

• CBC reference intervals vary with age, pregnancy and laboratory; trends may be more informative than one isolated value.

Microcytic and iron deficiency

• Low ferritin supports iron deficiency, but ferritin may be falsely elevated with inflammation; transferrin saturation can help in context.

• Common microcytic causes include iron deficiency, thalassemia trait and anemia of inflammation.

• Investigate cause of iron deficiency, particularly GI bleeding in appropriate risk groups; iron replacement without etiology workup may miss pathology.

• Thalassemia trait may show marked microcytosis with relatively preserved RBC count; avoid labeling it iron deficiency without iron studies.

Macrocytic and normocytic anemia

• Macrocytosis may reflect B12 or folate deficiency, alcohol, liver disease, hypothyroidism, reticulocytosis or marrow pathology.

• Neurological signs suggest B12 deficiency; do not correct folate alone while failing to recognize B12-related neuropathy.

• Normocytic anemia includes acute blood loss, inflammation, renal disease, marrow disease or early iron deficiency.

• Consider hematology referral for unexplained cytopenias or abnormal smear/blasts.

Hemolysis and urgent findings

• Hemolysis pattern includes increased LDH and indirect bilirubin, low haptoglobin and reticulocytosis when marrow response is intact.

• Schistocytes with thrombocytopenia or renal/neuro abnormalities raises concern for microangiopathic hemolytic anemia needing urgent evaluation.

• Severe symptomatic anemia with syncope, chest pain, hemodynamic instability or active bleeding requires emergency assessment.

• Transfusion decisions depend on symptoms, comorbidity and institutional protocols, not hemoglobin alone.

Communication and follow-up

• Explain uncertainty when ferritin, inflammation and renal function produce mixed patterns.

• Communicate follow-up tests, response to replacement and indications to pursue bleeding sources.

• Document red-flag symptoms and escalation thresholds based on the clinical picture.

Applied clinical case

A 58-year-old reports fatigue; Hb 89 g/L, MCV 70 fL, ferritin 8 micrograms/L and no obvious menstrual source.

Model reasoning and disposition

Iron deficiency anemia is likely; evaluate possible blood loss including gastrointestinal sources as appropriate and address symptoms and iron replacement with clinician follow-up.

Self-check prompts

• What does a low ferritin mean?

• Why is etiology evaluation necessary?

• Which anemia findings suggest urgent referral?

Selected guidelines (open online)

Canadian Cardiovascular Society — Guidelines Library

Lesson 11 | Platelets, Leukocytes, Coagulation and Bleeding Disorders

Systematic evaluation of cytopenias, clotting tests, thrombocytopenia and DIC.

WBC abnormalities

• Neutrophilia can reflect infection, inflammation or medications; assess symptoms, smear and trends rather than the count alone.

• Neutropenia with fever is an emergency, particularly after chemotherapy or with profound neutrophil depletion.

• Persistent lymphocytosis, blasts or multiple cytopenias suggest possible marrow/hematologic disease requiring specialist assessment.

• Review medication effects and infection history in isolated count abnormalities.

Platelets and bleeding mechanism

• Thrombocytopenia can arise from reduced production, increased destruction, consumption, splenic pooling or dilution.

• Mucocutaneous bleeding, petechiae and easy bruising often suggest primary hemostatic/platelet dysfunction.

• Deep tissue bleeding and hemarthroses can suggest coagulation factor deficiencies.

• Pseudothrombocytopenia due to EDTA-related clumping should be considered in otherwise discordant low platelet counts.

PT/INR, aPTT, fibrinogen

• PT/INR primarily assesses extrinsic/common pathways and is used to monitor warfarin; aPTT assesses intrinsic/common pathways and certain heparin contexts.

• DOAC anticoagulant effects cannot reliably be excluded using normal PT/aPTT; drug-specific assays may be necessary in selected emergency situations.

• Prolonged PT/aPTT requires interpretation with liver function, medication, mixing studies and bleeding context.

• DIC may cause thrombocytopenia, prolonged PT/aPTT, elevated D-dimer and low fibrinogen in a critically ill patient.

Critical hematology presentations

• DIC is a syndrome of systemic coagulation activation and consumption; identify and urgently treat underlying triggers such as sepsis or obstetric catastrophe.

• Heparin-induced thrombocytopenia is a prothrombotic reaction typically occurring after heparin exposure and requires urgent specialist-directed management.

• Suspected thrombotic thrombocytopenic purpura with hemolysis and thrombocytopenia requires emergency care; avoid unnecessary delay for confirmatory tests.

• Acute leukemia may cause unexplained bleeding, infections, fatigue and blasts; expedite appropriate referral.

Patient-centred care

• Assess epistaxis, gum bleeding, menorrhagia, GI blood loss, drugs and supplements that affect platelets or coagulation.

• Warn patients with serious bleeding or neurological symptoms to seek emergency care rather than awaiting routine follow-up.

• Use shared documentation of suspected mechanism, serial laboratory trend and responsible follow-up clinician.

Applied clinical case

A hospitalized patient with sepsis develops oozing from IV sites, platelets 45 x10^9/L, prolonged PT/aPTT and low fibrinogen.

Model reasoning and disposition

DIC is a high-priority consideration; urgent treatment of sepsis and supportive hospital management are needed, not outpatient evaluation.

Self-check prompts

• Which tests support consumptive coagulopathy?

• What suggests platelet rather than factor bleeding?

• Why is fever with neutropenia urgent?

Selected guidelines (open online)

Canadian Cardiovascular Society — Guidelines Library

Lesson 12 | Integrated Cardiology Cases, Prescribing Safety and Clinical Communication

Cross-system reasoning, cardiovascular medicines, shared decisions, professional scope and exam integration.

Multimorbidity and treatment prioritization

• In a patient with CAD, CKD, diabetes and AF, prioritize the current threat to life, then assess long-term preventive treatment interactions and competing harms.

• Separate symptom control from disease-modifying care and distinguish anticoagulation from antiplatelet indications.

• Check pregnancy status, allergies, renal function, electrolyte risk, drug-drug interactions and access barriers before recommending prescriptions.

• Use a medication reconciliation method that includes OTC medicines, supplements, patches, topical agents and recent changes.

Focused case synthesis

• State the main problem representation in one sentence: age/risk, onset, defining features and key test findings.

• Rank must-not-miss diagnoses before common benign diagnoses; justify what argues for and against each.

• Select investigations by urgency and probability, not by how many tests are available.

• Define immediate disposition and a safety-net plan, then long-term prevention.

Medication classes and cautions

• Antiplatelets, anticoagulants, statins, antianginals, BP agents and HF medications have overlapping effects but distinct evidence-based indications.

• ACEi/ARBs and MRAs require attention to potassium and kidney function; diuretic adjustments require volume and electrolyte review.

• QT-prolonging agents and stimulants may aggravate arrhythmia risk; NSAIDs can worsen fluid retention and bleeding risk.

• Avoid treatment suggestions outside professional scope; escalate severe arrhythmias, ACS, stroke, shock and suspected major bleeding to emergency services.

Patient communication and informed consent

• Explain diagnosis, uncertainty, options, benefits, harms and alternatives using plain language and teach-back.

• When emergency transfer is recommended, state specific risks, need for ambulance and what has been arranged; document informed refusal respectfully if it occurs.

• For chronic disease use shared goals, affordable monitoring, reassessment dates and clear return precautions.

• Screen for anxiety, depression and social needs without missing physiologic causes of symptoms.

Exam capstone approach

• For each case answer in order: stability, differential, discriminating test, first safe action, referral, long-term prevention.

• Practice timing without relying on memorized single-test rules; troponin, ECG, D-dimer and BNP require context.

• Review common traps: normal ECG does not exclude ACS; transient deficits can be TIA; fast heart rate does not prove a primary tachyarrhythmia.

• Revise the course PDF checklists and repeat incorrectly answered questions as part of spaced practice.

Applied clinical case

A 72-year-old with HF, CKD and AF has progressive breathlessness, black stools and new dizziness. A medication list includes an anticoagulant and NSAID.

Model reasoning and disposition

Stabilize/assess urgently for clinically significant GI blood loss and possible HF deterioration, review anticoagulant/NSAID exposures and communicate an emergency handover.

Self-check prompts

• Which problem takes priority?

• Why are medicine reconciliation and bleeding history critical?

• What needs to be documented at handover?

Selected guidelines (open online)

Canadian Cardiovascular Society — Guidelines Library

AHA/ACC — Atrial Fibrillation Clinical Practice Guideline

Canadian Cardiovascular Society — 2025 Heart Failure Guideline

AHA — 2025 Adult Advanced Life Support

Educational use only. Not a substitute for current guidelines, local emergency protocols, or individualized clinical care. Course cases and practice questions are original educational examples.