Achieving Excellence in the OSCE

Part I

Cardiology, Endocrinology, Gastroenterology, Hepatology and Nephrology

This book complements
Achieving Excellence in the OSCE - Part II

A.B.R. Thomson
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ACHIEVING EXCELLENCE IN THE OSCE

Part 1

Cardiology to Nephrology

A.B.R. Thomson
THE WESTERN WAY
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OSCEs and the CANMED Objectives

Medical expert
The discussion of complex cases provides the participants with an opportunity to comment on additional focused history and physical examination. They would provide a complete and organized assessment. Participants are encouraged to identify key features, and they develop an approach to problem-solving.

The case discussions, as well as the discussion of cases around a diagnostic imaging, pathological or endoscopic base provides the means for the candidate to establish an appropriate management plan based on the best available evidence to clinical practice. Throughout, an attempt is made to develop strategies for diagnosis and development of clinical reasoning skills.

Communicator
The participants demonstrate their ability to communicate their knowledge, clinical findings, and management plan in a respectful, concise and interactive manner. When the participants play the role of examiners, they demonstrate their ability to listen actively and effectively, to ask questions in an open-ended manner, and to provide constructive, helpful feedback in a professional and non-intimidating manner.

Collaborator
The participants use the “you have a green consult card” technique of answering questions as fast as they are able, and then to interact with another health professional participant to move forward the discussion and problem solving. This helps the participants to build upon what they have already learned about the importance of collegial interaction.

Manager
Some of the material they must access demands that they use information technology effectively to access information that will help to facilitate the delineation of adequately broad differential diagnoses, as well as rational and cost effective management plans.
**Health advocate**
In the answering of the questions and case discussions, the participants are required to consider the risks, benefits, and costs and impacts of investigations and therapeutic alliances upon the patient and their loved ones.

**Scholar**
By committing to the pre- and post-study requirements, plus the intense three day active learning Practice Review with colleagues is a demonstration of commitment to personal education. Through the interactive nature of the discussions and the use of the “green consult card”, they reinforce their previous learning of the importance of collaborating and helping one another to learn.

**Professional**
The participants are coached how to interact verbally in a professional setting, being straightforward, clear and helpful. They learn to be honest when they cannot answer questions, make a diagnosis, or advance a management plan. They learn how to deal with aggressive or demotivated colleagues, how to deal with knowledge deficits, how to speculate on a missing knowledge byte by using first principals and deductive reasoning. In a safe and supportive setting they learn to seek and accept advice, to acknowledge awareness of personal limitations, and to give and take 360° feedback.

**Knowledge**
The basic science aspects of gastroenterology are considered in adequate detail to understand the mechanisms of disease, and the basis of investigations and treatment. In this way, the participants respect the importance of an adequate foundation in basic sciences, the basics of the design of clinical research studies to provide an evidence-based approach, the designing of clinical research studies to provide an evidence-based approach, the relevance of their management plans being patient-focused, and the need to add “compassionate” to the Three C’s of Medical Practice: competent, caring and compassionate.

“They may forget what you said, but they will never forget how you made them feel.”

Carl W. Buechner, on teaching.

“With competence, care for the patient. With compassion, care about the person.”

Alan B. R. Thomson, on being a physician.
Prologue

HREs, better known as, High Risk Examinations. After what is often two decades of study, sacrifice, long hours, dedication, ambition and drive, we who have chosen Internal Medicine, and possibly through this a subspecialty, have a HRE, the [Boards] Royal College Examinations. We have been evaluated almost daily by the sadly subjective preceptor based assessments, and now we face the fierce, competitive, winner-take-all objective testing through multiple choice questions (MCQs), and for some the equally challenging OSCE, the objective standardized clinical examination. Well we know that in the real life of providing competent, caring and compassionate care as physicians, as internists, that a patient is neither a MCQ or an OSCE. These examinations are to be passed, a process with which we may not necessarily agree. Yet this is the game in which we have thus far invested over half of our youthful lives. So let us know the rules, follow the rules, work with the rules, and succeed. So that we may move on to do what we have been trained to do, do what we may long to do, care for our patients.

The process by which we study for clinical examinations is so is different than for the MCQs: not trivia, but an approach to the big picture, with thoughtful and reasoned deduction towards a diagnosis. Not looking for the answer before us, but understanding the subtle aspects of the directed history and focused physical examination, yielding an informed series of hypotheses, a differential diagnosis to direct investigations of the highly sophisticated laboratory and imaging procedures now available to those who can wait, or pay.

This book provides clinically relevant questions of the process of taking a history and performing a physical examination, with sections on Useful background, and where available, evidence-based performance characteristics of the rendering of our clinical skills. Just for fun are included "So you want to be a such-and-such specialist!" to remind us that one if the greatest strengths we can possess to survive in these times, is to smile and even to laugh at ourselves.

Sincerely,

Emeritus Distinguished University Professor, U of A
Adjunct Professor, Western University
Dedication

To My Family

For your support, caring and love
During these challenging years
And always.

Mark 15:34
Luke 23:34
Domenichino 16:41
Corinthians 1:13
Acknowledgements

Patience and patients go hand in hand. So also does the interlocking of young and old, love and justice, equality and fairness. No author can have thoughts transformed into words, no teacher can make ideas become behaviour and wisdom and art, without those special people who turn our minds to the practical - of getting the job done!

Thank you, Robin, Naiyana and Duen for translating those terrible scribbles, called my handwriting, into the still magical legibility of the electronic age. Thank you, Sarah and Rebecca, for your creativity and hard work.

My most sincere and heartfelt thanks go to the excellent persons at JP Consulting, and CapStone Academic Publishers. Jessica, you are brilliant, dedicated and caring. Thank you.

When Rebecca, Maxwell, Megan Grace, Henry and Felix ask about their Grandad, I will depend on James and Anne, Matthew and Allison, Jessica and Matt, and Benjamin to be understanding and kind. For what I was trying to say and to do was to make my professional life focused on the three C’s - competence, caring, and compassion - and to make my very private personal life dedicated to family - to you all.
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- Bits and Bytes for Rounds in Internal Medicine
- Mastering the Boards and Clinical Examinations. Part I. Cardiology to Nephrology
- Mastering the Boards and Clinical Examinations. Part II. Neurology to Rheumatology
OSCE Questions in Cardiology chapter

1. Take a directed history for disease of the cardiovascular system.
2. Perform a focused physical examination for disease of the heart and cardiovascular system.
3. Take a directed history and perform a focused physical examination for congestive heart failure (CHF).
4. Perform a focused physical examination to distinguish between the presence of left-sided congestive heart failure (L-CHF and e.g. LV failure [LVF]), and right-sided congestive heart failure (R-CHF and e.g. RV failure [RVF]).
5. Perform a direct physical examination to distinguish between the JVP and carotid waveforms.
6. Take a focused history to determine the risk factors for coronary artery disease.
7. Take a directed history for chest pain.
8. Take a focused history of clinical features increasing the likelihood of an acute coronary syndrome (ACS).
9. Perform a focused physical examination to determine if the person with an acute coronary syndrome more likely had disease of the LAD versus the RCA.
10. Take a directed history to differentiate between intermittent claudication (from atherosclerosis and peripheral vascular disease) and pseudoclaudication (from spinal stenosis).
11. Take a directed history for lower leg ulcers.
12. Take a directed history and perform a focused physical examination of the four most common types of lower leg ulcer.
13. Take a directed history for and perform a focused physical examination for peripheral vascular disease (arterial and venous insufficiency) in the lower extremities.
14. Take a focused history and perform a directed physical examination to distinguish between chronic vs acute (critical) ischemia.
15. Take a directed history and perform a focused physical examination to differentiate between arterial vs venous insufficiency.
16. Take a directed history for the causes of postural hypotension.
17. Take a directed history to determine the cause of lower leg edema.
18. Perform a focused physical examination for pulsus paradoxus (exaggeration of normal fall [> 20 mm Hg] in SBP with inspiration).
19. Perform a focused physical examination to determine the cause of atrial fibrillation.

20. Take a directed history for the causes of sinus bradycardia and tachycardia.

21. Perform a focused physical examination for the causes of bradycardia/heart block.

22. Give the disease processes associated with a loud, variable or soft intensity of S1.

23. Perform a focused physical examination of the heart sounds. Explain what underlying cardiac abnormalities may be determined from this examination.

24. Perform an auscultatory physical examination for signs of pulmonary hypertension.

25. Perform a focused physical examination for the causes of fixed splitting of S2 (splitting of S2 which persists on supine → expiration).

26. Perform a directed physical examination of abnormal S2 splitting to detect the presence of associated pathological abnormalities.

27. Perform a focused physical examination of the precordium, and from the timing of the murmur, give the differential of the lesion.

28. Perform a focused physical examination of the precordium for the site of optimal auscultation of normal and abnormal heart sounds and murmurs.

29. Take a directed history for the cause of a patient’s cardiac murmur.

30. Take a direct history and perform a focused physical examination to determine if a systolic murmur is benign (i.e. non-pathological).

31. Perform a focused physical examination of the precordium, which will help to distinguish between

32. Perform a physical focused physical examination to determine the severity of mitral regurgitation (MR).

33. Take a direct history and perform a focused physical examination for mitral valve prolapse.

34. Take a directed history and perform a focused physical examination for rheumatic fever and rheumatic heart disease (RHD).

35. Perform a physical focused physical examination to distinguish between the murmur of pulmonary stenosis (PS) and aortic stenosis (AS).
36. Perform a directed physical examination to distinguish between the systolic murmur of aortic sclerosis (no pressure gradient; due to stiff or dilated aortic root) and aortic stenosis (AS).

37. Perform a directed physical examination to distinguish between the systolic murmur from HOCM (hypertrophic cardiomyopathy, particularly septal component) and aortic stenosis (AS).

38. Perform a focused physical assessment for prosthetic heart valves.

39. Perform a physical focused physical examination to distinguish between the systolic murmur from HOCM (hypertrophic cardiomyopathy, particularly septal component) and aortic stenosis (AS).

40. Take a directed history and focused physical examination for TR.

41. Perform a focused physical examination for aortic regurgitation.

42. Perform a focused physical examination to distinguish between the diastolic murmurs of mitral stenosis (MS) versus tricuspid stenosis (TS).

43. Perform a physical examination to determine the severity of aortic regurgitation.

44. Take a directed history for the causes of pericarditis.

45. Perform a focused physical examination for acute cardiac tamponade.

46. Take a directed history and perform a focused physical examination for chronic constrictive pericarditis.

47. Perform a focused physical examination to distinguish between the presence of chronic pericarditis with constriction constructive pericarditis (CP) and cardiac tamponade (CT).

48. Perform a focused physical examination to determine the cause of acyanotic, cyanose tardive, and cyanotic congenital cardiovascular disease.

49. Perform a focused physical examination for dextrocardia.

50. Take a directed history and perform a focused physical examination for Eisenmenger syndrome.

51. Perform a focused physical examination for a ventricular septal defect (VSD).

52. Perform a focused physical examination for tetralogy for Fallot's.

53. Perform a focused physical examination for syphilitic aortitis with saccular aneurysm of aorta.

54. Perform a focused physical examination for bacterial endocarditis.

55. Take a directed history for palpitations.
56. Perform a directed physical examination of the patient with systemic hypertension.

57. Take a directed history for the causes of systemic hypertension.

58. Take a focused history for complications of malignant hypertensive emergency.

59. Perform a focused physical examination for an abnormally widened pulse pressure.

60. Take a focused history and perform a directed physical examination for causes of hyperkinetic heart syndrome causing an abnormally wide pulse pressure (PP) (PP > 50% of systolic BP).

61. Take a focused history to determine the cardiac risk stratification for noncardiac surgical procedures.

**OSCE Questions in Endocrinology chapter**

1. Take a directed history for diabetes mellitus.

2. Perform a focused physical examination for diabetic nephropathy.

3. Perform a focused physical examination of the diabetic foot.

4. Perform a focused physical examination to differentiate DKA from HONC.

5. Take a directed history and perform a focused physical examination in the adult to determine the causes of hypoglycemia.

6. Take a directed history for thyroid disease.

7. Perform a focused physical examination for thyroid disease.

8. Take a directed history and perform a focused physical examination to determine if a thyroid nodule is likely to be malignant.

9. Take a directed history for the factors which increase the pretest probability of a goiter being present.

10. Perform a focused physical examination to distinguish between Grave’s disease (GD) and Toxic Nodular Goitre (TNG).

11. Perform a focused physical examination for hypothyroidism.

12. Perform a focused physical examination for hyperthyroidism.

13. Take a directed history for the causes of hypoadrenalism (Addison’s disease).

14. Perform a focused physical examination for hypoadrenalism (Addison’s Disease).
15. Perform a focused physical examination for pheochromocytoma (catecholamine-secreting tumour).

16. Perform a focused physical examination for Cushing’s syndrome.

17. Take a directed history to determine the causes of secondary hyperlipidemia.

18. Take a directed history for the causes of hypo- and hypercalcemia.

19. Take a directed history to determine the cause of osteoporosis.

20. Take a directed history and perform a focused physical examination to determine the causes of gynecomastia.

21. Take a directed history and perform a focused physical examination to determine the cause of amenorrhea.

22. Perform a focused physical examination for paraneoplastic syndromes and hormone producing cancers.

**OSCE Questions in Gastroenterology chapter**

1. Perform a focused physical examination to determine the causes of stomatitis.

2. Take a directed history to determine the causes of halitosis.

3. Perform a focused physical examination to determine the causes of salivary gland swelling.

4. Perform a focused physical examination to determine the causes of parotid gland enlargement.

5. Take a directed history for dysphagia.

6. Take a directed history to determine the causes of RUQ pain.

7. Take a directed history and perform a focused physical examination for appendicitis, and stratify the risk and need for surgery.

8. Perform a focused physical examination to determine the causes of abdominal masses.

9. Take a directed history and perform a focused physical examination for the cause of abdominal bruit.

10. Take a directed history for bowel obstruction.

11. Take a directed history for diarrhea.

12. Take a directed history to determine the cause of infertility in men with inflammatory bowel disease.

13. Take a directed history of alcohol abuse.
14. Perform a directed physical examination for alcohol withdrawal (SSH-DTs: shake, seize, hallucinates, DTs).

15. Perform a focused physical examination hepatosplenomegaly.

16. Perform a physical examination for acute liver disease (acute hepatitis and fulminant liver failure).

17. Perform a focused physical examination for signs of chronic liver disease (portal hypertension).

18. Take a directed history and perform a focused physical examination for ascites.

19. Perform a focused physical examination to determine the cause of pruritus.

20. Prepare a patient for informed consent for the use of steroids (GCS, glucorticosteroids) in a patient with IBD, explaining the adverse effects.

21. Provide a patient with informed consent prior to their having possible bariatric surgery.

22. Prepare a patient for informed consent for the use of nonsteroidal anti-inflammatory drugs, explaining the potential adverse effects.

23. Perform a directed examination of an abdominal x ray (‘flat plate’)

**OSCE Questions in Hematology chapter**

1. Take a directed history of thrombocytopenia

2. Take a directed history and perform a focused physical examination of the patient with lymphadenopathy:

3. Perform a directed physical examination for lymph nodes in the neck and axilla.

4. Perform a focused physical examination for pernicious anemia.

5. Take a directed history for causes of hemolytic anemia.

6. Perform a focused physical examination for anemia.

**OSCE Questions in Nephrology chapter**

1. Perform a directed physical examination systemic hypertension.

2. Take a directed history for the causes of systemic hypertension.

3. Take a focused history for complications of malignant hypertensive emergency.
4. Perform a focused physical examination for an abnormally widened pulse pressure.

5. Perform a directed history for the causes of renal colic.

6. Take a directed history of causes of acute interstitial nephritis.

7. Take a directed history to determine the causes of acute renal failure (ARF).

8. Perform a focused physical examination for the causes of acute renal failure.

9. Take a directed history to determine the causes of chronic renal failure.

10. Perform a focused physical examination for chronic renal failure and its causes.

11. Take a directed history for hyponatremia.

12. Perform a focused physical examination for dehydration (extracellular volume depletion).

13. Take a focused history and perform a focused physical examination for obstructive sleep apnea (aka Pickwickian Syndrome).

**OSCE Questions in Miscellaneous chapter**

1. Take a directed history for unexplained fever.

2. Perform a focused physical examination for fever of unknown origin.

3. Perform a directed physical examination for fever and infection in a patient in hospital.

4. Take a directed history and perform a focused physical examination for postoperative (post-op) fever.

5. Perform a directed physical examination for flushing.

6. Take a directed history for urinary tract infection.

7. Take a directed history of lifestyle issue.
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21. Perform a focused physical examination for the causes of bradycardia/heart block.

22. Give the disease processes associated with a loud, variable or soft intensity of S₁.

23. Perform a focused physical examination of the heart sounds. Explain what underlying cardiac abnormalities may be determined from this examination.

24. Perform an auscultatory physical examination for signs of pulmonary hypertension.

25. Perform a focused physical examination for the causes of fixed splitting of S₂ (splitting of S₂ which persists on supine → expiration).

26. Perform a directed physical examination of abnormal S₂ splitting to detect the presence of associated pathological abnormalities.

27. Perform a focused physical examination of the precordium, and from the timing of the murmur, give the differential of the lesion.

28. Perform a focused physical examination of the precordium for the site of optimal auscultatation of normal and abnormal heart sounds and murmurs.

29. Take a directed history for the cause of a patient’s cardiac murmur.

30. Take a direct history and perform a focused physical examination to determine if a systolic murmur is benign (i.e. non-pathological).

31. Perform a focused physical examination of the precordium, which will help to distinguish between.

32. Perform a physical focused physical examination to determine the severity of mitral regurgitation (MR).

33. Take a direct history and perform a focused physical examination for mitral valve prolapse.

34. Take a directed history and perform a focused physical examination for rheumatic fever and rheumatic heart disease (RHD).

35. Perform a physical focused physical examination to distinguish between the murmur of pulmonary stenosis (PS) and aortic stenosis (AS).
36. Perform a directed physical examination to distinguish between the systolic murmur of aortic sclerosis (no pressure gradient; due to stiff or dilated aortic root) and aortic stenosis (AS).

37. Perform a directed physical examination to distinguish between the systolic murmur from HOCM (hypertrophic cardiomyopathy, particularly septal component) and aortic stenosis (AS).

38. Perform a focused physical assessment for prosthetic heart valves.

39. Perform a physical focused physical examination to distinguish between the systolic murmur from HOCM (hypertrophic cardiomyopathy, particularly septal component) and aortic stenosis (AS).

40. Take a directed history and focused physical examination for TR.

41. Perform a focused physical examination for aortic regurgitation.

42. Perform a focused physical examination to distinguish between the diastolic murmurs of mitral stenosis (MS) versus tricuspid stenosis (TS).

43. Perform a physical examination to determine the severity of aortic regurgitation.

44. Take a directed history for the causes of pericarditis.

45. Perform a focused physical examination for acute cardiac tamponade.

46. Take a directed history and perform a focused physical examination for chronic constrictive pericarditis.

47. Perform a focused physical examination to distinguish between the presence of chronic pericarditis with constriction constructive pericarditis (CP) and cardiac tamponade (CT).

48. Perform a focused physical examination to determine the cause of acyanotic, cyanose tardive, and cyanotic congenital cardiovascular disease.

49. Perform a focused physical examination for dextrocardia.

50. Take a directed history and perform a focused physical examination for Eisenmenger syndrome.

51. Perform a focused physical examination for a ventricular septal defect (VSD).

52. Perform a focused physical examination for tetralogy for Fallot’s.

53. Perform a focused physical examination for syphilitic aortitis with saccular aneurysm of aorta.

54. Perform a focused physical examination for bacterial endocarditis.
55. Take a directed history for palpitations.
56. Perform a directed physical examination of the patient with systemic hypertension.
57. Take a directed history for the causes of systemic hypertension.
58. Take a focused history for complications of malignant hypertensive emergency.
59. Perform a focused physical examination for an abnormally widened pulse pressure.
60. Take a focused history and perform a directed physical examination for causes of hyperkinetic heart syndrome causing an abnormally wide pulse pressure (PP) (PP > 50% of systolic BP).
61. Take a focused history to determine the cardiac risk stratification for noncardiac surgical procedures.
General

Useful background: the “O to W” of any history

O = Onset and duration
P = Provoking and alleviation factors
Q = Quality of pain (e.g. “Is the pain sharp or dull? Is it throbbing?”)
R = Radiation of pain
S = Severity (on a scale from 1 to 10)
T = Timing and progression (e.g. “Is the pain constant or intermittent?”)
U = “How does it affect ‘U’ in your daily life?”
V = déjà Vù? (e.g. “Has it happened before?”)
W = ‘What do you think is causing it?’

Source: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, page 7.

<table>
<thead>
<tr>
<th>Sensitivity</th>
<th>PID</th>
<th>present in disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specificity</td>
<td>NIH</td>
<td>negative in health</td>
</tr>
</tbody>
</table>

PLR = sensitivity/ 1-sensitivity
NLR = 1-sensitivity/specificity

Accuracy of the history and physical exam

One study in general medical clinic found that 55% of patients had been assigned correct diagnosis at the end of the history, and that number rose to 73% by the end of the physical examination.


One study in general medical clinic found that 55% of patients had been assigned correct diagnosis at the end of the history, and that number rose to 73% by the end of the physical examination.

Source: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, page 5.

1. Take a directed history for disease of the cardiovascular system.

- Cardiovascular symptoms
  - Chest pain
  - Dyspnea – exertion, paroxysmal nocturnal dyspnea (PND), orthopnea
  - Cough
  - Palpitations
  - Ankle swelling
  - Intermittent claudication
Associated symptoms
  - Left side congestive heart failure (L-CHF)
    - Fatigue
    - SOB, SOBOE orthopnea
    - Cough, hemoptysis
    - Baseline exercise in tolerance
    - Cyanosis
    - Cool extremities
    - Palpations
    - Nausea, vomiting
  - Right side congestive heart failure (R-CHF)
    - Edema of ankles, sacrum
    - Tender hepatomegaly
    - Determine New York class of CHF
  - Syncope
  - Fatigue
  - Weight gain

Functional status (New York Heart Association Classification, Angina/ dyspnea activity on activity and relationship to exercise)
  - Class I – intense
  - Class II – ordinary
  - Class III – less than ordinary
  - Class IV – at rest

Associated conditions/risk factors
  - Hypertension
  - Hyperlipidemia
  - Hyperhomocysteinemia
  - Obesity
  - Diabetes
  - Physical inactivity
  - Smoking
  - Causes of L/R- CHF
  - Family history
  - Personal past history of CAD, PVD, rheumatic fever, cardiac murmur, cardiac surgery, cardiac events, medications
  - Risk factors for CAD

Abbreviations: CAD, coronary artery disease; CHF, congestive heart failure; L-CHF, left side congestive heart failure; PND, paroxysmal nocturnal dyspnea; PVD, peripheral vascular disease; R-CHF, right side congestive heart failure; SOB, shortness of breath; SOBOE, shortness of breath on exertion
2. Perform a focused physical examination for disease of the heart and cardiovascular system.

- **Inspection**
  - **General appearance**
    - Scleral icterus
    - Mitral facies (rosy cheeks with blue tinge from pulmonary hypertension [PHT] and low cardiac output [MS])
    - Palour
    - Wasting
    - Oxygen mask
    - Marfan’s syndrome (MS) (aortic and mitral regurgitation)
    - Down syndrome (DS) (congenital heart disease)
    - Turner’s syndrome (TS) (coarctation of the aorta)
  - **Mouth**
    - High arched palate (MS)
    - Diseased teeth
    - Tongue, lips – central cyanosis, petechiae
  - **Hands, feet**
    - Clubbing
    - Splinter hemorrhages in nail beds
    - Osler nodes (Raised, red, tender nodules on the pulps of the fingers or toes, or on the thenar or hypothenar eminences)
    - Janeway lesions (Raised, red, non-tender nodules on the pulps of the fingers or on the palms)
    - Short, broad hands (DS)
    - Single palmar crease (DS)
    - Incurving fifth finger (DS)
    - Hyperflexible joints (DS)
      - Lymfedema (TS)
      - Short 4th metacarpal bone (TS)
      - Increased carrying angle of elbow (TS)
    - Aracanydactyly (spider fingers) (MS)
    - Periocular xanthelasma
  - **Neck**
    - Carotid arteries
    - Jugular venous pressure (JVP) elevated
    - Webbing, low hairline, redundant skin folds on back of neck (TS)
  - **Chest**
    - Funnel shaped chest
    - Widely spaced nipples (TS)
  - **Vital signs**
    - PR, BP, RR, % 0₂ saturation
    - Colour – white, blue, grey
    - Distress
    - Chest incisions, pacemakers
- Signs of peripheral vascular disease
- Fundic vessel abnormalities (hypertension, diabetes)
  o Palpation
    - PMI (apex beat)
    - Thrills and heaves
    - Reduced peripheral pulses
  o Percussion
    - Cardiomegaly (pulmonary edema)
    - Pleural effusion
  o Auscultation
    - Supine and upright, 5 areas, bell and diaphragm for S1/S2
    - L. lateral decubitus bell for S3/S4
    - Base of heart, lean forward, bell for diastolic murmur
    - Auscultate carotids (axilla)

➤ Signs of left side congestive heart failure (L-CHF)
  o Dyspnea, cough, hemoptysis
  o Basal crepitations
  o Cyanosis
  o Hypotension
  o Cold extremities
  o Fever, sweating

➤ Signs of right side congestive heart failure (R-CHF)
  o ↑ JVP
  o Hepatojugular reflux
  o Tender hepatomegaly
  o Pulsatile murmur
  o Hepatic bruit

➤ Signs of other causes of CHF
  o Hypertension
  o Vascular disease
  o Endocarditis
  o Constrictive pericarditis
  o Arrhythmia
  o Anemia
  o Hyperthyroidism, pheochromocytoma
  o Pregnancy
  o Heat stroke
  o Non compliance with other medications
  o PE, AE-COPD, pneumonia
  o High salt diet (salt shaker at bedside)
  o Acute/chronic renal failure
Nephrotic syndrome

➤ MAYO precipitating factors in heart failure
  o Diet (excessive sodium or fluid intake, alcohol)
  o Non-compliance with medication or inadequate dosing
  o Sodium retaining medications (NSAIDs)
  o Infection (bacterial or viral)
  o Myocardial ischemia or infarction
  o Arrhythmia (atrial fibrillation, bradycardia)
  o Breathing disorders of sleep
  o Worsening renal function
  o Anemia
  o Metabolic (hyperthyroidism, hypothyroidism)
  o Pulmonary embolus

➤ Signs of other causes of chest pain
  o Chest wall
    - Muscle strains
    - Myositis
    - Rib fracture or tumour
    - Infection (Coxsackie B)
  o Heart
    - Aortic aneurysm, pericarditis
  o Lung
    - PE, pleurisy, pneumonia, pneumothorax
  o GI
    - GERD, NCCP, PUD, pancreatitis, cholecystitis
  o MSK
    - Costochondritis
  o Skin
    - Herpes zoster
  o Psycho
    - Anxiety

Abbreviations: AE-COPD, Acute exacerbation of chronic pulmonary disease; BP, blood pressure; CHF, congestive heart failure; DS, Down syndrome; GERD, gastroesophageal reflux disease; GI, gastrointestinal; JVP, Jugular venous pressure; L-CHF, left side congestive heart failure; MS, Marfan’s syndrome; MSK, musculoskeletal; NCCP, Non cardiac chest pain; PE, pulmonary embolus; PHT, pulmonary hypertension; PR, pulse rate; PUD, peptic ulcer disease; R-CHF, right side congestive heart failure; TS, Turner’s syndrome;

Congestive heart failure

Useful terms:

- **Bigeminal pulse**
  - Irregular rhythm, alternating strong and weak beats, due to premature contraction opening aortic valve, premature contraction not opening aortic valve, 3:2 heartblock

- **Buerger’s test for PVD**
  - Elevate legs 45˚ → pallor; lower legs 90˚ → cyanosis

- **Campbell’s sign**
  - Trachea descends with inspiration; seen in acute respiratory distress, COPD, or other causes of severe airway obstruction

- **Hamman’s sign**
  - Mediastinal crunch, timed with systolic and diastolic components of heart beat, due to mediasintal air, such as with a pneumothorax

- **Kussmaul respiration**
  - ↑ Rate and depth of breathing is caused by anion-gab metabolic acidosis (MAKE UPL):
    - Methanol
    - ASA
    - Ketoacidosis
    - Ethylete glycol
    - Uremia
    - Paraldehyde
    - Lactic acidosis

- **Kussmaul’s sign**
  - ↑ JVP on inspiration, in RV failure when JVP is ↑ (on inspiration, JVP normally falls). On inspiration, normally BP ↓, PR↑

- **Pulsus alternans**
  - Regular rhythm, alternative strong and weak beats

- **Pulsus paradoxus**
  - Systolic blood pressure > 10-12 mmHg with inspiration (common is cardiac tamponade [ 98% prevalence] and acute asthma [ < 50% prevalence])

- **Sinus arrhythmia**
  - Normal ↓ PR on expiration

Abbreviations: BP, blood pressure; COPD, chronic obstructive pulmonary disease; JVP, jugular venous pressure; PR, pulse rate; PVD, peripheral vascular disease; RV, right ventricle

Useful background:

- Useful terms

  ➢ Odds that a given symptom or sign is present in a person without the targeted disorder.
    - Sensitivity (SENS) - Likelihood of finding a sign or symptom when the target disorder is present (pid – positive in disease)
    - Specificity (SPEC) - Likelihood of not finding a sign or symptom when the target disorder is not present (nih- negative in health)

  ➢ Odds that a given symptom or sign is present in person with the target disorder (likelihood ratio)
    - LR (>1) = SENS/ 1-SPEC of a present finding in a person with the target disorder
    - LR (<1) of an absent finding in a person with the target disorder = 1-SENS/SPEC

  ➢ Sen N out – Sensitive test; when negative, rules out disease
  ➢ Sp P in – Specific test; when positive, rules in disease

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, page 25.

Useful background: Performance characteristics of findings on history and physical examination in emergency department patients

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial clinical judgment</td>
<td>4.4</td>
<td>0.45</td>
</tr>
<tr>
<td>Past History</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heart failure</td>
<td>5.8</td>
<td>0.45</td>
</tr>
<tr>
<td>Myocardial infarction</td>
<td>3.1</td>
<td>0.69</td>
</tr>
<tr>
<td>Symptoms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Paroxysmal nocturnal dyspnea</td>
<td>2.6</td>
<td>0.70</td>
</tr>
<tr>
<td>Orthopnea</td>
<td>2.2</td>
<td>0.65</td>
</tr>
<tr>
<td>Edema</td>
<td>2.1</td>
<td>0.64</td>
</tr>
<tr>
<td>Physical examination</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Third heart sound (ventricular filling gallop)</td>
<td>11</td>
<td>0.88</td>
</tr>
<tr>
<td>Abdominojugular reflux</td>
<td>6.4</td>
<td>0.79</td>
</tr>
<tr>
<td>Jugular venous distention (JVP)</td>
<td>5.1</td>
<td>0.66</td>
</tr>
</tbody>
</table>
• Rales 2.8 0.51
• Any murmur 2.6 0.81
• Lower extremity edema 2.3 0.64
• Valsalva manoeuvre 2.1 0.41
• Systolic blood pressure < 100 mm Hg 2.0 0.97

Abbreviations: JVP, jugular venous pressure; PLR, positive likelihood ratio; NLR, negative likelihood ratio

Note that many historical points, symptoms and signs on physical examination have a PLR <2 (and are not included here)

➤ Remember the probability (%)

<table>
<thead>
<tr>
<th>PLR</th>
<th>Increase</th>
<th>NLR</th>
<th>Decrease</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>15%</td>
<td>0.5</td>
<td>-15%</td>
</tr>
<tr>
<td>5</td>
<td>30%</td>
<td>0.2</td>
<td>-30%</td>
</tr>
<tr>
<td>10</td>
<td>45%</td>
<td>0.1</td>
<td>-45%</td>
</tr>
</tbody>
</table>

Abbreviations: PLR, positive likelihood ratio; NLR, negative likelihood ratio;

Adapted from: Simel David L, et al. JAMA 2009, Table 16-6.

Useful background: Causes of congestive heart failure (CHF)

➤ LV failure (L-CHF, systolic dysfunction)
  • Inadequate LV filling
    - Mitral stenosis
    - LV diastolic dysfunction (e.g. LVH)
    - Pericardial constriction
  • Pressure overload
    - Aortic stenosis
    - Systemic hypertension
    - Pulmonary hypertension
  • Volume overload
    - Aortic or mitral regurgitation
    - High output heart failure e.g. beri beri, thyrotoxicosis, Paget disease, AV fistula
  • LV muscle disease
    - Myocardial infarction
    - Cardiomyopathy – hypertrophic, dilated, restrictive
    - Myocarditis
RV failure (R-CHF, diastolic dysfunction)
  o Secondary to L-CHF
  o Secondary to pulmonary hypertension ([cor pulmonate] e.g. PEs, chronic lung disease)
  o Mitral stenosis
  o Tricuspid regurgitation
  o Atrial myxoma
  o Congenital heart disease (atrial septal defect)

Abbreviation: AV, aortic valve; CHF, congestive heart failure; L-CHF, left sided congestive heart failure; LV, left ventricle; NSAIDs, nonsteroidal anti-inflammatory drugs; PE, pulmonary embolus; R-CHF, right-sided congestive heart failure; RV, right ventricle

Adapted from: Ghosh A.K.  *Mayo Clinic Scientific Press* 2008, Table 3-36; and Burton J.L. *Churchill Livingstone* 1971.

Useful background: The major and minor Framingham criteria for clinical diagnosis of congestive heart failure.

<table>
<thead>
<tr>
<th>Major</th>
<th>Minor</th>
</tr>
</thead>
<tbody>
<tr>
<td>o PND</td>
<td>o Peripheral edema</td>
</tr>
<tr>
<td>o Orthopnea</td>
<td>o Night cough</td>
</tr>
<tr>
<td>o Increased JVP</td>
<td>o DOE</td>
</tr>
<tr>
<td>o Rales</td>
<td>o Hepatomegaly</td>
</tr>
<tr>
<td>o Third heart sound</td>
<td>o Pleural effusion</td>
</tr>
<tr>
<td>o Chest radiography</td>
<td>o Heart rate &gt;120 beats per minute</td>
</tr>
<tr>
<td>- Cardiomegaly</td>
<td>o Weight loss ≥4.5 kg in 5 days with diuretic</td>
</tr>
<tr>
<td>- Pulmonary edema</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: DOE, dyspnea on exertion; JVP, jugular venous pressure; PND, paroxysmal nocturnal dyspnea


3. Take a directed history and perform a focused physical examination for congestive heart failure (CHF).

 Symptoms
  o Paroxysmal nocturnal dyspnea (PND)
  o Orthopnea (SOB, ie shortness of breath)
  o Dyspnea on exertion (SOBOE, shortness of breath on exertion)
Physical examination
- Jugular venous pressure (JVP) assess
- Peripheral and sacral edema
- S3 (ventricular filling gallop)
- Rales and wheezes
- Cardiac murmur

Chest radiograph
- Pulmonary venous congestion
- Interstitial edema
- Cardiomegaly
- Pleural effusion(s)

Electrocardiogram findings
- Any abnormal result
- Atrial fibrillation

Brain natriuretic peptide
- Most useful when < 100 pg/ml for decreasing the likelihood of CHF

Abbreviations: CHF, congestive heart failure; LVH, left ventricular hypertrophy; LV, left ventricle; MI, myocardial infarction; PE, pulmonary embolus


Useful background: New York heart association (NYHA) functional classification of congestive heart failure

<table>
<thead>
<tr>
<th>Class</th>
<th>Activity evoking angina</th>
<th>Limits to physical activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>II</td>
<td>Ordinary physical activity</td>
<td>Slight</td>
</tr>
<tr>
<td>III</td>
<td>Walking &lt; 2 blocks or &lt; 1 flight of stairs</td>
<td>Marked</td>
</tr>
<tr>
<td>IV</td>
<td>Minimal or at rest</td>
<td>Severe</td>
</tr>
</tbody>
</table>

Abbreviation: NYHA, New York heart association

Source: Filate W., et al. Medical Society, Faculty of Medicine, University of Toronto 2005, Table 3, page 55.
Useful background: Performance characteristics of physical examination for congestive heart failure (CHF).

The physical examination is very useful for the diagnosis of CHF. Relating to the heart rate, tachycardia (> 10 bpm at rest) and an abnormal valsalva response yield positive likelihood ratios (PLR) of 5.5 and 7.6, respectively.

While displacement of the PMI has a PLR of 5.8 and the $S_3$ gallop of 5.7, the age-old JVP has considerable merit, with an $\uparrow$ JVP having a PLR of 3.9, and abdominojugular (aka hepatojugular reflex) of 8.0.

### Elevated Left Heart Filling Pressures Finding

<table>
<thead>
<tr>
<th>Findings</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital signs</td>
<td></td>
</tr>
<tr>
<td>- HR &gt; 100/bpm at rest</td>
<td>5.5</td>
</tr>
<tr>
<td>- Abnormal Valsalva response</td>
<td>7.6</td>
</tr>
<tr>
<td>Lung examination</td>
<td></td>
</tr>
<tr>
<td>- Crackles</td>
<td>NS</td>
</tr>
<tr>
<td>Heart examination</td>
<td></td>
</tr>
<tr>
<td>- Elevated jugular venous pressure</td>
<td>3.9</td>
</tr>
<tr>
<td>- Positive abdominojugular test</td>
<td>8.0</td>
</tr>
<tr>
<td>- Supine, apical impulse lateral to MCL</td>
<td>5.8</td>
</tr>
<tr>
<td>- $S_3$ gallop</td>
<td>5.7</td>
</tr>
<tr>
<td>Legs, sacrum</td>
<td></td>
</tr>
<tr>
<td>- Edema</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Diagnostic standard: For elevated left heart filling pressures, *pulmonary capillary wedge pressure* > 12 mmHg or > 15 mmHg, or left ventricular end diastolic pressure > 15 mmHg

Abbreviations: CHF, congestive heart failure; HJ, hepatojugular; HR, heart rate; PLR, positive likelihood ratio; NLR, negative likelihood ratio; MCL, midclavicular line


### Diagnosis of left ventricular dysfunction (L-CHF)

<table>
<thead>
<tr>
<th>Medical inpatients, including post myocardial infarction</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical diagnosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- ECG abnormal</td>
<td>2.0-3.1</td>
<td>0.41-0.62</td>
</tr>
<tr>
<td>- Outpatients</td>
<td>2.8</td>
<td>0.03</td>
</tr>
</tbody>
</table>
The breathless ER patient’s history
- Patient history
  - Heart failure 5.8 0.45
  - Myocardial infarction 3.1 0.69

Physical examination
- Third heart sound (S₃) 11 0.88
- Abdominojugular reflux 6.4 0.79
- Jugular venous distention 5.1 0.66
- Rales 2.8 0.51

Chest radiograph
- Pulmonary venous congestion 12 0.48
- Interstitial edema 12 0.68
- Alveolar edema 6.0 0.95
- Cardiomegaly 3.3 0.33
- Pleural effusion(s) 3.2 0.81

Electrocardiogram
- Atrial fibrillation 3.8 0.79
- New T wave changes 3.0 0.83
- Any abnormal finding 2.2 0.64

Brain natriuretic peptide (pg/mL)
- >250 4.6
- >150 2.7
- >50 1.7
- <50 0.06

Overall clinical impression
- Initial clinical judgment that the patient is in CHF 4.4 0.45

Abbreviations: CHF, congestive heart failure; ECG, electrocardiogram; HJ, hepatojugular; JVP, jugular venous pressure; L-CHF, left side congestive heart failure; PLR positive likelihood ratio; NLR, negative likelihood ratio.

Adapted from: Simel David L, et al. JAMA 2009, Table 16-12, page 213.
Useful background: Distinguishing diastolic dysfunction from systolic dysfunction

<table>
<thead>
<tr>
<th>Finding</th>
<th>LR for diastolic dysfunction</th>
<th>LR for systolic dysfunction (EF &lt; 45%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Favor of normal systolic function</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Female sex</td>
<td>1.6</td>
<td>0.62</td>
</tr>
<tr>
<td>o Systolic blood pressure ≥160 mm Hg</td>
<td>1.8</td>
<td>0.55</td>
</tr>
<tr>
<td>➢ Favor of systolic dysfunction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Heart rate ≥ 100/min</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Left atrial ECG abnormality</td>
<td>0.42</td>
<td>2.4</td>
</tr>
</tbody>
</table>

Abbreviations: ECG, electrocardiogram; EF, ejection fraction; LR, likelihood ratio


Useful background: Performance characteristics of chest radiograph and electrocardiogram in emergency department patients in CHF

<table>
<thead>
<tr>
<th></th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Chest radiograph</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Pulmonary venous congestion</td>
<td>12</td>
<td>0.48</td>
</tr>
<tr>
<td>o Interstitial edema</td>
<td>12</td>
<td>0.68</td>
</tr>
<tr>
<td>o Alveolar edema</td>
<td>6.0</td>
<td>0.95</td>
</tr>
<tr>
<td>o Cardiomegaly</td>
<td>3.3</td>
<td>0.33</td>
</tr>
<tr>
<td>o Pleural effusion(s)</td>
<td>3.2</td>
<td>0.81</td>
</tr>
<tr>
<td>o Any edema</td>
<td>3.1</td>
<td>0.38</td>
</tr>
</tbody>
</table>

➢ ECG

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>o Atrial fibrillation</td>
<td>3.8</td>
<td>0.79</td>
</tr>
<tr>
<td>o New T wave changes</td>
<td>3.0</td>
<td>0.83</td>
</tr>
<tr>
<td>o Any abnormal findings</td>
<td>2.2</td>
<td>0.64</td>
</tr>
</tbody>
</table>

Note: The presence of pneumonia and hyperinflation on chest x-ray, as well as ECG evidence of either ST elevation or depression have PLRs < 2, are not included here.

Abbreviations: CHF, congestive heart failure; ECG, electrocardiogram; PLR, positive likelihood ratio; NLR, negative likelihood ratio.
Adapted from: Simel David L, et al. JAMA 2009, Table 16-7, page 201.
Useful background: Conditions that prompt hospitalization in heart failure

- Social
  - Lack of outpatient care

- CNS
  - Altered mentation

- Heart
  - Hypotension
  - Dyspnea at rest
  - Significant arrhythmias

- Kidney
  - Worsen renal function
  - Disturbed electrolytes


SO YOU WANT TO BE A CARDIOLOGIST!

Q1: When does the person with chronic left sided (L) congestive heart failure lose their orthopnea (preference to breathing in an upright position)?

A1: Once the L-CHF causes R-CHF, the failure of the RV causes unloading of the LV, relieving the pulmonary congestion.

Q2: 95% of persons with orthopnea will have heart disease, but what pulmonary disease makes up the remaining 5%?

A2: Bilateral, apical bullous disease (COPD)

What is “the best”? The “best signs” on physical examination for CHF are tachycardia, abnormal valsalva response, ↑JVP and positive HJ test, S₃, displaced apex and S₃ gallops.
4. Perform a focused physical examination to distinguish between the presence of left-sided congestive heart failure (L-CHF and e.g. LV failure [LVF]), and right-sided congestive heart failure (R-CHF and e.g. RV failure [RVF]).

<table>
<thead>
<tr>
<th>LVF (L-CHF)</th>
<th>RVF (R-CHF, PHT, cor pulmonale)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Inspection</strong></td>
<td><strong>Tachypnea</strong></td>
</tr>
<tr>
<td>- Dyspnea, tachypnea, orthopnea</td>
<td>- Peripheral cyanosis</td>
</tr>
<tr>
<td>- Pulmonary edema and peripheral cyanosis (low cardiac output)</td>
<td>- Rosy cheeks with blue tinge (mitral facies)</td>
</tr>
<tr>
<td>- Chgyne-Stokes respiration</td>
<td>- Hoarse (PHT-PA compression on L↓ recurrent laryngeal nerve)</td>
</tr>
<tr>
<td>- Caechexia</td>
<td><strong>Palpation</strong></td>
</tr>
<tr>
<td><strong>Blood pressure</strong></td>
<td><strong>Palpation</strong></td>
</tr>
<tr>
<td>- May be reduced</td>
<td>- ↑ JVP, ↑ v waves, HJR</td>
</tr>
<tr>
<td>- Displaced</td>
<td>- JVP, ↑ a waves (PHT)</td>
</tr>
<tr>
<td>- Dyskinetic</td>
<td>- Low volume</td>
</tr>
<tr>
<td>- Palpable gallop</td>
<td>- Kussmaul’s sign</td>
</tr>
<tr>
<td><strong>Auscultation</strong></td>
<td><strong>Auscultation</strong></td>
</tr>
<tr>
<td>- Heart- LV, S₃</td>
<td>- RV – S₃</td>
</tr>
<tr>
<td>- Lungs – Crackles and wheezes</td>
<td>- ↑ P₂ (PHT)</td>
</tr>
<tr>
<td>- Tricuspid regurgitation</td>
<td><strong>Associations</strong></td>
</tr>
<tr>
<td>- RVF, PHT</td>
<td>- Systolic ejection click (PHT)</td>
</tr>
<tr>
<td>- Systemic hypertension</td>
<td>- Systolic ejection murmur (PHT)</td>
</tr>
<tr>
<td>- Valvular disease</td>
<td>- Pulmonary regurgitation (dilation of PA, PHT)</td>
</tr>
<tr>
<td>- Arrhythmia (e.g. AF)</td>
<td>- Peripheral edema</td>
</tr>
<tr>
<td>- Volume depletion</td>
<td>- Tender hepatomegaly</td>
</tr>
<tr>
<td>- Anemia</td>
<td>- Positive HJR</td>
</tr>
<tr>
<td>- Hyperthyroidism</td>
<td>- LVF, volume overload, CAD</td>
</tr>
</tbody>
</table>
Central and jugular venous pressure

Useful background: JVP

- **Suggestions for the physical examination for increased jugular venous pressure (JVP) as a surrogate marker for increased central venous pressure (CVP)**
  - Position 45° upright, neck turned 45° to the side
  - Sternomastoid medial, internal jugular vein (JVP) posterior to carotid artery; lateral, external
  - Height > 3 cm (abnormal)
  - Right-sided pulsations are preferred over left-sided ones when reading the JVP, HJR, Kussmaul’s sign; left sided measurements may be falsely elevated because of kinking of the innominate vein.

- **What features of the JVP does one describe in its assessment?**
  - Height, character of waveform, and results of abdominojugular reflux

- **What is a normal range for JVP?**
  - Normal range is 4-5 cm above the sternal angle.

- **When is the abdominojugular reflux considered abnormal?**
  - Abnormality is indicated when there is a sustained rise in JVP >4 cm after applying abdominal pressure for a minimum of 15-30 seconds.

- **If you knew a person had a very elevated JVP but still wanted to evaluate the JVP pulsations, what could you do?**
  - Use a higher elevation of the bed (>30°) until pulsations are seen.

- **Effect of inspiration on CVP (JVP)**
  - Normal inspiration - ↓JVP

- **Kussmaul’s sign - ↑ JVP on inspiration** (opposite to normal, ie abnormal inspiratory increase in JVP). It occurs because the heart is unable to accommodate the increase in the venous return that accompanies the inspiratory fall in intrathoracic pressure.
  - R-CHF
  - SVC obstruction
TS
Constrictive pericarditis
Restrictive cardiomyopathy

Abbreviations: JVP, jugular venous pressure; R-CHF, right sided congestive heart failure; SVC, superior vena cava; TS, tricuspid stenosis

Adapted from: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, page 57.

Useful background: The physiology of JVP ascents and descents

- **A wave**
  - From right atrium (RA) contraction
  - S₁ and carotid upstroke
  - Coincides with S₄
  - Follows p wave on ECG
  - More permanent than V wave

- **C wave**
  - Poorly visible
  - From both bulging of tricuspid cusps into right atrium, as well as from transmitted carotid pulsation
  - Coincide with ventricular contraction
  - Interval between a and c wave of JVP coincides with P-R interval or RV coinciding with RV contraction
  - Occurs between S₁ and S₂
  - More prominent than Y descent

- **V wave**
  - At end of ventricular systole and at the early phase of ventricular diastole
  - Less prominent than A wave

- **Y descent**
  - At beginning of ventricular diastole
  - Caused by opening of the tricuspid valve and emptying of R. Atrium
  - Corresponds to S₃
  - Less prominent than X descent

Useful background: Causes of Abdomino-(hepato-) jugular reflex (AJR); (Sustained ↑ JVP ≥ 4 cm)

- R-CHF (not in L-CHF)
- Tricuspid regurgitation or stenosis
- Constrictive pericarditis or pericardial tamponade
- IVC obstruction
- Hypervolemia

AJR has 66% sensitivity and 100% specificity distinguishing Tricuspid (TR) from mitral regurgitation (MR) (Note: AJR+ in TR, AJR- in MR).


Useful background: Jugular venous pressure, wave forms, and their relationship to the normal heart sounds

Abbreviations: CCF, congestive cardiac failure; CVP, central venous pressure; HJR, hepatojugular reflex; JVP, jugular venous pressure

Source: Talley N. J., et al. *Maclennan & Petty Pty Limited* 2003, Figure 3.12, page 47.
5. Perform a direct physical examination to distinguish between the JVP and carotid waveforms.

- JVP is defined as the pressure of the internal jugular system and is a direct assessment of the pressure in the right atrium of the heart.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Venous Pulse</th>
<th>Carotid Pulse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Location</td>
<td>Low in neck and lateral</td>
<td>Deep in neck and medial</td>
</tr>
<tr>
<td>Contour</td>
<td>Double-peaked and diffuse</td>
<td>Single-peaked and sharp</td>
</tr>
<tr>
<td>Character</td>
<td>Undulant, not palpable</td>
<td>Force, brisk, easily felt</td>
</tr>
<tr>
<td>Waveform</td>
<td>Diffuse biphasic</td>
<td>Single sharp</td>
</tr>
<tr>
<td>Positional change</td>
<td>Varies with position</td>
<td>No variation</td>
</tr>
<tr>
<td>Respiratory variation</td>
<td>Height falls on inspiration</td>
<td>No variation</td>
</tr>
<tr>
<td>Effect of palpation</td>
<td>Wave nonpalpable, pressure obliterate pulse, vein fills</td>
<td>Pulse palpable, not compressible</td>
</tr>
<tr>
<td>Abdominal pressure</td>
<td>Displaces pulse upward</td>
<td>Pulse unchanged</td>
</tr>
</tbody>
</table>

Abbreviation: JVP, jugular venous pressure


Useful background: Causes of elevation of jugular venous pulse (JVP)

- SVC
  - SVC obstruction

- RA
  - RA thrombus, tumour, Bernheim effect
  - ↑ RA filling pressure

- TR
  - Tricuspid stenosis
  - Giant 'a' waves, cannon waves, tricuspid incompetence

- RV
  - ↓ RV filling
    - RV failure
    - RV infarction
    - Constrictive pericarditis
    - Cardiac tamponade
  - ↓ RV compliance
Lung  
- Coughing, valsalva manoeuvre  
- Pleural or pericardial effusion

Circulation  
- Increased blood volume  
- Bradycardia  
- Hyperdynamic circulation

Abbreviations: RA, right atrium; RV, right ventricle; SVC, superior vena cava

Useful background: Abnormalities of the venous waveforms

<table>
<thead>
<tr>
<th>Waveform</th>
<th>Cardiac Condition</th>
</tr>
</thead>
</table>
| a waves  | - Absent - Atrial fibrillation, sinus tachycardia  
|          | - Flutter - Atrial flutter  
|          | - Prominent - First degree AV block  
|          | - Large - TS  
|          | - Right atrial myxoma  
|          | - PHT  
|          | - PS  
|          | - Cannon - AV dissociation  
|          | - Ventricular tachycardia |
| X descent | - Absent - TR  
|          | - Prominent - Conditions causing enlarged a waves  
|          | - Large cv waves - TR  
|          | - Constrictive pericarditis |
| Y descent | - Slow - TS  
|          | - Rapid - Right atrial myxoma  
|          | - Constrictive pericarditis  
|          | - Severe R-CHF  
|          | - TR  
|          | - ASD  
|          | - Absent - Cardiac tamponade |

Abbreviations: ASD, atrial septal defect; AV, atrioventricular; PHT, pulmonary hypertension; PS, pulmonic stenosis; R-CHF, right sided congestive heart failure; TR, tricuspid regurgitation, TS, tricuspid stenosis

Useful background: Kussmaul’s sign

- Paradoxical increase in JVP with inspiration (normally with inspiration JVP falls)

- Causes
  - R-CHF
  - Restrictive cardiomyopathy (eg. sarcocidosis, hemochromatosis, amyloidosis)
  - Tricuspid stenosis
  - SVC syndrome
  - RV infarction (33-100%)

Abbreviations: RV, right ventricle; SVC, superior vena cava

Useful background: Performance characteristics of inspection of the neck veins.

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated venous pressure at the bedside</td>
<td></td>
</tr>
<tr>
<td>o Detecting measured CVP &gt; 8 cm H₂O</td>
<td>9.0</td>
</tr>
<tr>
<td>o Detecting measured CVP &gt; 12 cm H₂O</td>
<td>10.4</td>
</tr>
<tr>
<td>o Detecting elevated left heart diastolic pressures</td>
<td>3.9</td>
</tr>
<tr>
<td>o Detecting low left ventricular ejection fraction</td>
<td>7.9</td>
</tr>
<tr>
<td>o Predicting postoperative pulmonary edema</td>
<td>11.3</td>
</tr>
<tr>
<td>o Predicting post-operative myocardial infarction or cardiac death</td>
<td>9.4</td>
</tr>
</tbody>
</table>

- Positive abdominojugular test
  - Detecting elevated left heart diastolic pressures                    | 8.0 |

Abbreviation: CVP, central venous pressure; PLR, positive likelihood ratio; NLR, negative likelihood ratio; NS, not significant


**Acute coronary syndromes**

6. Take a focused history to determine the risk factors for coronary artery disease.

- Patient
  - Demography
    - Age
    - Male sex
    - Family history of premature CAD (<55 in men, <65 for...
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- Hypertension
- Diabetes
- Metabolic syndrome
- Stress and depression
- Socioeconomic factors

- Smoking
- Sedentary lifestyle
- Obesity

- Increased LDL: cholesterol level
- Low HDL cholesterol level
- Inflammatory markers (e.g. CRP, C-reactive protein)
- Small, dense LDL
- Lipoprotein (a)
- Homocysteine
- Fibrinogen

Abbreviations: CAD, coronary artery disease; HDL, high-density lipoprotein; LDL, low-density lipoprotein


Useful background: Very rare coronary artery causes of MI

- Embolism (thrombus, infected vegetation)
- Thrombosis (spontaneous, prothrombotic states)
- Aneurysm (e.g. Kawasaki disease as a child)
- Spasm (drugs e.g. cocaine)
- Arteritis (SLE, PAN, Takayasu)
- Anomalous coronary artery
- Dissection, spontaneous

Abbreviations: MI, myocardial infarction; SLE, systemic lupus erythematositis; PAN, polyarteritis nodosa.

Adapted from: Davey P. Wiley-Blackwell 2006, pages 150 and 156.
Useful background: Chest pain ‘PQRSTU-A’

- **Position** /location of pain
- **Quality**: crushing (e.g. ‘like someone standing on my chest’), dull, burning (suggests epigastric origin), pressing, squeezing, throbbing, knife like (sharp pain suggests chest wall/MSK pain)
- **Radiation** (i.e. does the pain radiate? Where?)
- **Severity**: scale of 1-10 (1=mild discomfort, 10= worst pain ever had)
- **Timing**: onset, duration and course
- **Uniqueness** of recent symptoms (i.e. inquire if there was anything different that prompted the patient to seek help e.g. increased duration or severity) precipitating and alleviating factors
- **Associated symptoms**: dyspnea, nausea, cough, palpitations, sweating

Abbreviation: MSK, musculoskeletal

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, page 123.

7. Take a directed history for chest pain.

- **Cardiovascular**
  - Coronary artery disease (angina; acute myocardial infarction [STEMI], acute coronary syndrome [ACS; anstable angina, NSTEMI]), aortic aneurysm/dissection, pericarditis (including constrictive pericarditis, cardiac tamponade)

- **Pulmonary**
  - Pneumothorax, pleurisy, pulmonary embolus, pneumonia

- **Gastrointestinal**
  - Esophagitis, peptic ulcer, pancreatitis, cholecystitis

- **Musculoskeletal**
  - Costochondrodynia, muscle spasm, nonspecific chest wall pain

- **Other**
  - Anxiety, herpes zoster

Abbreviation: MI, myocardial infarction; STEMI, ST elevation MI; NSTEMI, non ST elevation MI

Useful background: CCS functional classification of angina

<table>
<thead>
<tr>
<th>Class</th>
<th>Activity evoking angina</th>
<th>Limits to physical activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Prolonged exertion</td>
<td>None</td>
</tr>
<tr>
<td>II</td>
<td>Walking &gt; 2 blocks or &gt; 1 flight of stairs</td>
<td>Slight</td>
</tr>
<tr>
<td>III</td>
<td>Walking &lt; 2 blocks or &lt; 1 flight of stairs</td>
<td>Marked</td>
</tr>
<tr>
<td>IV</td>
<td>Minimal or at rest</td>
<td>Severe</td>
</tr>
</tbody>
</table>

Abbreviation: CCS, Canadian Cardiovascular Society

Source: Simel David L, et al. JAMA 2009, Table 2, page 55.

8. Take a focused history of clinical features increasing the likelihood of an acute coronary syndrome (ACS).

- **History**
  - Profile male over 70 y
  - Pain
    - Accelerating ischemic symptoms over 48 h
    - Ongoing rest pain for > 20 min
    - Recurrent ischemic pain during observation
  - Diabetes

- **Physical examination**
  - S3
  - Hypotension
  - Pulmonary edema
  - Peripheral vascular disease
  - Severe arrhythmia
  - Sweating
  - Nausea/vomiting

- **Investigation finding would include**
  - Pathologic Q waves; abnormal ST segments; T wave inversion >0.02 mV; ST-segment depression > 0.05 mV and increased cardiac biomarkers

Adapted from: Ghosh A.K. Mayo Clinic Scientific Press 2008, Table 3-25 and Table 3-26, page 101.
9. Perform a focused physical examination to determine if the person with an acute coronary syndrome more likely had disease of the LAD versus the RCA.

- **LAD**
  - Lung
    - Dyspnea
    - Orthopnea
    - Basal crackles
    - Cough
    - Hemoptysis
  - CNS
    - Fatigue
    - Syncope
  - Periphery
    - Hypotension
    - Cool extremities
    - Peripheral cyanosis

- **RCA**
  - JVP
    - Elevated JVP
    - Positive hepatojugular reflux
  - Liver
    - Hepatic tenderness
    - Hepatomegaly
    - Pulsatile liver
  - Periphery
    - Peripheral edema

Abbreviations: JVP, jugular venous pressure; LAD, left anterior descending artery; L-CHF, left side congestive heart failure; RCA, right coronary artery; R-CHF, right side congestive heart failure.


---

**SO YOU WANT TO BE A CARDIOLOGIST!**

**Q:** In the context of peripheral vascular disease, what is Buerger’s test?

**A:** Blanching” upon raising legs and “rubor” on dependency
Useful background: Clinical features that increase probability of myocardial infarct (MI)

PLR

➢ History
  o Pain in chest or left arm 2.7
  o Chest pain radiating to
    - Right shoulder 2.9
    - Left arm 2.3
    - Left and right arm 7.1
  o Chest pain most important symptom 2.0
  o Previous history of MI 1.5-3.0
  o Pleuritic chest pain
    Chest pain sharp or stabbing 0.3
  o Positional chest pain 0.3
  o Chest pain reported by palpation 0.2-0.4
  o Nausea or vomiting 1.9
  o Diaphoresis 2.0

➢ Physical exam
  o Third heart sound on auscultation 3.2
  o Hypotension (systolic BP <80 mm Hg) 3.1
    Pulmonary crackles on auscultation 2.1

➢ ECG
  o New ST segment elevation > 1 mm 5.7-53.9
  o New Q wave 5.3-24.8
  o Any ST segment elevation 11.2
  o New conduction deficit 6.3
  o New ST segment depression 3.0-5.2
  o Any Q wave 3.9
  o Any ST segment depression 3.2
  o T wave peaking and/or inversion >1 mm 3.1
  o New T wave inversion 2.4-2.8
  o Any conduction defect 2.7

Abbreviations: ECG, electrocardiography; PLR, positive likelihood ratio; MI, myocardial infarct

Adapted from: Panju AA et al. JAMA 1998; 280:1256-63; Table 35.11, page 413.; Simel David L, et al. JAMA 2009, Chapter 35, Table 35-5 and Table 35-6, page 467 and Table 35-8, page 472.
**Peripheral vascular disease**

Useful background: Common causes of leg pain.

---

### Claudication vs. Pseudoclaudication

<table>
<thead>
<tr>
<th>Character</th>
<th>Claudication</th>
<th>Pseudoclaudication</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cramp, ache</td>
<td>“Parasthetic” pins and needles</td>
</tr>
<tr>
<td>Bilateral</td>
<td>+/-</td>
<td>+</td>
</tr>
<tr>
<td>Onset</td>
<td>Walking</td>
<td>Walking &amp; Standing</td>
</tr>
<tr>
<td>Walking distance</td>
<td>Constant</td>
<td>Variable</td>
</tr>
<tr>
<td>Relief</td>
<td>Standing still</td>
<td>Sitting down, leaning forward</td>
</tr>
</tbody>
</table>


---

"Science is never cast in stone and ideas are written with a finger on shifting sand."

Anonymous
11. Take a directed history for lower leg ulcers.

- **Vein**
  - Stasis, with pigmentation and stasis eczema around lateral malleoli

- **Artery**
  - Large vessel: atherosclerosis, thrombangitis obliterans
  - Small vessel: vasculitis (diabetes mellitus [DM], rheumatoid arthritis [RA], sickle cell disease)
  - Nerve: Peripheral neuropathy: DM, syphilis

- **Skin**
  - Benign: Pyoderma gangrenosum, staph. aureus, TB, fungus
  - Malignant: basal cell, squamous, lymphoma, melanoma, Kaposi’s sarcoma

Abbreviations: DM, diabetes mellitus; RA, rheumatoid arthritis; TB, tuberculosis


12. Take a directed history and perform a focused physical examination of the four most common types of lower leg ulcer.

<table>
<thead>
<tr>
<th>Feature</th>
<th>Venous</th>
<th>Arterial</th>
<th>Arteriolar</th>
<th>Neurotrophic</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Onset</td>
<td>Trauma +/-</td>
<td>Trauma</td>
<td>Spontaneous</td>
<td>Trauma</td>
</tr>
<tr>
<td>Course</td>
<td>Chronic</td>
<td>Progressive</td>
<td>Progressive</td>
<td>Progressive</td>
</tr>
<tr>
<td>Pain</td>
<td>No (unless infected)</td>
<td>Yes</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Physical (ulcer)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Location</td>
<td>Medial aspects of leg</td>
<td>Toe, heel, foot</td>
<td>Lateral, posterior aspect of foot</td>
<td>Plantar</td>
</tr>
<tr>
<td>Surrounding skin</td>
<td>Stasis changes</td>
<td>Atrophic</td>
<td>Normal</td>
<td>Callous</td>
</tr>
<tr>
<td>Ulcer edges</td>
<td>Shaggy</td>
<td>Discrete</td>
<td>Serpiginous</td>
<td>Discrete</td>
</tr>
<tr>
<td>Ulcer base</td>
<td>Healthy</td>
<td>Eschar, pale</td>
<td>Healthy or pale</td>
<td></td>
</tr>
</tbody>
</table>

13. Take a directed history for and perform a focused physical examination for peripheral vascular disease (arterial and venous insufficiency) in the lower extremities.

- **History**
  - **Claudication**
    - Leg claudication
    - Location and severity of pain at rest, on exertion, at night
    - Onset/offset
    - Distance to develop claudication
    - Aching in lower legs, especially when dependent
    - Parathesia
  - **Impotence**
  - **Associated conditions/risk factors**
    - Hypertension
    - Hyperlipidemia
    - Hyperhomocysteinemia
    - Obesity
    - Diabetes
    - Physical inactivity
    - Smoking
    - Causes of L/R - CHF
    - Family history
    - Personal past history of CAD, PVD, rheumatic fever, cardiac murmur, cardiac surgery, cardiac events, medications

- **Physical examination**
  - **Inspection**
    - Pulses
      - Compare femoral, popliteal, tibial, dorsalis, pedis pulses; carotid, radial, brachial, abdominal aorta and renal arteries
      - Asymmetrical foot coolness
      - Pallor on leg elevation
      - Redness on leg dependency (positive Buerger's test)
      - Muscle atrophy
      - Bruits, thrills of abdominal aorta and femoral arteries
    - Limbs
      - Size
      - Symmetry
      - Edema
      - Muscle atrophy
    - Skin
      - Colour/pigmentation
      - Texture
      - Loss of hair on toes
- Ulcers/scars
- Gangrene
- Nails (colour, texture)
- Venous distribution (engorgement, varicosities)

- **Palpation**
  - Temperature (compares both limbs)
  - Capillary refill (compares both limbs)
  - Edema (compares both limbs)
  - Pulses (rate, rhythm, amplitude, waveform)
  - (Examined: carotid, radial, brachial, abdominal aorta; renal, femoral, popliteal, dorsalis pedis, tibial)
  - Pitting edema

- **Auscultation**
  - Bruits (carotid, abdominal aorta, renal, iliac, femoral)

- **Special manoeuvres**
  - Leg elevation for pallor
  - Dependency test for dusky rubor

- **Tests of arterial insufficiency**
  - Ankle/brachial index – compare palpated systolic BP values in brachial and either dorsalis pedis or posterior tibial arteries (normal A/B >1)
  - Capillary refill time
  - Venous filling time
  - Auscultatory bruit

Abbreviations: BP, blood pressure; CAD, coronary artery disease; L/R-CHF, left-/ right-sided congestive cardiac failure; PVD, peripheral vascular disease


Useful background: Grading system for lower extremity arterial occlusive disease (AOD), using ankle – to – brachial systolic pressure index

<table>
<thead>
<tr>
<th>Grade of AOD</th>
<th>Supine Resting</th>
<th>Post exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>1.0-1.4</td>
<td>No change or increase</td>
</tr>
<tr>
<td>Mild disease</td>
<td>0.8-0.9</td>
<td>&gt; 0.5</td>
</tr>
<tr>
<td>Moderate disease</td>
<td>0.5-0.8</td>
<td>&gt; 0.2</td>
</tr>
<tr>
<td>Severe disease</td>
<td>&lt; 0.5</td>
<td>&lt; 0.2</td>
</tr>
</tbody>
</table>

14. Take a focused history and perform a directed physical examination to distinguish between chronic vs acute (critical) ischemia.

<table>
<thead>
<tr>
<th>Pain</th>
<th>Acute</th>
<th>Chronic</th>
</tr>
</thead>
<tbody>
<tr>
<td>At rest</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>With exercise</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Predictable distance</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Relief with rest</td>
<td>-</td>
<td>+</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Examination</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Ulcers</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Gangrene</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Bruits</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>


Useful background: Performance characteristics of physical examination for peripheral vascular disease (PVD)

- While the previously taught physical findings of atrophic skin, absent lower limb hair and capillary refill time ≥ 5 seconds all have positive likelihood ratios of < 2, other traditional signs have considerable merit.

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inspection</td>
<td></td>
</tr>
<tr>
<td>Wounds or sores on foot</td>
<td>7.0</td>
</tr>
<tr>
<td>Foot colour abnormally pale, red, or blue</td>
<td>2.8</td>
</tr>
<tr>
<td>Atrophic skin</td>
<td>1.7</td>
</tr>
<tr>
<td>Absent lower limb hair</td>
<td>1.7</td>
</tr>
<tr>
<td>Palpation</td>
<td></td>
</tr>
<tr>
<td>Foot asymmetrically cooler</td>
<td>6.1</td>
</tr>
<tr>
<td>Absent femoral pulse</td>
<td>6.1</td>
</tr>
<tr>
<td>Absent posterior tibial and dorsalis pedis pulses</td>
<td>14.9</td>
</tr>
<tr>
<td>At least one pedal pulse present</td>
<td></td>
</tr>
<tr>
<td>Auscultation</td>
<td></td>
</tr>
<tr>
<td>Limb bruit present</td>
<td>7.3</td>
</tr>
<tr>
<td>Ancillary tests</td>
<td></td>
</tr>
<tr>
<td>Capillary refill time ≥5 seconds</td>
<td>1.9</td>
</tr>
<tr>
<td>Venous filling time &gt; 20 seconds</td>
<td>3.6</td>
</tr>
</tbody>
</table>

Abbreviations: PLR, positive likelihood ratio; NLR, negative likelihood ratio

Useful background: How pressure sores are graded.

- Grade I: erythema, skin intact.
- Grade II: skin loss, epidermis or dermis (abrasion, blister, shallow crater).
- Grade III: full thickness loss and damage to subcutaneous tissues.
- Grade IV: extensive destruction, tissue necrosis or damage to the underlying muscle or bone.


15. Take a directed history and perform a focused physical examination to differentiate between arterial vs venous insufficiency.

<table>
<thead>
<tr>
<th></th>
<th>Arterial insufficiency</th>
<th>Venous insufficiency</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>History of pain</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Location</td>
<td>Toes, points of previous trauma, lateral malleoli</td>
<td>Medial and lateral malleoli</td>
</tr>
<tr>
<td>o Pain</td>
<td>Intermittent claudication (exercise pain), rest pain</td>
<td>None, or ache in lower legs on dependency</td>
</tr>
<tr>
<td>o Paraesthesia</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>o Paralysis</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td><strong>Physical examination</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Skin</td>
<td>Shiny, atrophic</td>
<td>Brown pigment</td>
</tr>
<tr>
<td></td>
<td>No hair</td>
<td>Thick skin</td>
</tr>
<tr>
<td></td>
<td>Gangrene</td>
<td>(scarring of skin)</td>
</tr>
<tr>
<td></td>
<td>Thick, ridged nails</td>
<td>Skinny leg</td>
</tr>
<tr>
<td>o Palor</td>
<td>White (leg up), red (leg down)</td>
<td>Normal, or blue (leg down)</td>
</tr>
<tr>
<td>o Palor (cold)</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>o Pitting edema</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>o Pulses</td>
<td>↓</td>
<td>Normal</td>
</tr>
<tr>
<td>o Bruit</td>
<td>Yes</td>
<td>No</td>
</tr>
</tbody>
</table>


What is the best? The “best tests” for diagnosing PVD from physical examination are wounds or sores on foot, abnormal foot colour or coolness, absent pulses, limb bruit and venous filling time >20 seconds.
Useful background: Interpretation of findings

<table>
<thead>
<tr>
<th>Possible pathology</th>
<th>Finding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial</td>
<td></td>
</tr>
<tr>
<td>Raynaud’s disease</td>
<td>- Sharply demarcated pallor in fingers that changes over several minutes</td>
</tr>
<tr>
<td></td>
<td>- Normal wrist pulses</td>
</tr>
<tr>
<td>Arterial insufficiency</td>
<td>- Ulcers:</td>
</tr>
<tr>
<td></td>
<td>- Distal aspects of foot</td>
</tr>
<tr>
<td></td>
<td>- Painful</td>
</tr>
<tr>
<td></td>
<td>- Rapidly developing</td>
</tr>
<tr>
<td></td>
<td>- Often erythematous when infected</td>
</tr>
<tr>
<td>Chronic arterial</td>
<td>- Cool, pale extremity with hair loss</td>
</tr>
<tr>
<td>insufficiency</td>
<td></td>
</tr>
<tr>
<td>Vasculitis</td>
<td>- Headache, temple soreness</td>
</tr>
<tr>
<td></td>
<td>- Changes in skin colour and temperature</td>
</tr>
<tr>
<td></td>
<td>- Swelling</td>
</tr>
<tr>
<td>Venous</td>
<td></td>
</tr>
<tr>
<td>Superficial phlebitis</td>
<td>- Warmth, painful to touch</td>
</tr>
<tr>
<td></td>
<td>- Erythema due to inflammation of tissue around the vein</td>
</tr>
<tr>
<td>Acute DVT</td>
<td>- Pain secondary to inflammation in the absence of superficial changes</td>
</tr>
<tr>
<td></td>
<td>- Swelling of distal part of the extremity</td>
</tr>
<tr>
<td>Venous obstruction</td>
<td>- Prominent veins in an edematous limb</td>
</tr>
<tr>
<td>Chronic venous</td>
<td>- Skin:</td>
</tr>
<tr>
<td>insufficiency</td>
<td>- Warm and erythematous</td>
</tr>
<tr>
<td></td>
<td>- Thickened skin (woody)</td>
</tr>
<tr>
<td></td>
<td>- Increased pigmentation</td>
</tr>
<tr>
<td></td>
<td>- May have brownish ulcers around the ankles</td>
</tr>
</tbody>
</table>

Abbreviation: DVT, deep vein thrombosis

Printed with permission: McGee S. R. *Saunders/Elsevier* 2007, Table 1, page 249.
Postural orthostatic hypotension and hypovolemia

Useful background: Postural (orthostatic) hypotension

- With a change in body position
- Systolic BP decreases (>15 mmHg)
- Diastolic BP decreases (>0-10 mmHg)
- And/or heart rate increases (>20 bpm).
- Seen in conditions of autonomic dysfunction or volume depletion.

Abbreviation: BP, blood pressure


To examine a patient for orthostatic hypotension measure BP in supine patient, then have the patient sit up with the legs down or have patient stand for 2 minutes before reassessing BP.

16. Take a directed history for the causes of postural hypotension.

- Hypovolemia
  - Bleeding, dehydration
- Drugs
  - Vasodilators, diuretics, anticholinergics (including TCAs)
- Endocrine
  - Diabetes, Addison’s disease, hypopituitarism
- Autonomic neuropathy
  - Diabetes, amyloidosis, Shy-Drager syndrome
- Idiopathic

Abbreviation: TCAs, tricyclic antidepressants.

Adapted from: Talley N. J., et al. Maclellan & Petty Pty Limited 2003, Table 3.6, page 44.
Achieving Excellence in the OSCE Part 1 © A.B.R Thomson

Useful background: predicting the severity of blood loss causing hypovolemia.

<table>
<thead>
<tr>
<th>Supine to standing, PR ↑ 30 bpm</th>
<th>Sensitivity %</th>
<th>Specificity %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate blood loss &lt; 630 mL</td>
<td>22</td>
<td>98</td>
</tr>
<tr>
<td>Severe blood loss &gt; 630 mL</td>
<td>97</td>
<td>-</td>
</tr>
</tbody>
</table>

Abbreviation: bpm, beats per minute


**Peripheral edema**

Useful background: Causes of peripheral edema

- Pitting bilateral lower limb edema
  - Cardiac
    - Congestive heart failure
    - Constrictive pericarditis
  - Hepatic
    - Cirrhosis
  - Renal
    - Renal failure
    - Nephrotic syndrome
  - Gastrointestinal tract
    - Malabsorption
    - Starvation
    - Protein losing enteropathy
  - Beri Beri (wet)
  - Cyclic edema
  - Drugs
    - NSAIDs
    - Calcium channel blockers

- Unilateral
  - Deep venous thrombosis
  - Compression of large veins by tumour or lymph nodes
  - Lymphatic obstruction
  - Venous obstruction (usually DVT; rarely, external compression)
  - Venous valve incompetence from previous DVT
  - Cellulitis
  - Ruptured Baker’s cyst
  - Localized immobility (e.g. hemiparesis)

- Non-pitting lower limb edema
  - Hypothyroidism
Lymphedema
- Infectious (e.g. filariasis)
- Malignant (tumour invasion of lymphatics)
- Congenital (lymphatic development arrest)
- Allergy

Idiopathic
- Milroy’s disease (unexplained lymphedema which appears at puberty and is more common in females)

Abbreviations: DVT, deep vein thrombosis; NSAIDs, nonsteroidal anti-inflammatory drugs.


17. Take a directed history to determine the cause of lower leg edema.

- Heart
  - R-CCF, constrictive pericarditis, Beri Beri

- Thyroid
  - Hypothyroidism (myxedema)

- GI lumen
  - Maldigestion/malabsorption, or protein-losing enteropathy

- Liver
  - Portal hypertension with hypoalbuminemia

- Lymphatic obstruction
  - Tumor, allergy, infection, idiopathic (Milroy’s disease)

- Deep vein obstruction

Abbreviation: GI, gastrointestinal; R-CCF, right-sided congestive cardiac failure


- Perform a focused physical examination to differentiate between venous edema versus lymphedema.
Useful background: Differential diagnosis of types of edema

<table>
<thead>
<tr>
<th>Feature</th>
<th>Venous</th>
<th>Lymphedema</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bilateral</td>
<td>+/-</td>
<td>+/-</td>
</tr>
<tr>
<td>Foot involved</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Toes involved</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td>Thicken skin</td>
<td>0</td>
<td>+</td>
</tr>
<tr>
<td>Stasis</td>
<td>+</td>
<td>0</td>
</tr>
</tbody>
</table>


**Peripheral pulses**

Useful background: Description of characteristic pulses

<table>
<thead>
<tr>
<th>Pulse</th>
<th>Description</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulsus parvus</td>
<td>Small volume, weak pulse from ↓ LVS V</td>
<td>- Hypovolemia</td>
</tr>
<tr>
<td>(Hypokinetic pulse)</td>
<td></td>
<td>- LV failure</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Shock</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- MI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Restrictive pericardial disease</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Arrhythmia</td>
</tr>
<tr>
<td>Pulsus tardus</td>
<td>Small volume, slowly rising pulse</td>
<td>- Aortic stenosis</td>
</tr>
<tr>
<td></td>
<td>Delayed with respect to heart sounds</td>
<td></td>
</tr>
<tr>
<td>Hyperkinetic pulse</td>
<td>Strong, bounding pulse</td>
<td>- Heart block</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Hyperdynamic circulation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Fever</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Anemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Exercise</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Anxiety</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Reduced peripheral resistance</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Patent ductus arteriosus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Arteriovenous fistula</td>
</tr>
</tbody>
</table>
Collapsing
- Quick rise, quick fall
- ↑ CO

Waterhammer
- Quick rise, full expansion, quick fall
- AR

Bisferiens
- Double peaked pulse, mid systolic dip
- AR, AS
- Hypertrophic cardiomyopathy

Alternans
- Alternating amplitude of pulses
  (More easily detected in conjunction with blood pressure measurement)
- CHF

Abbreviations: LVSV, left ventricular stroke volume; LV, left ventricle; MI, myocardial infarction; SV, stroke volume; CHF, congestive heart failure; CO, cardiac output; AR, Aortic regurgitation; AS, Aortic stenosis

Source: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto 2005, Table 3 and Table 4, pages 251 and 252.

18. Perform a focused physical examination for pulsus paradoxus (exaggeration of normal fall [> 20 mm Hg] in SBP with inspiration).

Heart
- L-/R- CHF
- Pericarditis ± tamponade
- AR
- ASD

Lung
- COPD

Abbreviation CHF, congestive heart failure; AR, Aortic regurgitation; ASD, atrial septal defect

Adapted from: Mangione S. Hanley & Belfus 2000, pages 30 to 31, 63 to 66.
SO YOU WANT TO BE A CARDIOLOGIST!

Q: What is a “Spike and dome bifid pulse”?
A:  
  o First peak from rapid early-systolic emptying of ventricle, then an obstruction, followed by another emptying (second peak).
  o Association with severe HOCM


Useful background: Abnormal arterial pulse patterns

[Diagram showing various pulse patterns: ECG, Normal, Waterhammer, Bisferiens, Alternans, Anacrotic (Pulsus Parvus et Tardus), Paradoxical]

Source: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, page 252.

Useful background: Causes of absent radial pulse

- Aberrant radial artery or congenital anomaly (check the brachials and blood pressure)
- Artery tied off at surgery or previous surgical cut-down
- Catheterization of the brachial artery with poor technique
- Following a radial artery line for monitoring of blood gases or arterial pressure
- Blalock-Taussig shunt on that side (shunt from subclavian to pulmonary artery)
- Embolism into the radial artery (usually due to atrial fibrillation)

Arrhythmias

Useful background: Abnormalities in the pulse rate

- **Bradyarrhythmias** -
  - Sinus bradycardia
  - Sick sinus syndrome
  - Junctional and ventricular escape rhythms

- **Conduction delays** -
  - 1°, 2°, or 3° AV nodal block
  - Fascicular block
  - Bundle branch block

- **Irregular Tachyarrhythmias** -
  - Sinus arrhythmia
  - Atrial fibrillation
  - Multifactorial atrial tachycardia
  - Atrial flutter with variable block
  - Atrial or ventricular premature beats
  - Extrasystoles (ventricular or supra-ventricular)
  - 2° heart block
  - Ventricular fibrillation
  - Irregularity of volume also occurs in pulsus paradoxus and pulsus alternans

- **Regular tachyarrhythmias - narrow complex:** -
  - Supraventricular tachycardia
  - Atrial flutter
  - Wolfe-Parkinson-White syndrome
  - AV node re entry tract

- **Wide complex:** -
  - Supraventricular tachycardia with aberrance or bundle branch block
  - Ventricular tachycardia, torsades de pointes

- **Unstable arrhythmia** -
  - Arrhythmia plus hypotension, dyspnea, chest pain, presyncope, or syncope.

Abbreviation: AV, atrioventricular; 1°/2°/3° – first, second or third degree heart block

19. Perform a focused physical examination to determine the cause of atrial fibrillation.

- **Heart**
  - Mitral valve disease
  - IHD
  - HBP
  - Constrictive pericarditis
  - Cardiomyopathy
  - ASD
  - Ebstein's anomaly

- **Lung**
  - Chronic pulmonary disease

- **Thyroid**
  - Thyrotoxicosis

- **Ideopathic**
  - 'Lone fibrillation'

Abbreviation: IHD, ischemic heart disease; HBP, hypertension (systemic); ASD, atrial septal defect


---

**SO YOU WANT TO BE A CARDIOLOGIST!**

Q: In which conditions may the pulse rate in one arm differ from that in the other?

A: Usually, slowing of the pulse on one side occurs distal to the aneurysmal sac. Thus, an aneurysm of the transverse or descending aortic arch causes a retardation of the left radial pulse. Also, the artery feels smaller and is more easily compresses than usual. An aneurysm of the ascending aorta or common carotid artery may result in similar changes in the right radial pulse.

Useful background: The effect of exercise and vagal stimulation on tachyarrhythmias

<table>
<thead>
<tr>
<th>Atrial</th>
<th>Vagal stimulation</th>
<th>Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>o flutter</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>o Fibrillation</td>
<td>-</td>
<td>↑</td>
</tr>
<tr>
<td>o PAT</td>
<td>↓</td>
<td></td>
</tr>
<tr>
<td>o APC</td>
<td>-</td>
<td>↓</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Sinus</th>
<th>Vagal stimulation</th>
<th>Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>o Bradycardia</td>
<td>-</td>
<td>↑</td>
</tr>
<tr>
<td>o Arrhythmia</td>
<td>-</td>
<td>↓</td>
</tr>
<tr>
<td>o 2⁰ HB</td>
<td>-</td>
<td>↑</td>
</tr>
<tr>
<td>o 3⁰ HB</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: APC, atrial premature contraction; HR, heart rate; PAT, paroxysmal atrial tachycardia; 2⁰/3⁰ HB second/third degree heart block


SO YOU WANT TO BE A CARDIOLOGIST!

Q1: What is the difference between the bronchial pulse in ventricular versus supraventricular aortic stenosis (AS)焗?
A1: In ventricular AS, R=L brachial pulse. In supraventricular AS, the left brachial pulse has slow uptake (pulsus tardus), but the right brachial pulse is normal (L < R).


Q2: What causes a rapid arterial upstroke when input and cardiac pulse pressure are normal?焗
A2: o VSD, mitral regurgitation
    o HOCM (hypertrophic obstructive cardiomyopathy)

A rapid arterial upstroke occurs with high output states (eg. anemia, exercise, thyrotoxicosis, pregnancy, beriberi, Paget’s disease; AV fistulas
20. Perform a focused physical examination for the causes of bradycardia/heart block.

- **Classification**
  - Sinoatrial block
  - AV block:
    - 1°, P.R>0.2 sec
    - 2° Mobitz (fixed PR)
    - Wenkebach (varying PR)
    - High grade (2:1, 3:1, etc)
    - 3° complete
  - Bundle branch block
  - Short PR with long QRS (Wolff-Parkinson-White)

- **Heart**
  - Athletes
  - Sleep
  - Apparent (pulse deficit [PR<HR] in AF, ventricular bigeminy)
  - Block (3° AV block, or type I or II 2° AV block)
  - Myocardial infarction (MI; ischemic heart disease)
  - Vasovagal episode
  - Cardiomyopathy
  - Sinus arrhythmia (↓PR with expiration)

- **MSK**
  - Collagen vascular disease

- **Hypothermia**

- **Hypothyroidism**

- **Hepatic**
  - Severe jaundice

- **Head**
  - Increased intracranial pressure
  - Trauma

- **Drugs**
  - B-blockers, digoxin, amiodarone

- **Tachycardia**

- **Heart**
  - CCF
    - Constrictive pericarditis
    - Myocardial infarction (MI)
    - Myocarditis
- Aortic stenosis
- Hypertension

○ Rhythm
  - Supraventricular tachycardia
  - Ventricular tachycardia – hyperthyroidism, acute hypoxia or hypercapnea, sick sinus syndrome
  - Atrial flutter with 2:1 AV block, or with variable block
  - Multifocal atrial tachycardia

○ Hyperdynamic circulation
  - Exercise
  - Emotion
  - Fever
  - Hypolemia
  - Anemia
  - Hyperthyroidism
  - Pregnancy
  - AV fistula (Paget’s disease)
  - Beriberi

○ Drugs
  - Anticholinergics
  - Sympathomimetics

Abbreviations: AF, arterial fibrillation; AV, atrioventricular; CCF, congestive cardiac failure; HR, heart rate; MI, myocardial infarction; PR, pulse rate;


SO YOU WANT TO BE A CARDIOLOGIST!

Q: In the context of peripheral vascular disease, what is De Weese’s test?
A: Disappearance of palpable distal pulses after exercise
Palpation of precordium

Useful background: Apical (pericardial) impulse (PMI, point of maximal impulse)

- Double or even triple apical impulses occur in HOCM
- Pericardial impulse in mitral stenosis represents palpable S1 and S2 (from P2 components), opening snap, and diastolic thrill (patient in left lateral decubitus position).
- Precordial impulse in tricuspid regurgitation: palpable S2 (from P2 component) over pulmonic area, RV parasternal, pulsatile synchrony with each cardiac systole.
- Ectopic apical impulse (superior and medially): angina/previous MI, LV aneurysm, LV dyskinesia.


SO YOU WANT TO BE A CARDIOLOGIST!

Q1: What is the difference between pulsus alternans, pulsus bisferiens, and pulsus parvus?
A1: 
- Pulsus alternans: strong-weak, strong-weak peripheral artery strength due to severe LV dydfunction.
  - Pulsus bisferien’s are palpable peaks in systole, with fast upstroke and downstroke, with high pulse amplitude.
    - Occurs in severe aortic regurgitation (AR), and may be associated with concurrent aortic stenosis (AR+AS)
    - Pulsus bisforien’s once LV dydfunction occurs
  - Pulsus parvus (hypokinetic pulse of the low amplitude)
    - Aortic stenosis
    - Mitral stenosis
    - Cardiomyopathy
    - ↓ LV filling or contraction

Q2: What is the difference between pulsus parvus plus tardus, versus hyperkinetic pulse?
A2: 
- Pulsus parvus plus pulsus tardus (low amplitude plus slow upstroke) usually means presence of aortic stenosis
  - Hyperkinetic pulse (rapid upstroke, high amplitude): wide pulse pressure, aortic regurgitation. Normal pulse pressure, mitral regurgitation
Useful background: Performance characteristics of size and position of palpable apical impulse

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>POSITION OF APICAL BEAT</td>
<td></td>
</tr>
<tr>
<td>Supine apical impulse lateral to MCL</td>
<td></td>
</tr>
<tr>
<td>o Detecting cardiothoracic ratio &gt;0.5</td>
<td>3.4</td>
</tr>
<tr>
<td>o Detecting low ejection fraction</td>
<td>10.1</td>
</tr>
<tr>
<td>o Detecting increased left ventricular end-diastolic volume</td>
<td>8.0</td>
</tr>
<tr>
<td>o Detecting pulmonary capillary wedge pressure &gt;12 mmHg</td>
<td>5.8</td>
</tr>
<tr>
<td>o Detecting increased left ventricular end-diastolic volume</td>
<td>4.7</td>
</tr>
</tbody>
</table>


SO YOU WANT TO BE A CARDIOLOGIST

Q: What is the difference between “a snap”, “a click”, “a knock” and “a rub”?
A: o “Snap” – diastole, abnormal opening of the leaflets.
   o “Click” – systole, prolapse and backward ballooning of valve leaflet(s).
   o “Knock” (pericardial) - Louder and higher-pitched form of S3 (caused by early ventricular filling), and sudden stretching of the LV against a thick, calcified pericardium, or also heard in constrictive pericarditis. Occur in chronic calcified or constrictive pericarditis
   o “Rub” (pericardial) - High-pitched, scratchy systolic and diastolic sounds, best heard with firm pressure of diaphragm, heard best at lower (3/4 interspaces) sterna border during inspiration, due to acute pericarditis

Useful background: Performance characteristics of palpation of the precordium

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperkinetic apical movement</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Detecting associated mitral regurgitation or aortic valve disease in patients with mitral stenosis</td>
<td>11.2</td>
<td>0.3</td>
</tr>
<tr>
<td>Sustained apical movement</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Detecting severe aortic stenosis in patients with aortic flow murmurs</td>
<td>4.1</td>
<td>0.3</td>
</tr>
<tr>
<td>o Detecting moderate-to-severe aortic regurgitation in patients with basal early diastolic murmurs</td>
<td>2.4</td>
<td>0.1</td>
</tr>
<tr>
<td>o Detecting right ventricular peak pressure &gt; 50 mm Hg</td>
<td>3.6</td>
<td>0.4</td>
</tr>
<tr>
<td>o Palpable P$_2$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Detecting pulmonary hypertension in patients with mitral stenosis</td>
<td>3.6</td>
<td>0.05</td>
</tr>
<tr>
<td>o Absence of palpable P2</td>
<td></td>
<td>0.05</td>
</tr>
<tr>
<td>Varying intensity of S1, detecting AV dissociation in the presence of tachycardia</td>
<td>24.4 (probability &gt; 50%)</td>
<td>0.4</td>
</tr>
<tr>
<td>Palpable P2, detecting PHT</td>
<td>3.6 (probability ~25% ↑)</td>
<td></td>
</tr>
<tr>
<td>Position of apical beat - Supine apical impulse lateral to MCL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Detecting cardiothoracic ratio &gt;0.5</td>
<td>3.4</td>
<td>0.6</td>
</tr>
<tr>
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<tr>
<td>o Detecting increased left ventricular end diastolic volume</td>
<td>8.0</td>
<td>0.7</td>
</tr>
<tr>
<td>o Detecting pulmonary capillary wedge pressure &gt;12 mm Hg</td>
<td>5.8</td>
<td>NS</td>
</tr>
<tr>
<td>Size of apical beat - Apical beat diameter ≥4 cm in left lateral decubitus position at 45 degrees</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Detecting increased left ventricular end diastolic volume</td>
<td>4.7</td>
<td>NS</td>
</tr>
</tbody>
</table>
Abbreviations: AV, atrioventricular; EF, ejection fraction; PLR, positive likelihood ratio; NLR, negative likelihood ratio; MCL, midclavicular line; NS, not significant; PHT, pulmonary hypertension


*Note that a supine displaced apical impulse has a PLR < 2, and is not included here.

**Heart sounds**

➤ S1 and S2

Useful background: The first and second heart sounds.

Useful background: Special positions and manoeuvres for optimal auscultation of heart sounds

<table>
<thead>
<tr>
<th>Position</th>
<th>Effect on heart sounds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting upright, leaning forward, holding exhalation</td>
<td>↑AS, AR, pericardial rubs</td>
</tr>
<tr>
<td>Left lateral decubitus (LLD) (use bell of stethoscope)</td>
<td>S₃, S₄, MS</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Manoeuvre</th>
<th>Physiological effect</th>
<th>Effect on heart sounds</th>
</tr>
</thead>
<tbody>
<tr>
<td>Leg elevation</td>
<td>↑Venous return</td>
<td>↑Right sided murmurs, TR, PS</td>
</tr>
<tr>
<td>Fist clenching</td>
<td>↑Systemic arterial resistance</td>
<td>↑Some left sided murmurs MR, AR, VSD; ↓ AS</td>
</tr>
<tr>
<td>Squatting</td>
<td>↑Venous return, ↑vascular tone</td>
<td>↓MVP, HCM; ↑ AS</td>
</tr>
<tr>
<td>Standing (opposite to squatting)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: AS, aortic stenosis; AR, aortic regurgitation; HCM, hypertrophic cardiomyopathy; MR, mitral regurgitation; MS, mitral stenosis; MVP, mitral valve prolapse; PS, Pulmonary stenosis; TR, tricuspid regurgitation; VSD, ventricular septal defect

Adapted from: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, Table 12, page 63.

21. Give the disease processes associated with a loud, variable or soft intensity of S₁.

- General concepts
  - Normal closure of MV and TV
  - Sequence: mitral (M₁) and then tricuspid (T₁) close, pulmonary and then aortic valves open
  - Apex high pitched
○ ↑ S₁
  - ↑ HR
  - Hyperkinetic heart (eg. AR, PDA, AV fistulas, fever, anemia, thyrotoxicosis, beriberi, Paget’s disease)
  - ↑ LAP (early MS)
  - ↓ PR interval (S₁ becomes softer with AV blocks; pre-excitation syndromes such as WPW [Wolff-Parkinson-White]
  - ↓ PR interval (<160 msec)
  - ↑ Thickness of AV or TV leaflets (when leaflets later become rigid or fixed, S₁ becomes softer or absent)
  - ↑ AV pressure gradient

○ ↓ S₁
  - ↑ PR interval (> 20 msec)
  - LV dysfunction
  - LBBB
  - 1st degree heart block
  - Calcified AV
  - Acute AI (premature closure of MV)
  - MI, TI
  - CHF

○ Variable S₁
  - AF
  - Conduction heart block
  - Progressive increase in duration of PR interval (Wenckeback phenomenon) with mitral stenosis, mitral valve prolapsed with regurgitation with
    - MS
    - MV prolapsed with regurgitation

○ Split S₁: RBBB/LBBB
  - Listen with diaphragm over apex for mitral component (M₁), and over epigastric/subxiphoid area for less important tricuspid component (T₁)
  - S₁ should be louder than S₂ in these locations

Abbreviation: AF, Atrial fibrillation; AI, aortic insufficiency; AV, atrio-ventricular; CHF, congestive heart failure; HB, heart block; LAP, left atrial pressure; MS, mitral stenosis; MV, mitral valve; PDA, patent ductus arteriosus; TV, tricuspid valve

Adapted from: Mangione S. Hanley & Belfus 2000, page 207.
- Variable intensity of $S_1$

- Variations of intensity (loudness) are what is the most useful clinically

- Second-degree heart block (type 1 Wenckebach phenomenon; $S_2$ remains of constant intensity, $S_1$ progressively softens)

- Third-degree (complete) AV block (change in intensity of $S_1$ is random and chaotic; rhythm is slow and regular).

- Atrial fibrillation (rhythm is regularity irregular)

- Pulsus alternans

- Soft intensity of $S_1$
  - Calcified mitral stenosis
  - Long P-R interval
  - L-CHF
  - Severe aortic or mitral regurgitation
  - Myocardial infarction
  - LBBB

- Wide splitting of $S_1$
  - Best heard over left lower sternal border
  - Usually from delayed closure of tricuspid valve ($T_1$), such as from RBBB
  - Wide splitting of $S_2$ may also occur with wide splitting of $S_1$

- Pseudosplitting of $S_1$
  - Normal $S_1$ proceeded by $S_4$ (heard only at apex, low-pitched, soft heard best with bell of stethoscope)
  - $S_1$ may be followed by early systolic ejection click (loudest over the base, high-pitched, loud heard best with diaphragm).

Useful background: Second Heart Sound, $S_2$ (“dub”)

- **General concepts**
  - Heard best with the diaphragm over base of heart in the pulmonary area (Left 2nd/3rd parasternal intercostals spaces).
  - Produced by the sudden slowing of blood from with closure of the aortic ($A_2$) and then pulmonary ($P_2$) valves; $P_2$ can be heard normally only a few centimetres to the left of the upper sternal border.
  - Splitting of $S_2$ and the various forms of splitting is not useful clinically; heard best (because of hearing $P_2$) at the 2-3 left interspace.
  - Heard best a few centimetres to the left of the sternal border.

- **Loudness:** a loud $A_2$, or a loud $P_2$
  - Pulmonary or systemic or systemic hypertension
  - Correlation of the aorta
  - High-output states
  - a soft $A_2$, or soft $P_2$ – aortic or pulmonary valve stenosis
  - $P_2$ louder than $A_2$ – aortic or pulmonary valve stenosis
  - $S_2$ louder than $S_1$ at apex – pulmonary or systemic hypertension

- **Single splitting of $S_2$** ($A_2$ and $P_2$ cannot be head as distinct sounds, so merge into a single sound)
  - Paradoxical (reversed) splitting (splitting in expiration)
  - Pulmonary hypertension
  - Emphysema (hyperinflated lungs muffle $P_2$, so only $A_2$ is heart; $P_2$ is however still produced, so this is “pseudoparadoxical” splitting)


Useful background: The performance characteristics of $S_1$ and $S_2$ findings

- **Paradoxic splitting** is of no significance in detecting aortic stenosis, and a loud $P_2$ is not a significant test to detect a mean pulmonary arterial pressure $\geq 50$ mm Hg (pulmonary hypertension, PHT)

- $S_1$ of varying intensity has a positive likelihood ratio (PLR) of 24.4 to detect AV dissociation. $S_2$ with fixed wide splitting has a PLR of .6 for detecting ASD, whereas a palpable $P_2$ has a PLR of 3.6 for detecting PHT
Finding  PLR

- **S₁**
  - Varying intensity, detecting AV dissociation 24.4

- **S₂**
  - Fixed wide splitting, detecting ASD 2.6
  - Paradoxic splitting of S₂, detecting AS (peak gradient >50 mm Hg); and
  - Loud P₂, detecting PHT (mean PAP ≥50 mm Hg) *
  - Palpable P₂, detecting PHT 3.6

Note: *Not mentioned here because their PLR was < 2

Abbreviations; AS, aortic stenosis; ASD, atrial septal defect; AV, Atrioventricular; PLR, positive likelihood ratio; NLR, negative likelihood ratio; PAP, pulmonary arterial pressure; PHT, pulmonary hypertension; PLR, positive likelihood ratio; S₁, first heart sound; S₂, second heart sound


22. Perform a focused physical examination of the heart sounds. Explain what underlying cardiac abnormalities may be determined from this examination.

- **S₁**
  - Mitral and tricuspid valve closure (MV before TV); synchronous with cardiac impulse beat or carotid impulse
  - Beginning of LV and RV systole
  - Loud in MS and TS, tachycardia, hypertrophy, 3˚ heart block;
  - Soft in MR, 1˚ HB, LBBB, CCF, shock; variation in intensity with any cardiac arrhythmia
  - Normally split in tricuspid area on inspiration
  - Splitting (wider on inspiration) – RBBB

- **S₂**
  - Aortic and pulmonary valve closure (AV closes before PV, but PV opens before AV) end of LV and RV systole, beginning of diastole
  - Loud A₂ in HBP, loud P₂ in PHT; soft A₂ when AV calcified or in AR
  - Normal splitting of S₂ on inspiration (A₂, AV; P₂); pathological splitting: PS, MR, RBBB, VSD, ASD
  - P₂ > A₂ in youth; A₂ > P₂ in old age
o Fixed splitting of $S_2$ (not increasing normally on inspiration – ASD sudden opening of valve in MS or TS after $S_2$

o Normally split on inspiration (especially in children)

o Loud narrowly split P2 in pulmonary hypertension

o Soft widely split P2 in pulmonary stenosis

o Widely split in RBBB

o Widely split fixed P2 in ASD

o Paradoxically split P2 (narrows on inspiration) in LBBB and rarely in aortic stenosis and L and R shunts

➢ Opening snap

  o Indicates mobile AV valve and LA pressure

  o Mitral opening snap in mitral stenosis is maximal internal to apex, louder during expiration, thereby differentiated from split P2

➢ LV – $S_3$

  o Mild diastolic gallop, louder on expiration: physiological; young persons due to rapid diastolic filing; pathological: (↓ RV compliance)

  o LVF, AR, MR, VSD, PDA

➢ LV – $S_4$

  o Late diastole, from atrial contraction (disappears in AF)

  o AS, MR, HBP, CAD, old age

➢ RV – $S_4$

  o PHT, PS (↓ RV compliance)

➢ $S_3 + S_4$

  o Summation gallop (when HR > 120 bpm)

➢ Artificial

  o Prosthetic heart valves, pacemaker sounds

➢ $P_2$ increased in pulmonary hypertension (e.g.; MS, MR, L-CHF, PE, pulmonary fibrosis)

➢ $A_2$ increased in systemic hypertension, aortic sclerosis, arteriosclerosis, syphilitic aortitis; decreased in R-CCF, hypotension, severe anemia

➢ Diaphragm or bell

  o Diaphragm: High pitched, high pressure gradient across a small surface (AR)

  o Bell: Low pitched, low pressure gradient across a wide surface (MS)
Pansystolic murmurs
  o MR, TR; VSD (L-4th ICS); PDA) L-2nd ICS)

Abbreviations: A2, aortic part of S2; AI, aortic incompetence; AR, aortic regurgitation; AS, aortic stenosis; ASD, atrial septal defect; AV, aortic valve; CAD, coronary artery disease; CCF, congestive cardiac failure; HB, heart block; HBP, hypertension (systemic); HR, heart rate; ICS, intercostal space; LA, left atrium; LBBB, left bundle block; L-CCF, left-sided congestive cardiac failure; L-CHF, left side congestive heart failure; LV, left ventricle; LVF, left ventricular failure; MI, mitral incompetence; MR, mitral regurgitation; MS, mitral stenosis; MV, mitral valve; P2, pulmonary part of P2; PDA, patent ductus arteriosis; PE, pulmonary embolus; PHT, pulmonary hypertension; PS, pulmonary stenosis; PV, pulmonary valve; RBBB, right bundle branch block; R-CCF, right-sided congestive cardiac failure; RV, right ventricular; S1, first heart sound; S2, second heart sounds; TS, tricuspid stenosis; TV, tricuspid valve; VSD, ventricular septal defect


23. Perform an auscultatory physical examination for signs of pulmonary hypertension.

- Loud (even palpable) P2 over pulmonic area
- Loud S4, right side
- Pulmonary ejection sound
- Tricuspid regurgitation
- Inspiration widens the interval between closure of aortic and pulmonary valves
- Inspiration increases the distance between A2 and P2, and thus causes physiological (normal) splitting of S2.
- Physiological splitting of S2 is increased by lying down. Physiological splitting of S2 occurs in about half of adults.
- Paradoxical splitting of S2: A split S2 in the sitting/standing patient who breathes out (expiratory) is likely to be pathological (i.e. not physiological); however wide splitting of S2 may be normal in young adults.
  - ASD, VSD with pulmonary hypertension
  - Pulmonary stenosis
  - Pulmonary hypertension
  - Massive pulmonary embolism

24. Perform a focused physical examination for the causes of fixed splitting of S₂ (splitting of S₂ which persists on supine → expiration).

25. Perform a directed physical examination of abnormal S₂ splitting to detect the presence of associated pathological abnormalities.

<table>
<thead>
<tr>
<th>Splitting and pathogenesis</th>
<th>Associations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wide physiologic</td>
<td></td>
</tr>
<tr>
<td>o P₂ late</td>
<td></td>
</tr>
<tr>
<td>- Electrical delay of RV systole</td>
<td>RBBB</td>
</tr>
<tr>
<td>- Prolongation of RV systole</td>
<td>Acute cor pulmonale</td>
</tr>
<tr>
<td>- Increased hangout interval</td>
<td>Dilation of PA</td>
</tr>
<tr>
<td>o A₂ early</td>
<td></td>
</tr>
<tr>
<td>- Shortening of LV systole</td>
<td>MR</td>
</tr>
<tr>
<td>Wide and fixed</td>
<td></td>
</tr>
<tr>
<td>o Increased hangout interval or prolongation of RV systole</td>
<td>ASD</td>
</tr>
<tr>
<td>o Prolongation of RV systole</td>
<td>R-CHF</td>
</tr>
<tr>
<td>Paradoxic</td>
<td></td>
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<tr>
<td>o A₂ late</td>
<td></td>
</tr>
<tr>
<td>- Electrical delay of LV systole</td>
<td>LBBB</td>
</tr>
<tr>
<td>- Prolonged LV systole</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: AS, aortic stenosis; ASD, atrial septal defect; IHD, ischemic heart disease; LBBB, left bundle branch block; L-CHF, left side congestive heart failure; LV, left ventricular; MR, mitral regurgitation; RBBB, right bundle branch block; PA, pulmonary artery; PS, pulmonary stenosis; R-CHF, right side congestive heart failure; RV, right ventricular; RV systole and LV systole refer to the duration of right and left ventricular contraction.

SO YOU WANT TO BE A CARDIOLOGIST!

Q: Distinguish between RBBB and LBBB, by listening to the heart sounds(!)
A:  
  o RBBB: $A_2$-$P_2$ – when moving stethoscope from cardiac base to apex, the second component of $S_2$ dissappears; associated with wide splitting of $S_2$.
  
  o LBBB: $P_2$-$A_2$ – first component of $S_2$ becomes softer when moving stethoscope from the base to the apex of the heart ($A_2$ and not $P_2$ is heard at the apex).

SO YOU WANT TO BE A CARDIOLOGIST!

Q: How do you differentiate between the fourth heart sound, a split first heart sound and an ejection click?
A: The fourth heart sound is not heard when pressure is applied on the chest piece of the strethoscope, but pressure does not eliminate the ejection sound or the slitting of the first heart sound.

SO YOU WANT TO BE A CARDIOLOGIST!

Q: What are the implications of the third heart sound in patients with valvular heart disease?
A:  
  o In patients with mitral regurgitation, they are common but do not necessarily reflect ventricular systolic dysfunction or increase filling pressure.
  
  o In patients with aortic stenosis, third heart sounds are uncommon but usually indicate the presence of systolic dysfunction and raised filling pressure.
Pathological heart sounds

- S3 and S4

Useful background: Miscellaneous Heart Sounds.

> S3 and S4

- S3/S4 are best heard and felt at the apex (or PMI) with patient in left lateral decubitus position and accentuated by sitting, standing, exercise, leg elevation, abdominal pressure.
- Early diastolic snap of MS/TS; opening plop of myxoma of mitral or tricuspid valve; (varies from cycle to cycle) and pericardial knock

> All about S3

- Caused by sudden and abnormal deceleration in left ventricular flow at the end of its rapid filling phase, reflecting early and passive LV filling.
- May be physiologic under the age of 45, or with tachycardia, fever, exercise, anemia, hyperthyroidism, pregnancy, anxiety.
- A pathologic S3 “keeps bad company”
- Ken-tu’cky: S3 gallops (S₃+S₁/S₂); Ten-ne-ss’es: S4 gallop (S₄+S₁/S₂)
- Pathological S3 is due to diastolic overload (increased LV preload), decreased myocardial contractility, or loe ejection fraction (low-output failure)
- An S₃ (pathologic) plus an early diastolic rumble suggests sudden increased flow across the mitral valve.
- The pressure of S₃ rules out mitral stenosis; opening snap of MS is left sterna border, S₃ loudest at apex.

- $S_3$ predicts post-surgical development of CHF, predicts cardiac risk during non-cardiac surgery, predicts response to digitalis in treatment of CHF hypertension.
- Increased LV preload (diastolic overload): VSD, PDA ($S_3$ softens with the development of increased pulmonary mitral regurgitation)

Source: Mangione S. *Hanley & Belfus* 2000, pages 221 to 224.

- **All about $S_4$**
  - Late diastolic, low-pitched extra sound due to progressive loss of compliance of ventricles, corresponding to atrial and sudden tension of the AV valve ventricle with atrial contraction and stronger atrial boost, with an increased LV diastolic pressure
  - $S_4$ disappears in atrial fibulation or flutter, or with development of L-CHF
  - Causes include
    - Systemic or pulmonary hypertension
    - Aortic stenosis, particularly with a high gradient
    - Coarctation of the aorta
    - HOCM (almost always associated with $S_4$)
    - Coronary artery disease, including during myocardial infarction (~ 90%)
  - An audible $S_4$ may be physiologic, but a palpable $S_4$ is always pathologic


- **Opening Snap**
  - An early diastolic sound caused by opening and stretching of a stenotic mitral or tricuspid valve
  - The earlier the opening snap (OS) of mitral stenosis, the worse the stenosis; tachycardia also makes the OS earlier
  - Softening in intensity of the opening snap
    - Severe mitral stenosis (OS is present in 75-90% of MS)
    - CHF
    - Large right atrium (eg. pulmonary hypertention)
    - $P_2$ is loudest at the base, OS is loudest at the apex: if you think you hear an OS at the base, it's likely a $P_2$ (split $S_2$); if the sound becomes wider and louder on breathing out, you are probably hearing an OS, not a split $S_2$ or loud $P_2$ from PHT

- **Mitral or Tricuspid Valve Myxoma**
  - Diastolic prolapsed of a left atrial myxoma through an open valve, or a right atrial prolapsed through an open tricuspid valve
  - Vary in intensity and quality from cycle to cycle
Ejection Sounds

- Best hear at apex, sitting position or expiration
- Hyperdynamic heart syndrome – fever, anemia, pregnancy, shunts, hyperthyroidism
- Stenosis semilunar valve (valvular, not sub – or supraventricular aortic or pulmonary stenosis) or bicuspid valve
- Sudden early ventricular systolic distention of aorta or pulmonary artery
- Ejection sound plus an ejection murmur occurs with stenosis of semilunar bicuspid valve with post stenosis dilation of aorta or root of pulmonary artery
- Best heard at the apex
- Loss of click with aortic stenosis reflects progressive fibrosis and calcification of aortic valve, with increasing gradient across aortic valve.


Useful background: Performance characteristics of the third (S₃) and fourth (S₄) heart sounds

- S₃ has good performance characteristics in terms of its positive likelihood ratio for detecting or predicting a range of cardiac abnormalities ranging from ↑ LV filling pressures or reduced ejection fraction, to predicting a myocardial infarction or postoperative cardiac death.
  
  In contrast, the S₄ does not have a significant use to detect ↑ LV filling pressure or aortic stenosis, but has a PLR of 3.2 for predicting 5-year mortality in patients after a myocardial infarction.

<table>
<thead>
<tr>
<th>Finding (Ref)</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>The third heart sound – S₃</td>
<td></td>
</tr>
<tr>
<td>- Detecting ejection fraction &lt;0.5</td>
<td>3.4</td>
</tr>
<tr>
<td>- Detecting ejection fraction &lt;0.3</td>
<td>4.1</td>
</tr>
<tr>
<td>- Detecting elevated left heart filling pressures</td>
<td>5.7</td>
</tr>
<tr>
<td>- Detecting elevated BNP level</td>
<td>10.0</td>
</tr>
<tr>
<td>- Detecting myocardial infarction in patients with acute chest pain</td>
<td>3.2</td>
</tr>
<tr>
<td>- Predicting postoperative pulmonary edema</td>
<td>14.6</td>
</tr>
<tr>
<td>- Predicting postoperative myocardial infarction or cardiac death</td>
<td>8.0</td>
</tr>
<tr>
<td>The fourth heart sound – S₄</td>
<td></td>
</tr>
<tr>
<td>- Predicting 5-year mortality in patients after myocardial infarction</td>
<td>3.2</td>
</tr>
</tbody>
</table>
Detecting elevated left heart filling pressures, or
Detecting severe aortic stenosis

Note: *Not mentioned here because their PLR was < 2

Abbreviation: PLR, positive likelihood ratio


Useful background: Heart sounds

<table>
<thead>
<tr>
<th>Sound</th>
<th>Location, pitch</th>
<th>Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Early ejection sound</td>
<td>o Aortic: apex &amp; base</td>
<td>o Congenital AS,</td>
</tr>
<tr>
<td></td>
<td>o Pulmonic: base (high</td>
<td>bicuspid AV, congenital PS, Ao.</td>
</tr>
<tr>
<td></td>
<td>pitched)</td>
<td>Root or PA dilation,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>physiologic (flow murmur)</td>
</tr>
<tr>
<td>➢ Mid to late ejection click</td>
<td>o Mitral: apex</td>
<td>o MV or TV prolapse</td>
</tr>
<tr>
<td></td>
<td>o Tricuspid: LLSB (high</td>
<td></td>
</tr>
<tr>
<td></td>
<td>pitched)</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: AF, arterial fibrillation; AS, aortic stenosis; AV, aortic valve; HR, heart rate; LAP, left arterial pressure; LBBB, left bundle branch block; LLSB, left lower sternal border; MS, mitral stenosis; MV, mitral valve; PA, pulmonary artery; PR, pulse rate; PS, pulmonary stenosis; RBBB, right bundle branch block; TV, tricuspid valve

Source: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, pages 59 and 60.

SO YOU WANT TO BE A CARDIOLOGIST!
Q: Does the fourth heart sound denote heart failure, like the S3 gallop does?
A: No
Useful background: Systolic and diastolic normal heart sounds

<table>
<thead>
<tr>
<th>Timing</th>
<th>Systolic Name</th>
<th>Diastolic Name</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early systolic</td>
<td>Ejection sounds (aortic or pulmonary)</td>
<td>Early diastolic</td>
</tr>
<tr>
<td></td>
<td>Click (mitral or tricuspid)</td>
<td>Opening snap (mitral or tricuspid)</td>
</tr>
<tr>
<td></td>
<td>Aortic prosthetic valve sounds</td>
<td>Early S3</td>
</tr>
<tr>
<td>Mid-to-late systolic</td>
<td>Click (mitral or tricuspid)</td>
<td>Mid diastolic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>S3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Summation sound (S3 + S4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>S4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pacemaker sound</td>
</tr>
</tbody>
</table>


**Heart murmurs**

Useful background: Golden, Silver, Bronze and Tin Rules of Cardiac Murmurs

- **Gold**
  - Judge murmurs by the company they keep
  - A soft or absent S2 is pathological

- **Silver**: Pathologic murmurs
  - All diastolic murmurs
  - All holosystolic or late systolic murmurs
  - All continuous murmurs (span the entire cardiac cycle)

- **Bronze**: mechanism of production of murmurs
  - Abnormal size, shape, edge of the area through which flow is occurring
  - Low blood viscosity
  - Hyperdynamic heart syndrome

- **Tin**
Murmur that extends into S2 is usually pathologic; a murmur in early or mid systole is usually benign and due to ejection through semilunar aortic and pulmonary valves


Useful background: Likelihood ratios of individual findings for identifying systolic murmurs that are clinically significant*

<table>
<thead>
<tr>
<th>Clinical Sign</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic thrill</td>
<td>12</td>
<td>0.73</td>
</tr>
<tr>
<td>Holosystolic murmur</td>
<td>8.7</td>
<td>0.19</td>
</tr>
<tr>
<td>Loud murmur</td>
<td>6.5</td>
<td>0.08</td>
</tr>
<tr>
<td>Plateau-shaped murmur</td>
<td>4.1</td>
<td>0.48</td>
</tr>
<tr>
<td>Loudest at the apex</td>
<td>2.5</td>
<td>0.84</td>
</tr>
</tbody>
</table>

Abbreviations: CI, confidence interval; LR, likelihood ratio; PLR, positive likelihood ratio, NLR, negative likelihood ratio.

*Moderate to severe aortic stenosis or mitral regurgitation, congenital shunt, or intraventricular pressure gradient.

Note that radiation of the carotids have a PLR < 2, and is not included here.

Adapted from: Simel David L, et al. JAMA 2009, Table 33-14, page 96.

SO YOU WANT TO BE A CARDIOLOGIST!

Q1: What are the causes of the third heart sound (S3)?
A1:  
   ➢ Physiological: in normal children and young adults
   ➢ Pathological
      o Heart failure
      o Left ventricular dilatation without failure: mitral regurgitation, ventricular septal defect, patent ductus arteriosus
      o Right ventricular S3 in right ventricular failure, tricuspid regurgitation

Useful background: Murmurs

- The length and intensity of a murmur do not necessarily reflect severity
- Diastolic murmurs usually represent valvular disease
- Systolic murmurs are caused by:
  - Structural valve disease
  - Dilation of heart valve (e.g., LV dilation – AR, MR), or dilation of large vessel (dilation of aorta from atherosclerosis; dilation of pulmonary artery from PHT)
  - Pressure difference
  - Rapid flow
  - Ruptured papillary muscle, or the (VSD)
  - Floating tissue – bacterial endocarditis
- Intensity
  - High pitched - large pressure difference across small orifice (e.g. AR)
  - Low pitched - small pressure difference across large orifice (e.g. MS)

Abbreviations: AR, aortic regurgitation; LV, left ventricle; MR, mitral regurgitation; MS, mitral stenosis; PHT, pulmonary hypertension; VSD, ventricular septal defect

Useful background: Murmur grades

<table>
<thead>
<tr>
<th>Grade (out of &quot;6&quot;)</th>
<th>Intensity</th>
<th>Thrill</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Very faint, often not audible in all positions or by beginners</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>Quiet, usually audible by all listeners</td>
<td>-</td>
</tr>
<tr>
<td>3</td>
<td>Moderately loud</td>
<td>-</td>
</tr>
<tr>
<td>4</td>
<td>Loud</td>
<td>+</td>
</tr>
<tr>
<td>5</td>
<td>Very loud, audible with stethoscope partly off chest</td>
<td>+</td>
</tr>
<tr>
<td>6</td>
<td>Loudest, audible with stethoscope removed from contact with chest</td>
<td>+</td>
</tr>
</tbody>
</table>

Source: Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, Table 9, page 61.

26. Perform a focused physical examination of the precordium, and from the timing of the murmur, give the differential of the lesion.

<table>
<thead>
<tr>
<th>Timing of murmur</th>
<th>Differential of the lesion</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Systolic</td>
<td></td>
</tr>
<tr>
<td>➢ Pan</td>
<td>o MR</td>
</tr>
<tr>
<td></td>
<td>o TR</td>
</tr>
<tr>
<td></td>
<td>o VSD</td>
</tr>
<tr>
<td></td>
<td>o Aortopulmonary shunts</td>
</tr>
<tr>
<td>➢ Mid</td>
<td>o AS</td>
</tr>
<tr>
<td></td>
<td>o Pulmonary stenosis (PS)</td>
</tr>
<tr>
<td></td>
<td>o Hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td></td>
<td>o ASD</td>
</tr>
<tr>
<td>➢ Late</td>
<td>o Mitral valve prolapse</td>
</tr>
<tr>
<td></td>
<td>o Papillary muscle dysfunction (due usually to ischemia or hypertrophic cardiomyopathy)</td>
</tr>
<tr>
<td>• Diastolic</td>
<td></td>
</tr>
<tr>
<td>➢ Early</td>
<td>o AR</td>
</tr>
<tr>
<td></td>
<td>o Pulmonary regurgitation (PR)</td>
</tr>
<tr>
<td>➢ Mid</td>
<td>o MS</td>
</tr>
<tr>
<td></td>
<td>o TS</td>
</tr>
<tr>
<td></td>
<td>o Atrial myxoma</td>
</tr>
<tr>
<td></td>
<td>o Austin Flint murmur of AR</td>
</tr>
</tbody>
</table>

Achieving Excellence in the OSCE Part 1 © A.B.R Thomson
Carey Coombs† murmur of acute rheumatic fever

- Late (Presystolic)
  - MS
  - TS
  - Atrial myxoma

- Continuous
  - PDA
  - AS+AR
  - MS+AR
  - MS+PR
  - Venous hum
  - Aortopulmonary septal defect
  - Rupture of sinus of Valsalva into RV or RA
  - ‘Mammary soufle’ (in late pregnancy or early postpartum period)
  - Bronchial artery anastamosis in pulmonary atresia
  - Pericardial friction rub

NB: The combined murmurs of aortic stenosis and aortic regurgitation, or mitral stenosis and mitral regurgitation, may sound as if they fill the entire cardiac cycle, but are not continuous murmurs by definition.

Venous hum: above clavicle and down over upper sternum: ↑ by turning head, ↓ by jugular compression; distinguish from PDA (patient ductus arteriosus)

Abbreviations: AR, aortic regurgitation; AS, aortic stenosis; ASD, atrial septal defect; MR, mitral regurgitation; MS, mitral stenosis; PDA, patent ductus arteriosus; PR, pulmonary regurgitation; RA, right atrium; RV, right ventricle; TR, tricuspid regurgitation; TS, tricuspid stenosis; VSD, ventricular septal defect


“The difference between how a person treats the powerless versus the powerful is as good a measure of human character as I know.”

Robert Sutton
Useful background: Heart sounds of non-valvular origin (continuous murmurs)

<table>
<thead>
<tr>
<th>Murmur</th>
<th>Location</th>
<th>Radiation</th>
<th>Quality</th>
<th>Pitch</th>
<th>Associated signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pericardial friction rub</td>
<td>Variable, 3rd ICS</td>
<td>Little</td>
<td>Scratchy</td>
<td>High</td>
<td>3 phases: mid-systolic, mid diastolic, pre systolic</td>
</tr>
<tr>
<td>Patent ductus arteriosus</td>
<td>2nd LICS</td>
<td>Left clavicle</td>
<td>Harsh, machinery like</td>
<td>Medium</td>
<td>Loudest in late systole fades in diastole, often silent interval in late diastole</td>
</tr>
<tr>
<td>Venous hum</td>
<td>Above medial third of right clavicle</td>
<td>1st-2nd ICS</td>
<td>Humming, roaring</td>
<td>Low</td>
<td>Soft murmur without a silent interval, loudest in diastole</td>
</tr>
</tbody>
</table>

Abbreviations: ICS et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, Table 11, page 62.

*The secret of the care of the patient is in the caring for the patient.*

27. Perform a focused physical examination of the precordium for the site of optimal auscultation of normal and abnormal heart sounds and murmurs.

- **Aortic area**
  - AS, AR
  - A2, Aortic ejection click
  - Systemic hypertension
  - Dilated aortic aneurysm

- **Pulmonary area**
  - PS, PR, flow, PDA
  - P2, pulmonary ejection click
  - PHT

- **Mitral area**
  - MS, MR, AS, AR, HSS, functional jid-diastolic rumble
  - A2, S3, S4

- **Tricuspid area**
  - TS, TR, PR, VSD
  - RV S3, S4
  - TV opening sanp

- **Descending thoracic area**
  - Coarctation of aorta
  - Aortic aneurysms
Abbreviations: AO, aorta; AR, Aortic regurgitation; AS, aortic stenosis; ICS, intercostal space; IHSS, idiopathic hypertrophic subaortic stenosis; L-CHF, left sided congestive heart failure; LA, left atrium; LICS, left intercostal space; LV, left ventricle; LVE, left ventricular enlargement; MR, mitral regurgitation; MS, mitral stenosis; PA, pulmonary artery; PDA, patent ductus arteriosus; PHT, pulmonary hypertension; PS, pulmonary stenosis; RA, right atrium; RICS, right intercostal space; RV, right ventricular; RVE, right ventricular enlargement; TR, Tricuspid regurgitation; TS, tricuspid stenosis; TV, tricuspid valves; VSD, ventricular septal defect

Adapted from: Mangione S. Hanley & Belfus 2000, pages 239-240; Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, Table 7, page 58.

28. Take a directed history for the cause of a patient’s cardiac murmur.

- **Idiopathic**
  - Flow, dilation, distortion, anemia, hyperthyroidism
  - Dissecting aneurysm
  - Hypertrophic obstructive cardiomyopathy (HOCM)

- **Inherited**
  - Cyanotic, acyanotic
  - Marfan’s, Turner’s, Down syndrome

- **Infection**
  - Syphilis
  - Subacute bacterial endocarditis (SBE)

- **Immune**
  - Lupus (Libman Sachs murmur)
  - Ankylosing spondylitis

- **Infiltration**
  - Carcinoid
  - Tumor
  - Atrial myxoma

- **Trauma**

- **Inflammation**
  - Rheumatic heart disease

- **Metabolic**
  - Coronary artery disease (CAD)
Papillary muscle rupture, ventricular septal defect (VSD)

Abbreviations: CAD, coronary artery disease; HOCM, hypertrophic obstructive cardiomyopathy; SBE, subacute bacterial endocarditis; VSD, ventricular septal defect


Useful background: Performance characteristics for physical examination for murmurs and valvular heart disease

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal heart examination</td>
<td></td>
</tr>
<tr>
<td>o Detecting any valvular heart disease</td>
<td>18.3</td>
</tr>
<tr>
<td>Characteristic systolic murmur</td>
<td></td>
</tr>
<tr>
<td>o Detecting AS</td>
<td>3.3</td>
</tr>
<tr>
<td>o Detecting mild MR or worse</td>
<td>5.4</td>
</tr>
<tr>
<td>o Detecting moderate to severe MR</td>
<td>3.3</td>
</tr>
<tr>
<td>o Detecting MVP</td>
<td>12.1</td>
</tr>
<tr>
<td>o Detecting mild TR or worse</td>
<td>14.6</td>
</tr>
<tr>
<td>o Moderate to severe TR</td>
<td>10.1</td>
</tr>
<tr>
<td>o Detecting VSD</td>
<td>24.9</td>
</tr>
<tr>
<td>Characteristic diastolic murmur</td>
<td></td>
</tr>
<tr>
<td>o Detecting mild AR or worse</td>
<td>9.9</td>
</tr>
<tr>
<td>o Moderate to severe AR</td>
<td>4.3</td>
</tr>
<tr>
<td>o Detecting PR</td>
<td>17.4</td>
</tr>
</tbody>
</table>

Abbreviations: AR, aortic regurgitation; AS, aortic stenosis; PLR, positive likelihood ratio; MR, mitral regurgitation; MVP, mitral valve prolapse; NS, not significant; PR, pulmonary regurgitation; TR, tricuspid regurgitation; VSD, ventricular septal defect


"Whoever said that old age was “The Golden Years” was already demented.”

Grandad
29. Take a direct history and perform a focused physical examination to
determine if a systolic murmur is benign (i.e. non-pathological).

- Pathophysiology: Due to rapid flow across the mitral or tricuspid valve or
to distension of the left ventricular wall

- History
  - Family history
    - Family members with heart disease
  - Past medical history
    - Ante- and perinatal history
    - Infancy and childhood
  - Personal history of
    - Central cyanosis
    - Feeding difficulties
    - Poor weight gain

- Cardiovascular exam

- Characteristics of murmur
  - Short, soft (grade III/IV, or less) diastolic murmur
  - Loudest at the apex or left sternal border
  - Follows immediately after a physiologic S3
    - Heard in normal young people
    - No supportive evidence of organic heart disease
    - Soft mid-diastolic murmur
  - Associated with Still’s murmur (precordial vibratory murmur)
  - Valsalva manoeuvre - both pulmonary ejection and Still’s murmur
    disappear

- Absence of
  - On palpitation
    - Systolic thrill, over the suprasternal area
    - Abnormal apical impulse
  - On auscultation
    - Holosystolic or diastolic murmur (a purely diastolic murmur
      should be considered organic until proved otherwise)
    - Presence of ejection clicks/sounds
    - Fixed splitting of $S_2$
- **Pulmonary ejection systolic murmur**

  - **Location**
    - Pulmonary (2nd/ICS; also can be heard over the aortic area, [left sternal border], apex, or neck [left side])
    - Loudest at the second or third left interspace
  - **Early systolic ejection murmur**
    - Early to mid systolic
  - **Short, soft, high frequency, blowing crescendo decrescendo murmur**
  - **Increased in supine position**
  - **Normal P2, no diastolic murmur, no clicks, heaves or thrills**
    - Most commonly heard in thin adolescents
  - **Differentiate from ASD and PS**
    - S2 normally split and of normal intensity

- **Precordial vibratory murmur (Still’s murmur): Most commonly heard between the ages of 2 and 6 years of age**

  - **Short, soft (I-II/VI) mid systolic murmur**
    - Low frequency, coarse, twangy
    - Starts after S1, left lower sternal border
    - Changes with position
    - Rarely radiate to the neck (rarely)
  - **Softens or disappears on standing, reappears on squatting**
  - **Differentiate from VSD**
Carotid arterial bruit

- Loudest over carotid artery, at base of the heart, or right supraclavicular area (opposite to aortic stenosis, which is loudest over the second right ICS)
- Harsh, crescendo-decrescendo, ejection systolic murmur
- No precordial or suprasternal notch thrill
- \( \uparrow \) with
  - Hyperextension of the shoulders toward the back
  - Compression of the subclavian artery
- Unaffected by Valsalva manoeuvre

Venous hum

- Maximal in early diastole
- Continuous murmur
- Location
  - Infraclavicular, loudest at the right supraclavicular area or upper right sternal border
- Loudest in upright position
- \( \uparrow \) with Sudden release of the jugular veins
- Disappears when lying down
  - \( \downarrow \) in supine position
  - Compression of the jugular veins
- Differentiate from PDA
  - Thrill palpable over the jugular veins

Mammary soufflé

- Usually present in late pregnancy or early lactation
  - Varies from day to day; disappears after lactation period
- Continuous murmur related to the cardiac cycle
  - In some patients may be primarily systolic
- Loudest at the third or fourth interspace (either side or bilateral)
- Unaffected by Valsalva murmur
- Straight back/pectus excavatum (due to close proximity of pulmonary artery)
  - Mid-systolic ejection murmur
  - Short, crescendo-decrescendo
    - Grade I-III/VI
  - Loudest at the left upper sternal border
  - Louder in held exhalation
  - S2 usually widely split
    - P2 (less commonly A2) can be loud
  - ASD or PS by diagnostic, but chest x-ray

Abbreviations: ASD, atrial septal defect; ICS, intercostal space; PDA, patent ductus arteriosus; PS, pulmonary stenosis; VSD, ventricular septal defect


Useful background: LR for a significant systolic murmur vs functional systolic murmur

<table>
<thead>
<tr>
<th>Clinical sign</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Holosystolic murmur</td>
<td>8.7</td>
<td>0.19</td>
</tr>
<tr>
<td>Loud murmur</td>
<td>6.5</td>
<td>0.08</td>
</tr>
<tr>
<td>Plateau-shaped murmur</td>
<td>4.1</td>
<td>0.48</td>
</tr>
<tr>
<td>Loudest at the apex</td>
<td>2.5</td>
<td>0.84</td>
</tr>
</tbody>
</table>

Abbreviation: NLR, negative likelihood ratio; PLR, positive likelihood ratio
Useful background: Performance characteristics of dynamic manoeuvres to help determine the nature of a murmur

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Respiration</td>
<td></td>
</tr>
<tr>
<td>o Louder during inspiration, detecting right-sided murmurs (TR or PS)</td>
<td>7.8</td>
</tr>
<tr>
<td>➢ Changing venous return</td>
<td></td>
</tr>
<tr>
<td>o Louder with Valsalva strain, detecting HC</td>
<td>14.0</td>
</tr>
<tr>
<td>o Louder with squatting-to-standing, detecting HC</td>
<td>6.0</td>
</tr>
<tr>
<td>o Softer with standing-to-squatting, detecting cardiac myopathy</td>
<td>7.6</td>
</tr>
<tr>
<td>o Softer with passive leg elevation, detecting HC</td>
<td>9.0</td>
</tr>
<tr>
<td>➢ Changing systemic vascular resistance (overload)</td>
<td></td>
</tr>
<tr>
<td>o Softer with isometric hand grip, detecting HC</td>
<td>3.6</td>
</tr>
<tr>
<td>o Louder with isometric hand grip, detecting MR or VSD</td>
<td>5.8</td>
</tr>
<tr>
<td>o Louder with transient arterial occlusion, detecting MR or VSD</td>
<td>48.7</td>
</tr>
<tr>
<td>o Softer with amyl nitrite inhalation (if tachycardia induced), detecting MR or VSD</td>
<td>10.5</td>
</tr>
</tbody>
</table>

Abbreviations: HC, hypertrophic cardiomyopathy; PLR, positive likelihood ratio; NLR, negative likelihood ratio; MR, mitral regurgitation; PR, pulse rate; TR, tricuspid regurgitation; VSD, ventricular septal defect


30. Perform a focused physical examination of the precordium, which will help to distinguish between.

➢ Pleural rub (vs pulmonary rales)
  o ↑ by pressure to chest wall with stethoscope
  o Present in inspiration and expiration
  o Not cleared by coughing

➢ Pericardial rub (vs cardiac murmur)
  o ↑ by pressure on chest wall
  o ↑ by expiration
  o ↑ by lying on L or R side
  o Sound changes from day –to- day

➢ Pericardial effusion (vs cardiac tamponade [restricted diastolic filling])
o ↑ dullness of precardium
o ↓ apex impulse
o ↓ heart sounds
o Pressure on adjacent structures
o Ewart’s sign (compression of lung near left scapula results in dullness, bronchial breathing, increased tactile vocal fremitus)

➢ Tamponade
  o ↑ JVP
  o ↓ SBP
  o Pulsus paradoxicus (shock, laboured breathing, ventilation)
  o Absence of cardiomegaly

Abbreviations: JVP, jugular venous pressure; SBP, systolic blood pressure

Useful background: Select features of systolic murmurs

➢ In the holosystolic murmur, the murmur begins just after the first heart sound (S1) and continues throughout the systole. In the late systolic murmur, the murmur begins at the middle of the systole or later and ends at the second heart sound (S2). In an early peaking murmur, peak intensity is before the middle of the systole. In a mid or late peaking murmur, peak intensity is at the middle of the systole or later.

Source: Simel David L, et al. JAMA 2009, Figure 33-1

Systolic murmurs

➢ Aortic
  o Aortic stenosis- confirmed by narrow pulse pressure and thrill (patient leaning forward in expiration)
  o Increased flow rate
  o Valve distortion without stenosis
  o Post valvar dilatation eg hypertension
  o Coarctation murmur is later in systole and may extend to 2nd sound

➢ Pulmonary
  o Functional, especially in young people
  o Pulmonary stenosis- may be very soft especially if associated with VSD
  o Increased flow rate ASD, VSD, TAPVD (total anomalous pulmonary venous drainage), hyperdynamic circulation
  o Post valvar dilatation, eg pulmonary hypertension
Pansystolic murmurs
- Continuous with the 2nd sound
- Mitral regurgitation – propagated into axilla
- Tricuspid regurgitation – increases with inspiration
- VSD - 3rd or 4th LICS. Thrill in 90 per cent
- PDA- usually in 2nd LICS. Murmur may be absent, pansystolic or continuous

Abbreviations: ASD, atrial septal defect; LICS, left intercostal space; PDA, patent ductus arteriosus; TAPVD, total anomalous pulmonary venous drainage; VSD, ventricular septal defect


SO YOU WANT TO BE A CARDIOLOGIST!

Q: How do you make the diagnosis of MR, in the absence of a murmur? (In the patient with a thick chest, well aerated lung tissue and a large RV, even severe MR may not have an audible murmur of MR)

A:  
- Large L-atrium and L-ventricle
- S₂ widely split

Useful background: Typical location of maximal intensity and radiation for various types of abnormal systolic murmurs

<table>
<thead>
<tr>
<th>Location of maximal intensity</th>
<th>Radiation</th>
<th>Typical for</th>
</tr>
</thead>
<tbody>
<tr>
<td>Second R.ICS</td>
<td>Right carotid artery, Right clavicle</td>
<td>- AS</td>
</tr>
<tr>
<td>Fifth or sixth L.ICS</td>
<td>Left anterior axillary line, Left axilla</td>
<td>- MV-P</td>
</tr>
<tr>
<td>Mid left thorax</td>
<td>Lower R.SB, Epigastrium, Fifth ICS, mid left thorax</td>
<td>- TR</td>
</tr>
<tr>
<td>Lower L.SB</td>
<td>Lower L.SB</td>
<td>- HCM</td>
</tr>
<tr>
<td>Fifth L.ICS</td>
<td>Lower L.SB</td>
<td>- HCM</td>
</tr>
</tbody>
</table>

Abbreviations: HCM, hypertrophic cardiomyopathy; ICS, intercostal space; MV-P, mitral valve prolapse; SB, sternal border; TR, tricuspid regurgitation

Mitral regurgitation (MR)

- Precordium
  - Apical systolic thrill,

- Apex
  - Displaced to left.

Adapted from: Talley N. J., et al. *Maclennan & Petty Pty Limited 2003*, Figure 3.29, page 75.

- Auscultation
  - Apical systolic regurgitant murmur following a $\downarrow S_1$ radiating
  - $S_1 \downarrow$ or absent (murmur may replace $S_1$).
  - $S_3$ due to increased left ventricular end diastolic volume. Auscultating an $S_3$ does not reflect the severity of MR, nor does a systolic regurgitant wave in the neck weins reflect the severity of TR.
  - Diastolic flow murmur in severe MR
  - In tall persons (eg. Marfan’s syndrome, maximal intensity of MR murmur is close to left sternal border)
  - Radiation
    - Left axilla or left interscapular area
    - With ruptured chordate tendinae
<table>
<thead>
<tr>
<th>Murmur</th>
<th>Location</th>
<th>Radiation</th>
<th>Quality</th>
<th>Pitch</th>
<th>Associated signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pansystolic murmurs</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>↓S₁; S₃, S₄ present, laterally displaced diffuse PMI</td>
</tr>
<tr>
<td>Mitral regurg.</td>
<td>Apex</td>
<td>L axilla</td>
<td>Blowing</td>
<td>High</td>
<td></td>
</tr>
</tbody>
</table>

**SO YOU WANT TO BE A CARDIOLOGIST!**

Q1: In persons with mitral regurgitation (MR), what is the meaning of a diastolic rumble?  
A1: Coexistent mitral stenosis (MS)

Q2: OK. In persons with MR, a diastolic rumble and a large left atrium, what is the interpretation?  
A2: No MS


31. Perform a physical focused physical examination to determine the severity of mitral regurgitation (MR).

- Displaced of apex (PMI) from large LV
- Louder and longer apical systolic murmur
- Loud S₃
- Early loud diastolic flow murmur after S₃
- S₂ widened, unless pulmonary hypertension is present and the S₂ split is narrow

Abbreviation: LV, left ventricle; PMI point of maximal impulse of apex of LV.

Causes (s)
- Rheumatic damage to the valves
- Pallilary muscle dysfunction (especially after acute MI)
- Rupture of chordate tendinae (usually with infective endocarditis)
- Myxomatous degeneration of valve
- Mitral valve replacement
- LV dilatation
- Coronary heart disease
- Annular calcification
- Infective endocarditis
- Cardiomyopathy
- Connective tissue disorders

Useful background: Performance characteristics of physical examination for determining the severity of characteristics systolic murmur of moderate to severe mitral and tricuspid regurgitation

- Auscultating the murmur of mitral regurgitation (MR) has a positive likelihood ratio (PLR) of 4.4 that the MR is moderate – to – severe, whereas for tricuspid regurgitation (TR), palpating a pulsative liver has a PLR of 3.9 that the regurgitation is moderate – to – severe.

- Auscultating an S₃ does not reflect the severity of MR, nor does a systolic regurgitant wave in the neck weins reflect the severity of TR.

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Mitral Regurgitation (MR)</td>
<td></td>
</tr>
<tr>
<td>o Detecting MR</td>
<td>4.4</td>
</tr>
<tr>
<td>➢ Tricuspid regurgitation (TR)</td>
<td></td>
</tr>
<tr>
<td>o Pulsatile liver</td>
<td>3.9</td>
</tr>
</tbody>
</table>

Abbreviations: PLR, positive likelihood ratio; MR, mitral regurgitation; TR, tricuspid regurgitation

Useful background: Causes of mitral regurgitation (MR)

- **Acute**
  - Rupture of the papillary muscle (often with MI)
  - Endocarditis (often due to perforation of the MV leaflet or the chordate)
  - Trauma
  - Myxomatous degeneration of the MV

- **Chronic**
  - Mitral valve leaflets
    - Annular calcification
    - Endocarditis
      - Chordal and papillary muscles
      - Rheumatic
      - Prolapse
      - Annular calcification
      - Connective tissue disease
      - Congenital cleft
      - Drug related
  - Heart
    - Cordial and papillary muscles
      - MV Prolapse
      - Rupture of chordae
      - Myocardial infarction
      - Papillary muscle rupture
    - Myocardium
      - Regional ischemia of infarctions
      - Dilated cardiomyopathy (coronary artery disease)
      - Hypertrophic cardiomyopathy
    - Left ventricular dilatation
  - Connective tissue disorders

**Abbreviations:** MI, myocardial infarction; MV, mitral valve

32. Take a direct history and perform a focused physical examination for mitral valve prolapse.

**History**
- Palpitations associated with mild tachyarrhythmias
- Adrenergic symptoms
- Chest pain
- Anxiety or fatigue

**Physical Examination**
- Mid-systolic click, followed by late or mid-systolic high pitched crescendo murmur
- Squatting will bring the click closer to the second heart sound and decrease the duration of the murmur
- ↑ with squatting
- ↓ with valsalva manoeuvre, standing, bradycardia

---

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Q1: What is the mechanism of the click in MVP?

A1: Clicks result from sudden tensing of the mitral valve apparatus as the leaflets prolapse into the left atrium during systole.

Q2: What are the types of pulmonary stenosis (PS)?

A2:
- Valvular
- Subvalvular: infundibular and subinfundibular
- Supravalvular

Murmur | Location | Radiation | Quality | Pitch | Associated signs
---|---|---|---|---|---
MV prolapse | Apex | Axilla | Mid-systolic

- Complications
  - Heart
    - Murmur
      - MR
      - Endocarditis
      - TR (myxomatous degeneration of TV)
    - Arrhythmias:
      - VT (Ventricular tachycardia)
      - PST (Paroxysmal supraventricular tachycardia)
      - VPC (ventricular premature contractions)
  - Atypical chest pain
  - Transient ischemic attacks (TIAs)
  - Sudden death

- Associated conditions
  - Chronic rheumatic heart disease
  - Ischemic heart disease
  - Marfan’s syndrome (high-arched palate, arm span greater than height, straight back, pectus excavatus, HOCM)
  - Cardiomyopathies
  - ASD (secundum type)
  - Ehlers-Danlos syndrome
  - Psoriatic arthritis
  - Ebstein’s anomaly
  - SLE

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Q: When does the absence of a mitral area murmur or a late systolic/ holosystolic murmur significantly reduce the likelihood of mitral regurgitation?

A: In the setting of an acute myocardial infarction.


Atrial septal defect (ASD)

- Normal pulse
- Parasternal lift over pulmonary artery
- Normal jugular pulse; systolic ejection murmur in pulmonic area
- Low pitched diastolic rumble over tricuspid area (at times); persistent wide splitting of S2


Useful background: Ventricular septal defect (VSD) (at the left sternal edge)

Source: Talley N. J., et al. MacLennan & Petty Pty Limited 2003, Figure 3.33, page 83.
EASY QUESTIONS FOR OUR WOULD-BE CARDIOLOGIST!

Q: In the context of PS, what are the features of the syndromes named after Noonan, Watson and Williams?

A:  
- Noonan’s syndrome: short stature, ptosis, downward slanting eyes, wide-spaced eyes (hypertelorism), low-set ears, webbed neck, mental retardation and low posterior hairline. About two-thirds of patients with Noonan’s syndrome have pulmonary stenosis due to valve dysplasia.
  - William’s syndrome: infantile hypercalcaemia, elfin facies and mental retardation, in addition to supravalvular pulmonary stenosis. Subvalvular pulmonary stenosis, which is caused by the narrowing of the right ventricular infundibulum or sub-infundibulum, usually occurs in association with a ventricular septal defect.


Rheumatic fever and rheumatic heart disease

33. Take a directed history and perform a focused physical examination for rheumatic fever and rheumatic heart disease (RHD).

- Heart
  - Endocarditis
  - Pericarditis
  - Valvulitis
  - Myocarditis (MV > AV)

- Skin
  - Rheumatic nodules
  - Non-tender, painless, subcutaneous lesions along tendons and over bony prominences on back of hands, elbows, knees, spine)
    - Erythema multiforme

- CNS
  - Chorea

- Systemic
  - Weight loss, fever

- Joints
  - Polyarthritis

- Blood
  - Anemia

- Lung
  - Pleursy, pneumonia

- GI
  - Peritonitis
Abbreviations: AV, aortic valve; CNS, central nervous system; GI, gastrointestinal; MV, mitral valve; RHD, rheumatic heart disease;


Useful background: Diagnosis of initial attack of Rheumatic Fever

- **Jones criteria**
  - Major manifestations
    - Carditis
    - Polyarthritis
    - Chorea
    - Erythema marginatum
    - Subcutaneous nodules
  - Minor manifestations
    - Clinical findings
      - Arthralgia
      - Fever
    - Laboratory findings
      - Elevated acute phase reactants (erythrocyte sedimentation rate, C-reactive protein)
      - Prolonged PR interval
  - Supporting evidence of antecedent group A streptococcal infection
    - Positive throat culture or rapid diagnostic test
    - Elevated or rising streptococcal antibody titer

- **Ducket Jones diagnostic criteria for Rheumatic Fever**

Rheumatic fever = 2 major or 1 major + 2 minor + evidence of recent streptococcal infection (Group A streptococcal (throat) infection can → immune reaction → acute rheumatic fever).

Abbreviations: CRP, C reactive protein; ESR, erythrocyte sedimentation rate.

Aortic stenosis

- Precordium-
  - Basal systolic thrill;

- Apex
  - Displaced anteriorly and laterally.

- Carotids
  - Slow upstroke to a delayed peak.

- Auscultation
  - A2 diminished or paradoxically ejection systolic murmur radiating to carotids.

- Cold extremities.

- Reversed S2: P2-A2 in severe AS (paradoxical splitting)

<table>
<thead>
<tr>
<th>Murmur</th>
<th>Location</th>
<th>Radiation</th>
<th>Quality</th>
<th>Pitch</th>
<th>Associated signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aortic stenosis</td>
<td>2nd RICS</td>
<td>Neck</td>
<td>Harsh</td>
<td>Medium</td>
<td>Ejection sound, ↓ S2, S4, narrow pulse pressure, slow rising and delayed pulse</td>
</tr>
</tbody>
</table>

Useful background: Accuracy of the physical examination for detecting aortic stenosis

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slow carotid upstroke</td>
<td>9.2</td>
<td>0.56</td>
</tr>
<tr>
<td>Murmur radiating to right carotid</td>
<td>8.1</td>
<td>0.29</td>
</tr>
<tr>
<td>Reduced or absent S2</td>
<td>7.5</td>
<td>0.50</td>
</tr>
<tr>
<td>Murmur over right clavicle</td>
<td>3.0</td>
<td>0.10</td>
</tr>
<tr>
<td>Any systolic murmur</td>
<td>2.6</td>
<td>0</td>
</tr>
<tr>
<td>Reduced carotid volume</td>
<td>2.0</td>
<td>0.64</td>
</tr>
</tbody>
</table>

Abbreviation: NLR, negative likelihood ratio; PLR, positive likelihood ratio.

34. Perform a physical focused physical examination to distinguish between the murmur of pulmonary stenosis (PS) and aortic stenosis (AS).

<table>
<thead>
<tr>
<th>Physical sign</th>
<th>AS</th>
<th>PS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Location of maximal intensity</td>
<td>2nd R-ICS, or apex</td>
<td>L-sternal border</td>
</tr>
<tr>
<td>Breathing-effect on murmur</td>
<td>Louder in expiration</td>
<td>Louder in inspiration</td>
</tr>
<tr>
<td>Effect on ejection click</td>
<td>No change during inspiration</td>
<td>Soften during inspiration</td>
</tr>
<tr>
<td>Effect of standing</td>
<td>Softer</td>
<td>Louder</td>
</tr>
<tr>
<td>Valsalva manoeuvre</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Staining phase</td>
<td>Softer</td>
<td>Softer</td>
</tr>
<tr>
<td>Releasing phase</td>
<td>Louder slowly</td>
<td>Louder quickly</td>
</tr>
<tr>
<td>S₂</td>
<td>Paradoxical splitting of S₂</td>
<td>Widened physiological S₂</td>
</tr>
<tr>
<td>S₄ inspiration</td>
<td>Expiration</td>
<td>Inspiration</td>
</tr>
</tbody>
</table>

Abbreviation: R, right; L, left; ICS intercostals space

Systolic Regurgitation causes: intital regurgitation (MR), tricuspid regurgitation (TR), ventricular septal defect (VSD), patent ductus arteriosus (PDA)

Adapted from: Mangione S. *Hanley & Belfus* 2000, pages 258 and 259.

“There’s always a taller mountain.”
Et al.
Useful background: Aortic stenosis (AS) (at the aortic area)

- Soft S2
- Reversed A2 / P2 (narrow or reverse split S2)
- S4
- Narrow pulse pressure
- Systolic thrill
- Heaving apex beat
- L- CHF

Adapted from: Talley N. J., et al. *Maclennan & Petty Pty Limited*, 2003, Figure 3.31, page 77.
Q: What does the S2 tell us in AS?
A:  
  o Normal: strong evidence against the presence of critical aortic stenosis
  o Soft S2: valvular stenosis (except in calcific stenosis of the elderly, where the margin of the leaflets usually maintain their mobility)
  o Single: second heart sound may be heard when there is fibrosis and fusion of the valve leaflets
  o Reverse splitting of the second sound: indicates mechanical or electrical prolongation of ventricular systole

SO YOU WANT TO BE A CARDIOLOGIST!


35. Perform a directed physical examination to distinguish between the systolic murmur of aortic sclerosis (no pressure gradient; due to stiff or dilated aortic root) and aortic stenosis (AS).

<table>
<thead>
<tr>
<th></th>
<th>Aortic Stenosis</th>
<th>Aortic Sclerosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>o Dizzy, syncopy, chest pain, dyspnea</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulse</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Slow, small volume</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Apex beat PMI</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Precordial thrill</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Heart sounds (S₂, A₂)</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Murmur</td>
<td></td>
<td>+</td>
</tr>
<tr>
<td>o Short peaks in first half of systole</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

36. Perform a directed physical examination to distinguish between the systolic murmur from HOCM (hypertrophic cardiomyopathy, particularly septal component) and aortic stenosis (AS).

<table>
<thead>
<tr>
<th></th>
<th>AS</th>
<th>HOCM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early after S1</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Midsystole</td>
<td>-</td>
<td>+</td>
</tr>
<tr>
<td>Intensity of murmur:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Valsalva manoeuvre</td>
<td>-</td>
<td>↑</td>
</tr>
<tr>
<td>Squatting</td>
<td>↑</td>
<td>↓</td>
</tr>
<tr>
<td>Associated murmur of MR</td>
<td>↓</td>
<td>+</td>
</tr>
<tr>
<td>Timing of onset systolic murmur</td>
<td>Immediately after S1</td>
<td>Begins in mid-systole</td>
</tr>
<tr>
<td>Valsalva manoeuvre</td>
<td>↓ intensity</td>
<td>↑ intensity</td>
</tr>
<tr>
<td>Squatting</td>
<td>↑ intensity</td>
<td>↓ intensity</td>
</tr>
<tr>
<td>Associated MR*</td>
<td>Rare</td>
<td>75%</td>
</tr>
</tbody>
</table>

**Murmur**

- **Aortic stenosis**: 2nd RICS, Neck, Harsh, Medium, Ejection sound, ↓ S2, S4, narrow pulse pressure, slow rising and delayed pulse
- **Hypertrophic cardiomyopathy**: 3rd-4th LICS, LLSB → apex, base, Harsh, Medium, S3, S4 sustained apical impulse, two palpable components, carotid pulse rises quickly

*MR murmur HOCM ends before A2, and may be from MVP (mitral valve prolapsed)
MR, initiated regurgitation; S1, first heart sound; A2 the A2 components of S2, the second sound

Abbreviations: AS, aortic stenosis; HOCM, hypertrophic cardiomyopathy; MR, mitral regurgitation

Adapted from: Mangione S. *Hanley & Belfus*, 2000, pages 256 to 258.
SO YOU WANT TO BE A CARDIOLOGIST!

Q: How does the patient’s age affect the likely cause of their aortic stenosis?
A: o Under the age of 60 years: rheumatic, congenital
  o Between 60 and 75 years: calcified bicuspid aortic valve, especially in men
  o Over the age of 75 years: degenerative calcificatio

Useful background: Causes of the combined aortic and mitral valve disease

- Rheumatic valvular disease
- Infective endocarditis
- Collagen degenerative disorder, e.g. Marfan’s syndrome
- Calcific changes in the aortic and mitral valve apparatus
- Auscultation: stenosis – longer, peaks in early systolic, normal or loud S2 (especially if there is associated systemic hypertension)

- Aortic Stenosis
  o Congenital, degenerative, rheumatic (bicuspid semiluminar valve)
  o Valvular, supravalvular, subvalvular
  o Arterial pulse
    - Valvular: small amplitude (parsus) with slow upstroke (tardus) may be associated thrill best heard over carotid artery
    - Supravalvular: amplitude of pulse higher R>L –sided vessels
    - Subvalvualr: brick pulse, with palpable double systolic impulse (pulsus bisferiens)

- Valvular Aortic Stenosis Impulse (Pulsus Bisferiens)
  o PMI – normal in AS, unless LV hypertrophy and L-CHF or AS plus aortic regurgitation
  o Precordial thrill – palpable, does not reflect severity of AS
  o Murmurudget in “sach area” 2nd right intercoatal space (aortic area) down to 5/6th intercoatal space at the left mid clavicular line
  o Crescendo – decrescendo murmur; louder and longer murmur with later peak is more severe soft or absent A2 and audible or palpable S4, suggests more. If murmur becomes softer, suspect obesity or COPD, or CHF.

- Subvalvular Aortic Stenosis (HOCM)
- Supravalvular Aortic Stenosis
  o Males, with associated congenital abnormalities
    - Typical Facies
- Patulus lip
- Deep, husky voice
- Hypercalcemia
  - Pulse and BP stronger on right than left side (R > L)
  - No aortic ejection click
  - May be an associated murmur of aortic regurgitation

Adapted from: Mangione S. *Hanley & Belfus* 2000, pages 254 to 259.

Useful background:

- The complications of aortic stenosis
  - Sudden death occurs in 10-20% of adults and 1% of children. It has been rarely documented to occur without prior symptoms.
  - Ventricular arrhythmias (more common than supraventricular arrhythmias)
  - Heart block (may occur because of calcification of conducting tissues).
  - Systemic embolization (disintegration of the aortic valve or concomitant aortic atheroma).
  - Infective endocarditis
  - Hemolytic anemia


37. Perform a focused physical assessment for prosthetic heart valves.

- Mitral Valve
  - Recognized by their site, metallic first heart sound, normal second heart sound and metallic opening snap
  - Systolic murmurs are often
  - Diastolic flow murmurs may be heard normally over the disc valves

- Aortic Valve
  - Be recognized by their site, normal first heart sound and metallic second heart sound

- Both mitral and aortic
  - Both the first and second heart sounds will be metallic
  - The presence of a systolic murmur does not indicate valve dysfunction
  - The presence of an early diastolic murmur indicates a malfunctioning aortic valve

- Complications
  - Thromboembolism
- Valve dysfunction, including valve leakage, valve dehiscence and valve obstruction due to thrombosis and clogging
- Bleeding (such as upper gastrointestinal hemorrhage) due to anticoagulants
- Hemolysis at the valve, causing anemia
- Endocarditis (prosthetic valve endocarditis)
  - <2 months of surgery: develops as a result of intraoperative contamination of the prosthetic valve or as a consequence of a postoperative nosocomial infection
  - >2 months of surgery: after transient bacteremia (minor skin or upper respiratory tract infections or following dental or urinary manipulations). The non-cardiac manifestations resemble those of native valve infective endocarditis
- Structural dysfunction
  - Fracture
  - Poppet escape
  - Cuspal tear
  - Calcification
- Non-structural dysfunction
  - Paravalvular leak
  - Suture/tissue entrapment
  - Noise

- Negative prognostic factors
  - Heart failure
  - Non-streptococcal endocarditis, especially *Staph. aureas*, fungal endocarditis
  - Infection of a prosthetic valve
  - Elderly patients
  - Valve ring or myocardial abscess

- Conditions that can simulate clinical manifestations of infective endocarditis
  - Atiral myxoma
  - Non-bacterial endocarditis
  - Systemic lupus erythematosus (SLE)
  - Sickle cell disease

Useful background: Performance characteristics of physical examination of severe aortic stenosis (AS)

The finding of S₄ gallop, a murmur best heard over the 2nd intercostal space, or a murmur radiating into the neck have no value to predict the presence of severe AS.

### Finding

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial pulse</td>
<td></td>
</tr>
<tr>
<td>o Delayed carotid artery upstroke</td>
<td>3.7</td>
</tr>
<tr>
<td>o Reduced carotid artery volume</td>
<td>2.3</td>
</tr>
<tr>
<td>o Brachioradial delay</td>
<td>2.5</td>
</tr>
<tr>
<td>Apical impulse</td>
<td></td>
</tr>
<tr>
<td>o Sustained apical impulse</td>
<td>4.1</td>
</tr>
<tr>
<td>o Apical carotid delay</td>
<td>2.6</td>
</tr>
<tr>
<td>Heart sounds</td>
<td></td>
</tr>
<tr>
<td>o Absent A2</td>
<td>4.5</td>
</tr>
<tr>
<td>o Absent or diminished A2</td>
<td>3.6</td>
</tr>
<tr>
<td>Murmur</td>
<td></td>
</tr>
<tr>
<td>o Late peaking (midsystole or beyond)</td>
<td>4.4</td>
</tr>
<tr>
<td>o Prolonged duration</td>
<td>3.9</td>
</tr>
</tbody>
</table>

Abbreviation: ICS, intercostal space; PLR, positive likelihood ratio

Note: The finding of S₄, murmur loudest over aortic area (2nd ICS), and transmission of the murmur to the neck are not mentioned here because their PLR are < 2.

38. Perform a physical focused physical examination to distinguish between the murmur of tricuspid regurgitation (TR) and mitral regurgitation (MR).

<table>
<thead>
<tr>
<th>Sign</th>
<th>Tricuspid regurgitation</th>
<th>Mitral regurgitation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pulse</strong></td>
<td>Normal</td>
<td>Jerky, or normal</td>
</tr>
<tr>
<td><strong>JVP</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>‘V’ wave</td>
<td>↑</td>
<td>Normal</td>
</tr>
<tr>
<td>Deep Y descents</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Lancisi’s sign)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Palpation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left parasternal heave</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td><strong>Auscultation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parasystolic murmur</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Intensity on inspiration</td>
<td>↑</td>
<td>↑↑</td>
</tr>
<tr>
<td>Valsava</td>
<td>in 3 sec</td>
<td>in 1 sec</td>
</tr>
<tr>
<td>Radiation</td>
<td>To liver</td>
<td>To axilla</td>
</tr>
<tr>
<td>AJR</td>
<td>+</td>
<td>-</td>
</tr>
</tbody>
</table>

Abbreviations: JVP, Jugular venous pressure; AJR, abdomino jugular reflux test


**Tricuspid regurgitation (TR)**

39. Take a directed history and focused physical examination for TR.

| **History**            |                         |                      |
| IV drug use            |                         |                      |
| Trauma to the chest    |                         |                      |
| Rheumatic fever        |                         |                      |
| Chronic obstructive pulmonary disease (COPD) | | |

| **Physical examination** |                         |                      |
| Skin                    | - Peripheral cyanosis   |                      |
|                         | - Ankle edema           |                      |
| JVP                     | - Large ‘v’ waves in the jugular venous pulse | |
| Pulse                   | - Atrial fibrillation   |                      |
| Heart                   | - Palpation             |                      |
|                         | ▪ Left parasternal heave|                      |
Murmur
Tricuspid regurg

- Palpable or loud P2
  - Auscultation loud P2, S3
- Pansystolic murmur at LLSB which ↑ in inspiration (Carvallo’s sign)
- Mid-diastolic murmur of mitral stenosis

<table>
<thead>
<tr>
<th>Location</th>
<th>Radiation</th>
<th>Quality</th>
<th>Pitch</th>
<th>Associated signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>LLSB</td>
<td>RLSB</td>
<td>Blowing</td>
<td>High</td>
<td>S3, ↑JVP</td>
</tr>
</tbody>
</table>

- Abdomen
  - Hepatomegaly systolic pulsations
  - Ascites

Causes
- Without pulmonary hypertension (PHT)
  - Congenital
  - Trauma
  - Endocarditis
- With (secondary to) PHT
  - Mitral stenosis plus PHT
  - ASD plus PHT
  - R-CHF plus PHT
  - R-CHF plus PHT, plus L-CHF

Abbreviation: ASD, atrial septal defect; LLSB, left lower sterna border; PHT, pulmonary hypertension; R-CHF, right-side congestive heart failure

Adapted from: Baliga R.R. Saunders/Elsevier 2007, page 64.

SO YOU WANT TO BE A CARDIOLOGIST!

Q: Under what circumstances is the blood pressure lower in the legs than arms (normal difference: 10-15 mm Hg higher in legs than arms)?; Abnormal difference (Hill’s sign, > 20 mm Hg; exaggeration of normal, indicating ↑ SV (stroke volume)’ such as from tachycardia)

A:
  - Atherosclerosis in the elderly
  - Aortic dissection
  - Aortic regurgitation (severe)

SO YOU WANT TO BE A CARDIOLOGIST!

Q1: In the context of a systolic murmur, what is the significance if after a long diastole (such as following a premature beat), the intensity of the murmur becomes louder at the base but not at the apex?

A1: Then the systolic murmur is likely comprised of both a regurgitation murmur plus an ejection murmur.

Q2: What is the exception to this general rule?

A2: The exception is mitral valve prolapse (MVP), in which, the murmur becomes softer after a long diastole.

Cardiomyopathy

Useful background: Causes of cardiomyopathy

- **Primary**
  - Idiopathic
  - Endomyocardial fibrosis or fibro-elastosis
  - Hypertrophic obstructive cardiomyopathy (HOCM)
  - Pregnancy and puerperium

- **Secondary**
  - Toxins, drugs – alcohol, anthracyclines
  - Anemia
  - Endocrine
    - Pheochromocytoma
    - Thyrotoxicosis
    - Hypothyroidism
    - Hypoadrenalism
  - Infection- viral (Coxsachie)
    - Toxoplasmosis
    - Schistosomiasis
    - Chagas disease (trypanosoma)
    - Sarcoidosis
    - TB
  - Infiltration
    - Amyloidosis
    - Leukemia
    - Hemochromatosis
  - Metabolic
    - Beri-beri
- Iron overload
- Carcinoid
- Porphyria
- Glycogen storage disease
- Cobalt or antimony poisoning
  o ‘Collagen-vascular’ disease
    - SLE
    - Arteritis
    - Systemic sclerosis
    - Rheumatoid disease
    - Ankylosing spondylitis
  o Neuromuscular
    - Friedreich’s ataxia
    - Myopathies (eg dystrophia myotonica, Duchenne)

Abbreviations: HOCM, hypertrophic obstructive cardiomyopathy; SLE, systemic lupus erythematosus; TB, tuberculosis


SO YOU WANT TO BE A CARDIOLOGIST!

Q1: What are the complications of hypertrophic cardiomyopathy?
A1:
  o Sudden death
  o Atrial fibrillation
  o Infective endocarditis
  o Systemic embolization

Q2: What is the most characteristic pathophysiological abnormality in hypertrophic cardiomyopathy?
A2: Diastolic dysfunction

Useful background: Effort of inspiration on cardiac murmurs

- Increases: TS, TR, PS
- Decreases: MS, MR, AS, AR, L → R shunts

Abbreviations: AS, aortic stenosis; AR, aortic regurgitation; MS, mitral stenosis; MR, mitral regurgitation; PS, pulmonary stenosis; TS, tricuspid stenosis; TR, tricuspid regurgitation.


**Diastolic murmurs**

Useful background: Diastolic murmurs are classified according to the time of onset of the murmur

- **Early**
  - Begins with the second heart sound (S2).
  - Decrease in intensity (decrescendo) and disappear before the first heart sound (S1).
  - Can continue through diastole.

- **Mid**
  - Begins clearly after S2 (in mitral stenosis, classically after an opening snap [OS]).

- **Late**
  - Begins in the interval immediately before S1.
  - In mitral stenosis, the mid diastolic murmur may merge with the late diastolic (presystolic) murmur.

Abbreviations: OS, opening snap

Source: Simel David L, *et al.* JAMA 2009, Figure 32-2, page 421.

- **Characteristics of murmur**
  - Decrescendo diastolic murmur beginning immediately at S2 present in early-to-mid diastole, or all diastole.
  - Heard but in expiration with diaphragm and patient sitting up and bending forward, or with squatting, at Erb’s point (3/4th ICS intercostals space).
  - Mid-to-late diastolic (does not start immediately after S2)
  - Opening snap, followed by a short pause, then the typical initial crescendo pattern of MS murmur
- Initial diastolic crescendo, the decrescendo, then late crescendo (presystolic accentuation of the diastolic rumble)
- Low pitched (listen with bell of stethoscope)
- Maneuvres to create louder murmur:
  - left lateral decubitis position
  - expiration
  - squatting during hand-grip maneuvre
  - leg raising
  - after coughing
  - release phase of Valsalva maneuvre
  - after exercise

➤ Apex
  - displaced laterally and anteriorly
  - thrill often palpable along left sternal border and in the jugular notch.

➤ Carotids
  - double systolic wave.

➤ Auscultation
  - Decrescendo diastolic murmur along left sternal border; S1 and A2 are increased.
  - Length of murmur is proportional to severity

➤ Associations
  - Marfan’s syndrome,
  - rheumatoid spondylitis.

➤ Causes of acute aortic regurgitation
  - Infective endocarditis
  - Aortic dissection
  - Trauma
  - Failure of prosthetic valve
  - Rupture of sinus of Valsalva
<table>
<thead>
<tr>
<th>Murmur</th>
<th>Location</th>
<th>Radiation</th>
<th>Quality</th>
<th>Pitch</th>
<th>Associated signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diastolic murmurs</td>
<td>OS</td>
<td></td>
<td></td>
<td></td>
<td>Ejection sound, S₃, S₄, laterally displaced PMI, wide pulse pressure, bounding pulse, midsystolic flow murmur or Austin Flint murmur</td>
</tr>
<tr>
<td>Aortic regurg.</td>
<td>2ⁿᵈ-₄ᵗʰ</td>
<td>Apex, RSB</td>
<td>Blowing</td>
<td>High</td>
<td></td>
</tr>
<tr>
<td></td>
<td>LICS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


- MSK
  - Reiter’s syndrome
  - Ankylosing spondylitis

- Rheumatic fever

- Endocarditis

- Conditions associated with aortic valve leaflet abnormalities
  - Marfan syndrome
  - Rheumatoid arthritis
  - Ankylosing spondylitis

- Diseases that affect the aortic root
  - Hypertension
  - Syphilis
  - Inherited connective tissue disorders
  - Aortic aneurysm (dissection of descending aorta)

- Misdiagnosed AR
  - Easily confused with similar murmur of pulmonary regurgitation or tricuspid regurgitation (Graham Steell murmur): early diastole, 2ⁿᵈ L-ICS due to pulmonary hypertension.
Sudden onset AR is different from chronic AR by having a soft/absent S1, and a diastolic murmur which is never holodiastolic (only early-to-mid diastole)

Abbreviation: AR, aortic regurgitation


SO YOU WANT TO APPLY FOR A CARDIOLOGY RESIDENCY!

Stump Your Staff – See if they know more than four of the eponymous signs of aortic regurgitation

- Quincke’s sign: capillary pulsation in the nail beds - it is of no value, as this sign occurs normally
- Corrigan’s sign: prominent carotid pulsations
- De Musset’s sign: head nodding in time with the heartbeat
- Hill’s sign: increased blood pressure in the legs compared with the arms
- Mueller’s sign: pulsation of the uvula in time with the heartbeat
- Duroziez’s sign: systolic and diastolic murmurs over the femoral artery on gradual compression of the vessel
- Traube’s sign: a double sound heard over the femoral artery on compressing the vessel distally; this is not a ‘pistol shot’ sound that may be heard over the femoral
- Corrigans neck pulsation
- De Mussetts head nodding
- Duroziez’s femoral diastolic murmur
- Quincke’s capillary pulsation (nails)

SO YOU WANT TO BE A CARDIOLOGIST!

Q1: What is the effect of pregnancy on MS?
A1:  
- Patient usually become symptomatic in T2 of pregnancy
- Blood volume increases pulmonary blood volume diminishes late in the third trimester, the symptom improve

Q2: In the context of MS, what is Ortner’s syndrome?
A2:  
- Hoarseness of voice caused by left vocal cord paralysis associated with enlarged left atrium in mitral stenosis


Q3: What causes a systolic murmur to accompany to the typical diastolic murmur of AR?
A3: Severe AR, or concurrent AS (i.e. AR+AS)

Q4: In which cardiac phases are the diagnosis and severity of AR and MR made?
A4: Curious comparison: AR is diagnosed in diastole, but its severity assessed in systole; MR is diagnosed in systole, but its severity is assessed in diastole

Q5: In the context of aortic regurgitation (AR), what is Landolfi’s sign?
A5: The pupil contracts in systole, and dilates in diastole

Mitral stenosis:

- Precordium
  - tapping
- Apex beat
  - diastolic thrill at apex; parasternal lift.
- Auscultation
  - loud S1,
  - P2 diastolic opening snap followed by rumble with presystolic accentuation.
Murmur | Location | Radiation | Quality | Pitch | Associated signs
---|---|---|---|---|---
Mitral stenosis | Apex | Little/None | Rumbling | Low | ↑S1, OS after S2, RV impulse, often assoc. with AV disease
Atrial fibrillation may be pulse pattern. | | | | | Mitral or tricuspid distortion eg Carey-Coombs murmur of active rheumatic carditis
Cold extremities. | | | | | This distance is inversely proportional to the severity of the stenosis;
| | | | | Presystolic accentuation is present only if patient is in sinus rhythm.

Useful background: Physical examination for MS

Assessment of severity
- The louder the murmur and the thrill, the more severe the MS
- Conditions making murmur louder without necessarily a greater pressure gradient (false positive), unless there is associated pulmonary hypertension which softens the murmur of severe MS, and unless there is associated mitral regurgitation (which increases diastolic flow across the mitral valve making a louder murmur of MS)
- Conditions making murmur softer, causing overestimation of severity (false negative)
  - Emphysema
  - Very severe stenosis mitral or tricuspid valve
  - Severe pulmonary hypertension
  - Atrial fibrillation
Useful background: Performance characteristics of other cardiac findings in mitral stenosis

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
</table>
| ➢ Graham Steell murmur  
  o Detecting pulmonary hypertension | 4.2 |
| ➢ Hyperkinetic apical movement  
  o Detecting associated mitral regurgitation or aortic valve disease | 11.2 |
| ➢ Hyperkinetic arterial pulse  
  o Detecting associated mitral regurgitation | 14.2 |


➢ Causes  
  o Rheumatic fever  
  o Rheumatoid arthritis  
  o Systemic lupus erythematosus  
  o Malignant carcinoid (carcinoid syndrome)  
  o Congenital: parachute mitral valve with one papillary muscle having chordate attached to both leaflets of mitral valve  
  o Myxoma of left atrium  
  o Calcified bacterial vegetation of mitral valve  
    - Mitral annulus and leaflets  
  o Mimics  
    - Severe MR  
    - VSD  
    - Austin Flint murmur* of severe AR  
    - Ball valve thrombus in LA  
    - Cor triatriatum (a rare congenital heart condition where a thin membrane across the left atrium obstructs pulmonary venous flow)

Abbreviations: LA, left atrium; MR, mitral regurgitation; VSD, ventricular septal defect
*Definition: Austint Flint murmur: diastolic murmur (rumble) caused by functional closure of the anterior leaflet of mitral valve when there is moderate-to-severe aortic regurgitation, Distinguish from MS by the absence of an opening snap in a non-calcified valve, and the presence of S3 (rare in mitral stenosis).


40. Perform a focused physical examination to distinguish between the diastolic murmurs of mitral stenosis (MS) versus tricuspid stenosis (TS).

<table>
<thead>
<tr>
<th>Physical finding</th>
<th>TS</th>
<th>MS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Location</td>
<td>o epigastric and R/L parastenal area</td>
<td></td>
</tr>
<tr>
<td></td>
<td>o over apex if RV is very large</td>
<td></td>
</tr>
<tr>
<td>effect on murmur of inspiration</td>
<td>Louder</td>
<td>softer</td>
</tr>
<tr>
<td>position to best hear murmur</td>
<td>R lateral decubitus</td>
<td>L lateral decubitus</td>
</tr>
<tr>
<td>quality</td>
<td>“scratchy”</td>
<td>Low pitch</td>
</tr>
<tr>
<td>presystolic accentuation</td>
<td>Absent</td>
<td>present</td>
</tr>
</tbody>
</table>

Source: Mangione S. *Hanley & Belfus* 2000. pages 266 to 269.

*Let's see if there is mechanistic information that we can tease out.*

Grandad
Aortic regurgitation

- Often associated with Marfan’s syndrome, rheumatoid spondylitis.
- Precordium – Apex displaced laterally and anteriorly
- Thrill often plapable along left sterna border and in the jugular notch.
- Carotids – Double systolic wave
- Auscultation – Decrescendo diastolic murmur along left sternal border
- M₁ and A₂ are increased

41. Perform a physical examination to determine the severity of aortic regurgitation.

- Wide pulse pressure
- Soft S₂
- Duration of the decrescendo diastolic murmur
- Austin Flint murmur (an apical, low pitched, diastolic murmur caused by vibration of the anterior mitral cusp in the regurgitation jet, and is heard at the apex)
- Signs of left ventricular failure
- Hill’s sign


---

**SO YOU WANT TO BE A CARDIOLOGIST!**

**Q1:** In the context of Aortic regurgitation, what is “Hill’s” sign?

**A1:**
- Higher systolic pressure in the leg than in the arm, and
- An indicator of severity of aortic regurgitation

**Q2:** In the context of wanting to pass the cardiology fellowship examination, what is “cor-bovinum”?

**A2:** Slow and progressive left ventricular dilatation and hypertrophy in an attempt to normalize wall stress. The heart may thus become larger and heavier than in any other form of chronic heart disease – cor bovinum (bovine or ox heart).
Useful background: Performance characteristics for aortic regurgitation (AR)

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
</table>
| ➢ Characteristic diastolic murmur  
  o Detecting mild aortic regurgitation or worse | 9.9 |
| o Detecting moderate-to-severe aortic regurgitation | 4.3 |
| ➢ Early diastolic murmur loudest on right side of sternum  
  o Detecting dilated aortic root or endocarditis | 8.2 |
| ➢ The finding of an early diastolic murmur which becomes softer with amyl nitrite inhalation is not of significant use to distinguish AR versus Graham Steell murmur | NS |


➢ Characteristics of moderate-to-severe AR

<table>
<thead>
<tr>
<th>Finding</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PPLR</th>
<th>NLR</th>
</tr>
</thead>
</table>
| ➢ Diastolic murmur  
  o Murmur grade 3 or louder | 30-61 | 86-98 | 8.2 | 0.6 |

Only when the diastolic blood pressure is \( \leq 50 \text{ mm Hg} \), the pulse pessure is \( \geq 80 \text{ mm Hg} \), or Hill’s test is \( \geq 60 \text{ mm Hg} \) are these findings have a positive likelihood ratio (PLR) which makes then clinically useful (PLR, 19.3, 10.9 and 17.3, respectively). Duroziez’s sign, femoral pistol shot bruit, and the water hammer pulse do not signify severe AR. Modest PLRs are associated with S3 gallop (5.9), and enlarged or sustained apical impulse (2.4).

Likelihood ratios of the physical examination for detecting aortic regurgitation

<table>
<thead>
<tr>
<th>Patient Population</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall cardiac examination</td>
<td>5.1</td>
<td>0.82</td>
</tr>
<tr>
<td>Referred for evaluation of systolic murmur</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Third heart sound (to identify severe AR)</td>
<td>5.9</td>
<td>0.83</td>
</tr>
<tr>
<td>Patients with isolated aortic insufficiency, referred for echocardiography</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: AR, aortic regurgitation; CI, confidence interval; PLR, positive likelihood ratio; NLR, negative likelihood ratio.


**SO YOU WANT TO BE A CARDIOLOGIST!**

Q: In the context of aortic regurgitation (AR), what is Duroziez’ double murmur?

A:  
- Application of gradual pressure over femoral artery in persons with aortic regurgitation (AR) results in a to-and-fro systolic and diastolic murmur.
- Sensitivity of 58-100% for AR (false positive in all high output states).

**Causes of collapsing pulse: Hyperdynamic circulation due to**
- Aortic regurgitation
- Thyrotoxicosis
- Severe anaemia
- Paget’s disease
- Complete heart block

Source: Baliga R.R. Saunders/Elsevier 2007, page 78
Useful background: Aortic regurgitation

- If the examiner does NOT hear an AR murmur, the likelihood that the patient has AR is diminished as follows:
  - NLR 0.1 for moderate or greater AR
  - NLR 0.2 to 0.3 for mild or greater AR

- If the examiner DOES hear an AR murmur, the likelihood that the patient has AR is increased as follows:
  - PLR 4.0 to 8.3 for moderate or greater AR
  - PLR 8.8 to 32.0 for mild or greater AR

Abbreviations: AR, aortic regurgitation; PLR, positive likelihood ratio; NLR, negative likelihood ratio


**Pericardial diseases**

Useful background: Pericardial friction rubs

- Systolic as well as diastolic components (one near $S_3$, early in diastole; the second diastolic component is at time of $S_4$)
- May resemble a $S_3$ gallop
- Best heard 3/4 th LSB (left sterna boarder); sitting up, leaning forward, holding breath in inspiration
- Caused by pericarditis:
  - All pericardium
    - Viral
    - Lupus
    - Uremia
  - Focal pericarditis
    - Myocardial infarction
    - Tumor

**Pericarditis**: Tachycardia; friction rub; diminished heart sounds and enlarged heart to percussion (with effusion); pulsus paradoxicus; neck vein distention, narrow pulse pressure and hypotension (with tamponade).

Abbreviations: MVP, mitral valve prolapse;

Source: Mangione S. *Hanley & Belfus*, 2000, pages 246-251; pages 74 and 75.

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**SO YOU WANT TO BE A CARDIOLOGIST!**

**Q1**: In the context of cardiac tamponade, what is Beck’s triad?

**A1**:  
- Low arterial blood pressure
- High venous pressure
- Absent apex in cardiac tamponade is known as Beck’s triad

**Q2**: What is Dressler’s syndrome?

**A2**: Persistent pyrexia, pericarditis and pleurisy, post-myocardial infarction

---

**Pericarditis**

42. Take a directed history for the causes of pericarditis.

- **Immune**
  - Collagen vascular disease

- **Metabolic**
  - Rh. Fever
  - Uremia
  - Myxedema

- **Tumor**
  - Neoplasm
  - Radiation

- **Trauma**
  - Post – surgery
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- Idiopathic
  - Endomyocardial fibrosis (EMF)

- Drug (phenylbutazone)

- Infective
  - Viral
  - Bacterial (pyogenic or TB)
  - Mycotic
  - Parasitic
  - Post–purulent constriction

- Post-myocardial infarction (Dressler’s syndrome; pericarditis may be focal)

Abbreviations: EMF, endomyocardial fibrosis; TB, tuberculosis


Useful background: Causes of constrictive pericarditis

- Tuberculosis (<15% of patients)
- Connective tissue disorder
- Neoplastic infiltration
- Radiation therapy (often years earlier)
- Postpurulent pericarditis
- Hemopericardium after surgery
- Chronic renal failure

43. Perform a focused physical examination for acute cardiac tamponade.

- Inspection
  - Tachypnea
    - Anxiety
    - Restlessness
    - Syncope
  - JVP ↑, Kussmaul’s sign, prominent x but absent y descent
  - Pulse and blood pressure
    - Rapid pulse rate
    - Pulsus paradoxus
    - Hypotension
  - Apex beat: not palpable
Auscultation: reduced heart sounds

Lungs: dullness and bronchial breathing at the left base (due to lung compression by the distended pericardial sac).

Abbreviation: JVP, jugular venous pressure


44. Take a directed history and perform a focused physical examination for chronic constrictive pericarditis.

History
   - Tiredness
   - Exertional dyspnoea
   - Effort syncope
   - Symptoms due to ascites
   - Ankle swelling
   - Nausea, vomiting, dizziness, cough

Physical signs: cachexia
   - Pulse and blood pressure: Pulsus paradoxus (more than the normal 10 mm Hg fall in the arterial pulse pressure on inspiration, because increased right ventricular filling compresses the left ventricle); low blood pressure
   - The JVP ↑, Kussmaul’s sign (uncommon); prominent x and y descents
   - Apex beat: not palpable
   - Auscultation: Heart sounds distant, early S3, early pericardial knock (rapid ventricular filling abruptly halted)
   - Abdomen: hepatomegaly, splenomegaly, ascites
   - Peripheral edema
   - Causes of chronic constrictive pericarditis:
     - Cardiac operation or trauma
     - Tuberculosis
- Histoplasmosis or pyogenic infection
- Neoplastic disease
- Mediastinal irradiation
- Connective tissue disease (especially rheumatoid arthritis)
- Chronic renal failure.

Abbreviation: JVP, jugular venous pressure


45. Perform a focused physical examination to distinguish between the presence of chronic pericarditis with constriction constructive pericarditis (CP) and cardiac tamponade (CT).

- **Inspection**
  - Cachexia, in chronic tachypnea
  - Anxiety, restless (CT)
  - Syncope (CT)

- **Palpation**
  - **Pulse**
    - Increased rate
    - Pulsus paradoxus
    - (↓ BP on inspiration > 10 mmHg)
    - (JVP ↑, Kussmaul’s sign, prominent x and y descents)
  - **Blood pressure**
    - Reduced
  - **Apex beat**
    - Difficult to feel

- **Auscultation**
  - Difficult to hear heart sounds
  - Early S₃
  - Pericardial knock

- **Associations**
  - Hepatosplenomegaly
  - Peripheral edema
  - Left lung base dull, with bronchial breathing due to lung compression (CT)

- **Signs of pericardial disease:** ↑ JVP, dyspnea, pericardial rub or effusion

- **Signs of cardiac tamponade:** pulsus paradoxus: ASA, severe LV dysfunction (especially those with uremic pericarditis), regional tamponade (tamponade affecting 1 or 2 heart chambers, such as following heart surgery), severe hypotension, aortic regurgitation.

- **Cardiac tamponade without pulsus paradoxus:** ASD, severe LV dysfunction (especially those with uremic pericarditis), regional
tamponade (tamponade affecting 1 or 2 heart chambers, such as following heart surgery), severe hypotension, aortic regurgitation

Abbreviations: ASA, American Society of Anesthesiologists; ASD, atrial septal defect; CPC, chronic pericarditis with constriction; CT, cardiac tamponade; JVP, jugular venous pressure; LV, left ventricle


SO YOU WANT TO BE A CARDIOLOGIST!

Q: In the context of the patient with constrictive pericarditis, what is Broadbent’s sign?
A: Intercostal indrawing during systole.

Useful background: Causes of pericardial effusion

- Exudates
  - Acute pericarditis
  - Metastatic malignancy

- Transudates
  - CHF
  - Liver failure
  - Nephrotic syndrome
  - Myxedema

- Hemopericardium
  - Aortic dissection
  - Trauma

Useful background: Performance characteristics of the physical examination for constrictive pericarditis (CP) and cardiac tamponade (CT)

<table>
<thead>
<tr>
<th>Physical finding</th>
<th>Frequency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>JVP ↑</td>
<td></td>
</tr>
<tr>
<td>o ↑ y descent</td>
<td>98</td>
</tr>
<tr>
<td>o (Friedrich’s sign)</td>
<td>57-94</td>
</tr>
<tr>
<td>o Kussmaul’s sign</td>
<td>50</td>
</tr>
<tr>
<td>Pulse</td>
<td></td>
</tr>
<tr>
<td>o AF tachycardia &gt; 100 bpm</td>
<td>36-70</td>
</tr>
<tr>
<td>BP</td>
<td></td>
</tr>
<tr>
<td>o Pulsus &gt; 10 mmHg</td>
<td>17-43</td>
</tr>
<tr>
<td>o Paradoxicus &gt; 20</td>
<td>&gt;20</td>
</tr>
<tr>
<td></td>
<td>&gt;30</td>
</tr>
<tr>
<td></td>
<td>&gt;40</td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Auscultation of presordium</td>
<td></td>
</tr>
<tr>
<td>o Total paradox</td>
<td>23</td>
</tr>
<tr>
<td>o Pericardial knock</td>
<td>28-94</td>
</tr>
<tr>
<td>o Rub</td>
<td>4</td>
</tr>
<tr>
<td>o ↓ heart sounds</td>
<td>-</td>
</tr>
<tr>
<td>Other</td>
<td></td>
</tr>
<tr>
<td>o Hepatomegaly</td>
<td>87-100</td>
</tr>
<tr>
<td>o Edema</td>
<td>63</td>
</tr>
<tr>
<td>o Ascites</td>
<td>53-89</td>
</tr>
</tbody>
</table>

Abbreviations: AF, arterial fibrillation; BP, blood pressure; CP, constrictive pericarditis; CT, cardiac tamponade; JVP, jugular venous pressure

**Congenital heart disease**

**SO YOU WANT TO BE A PEDIATRIC CARDIOLOGIST!**

**Q: What are the types of VSD?**

**A:**

- The supracristal type (above the crista supraventricularis):
  - A high defect just below the pulmonary valve and the right coronary cusp of the aortic valve
  - The latter may not be adequately supported, resulting in aortic regurgitation
  - In Fallot’s tetralogy this defect is associated with the rightward shift of the interventricular septum
  - In double-outlet left ventricle with subaortic stenosis the supracristal defects associated with a leftward shift of the septum

- The infracristal defect, which may be in either the upper membranous portion of the interventricular septum, or the lower muscular part
  - Small defects (maladie de Roger; curiously, a very loud murmur)
  - Swiss cheese appearance (multiple small defects)
  - Large defects
  - Gerbode defect (defect opening into the right atrium)


**Useful background: Anatomic classification of congenital heart disease**

- **Shunts**
  - R and L heart shunts \((R \rightarrow L \text{ or } L \rightarrow R)\)

- **Valvular defects**
  - Aortic or pulmonary stenosis
  - Bicuspid aortic valve (predisposes to later aortic stenosis)
  - Tricuspid atresia

- **Complex lesions**
  - Fallot’s tetralogy
  - Transposition of the great vessels
  - Ebstein’s anomaly
  - Combinations of defects

Physiological classification of congenital heart disease based on the presence or absence of cyanosis

- **Acyanotic**
  - With L → R shunt
    - Ventricular septal defect (VSD)
    - Atrial septal defect (ASD)
    - Patent ductus arteriosus (PDA)
  - With no shunt
    - Bicuspid aortic valve, congenital aortic stenosis
    - Coarctation of aorta
    - Dextrocardia
    - Pulmonary stenosis, tricuspid stenosis
    - Ebstein’s anomaly

- **Cyanotic**
  - Ebstein’s anomaly (if an atrial septal defect (ASD) and R → L shunt are also present)
  - Truncus arteriosus
  - Transposition of the great vessels
  - Tricuspid atresia
  - Total anomalous pulmonary venous drainage
  - Eisenmenger’s syndrome (pulmonary hypertension and a right – to - left shunt)
  - Tetralogy of Fallot


Useful background: Causes of cyanosis

- **Central cyanosis**
  - Decreased arterial oxygen saturation
    - Decreased concentration of inspired oxygen: high altitude
    - Reduced cardiac output: left ventricular failure or shock
    - Lung disease: chronic obstructive pulmonary disease with cor pulmonale, massive pulmonary embolism
    - Right-to-left cardiac shunt (cyanotic congenital heart disease)
  - Polycythemia
  - Hemoglobin abnormalities (rare): methemoglobinemia, sulphemoglobinemia

- **Peripheral cyanosis**
  - All causes of central cyanosis cause peripheral cyanosis
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- Exposure to cold
- Arterial or venous obstruction


46. Perform a focused physical examination to determine the cause of acyanotic, cyanose tardive, and cyanotic congenital cardiovascular disease.

- **Acyanotic** – coarctation of the aorta; aortic stenosis (valve or infundibulum 1-2 cm below the valve); dextracardia

- **Cyanose tardive** - (< 30% R→L shunt; > 30% with exercise, CCF) ASD, VSD (Roger’s disease; Eisenmenger complex), PFA, PDA, PS

- **Cyanotic** – tetrology of Fallot, transposition of great vessels

- **Coarctation of the aorta**
  - Visible or palpable pulsation above the episternal notch
  - Systolic murmur (front, back)
  - SBP in arms > legs
  - Collateral circulation (internal mammary, interstitial, scapillary arteries)
  - Chest x-ray
    - ↓ aortic knuckle
    - Indentation of aortic shadow at point of contraction
    - Notching of lower borders of ribs (erosion by dilated intercostal arteries)

- **Patent foramen ovale (PFA)**
  - Persistent valve-like opening covered by a membrane on the left side
  - May cause paradoxic embolus (peripheral vein or RA embolus goes to systolic circulation, e.g. brain, kidney rather than lung
  - With RAH and LAH, large defect produced like ASD

- **Atrial septal defect (ASD)**
  - Early LA → RA: no cyanosis or clubbing (one of the few causes of R-side enlargement without cyanosis)
  - Murmur of PS (↑ flow)
  - ↑P₂
  - Late RA→LA: cyanosis, clubbing

- **Chest x-ray**
  - RVH
  - Prominent, pulsating pulmonary arteries
- Hilar dance (prominent, pulsating hilar shadows)
- Small aorta

- Lutenbacher syndrome
  - ASA plus MS (mitral stenosis)

- Interventricular defects (IVD)
  - Roger’s disease
    - Small IVD
    - Harsh systolic murmur, L-3rd, ICS
    - Thrill (in 90%)
  - Eisenmenger complex
    - Dextraposed aorta (partially arising from RV)
    - Large high VSD
    - Systolic murmur with a thrill; may have associated AR
    - Distinguish from tetrology of Fallot

- Patent ductus arteriosus (PDA)
  - Persistance of fetal connection between pulmonary artery into aorta
  - Continuous (systolic and diastolic) all over precordium, or medial to left midscapula
  - Thrill
  - LVH, RVH; late, RV → LV shunt, with cyanosis
  - ↑ PP due to ↓ DBP (PP = SBP - DBP)
  - Corrigan pulse (also seen in AR)

- Tetralogy of Fallot
  - VSD, RVH, PS, dextra posed aorta
  - Cyanosis, clubbing, retarded growth, polycythermia
  - $A_2 > P_2$
  - Distinguish from Eisenmenger complex

- Transposition of the great vessels
  - RV → aorta, LV → pulmonary artery
  - Survival only with a shunt: PFA, VSD, PDA

Abbreviations: AR, aortic regurgitation; ASD, atrial septal defect; CCF, congestive cardiac failure; DBP, diastolic blood pressure; IVD, interventricular defects; LA, left atrium; LV, left ventricle; LVH, left ventricular hypertrophy; MS, mitral stenosis; PDA, patent ductus arteriosus; PFA, patent forman ovale; PP, pulse pressure; PS, pulmonary stenosis; RA, right atrium; RV, right ventricle; RVH, right ventricle hypertrophy; SBP, systolic blood pressure; VSD, ventricular septal defect

47. Perform a focused physical examination for dextrocardia.

- Apex beat is on the right side.
- Heart sounds heard on the right side of the chest
- Liver dullness on the left side
- Possible bronchiectasis
- Mazinkowski’s sign – not named after this person!
  - Dextrocardia without evidence of situs inversus, usually associated with cardiac malformation
  - May occur with cardiac malformation in Turner’s syndrome


SO YOU WANT TO BE A CARDIOLOGIST!

Q1: Which cardiac lesions are dependent on PDA?
A1:  
  - Hypoplastic left heart syndrome
  - Complex coarctation of aorta
  - Critical congenital aortic stenosis

Q2: what happens to the continuous murmur of patent ductus arteriosus (PDA) in pulmonary hypertension?
A2:  
  - First the diastolic murmur, then the systolic murmur, becomes softer and shorter
  - P2 increases intensity

Source: Baliga R.R. Saunders/Elsevier 2007, page 81

Q3: What is Lutenbacher syndrome?
A3: ASD plus mitral stenosis.

48. Take a directed history and perform a focused physical examination for Eisenmenger syndrome.

- Definition
  - Pulmonary hypertension with a reverse or bidirectional shunt
  - Shunt (VSD, ASD, patent ductus arteriosus, persistent truncus arteriosus, single ventricle or common artroventricular canal

- History
- CNS
  - Cerebrovascular accidents (as a result of paradoxical embolization, venous thrombosis of cerebral vessels, or intracranial haemorrhage)
  - Sudden death
  - Brain abscess
- Lung
  - Pulmonary embolization
  - Hemoptysis
- Heart
  - Right ventricular failure
  - Paradoxical embolization
  - Infective endocarditis
- Kidney
  - Hyperuricemia

- Physical examination
  - Clubbing of fingers
  - Central cyanosis
  - JVP ‘a’ waves ; ‘v’ wave if tricuspid regurgitation is present
  - Left parasternal heave
  - Palpable P2
  - Loud P2
  - Pulmonary ejection click
  - Early diastolic murmur of pulmonary regurgitation (Graham Steell murmur)
  - Tricuspid regurgitation
  - S2
    - VSD: single second sound
    - ASD: fixed, wide split second sound
    - PDA: reverse split of second sound, and lower-limb cyanosis

Adapted from: Baliga R.R. Saunders/Elsevier 2007, pages 88 and 89.

49. Perform a focused physical examination for a ventricular septal defect (VSD).

- Murmur
  - Continuous in systole and diastole, with no interval of a silent pause
  - May cover entire systole, or be decrescendo, crescento, or mixed crescendo/decrescendo
  - Best heard L. LSB
  - If there is a crescendo pattern, it starts after S₁
  - Usually due to PDA (patent ductus arteriosus)
  - When pulmonary hypertension (PHT) develops, diastolic component disappears.
With further worsen PHT, the continuous murmur completely disappears.

<table>
<thead>
<tr>
<th>Murmur</th>
<th>Location</th>
<th>Radiation</th>
<th>Quality</th>
<th>Pitch</th>
<th>Associated signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>VSD</td>
<td>3rd-5th LICS</td>
<td>Wide</td>
<td>Harsh</td>
<td>High</td>
<td>Vary with severity</td>
</tr>
</tbody>
</table>

Complications
- CHF
- RV outflow obstruction (muscular infundibular obstruction)
- Aortic regurgitation
- Infective endocarditis
- Pulmonary hypertension and reversal of shunt L → R → R → L (Eisenmenger complex)

Causes (defect in the membranous portion of the interventricular septum)
- Congenital
- Rupture of the interventricular septum (eg. complication of myocardial infarction)

Associations
- Heart
  - Fallot’s tetralogy
  - Double-outlet right ventricle
  - AV canal defects
  - Patent ductus arteriosus (PDA)
  - Pulmonary stenosis
  - Secundum atrial septal defects
  - Tricuspid atresia
- Vessels
  - Truncus arteriosus
  - Coarctation of aorta
  - Transposition of the great arteries
  - Pulmonary atresia

Abbreviation: L. LSB, left lower sternal border

50. Perform a focused physical examination for tetralogy for Fallot’s.

- **Necessary finding**
  - VSD with R → L shunt
  - PS (infundibular or valvular)
  - RVH
  - Dextroposition of the aorta, with overriding of the VSD

- **Physical examination**

- **Skin/ hands**
  - Clubbing
  - Central cyanosis

- **Precordium**
  - Palpation
    - Left parasternal heave with normal left ventricular impulse
  - Auscultation
    - Ejection systolic murmur heard in the pulmonary area
  - After Blalock-Taussing shunt
    - Radial pulse L < R
    - The arm on the side of the anastomosis (usually the left) may be smaller than the other arm
    - Blood pressure difficult to obtain (narrow pulse pressure in the arm supplied by the collateral vessels)
    - Thoracotomy scar

- **Complications**
  - Cyanotic and syncopal spells
  - Cerebral abscess
  - Endocarditis

Abbreviation: PS, pulmonary stenosis; RVH, right ventricular hypertrophy


SO YOU WANT TO BE A CARDIOLOGIST!

Q1: What is Kartagener’s syndrome?
A1:  o Dextrocardia or situs inversus bronchiectasis
      o dysplasia of the frontal sinuses

Q2: Which other abnormality has been associated with dextrocardia?
A2:  o Asplenia (blood smear may show Heinz and Howell-Jolly bodies)
Q1: What is the Taussing-Bing syndrome?
A1:  
  o The aorta arises from the right ventricle  
  o The pulmonary trunk overrided both ventricles at the site of an interventricular septal defect


Q2: What is the effect of pregnancy in women with VSD?
A2: Small defects should be present no problems.

Q: What are the types of aortic coarctation?
A:  
  ➢ Common:  
    o Infantile or preductal where the aorta between the left subclavian artery and patent ductus arteriosus is narrowed. Its manifests in infancy with heart failure. Associated lesion include patent ductus arteriosus, aortic arch anomalies, transposition of the great arteries, ventricular septal defect.  
    o Adult type: the coarctation in the descending aorta is juxtaductal or slightly post ductal. It may be associated with bicuspid aortic valve or patent ductus arteriosus. It commonly between the age of 15 and 30 years.

  ➢ Rare  
    o Localized juxtaductal corctation  
    o Coarctation of the ascending thoracic aorta

SO YOU WANT TO BE A CARDIOLOGIST!

Q1: What do you understand by the term ‘situs inversus’?
A1: Right-sided cardiac apex, right stomach, right-sided descending aorta. The right atrium is on the left. The left lung has three lobes and the right lung has two.

Q2: What do you understand by the term ‘dextroversion’?
A2: Right-sided cardiac apex, left sided stomach and left-sided descending aorta.

Q3: What do you understand by the term “levoversion”?
A3: Left-sided apex, right-sided stomach and right descending aorta.


---

SO YOU WANT TO BE A PEDIATRIC CARDIOLOGIST!

Q1: You know all about Fallot and his tetralogy?
A1: Well, ASD, pulmonary stenosis and right ventricular hypertrophy.

Q2: What is Fallot’s pentatology?
A2: Fallot’s tetralogy with associated ASD is known as Fillot’s pentology.

Q3: In the contest of a systolic murmur, what is the ‘Gallavardin phenomenon’?
A3: The high-frequency components of the ejection systolic murmur may radiate to the apex, falsely suggesting mitral regurgitation.
SO YOU WANT TO BE A PEDIATRIC CARDIOLOGIST!

Q1: What causes rib notching?
A1:  
- Collateral flow through dilated, tortuous and pulsatile posterior intercostals arteries typically causes notching on the under surfaces of the posterior portions of the ribs.
- The anterior parts of the ribs are spared because the anterior intercostals arteries do not run in the costal grooves.
- Notching is seldom found above the third or below the ninth rib and rarely appears before the age of 6 years.
- Conditions in which rib notching is seen
  - Coarctation of aorta
  - Pulmonary oligaemia
  - Blalock-Taussig shunt
  - Subclavian artery obstruction
  - Superior vena caval syndrome
  - Neurofibromatosis
  - Arteriovenous malformations of the lung or the chest wall

Q2: What are the complications of aortic coarctation?
A2:  
- Severe hypertension and resulting complications:
  - Stroke
  - Premature coronary artery disease


Q3: What are the fundal findings in coarctation of aorta?
A3: Hypertension due to coarctation of aorta causes retinal arteries to be tortuous with frequent ‘U’ turns; curiously, the classical signs of hypertensive retinopathy are rarely seen.

Diseases of the aorta

51. Perform a focused physical examination for syphilitic aortitis with saccular aneurysm of aorta.

- **Nerves**
  - L. recurrent laryngeal nerve
    - Hoarseness or aphi\(\)nia
  - Cervical sympathetic nerve
    - Unequal pupils (Horner’s syndrome)

- **Trachea**
  - Tug, displacement, stridor

- **Bronchi**
  - Cough, atelectasis

- **Brachial artery**
  - Difference in BP in arms > 20 mmHg

- **Heart**
  - Aortic dilation, AR
  - Signs of MI, from obstruction of coronary arteries
  - Signs of myocarditis
  - Conduction defects (syphilitic gamma)

- **Bone**
  - Erosion of spine, pulsation in region of L. scapula, bulging of aortic area of chest wall

Abbreviations: AR, aortic regurgitation; BP, blood pressure; MI, myocardial infarction;

Useful background: Abdominal Aortic Aneurysm (AAA)

<table>
<thead>
<tr>
<th>Width of aorta by palpation</th>
<th>Sensitivity (%)</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥3.0 cm (all)</td>
<td>39</td>
<td>12.0</td>
<td>0.72</td>
</tr>
<tr>
<td>3.0 cm – 3.9 cm</td>
<td>29</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥4.0 cm</td>
<td>15.6</td>
<td>0.51</td>
<td></td>
</tr>
<tr>
<td>4.0 cm – 4.9 cm</td>
<td>50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≥5.0 cm</td>
<td>76</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Physical sign

<table>
<thead>
<tr>
<th>Physical sign</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definite pulsatile mass</td>
<td>28</td>
<td>97</td>
</tr>
<tr>
<td>Definite or suggestive pulsatile mass</td>
<td>50</td>
<td>91</td>
</tr>
<tr>
<td>Abdominal bruit</td>
<td>11</td>
<td>95</td>
</tr>
<tr>
<td>Femoral bruit</td>
<td>17</td>
<td>87</td>
</tr>
<tr>
<td>Femoral pulse deficit</td>
<td>22</td>
<td>91</td>
</tr>
</tbody>
</table>

The only physical exam manoeuvre of demonstrated value for diagnosis of an AAA is abdominal palpation to detect a widened aorta.

Abbreviation: AAA, abdominal aortic aneurysm

Source: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, page 259.

Useful background: Performance characteristics of clinical findings for thoracic aortic dissection

<table>
<thead>
<tr>
<th>Symptom or sign</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Focal neurologic deficit</td>
<td>6.6-33</td>
<td>0.7-0.9</td>
</tr>
<tr>
<td>Pulse deficit</td>
<td>5.7</td>
<td>0.7</td>
</tr>
<tr>
<td>Enlarged aorta or wide mediastinum</td>
<td>2.0</td>
<td>0.3</td>
</tr>
</tbody>
</table>

All often symptoms/signs (including pulse deficit, murmur of aortic insufficiency and widened mediastinum on chest X-ray have low sensitivity – [Simel David L, et al. JAMA 2009, Table 50-8, page 672.])

Abbreviations: PLR, positive likelihood ratio; NLR, negative likelihood ratio

*Note that many historical points, symptoms and signs on physical examination have a PLR < 2 (and are not included here)

Bacterial endocarditis

Useful background: Modified Duke criteria for the diagnosis of infective endocarditis

➢ Definite infective endocarditis

➢ Diagnosis accepted
  o Pathologic criteria
    - Microorganism on culture or histologic examination of a vegetation, a vegetation that has embolize, or an intracardiac abcess specimen; or
    - Pathological lesion; vegetation or intracardiac abcess confirmed by histologic examination showing active endocarditis
  o Clinical criteria—a
    - 2 major criteria, or
    - 1 major criterion and 3 minor criteria, or
    - 5 minor criteria
  o Possible infective endocarditis
    - 1 major criterion and 1 minor criterion, or
    - 3 minor criteria

➢ Diagnosis rejected
  o Firm alternative diagnosis explaining evidence of infective endocarditis, or
  o Resolution of infective endocarditis syndrome which antibiotic therapy for ≤ 4 days, or
  o No pathological evidence of infective endocarditis, as above


52. Perform a focused physical examination for bacterial endocarditis.

➢ Clinical features of subacute bacterial endocarditis
  o Fever, tachycardia, arthralgia, pallor with café au lait complexion
  o Microscopic hematuria
  o Osiers nodes- pink, tender, on digits, palms or soles
  o Splinter hemorrhages and petechiae
  o Clubbing
  o Splenomegaly
  o R- CHF or other endocardial damage

➢ Acute damage
  o Cusps
  o Chordae tendinæ
- Endocardium
  - Pneumococcus, haemolytic streptococcus, staphylococcus, gonococcus
- Pericardium
  - Pneumonia
  - Strep’
- Myocardium
  - Staph’
  - Affects either a previously normal or abnormal valve

- Subacute damage
  - Cusps, chordae tendinae, endocardium, aorta
  - Usually affects a previously abnormal valve
  - New murmur, or changing murmur
  - Fever
  - Joint pain and tenderness
  - Anemia
  - Petechial hemorrhage
  - Embolism
  - Clubbing

- Risk factors
  - Stratify risk of having bacterial endocarditis

  - High risk factors
    - Rheumatic fever
    - Artificial valves
    - Previous IE
    - IV drug users
    - Intravascular devices (e.g. arterial lines)
  - Pulmonary risk factors
    - Septic pulmonary embolism
  - Neurological risk factors
    - Immune mediated phenomena
    - Focal deficit
    - Headache
    - Meningitis

  - Moderate risk factors
    - Most congenital heart malformations
    - Valvular dysfunction
    - HCM
    - MVP with MR
  - Metastatic infection
    - Organ infarction
    - Embolic manifestations

  - Peripheral signs
    - Petechiae
    - Conjunctivae
    - Buccal mucosa
    - Palate
    - Splinter hemorrhages: linear dark red streaks (nails)
    - Janeway lesions: ~ 5mm non-tender hemorrhagic macules on

  - Ask the patient about:
    - Prosthetic heart valves
    - Recent surgeries
    - Indwelling catheters or hemodialysis
    - Recent IV drug use
Constitutional risk factors
- Fever
- Chills
- Malaise
- Night sweats
- Anorexia
- Arthralgias

Cardiac risk factors
- Murmur
- Palpation
- CHF

MSK
- Splenomegaly
- Synovitis
- Vasculitis
- Glomerulonephritis

Abbreviations: R-CHF, right side congestive heart failure; HCM, hypertrophic cardiomyopathy; IE, infectious endocarditis; MR, mitral regurgitation; MVP, mitral valve prolapse.

Adapted from: Burton J.L. Churchill Livingstone, 1971; and Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, Table 13, page 66.

Useful background: Valve disease predisposing to endocarditis

Patient
- Prosthetic valves*
- Previous endocarditis*

Valves
- Mitral or aortic regurgitation (MR, AR)*
- Ventricular septal defect (VSD)*
- Patent ductus arteriosus (PDA)*
- Aortic stenosis (AS)
- Hypertrophic cardiomyopathy
- Atrial septal defect (ASD)
- Pure mitral stenosis (MS)

*high risk

Palpitations

53. Take a directed history for palpitations.

- Definition: sensations of a rapid or irregular heartbeat occurring in normal, healthy people during exercise and states of anxiety.

- General
  - Presyncope/syncope
  - Malaise/fatigue
  - Fever/chills/night sweats
  - Diaphoresis

- Heart
  - Slow vs. rapid, regular vs. irregular
  - Frequency of bouts
  - Duration
  - Nature of onset/offset

- CNS
  - Numbness/paresthesia
  - Weakness
  - Visual/speech abnormalities

- Lung
  - Lung base crackles
  - Dyspnea/orthopnea/PND
  - Cough
  - Chest pain

- Ankles/sacrum edema

- Causes
  - Heart
    - Pericardial (pericarditis)
    - Myocardial (LVH, infarction, CHF, myxoma, ASD, amyloidosis)
    - Endocardial (infarction, sick sinus, valvular- MS/MR, AS/AR
  - Lung
    - Asthma, COPD
    - Pneumonia
    - Pulmonary embolism
  - Endocrine
    - Hyperthyroid
    - Pheochromocytosis
    - Hypoglycemia
  - Drugs and Toxins
    - EtOH (binge or withdrawal)
    - CO poisoning
    - Stimulants (caffeine, theophylline, amphetamines, cocaine)
  - Metabolic – electrolyte abnormalities
  - Infection – sepsis

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Systemic hypertension

Useful background: Definitions

- Asymptomatic: systolic BP >200 mm Hg +/- diastolic BP > 120 mm Hg: Needs therapy to prevent potential complications of a malignant hypertensive crisis.
- Malignant hypertension: Symptomatic accelerated hypertension (hypertension plus end organ damage)

Definition: SBP > 180 mm Hg, DBP > 100 mm Hg

- End-organ damage
  - Eye
    - Retinal hemorrhages
    - Optic nerve edema
    - Blurred vision
  - Brain
    - CVA (thrombosis; intracerebral / or subarachnoid hemorrhage)
    - Confusion
    - Headaches
    - Seizures
  - Heart
    - Ischemia
    - L-CHF
    - Dissecting aortic aneurysm
  - Lung
    - Acute pulmonary edema
  - Kidney
    - Rapid ↑Cr, ↓CCR
    - Acute renal failure

- May be a disconnect between BP and end-organ damage


Abbreviations: ASD, atrial septal defect; CHF, congestive heart failure; CO, carbon monoxide; COPD, chronic obstructive pulmonary disease; CVA, cerebro vascular accident; ETOH, ethanol; LVH, left ventricular hypertrophy; PND, paroxysmal nocturnal dyspnea; TIA, transient ischemic attack

Classification of blood pressure for adults 18 years of older

<table>
<thead>
<tr>
<th>Category</th>
<th>Blood Pressure Level, mm Hg</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Systolic</td>
<td>Diastolic</td>
</tr>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>&amp; &lt;80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120-139</td>
<td>or 80-89</td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 1</td>
<td>140-159</td>
<td>or 90-99</td>
</tr>
<tr>
<td>Stage 2</td>
<td>≥160</td>
<td>or ≥100</td>
</tr>
</tbody>
</table>

Source: Ghosh A.K. Mayo Clinic Scientific Press 2008, Table 14-1, page 425

54. Perform a directed physical examination of the patient with systemic hypertension.

- **Eye**
  - Vessels
    - Arteriolar narrowing
    - A-V nicking
    - Hemorrhages
    - Exudates
    - Papilledema

- **Retina**
  - Papilledema (blurring of disc margins, no venous pulsations)
  - Narrowing/irregularity of arterioles
  - AV nicking
  - Hemorrhages
  - Cotton wool spots

- **CNS**
  - Evidence of CVA (effect on cranial nerves, cerebellum, motor/sensory systems)

- **Neck**
  - Thyromegaly
  - Carotid bruits
  - ↑ JVP

- **Cardiovascular examination: evidence of**
  - Left ventricular hypertrophy
  - Congestive heart failure
Peripheral vasculature
- Bruits
  - Abdominal aorta
  - Arteries - Renal arteries, femoral arteries, popliteal arteries, posterior tibial artery, dorsalis pedis artery
- Absent or diminished peripheral pulses
- Ankle to brachial index (peripheral vascular disease or coarctation of the aorta)

Abdomen
- Skin
  - Striae of Cushing’s syndrome
- Kidney
  - Renal bruits to suggest renovascular hypertension
  - Renal masses to suggest PCK disease
- Arteries
  - Abnormal aortic pulsations
  - Femoral bruits to suggest peripheral vascular disease
  - Radio-femoral delay to suggest coarctation of the aorta

Abbreviations: AV, aortic valve; CHF, congestive heart failure; CNS, central nervous system; CVA, cerebrovascular accident; JVP, jugular venous pressure; LVH, left ventricular hypertrophy; PCK, polycystic kidney disease.


55. Take a directed history for the causes of systemic hypertension.

Primary

Secondary
- Renal
  - Ischemia
  - Pyelonephritis
  - Glomerulonephritis
  - Polycystic kidney disease
  - Hydronephrosis
  - Diabetes, gout, amyloidosis, nephrocalcinosis
  - Collagen vascular disease
- Metabolic
  - Pheochromocytoma
  - Cushing’s syndrome
  - Conn’s syndrome
- Toxemia of pregnancy
- Coarctation of aorta
- Neurogenic
- Bulbar polio
- Head injury
- Hypothalamic tumour


56. Take a focused history for complications of malignant hypertensive emergency.

- **CNS**
  - Confusion
  - Seizures
  - Headaches
  - Visual changes
  - Cerebral thrombosis
  - Intracerebral or subarachnoid hemorrhage

- **Heart**
  - Unstable angina
  - Myocardial infarction
  - Dissecting aortic aneurysm

- **Lung**
  - Acute pulmonary edema

- **Kidney**
  - Acute renal failure

- **Genitourinary**
  - Severe pre-eclampsia and eclampsia

- **Endocrine**
  - Pheochromocytoma

Abbreviations: CNS, central nervous system.


57. Perform a focused physical examination for an abnormally widened pulse pressure.

- **Definition:** pulse pressure > 50% of systolic blood pressure

- **Causes**
  - Hyperdynamic heart syndrome (↑SV, ↓PVR)
  - Aortic regurgitation
  - Patent ductus arteriosus (PDA)
Exercise
Anemia
Arteriovenous fistulas
Beriberi
Paget’s disease
Cirrhosis
Pregnancy
Thyrotoxicosis
Severe exfoliative dermatitis

Abbreviations: PVR, peripheral vascular resistance; SV, stroke volume.

SO YOU WANT TO BE A CARDIOLOGIST!

Q: Differentiate between Pseudohypertension and Pseudohypotension

A:  

- Pseudohypertension – Artery can be palpated when a blood pressure cuff is inflated to the point of obliterating the radial pulse, and the artery is still palpated as a firm tube in the absence of a pulse (Osler’s maneuvre). Positive Osler’s sign, indicating the presence of arterosclerosis, and both SBP and DBP be overestimated.

- Pseudohypotension – in conditions of high peripheral vascular resistance such as shock, Korotkoff sounds are difficult to use to measure accurately systolic or diastolic pressure.


Pulse pressure

58. Take a focused history and perform a directed physical examination for causes of hyperkinetic heart syndrome causing an abnormally wide pulse pressure (PP) (PP > 50% of systolic BP).

- Increased pulse pressure
  - Heart
    - Aortic regurgitation
    - Patent ductus arteriosus
    - AV fistula
  - Lung
    - Hypercapnia
  - Metabolic
- Fever
- Anemia
- Beriberi
- Hyperthyroidism
  - Bone – Paget’s disease
  - Liver – cirrhosis
  - Skin – severe exfoliative dermatitis
  - Pregnancy

- Reduced pulse pressure (PP; PP < 25% of SBP).
  - Aortic stenosis
  - Constrict percarditis
  - Cardiac tamponade
  - Tachycardia
  - Hypotension

Useful background: Cause of differences in blood pressure between arms or between arms or between the arms and legs?

- Heart
  - Patent ductus arteriosus
  - Supravalvular

- Aorta
  - Coarctation of the aorta
  - Dissecting aortic aneurysm
  - Aortic stenosis

- Artery
  - Occlusion or stenosis of the artery of any cuase
  - Thoracic outlet syndrome


SO YOU WANT TO BE A CARDIOLOGIST!

Q: In the context of increased pulse pressure in one limb (due to AV fistula), what is the area of the Branham sign? (compressing the area of suspected AV fistula causes ↓ HR).

A: Branham sign is bradycardia caused by inhibiting the ↑ RA pressure caused by the fistula, thereby inhibiting vagal and stimulating the sympathetic pathway [Bainbridge reflex]).
Cardiac risk stratification

Useful background: American Society of Anesthesiologist Classification of Anesthetic Mortality within 48 hours postoperatively

<table>
<thead>
<tr>
<th>Class</th>
<th>Physical Status</th>
<th>48-Hour Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Healthy persons younger than 80 y</td>
<td>0.07%</td>
</tr>
<tr>
<td>II</td>
<td>Mild systemic disease</td>
<td>0.24%</td>
</tr>
<tr>
<td>III</td>
<td>Severe but not incapacitating systemic disease</td>
<td>1.4%</td>
</tr>
<tr>
<td>IV</td>
<td>Incapacitating systemic disease that is constant threat to life</td>
<td>7.5%</td>
</tr>
<tr>
<td>V</td>
<td>Moribund patient not expected to survive 24 hours, regardless of surgery</td>
<td>8.1%</td>
</tr>
<tr>
<td>E</td>
<td>Suffix added to any class to indicate emergency procedure</td>
<td>Double risk</td>
</tr>
</tbody>
</table>


59. Take a focused history to determine the cardiac risk stratification for noncardiac surgical procedures.

<table>
<thead>
<tr>
<th>Cardiac risk</th>
<th>Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ High</td>
<td>o Emergency operations</td>
</tr>
<tr>
<td></td>
<td>o Elderly patient</td>
</tr>
<tr>
<td></td>
<td>o Aortic and other major vascular procedures</td>
</tr>
<tr>
<td></td>
<td>o Peripheral vascular procedures</td>
</tr>
<tr>
<td></td>
<td>o Prolonged surgical procedures associated with large fluid shifts or blood loss (or both)</td>
</tr>
<tr>
<td>➢ Intermediate</td>
<td>o Carotid endarterectomy</td>
</tr>
<tr>
<td></td>
<td>o Head and neck operations</td>
</tr>
<tr>
<td></td>
<td>o Abdominal and intrathoracic procedures</td>
</tr>
<tr>
<td></td>
<td>o Orthopedic procedures</td>
</tr>
<tr>
<td></td>
<td>o Prostate operations</td>
</tr>
<tr>
<td>➢ Low</td>
<td>o Cataract extraction</td>
</tr>
<tr>
<td></td>
<td>o Breast operation</td>
</tr>
<tr>
<td></td>
<td>o Endoscopic procedures</td>
</tr>
</tbody>
</table>

Adapted from: Ghosh A.K. Mayo Clinic Scientific Press 2008, Table 8.9, page 337.
Useful background: Odds ratios (OR) > 2 for perioperative pulmonary complications.

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>Odds ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Patient</td>
<td></td>
</tr>
<tr>
<td>o ASA class &gt; II</td>
<td>4.9</td>
</tr>
<tr>
<td>o Chronic obstructive pulmonary disease</td>
<td>2.4</td>
</tr>
<tr>
<td>o Age (2.3 for 60-69, increasing to 5.6 for ≥ 80 years)</td>
<td></td>
</tr>
<tr>
<td>o Total functional dependence</td>
<td>2.5</td>
</tr>
<tr>
<td>➢ Co-morbidities</td>
<td></td>
</tr>
<tr>
<td>o Congestive heart failure</td>
<td>2.9</td>
</tr>
<tr>
<td>o Serum albumin &lt;35 g/L</td>
<td>2.5</td>
</tr>
<tr>
<td>➢ Impaired sensorium</td>
<td></td>
</tr>
<tr>
<td>➢ Cigarette use</td>
<td></td>
</tr>
<tr>
<td>➢ Procedure-related</td>
<td></td>
</tr>
<tr>
<td>o Surgical site or type of procedure</td>
<td>6.9</td>
</tr>
<tr>
<td>o Open abdominal aortic repair</td>
<td>4.2</td>
</tr>
<tr>
<td>o Thoracic</td>
<td>3.1</td>
</tr>
<tr>
<td>o Abdominal</td>
<td>2.5</td>
</tr>
<tr>
<td>o Neurosurgical</td>
<td>2.2</td>
</tr>
<tr>
<td>o Head and Neck</td>
<td>2.1</td>
</tr>
<tr>
<td>o Vascular</td>
<td>2.5</td>
</tr>
<tr>
<td>o Emergency surgery</td>
<td>2.3</td>
</tr>
<tr>
<td>o Prolonged surgery (&gt;3 h)</td>
<td></td>
</tr>
<tr>
<td>➢ General anesthesia</td>
<td>2.4</td>
</tr>
</tbody>
</table>

Note that the OR < 2 for age 50-59; partial functional dependence, unpaired sensorium, smoking; blood transfusion > 4 units.

Abbreviation: ASA, American Society of Anesthesiologists

Useful background: Selected quality measures

<table>
<thead>
<tr>
<th>Clinical Condition</th>
<th>Selected Quality Measures</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Acute coronary syndrome</td>
<td>• Aspirin at arrival &amp; discharge</td>
</tr>
<tr>
<td></td>
<td>• B-blocker at arrival &amp; discharge</td>
</tr>
<tr>
<td></td>
<td>• ACE inhibitor for LVSD</td>
</tr>
<tr>
<td>➢ Congestive heart failure</td>
<td>• Left ventricular function assessment</td>
</tr>
<tr>
<td></td>
<td>• ACE inhibitor for LVSD</td>
</tr>
<tr>
<td></td>
<td>• Smoking cessation advice &amp; counseling</td>
</tr>
<tr>
<td>➢ Community-acquired pneumonia</td>
<td>• Oxygenation assessment within 24 h</td>
</tr>
<tr>
<td></td>
<td>• Pneumococcal screening &amp; vaccination</td>
</tr>
<tr>
<td></td>
<td>• Antibiotic timing (first dose in &lt; 4 h)</td>
</tr>
<tr>
<td></td>
<td>• Smoking cessation advice &amp; counseling</td>
</tr>
<tr>
<td>➢ CRC screening</td>
<td>• Risk: age, family history, IBA, smoking, obesity (sporadic, familial poly syndromes)</td>
</tr>
<tr>
<td></td>
<td>• Adequacy of preparation &amp; sedation</td>
</tr>
<tr>
<td></td>
<td>• Reporting of cecal landmarks</td>
</tr>
<tr>
<td></td>
<td>• Withdrawal time</td>
</tr>
<tr>
<td></td>
<td>• Personal polyp detection rates (M,25%, F,15%)</td>
</tr>
</tbody>
</table>

Abbreviation: ACE, angiotensin-converting enzyme; LVSD, left ventricular systolic dysfunction; CRC, colorectal cancer


"Science, like good diagnosis, represents incremental progress of small steps taken slowly on solid ground.”

Grandad
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OSCE Questions in Endocrinology Chapter

1. Take a directed history for diabetes mellitus.
2. Perform a focused physical examination for diabetic nephropathy.
3. Perform a focused physical examination of the diabetic foot.
4. Perform a focused physical examination to differentiate DKA from HONC.
5. Take a directed history and perform a focused physical examination in the adult to determine the causes of hypoglycemia.
6. Take a directed history for thyroid disease.
7. Perform a focused physical examination for thyroid disease.
8. Take a directed history and perform a focused physical examination to determine if a thyroid nodule is likely to be malignant.
9. Take a directed history for the factors which increase the pretest probability of a goiter being present.
10. Perform a focused physical examination to distinguish between Grave’s disease (GD) and Toxic Nodular Goitre (TNG).
11. Perform a focused physical examination for hypothyroidism.
12. Perform a focused physical examination for hyperthyroidism.
13. Take a directed history for the causes of hypoadrenalism (Addison’s disease).
14. Perform a focused physical examination for hypoadrenalism (Addison’s Disease).
15. Perform a focused physical examination for pheochromocytoma (catecholamine-secreting tumour).
16. Perform a focused physical examination for Cushing’s syndrome.
17. Take a directed history to determine the causes of secondary hyperlipidemia.
18. Take a directed history for the causes of hypo- and hypercalcemia.
19. Take a directed history to determine the cause of osteoporosis.
20. Take a directed history and perform a focused physical examination to determine the causes of gynecomastia.
21. Take a directed history and perform a focused physical examination to determine the cause of amenorrhea.
22. Perform a focused physical examination for paraneoplastic syndromes and hormone producing cancers.
Diabetes mellitus

1. Take a directed history for diabetes mellitus.

➤ Glucose control

- Method and frequency of glucose monitoring, and by whom (patient, caregiver, health care worker)
- Typical levels of HbA1c, blood glucose at different times
- Dietary pattern, spacing of meals and snacks, quality and quantity of intake (CDA diet/calories/day); alcohol.
- Hypoglycemic reactions (frequency, symptoms); anxiety, tremor, seizures, palpitations, sweating, hunger
- Hyperosomolar non-ketotic coma (HONC)
- Diabetic ketoacidosis (DKA)
  - Symptoms of hyperglycemia (polyuria, polyphagia, polydypsia)
  - Anorexia, nausea, vomiting, abdominal pain
  - Fatigue
  - Kussmaul breathing
  - Precipitation: D/C insulin, infection, altered exercise/diet
- Medications (type, dose, frequency and adverse effects) Insulin, oral hypoglycemics, antagonistic medications (thiazides, corticosteroids)

➤ Causes/associated conditions

- Hormone-induced states (rare)
  - Acromegaly
  - Cushing’s syndrome
  - Pheochromocytoma
  - Glucagonoma
- Drugs
  - Steroids
  - Oral contraceptive agents
  - Streptozotocin, diazoxide, phenytoin, thiazide diuretics
- Pancreatic disease
  - Chronic pancreatitis, carcinoma
  - Haemochromatosis
- Syndromes
  - Lipoatrophic diabetes (characterized by generalized lipoatrophy, hyperglycemia, hepatomegaly, hirsutism, acanthosis nigricans, hyperpigmentation and hyperlipidemia)
- Family history
- Having one sibling or parent with T2 diabetes increases the lifetime risk for developing T2 diabetes to 10-15 %

➤ Complications

  o Hyperglycemia
    - Polyphagia
    - Weight changes
    - Polydipsia (+/- nocturia)
    - Polyuria
    - Blurred vision
    - Yeast infections

  o Hypoglycemia (adrenergic symptoms and signs)
    - Hunger
    - Palpitations
    - Sweating
    - Anxiety
    - Tremors
    - Seizures

  o Neuropathy

  o ANS neuropathy

  o Impotence

  o Neurogenic bladder: retention overflow incontinence

  o Orthostasis hypotension (gastroparesis),

  o bowel dysmobility (diarrhea/constipation)

  o PC bloating, fullness

  o Autonomic neuropathy
    - Orthostatic hypotension, gastroparesis (nausea, vomiting, postprandial bloating and early satiety), diarrhea, constipation, neurogenic bladder (retention and overflow incontinence), and impotence

  o Sensory neuropathy
    - Vibration sense (first lost), proprioception and light touch in glove-and-stocking distribution, Charcot’s joints, foot ulcerations

  o Radiculopathy
    - Shooting or burning pain, often radiating down lower extremities

  o Mononeuropathy
    - Cranial nerve (CN) palsies; often CN III (but pupils spared), CN IV, CN VI

  o Amyotrophy
- Atrophy of the pelvic girdle and large leg muscles that can spontaneously remit. Often affects older males

  o Mononeuritis multiplex
    - Peripheral nerve palsies that can cause sensory or motor neuropathies such as foot drop

  o Peripheral neuropathy:
    - anesthetic/paresthetic/hyperesthetic feet
    - sensory: vibration, proprioception, light touch (glove-in stocking)
    - Charcot’s joints

  o Retinopathy
    - Fundoscopic examinations; visual acuity – blurred vision; cataracts

  o Nephropathy
    - Known renal disease or proteinuria, date of last urinalysis

  o Cardiovascular
    - Cardiac: angina, MI, Hx or symptoms of CHF or pulmonary edema
    - Peripheral vascular disease: claudication, rest pain, foot ulcers or infections, amputations, foot care

Abbreviation: CN, cranial nerve


What’s “the best”? The “best” clinical findings for diabetic foot in a diabetic patient are a foot ulcer >2cm, a foot ulcer with bone exposed, or a positive probe test.
2. Perform a focused physical examination for diabetic nephropathy.

- **Mononeuritis multiplex**
  - Commonly affects
  - Cranial nerve III (ocular sparing)

- **Autonomic neuropathy**
  - Postural hypotension

- **GI**
  - Gastroparesis
  - Diarrhea
  - Atonic bladder (UTI)

- **Distal motor neuropathy**
  - Wasted hand/foot muscles

- **Heart**
  - Loss of sinus arrhythmia
  - MI, CHF

- **Diabetic amyotrophy**
  - Wasted quadriceps muscle
  - + painful skin

- **Distal sensory neuropathy**
  - ‘stocking’ ± ‘glove’ sensory
  - Loss ± Charcot joint

Abbreviations: CHF, congestive heart failure; MI, myocardial infarction; UTI, urinary tract infection

Useful background: DKA and HONC

Diabetic ketoacidosis (DKA)
- Normal → drowsy
  → coma
  Ketotic ‘fetor’
- ↓ blood pressure
  Postural hypotension
- ↑ respiratory rate and depth (Kussmaul breathing)
- ↑ heart rate
- Gastroparesis (± succession splash)
- Urine
  • Ketones +++
  • Glucose +++

Mortality rate < 5%

Management of DKA
- IV fluids
- IV insulin
- IV KCl (after insulin and fluids)
- Treatment of underlying cause

Investigations
<table>
<thead>
<tr>
<th>DKA</th>
<th>HONC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose</td>
<td>↑</td>
</tr>
<tr>
<td>Na</td>
<td>→</td>
</tr>
<tr>
<td>Urea</td>
<td>↑</td>
</tr>
<tr>
<td>pH</td>
<td>↓</td>
</tr>
<tr>
<td>Serum osmolality</td>
<td>↑</td>
</tr>
</tbody>
</table>

Hyperosmolar non-ketotic coma (HONC)
- ↓↓ conscious level
  Cerebrovascular accident (CVA)
- Pulmonary embolism
  Myocardial infarction
- Arterial insufficiency
- Urine
  • Ketones 0
  • Glucose +++
- Deep vein thrombosis (DVT)

Mortality rate 20-40%

Abbreviations: CVA, cerebrovascular accident; DVT, deep vein thrombosis

Useful background: Risk and management

- Modifiable risk factors
  - Hypertension
  - Smoking
  - Hyperlipidemia
  - Obesity
  - Exercise
  - Substance abuse
  - Personal history of gestational diabetes

- Management
  - Diet
    - Caloric intake
    - Amount and types of fats, protein, fibre, and sugar
  - Lifestyle
    - Weight
    - Smoking
    - Alcohol or substance use
    - Exercise (type and amount)
  - Drug treatments
    - All medications
    - Insulin (type, amount, dosing schedule, side effects)
    - Hypoglycemic agents (type, frequency, side effects)
    - Monitoring (type [blood/urine], frequency, HbA₁c)
    - Adherence to recommendations
    - Family history of diabetes


3. Perform a focused physical examination of the diabetic foot.

- Skin (look between toes)
  - Dry cracked skin
  - Necrobiosis diabeticorum
  - Blisters, corns, bunions
  - Ingrown or dystrophic nails
  - Cellulitis
  - Ulcers
  - Infection between toes

- Vascular insufficiency
  - Pallor, red cool skin
  - Loss of hair
  - Ulcers, with or without infection
  - Gangrene or amputations
Reduced posterior tibial and dorsalis pedis pulses

- **Neuropathy**
  - **Sensory**
    - Reduced vibration sense (use 128 Hz fork)
    - Reduced pin prick/fine touch (5.07 monofilament)
    - Glove/stocking distribution in legs
  - **Motor**
    - Claw-hammer toes, foot drop, pes cavus, ankle deformity
  - **Reflex**
    - Ankle jerk reduced


Useful background: Performance characteristics of clinical tests for the diabetic foot

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Predictors of subsequent foot ulceration</strong></td>
<td></td>
</tr>
<tr>
<td>o Unable to sense the 5.07 monofilament</td>
<td>2.4</td>
</tr>
<tr>
<td><strong>Predictors of osteomyelitis, in patients with foot ulcers</strong></td>
<td></td>
</tr>
<tr>
<td>o Ulcer area &gt;2 cm</td>
<td>7.2</td>
</tr>
<tr>
<td>o Positive probe test</td>
<td>4.3</td>
</tr>
<tr>
<td>o Ulcer depth &gt;3 mm or bone exposed</td>
<td>3.6</td>
</tr>
</tbody>
</table>

4. Perform a focused physical examination to differentiate DKA from HONC.

<table>
<thead>
<tr>
<th>DKA</th>
<th>HONC</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ CNS</td>
<td></td>
</tr>
<tr>
<td>☐ Normal to coma</td>
<td>☐ Drowsy</td>
</tr>
<tr>
<td>☐ Drowsy</td>
<td></td>
</tr>
<tr>
<td>☐ Coma</td>
<td></td>
</tr>
<tr>
<td>➢ CVS</td>
<td></td>
</tr>
<tr>
<td>☐ Hypotension (including postural changes)</td>
<td>☐ Myocardial infarction</td>
</tr>
<tr>
<td>☐ Tachycardia</td>
<td>☐ Peripheral vascular disease</td>
</tr>
<tr>
<td>➢ Respiratory</td>
<td></td>
</tr>
<tr>
<td>☐ Rate ↑</td>
<td>☐ DVT</td>
</tr>
<tr>
<td>☐ Depth ↑ (kussmaul breathing)</td>
<td>☐ Pulmonary embolus</td>
</tr>
<tr>
<td>☐ Ketotic breath</td>
<td></td>
</tr>
<tr>
<td>➢ GI</td>
<td></td>
</tr>
<tr>
<td>☐ Succussion splash (gastroparesis)</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: CVA, cerebrovascular accident; DKA, diabetic ketoacidosis; DVT, deep vein thrombosis; HONC, hyperosmolar non-ketotic coma

**Hypoglycemia**

Useful background: Complications of hypoglycemia

Usually related to diabetic treatment

- **CNS**
  - Concentration
  - Personality change
  - Anxiety → seizures, coma

- **CVS**
  - Heart rate
  - Sweating
  - Tremor

- **Pancreas**
  - Weight in insulinoma

- **PNS**
  - Extensor plantar response

- Panhypopituitarism
- IGF-II producing tumor
- Hepatic failure
- Adrenal failure
- Renal failure

Abbreviations: CNS, central nervous system; CVS, cardiovascular system; PNS, peripheral nervous system

5. Take a directed history and perform a focused physical examination in the adult to determine the causes of hypoglycemia.

- Starvation and exercise

- Reaction to glucose ingestion
  - Functional
  - Early diabetes mellitus
  - Post gastrectomy

- B cell overactivity
  - Insulinoma
  - Hyperplasia

- Endocrine disease
  - Early diabetes mellitus
  - Hypothyroidism
  - Hypopituitarism
  - Hypoadrenalism
  - Insulinoma
  - B-cell hyperplasia

- Drugs
  - Insulin
  - Sulphonylureas and diabiotic acids
  - Salicylates
  - Antihistamines

- Sensitivity to:
  - Leucine
  - Galactose, fructose
  - Alcohol
  - Tobacco

- Liver disease
  - Glycogen storage disease
  - Hepatoma

- Fibrosarcoma

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 93.
Thyroid disease

6. Take a directed history for thyroid disease.

- **Condition**
  - Medications, allergies, smoking
  - History of major illnesses (especially autoimmune)
  - Hospitalizations/surgeries
  - Radiation exposure (especially neck)
  - Family history of multiple endocrine neoplasia, medullary cancer or other cancers/lumps
  - Person history of goiter or nodules
  - Family history of goitre or nodules

- **History of HIV status/risk factors, smoking, alcohol, drug usage.**

- **Causes /associations**
  - Diabetes mellitus
    - Polyuria, polydipsia, thirst, blurred vision, weakness, infections, groin itch, rash (pruritus vulvae, balanitis), weight loss, tiredness, lethargy, and disturbance of conscious state
  - Hypoglycemia
    - Morning headaches, weight gain, seizures, sweating
  - Primary adrenal insufficiency
    - Pigmentation, tiredness, loss of weight, anorexia, nausea, diarrhea, nocturia, mental changes, seizures (hypotension, hypoglycemia)
  - Acromegaly
    - Fatigue, weakness, increased sweating, heat intolerance, weight gain, enlarging hands and feet, enlarged and coarsened facial features, headaches, decreased vision, voice change, decreased libido, impotence.

- **Complications**
  - Goiter
    - Dysphagia
    - Neck swelling
    - Stridor
  - CNS
    - Fatigue, weakness, tremor
    - Dysphagia
  - GI
    - Diarrhea/constipation
    - Weight change
  - GU
    - Decreased menses or fertility
- Polyuria

○ Throtoxicosis
  - Preference for cooler weather
  - Weight loss
  - Increased appetite (polyphagia)
  - Palpitations
  - Increased sweating
  - Nervousness
  - Irritability
  - Diarrhea
  - Amenorrhea
  - Muscle weakness
  - Exertional dyspnea

○ Hypothyroidism (myxedema)
  - Fatigue
  - Cold intolerance
  - Slowing of mental and physical performance
  - Hoarseness
  - Enlarged tongue
  - Slow pulse
  - Pericardial effusion
  - Anorexia
  - Weight gain
  - Constipation
  - Paresthesia
  - Slow speech
  - Muscle cramps
  - Slow relaxation of reflexes
  - Menorrhagia
  - Amenorrhea
  - Anovulatory cycles
  - Periorbital edema
  - Rough skin
  - Dry coarse hair
  - Anemia

○ Differential diagnosis
  - Thyroid tumor (benign vs. malignant)
  - Goitre
  - Thyroid cyst
  - Thyroglossal duct cyst

7. Perform a focused physical examination for thyroid disease.

- **Neck mass**
  - Inspection
    - Nutritional status
    - General appearance
    - Anatomical landmarks of thyroid
    - Mass, tenderness
    - Effect of swallowing
  - Palpation
    - Positioning, with and without swallowing
    - Gland size, consistency, tenderness and nodularity
    - Module location, size and number, character, tenderness
  - Auscultation

- **Eyes**
  - Chemosis (conjuntival edema) and hyperemia, periorbital edema
  - Corneal exposure with ulceration
  - Widening of the palpebral fissure (thyroid stare)
  - Lid retraction (widening of palpebral fissure) or lid lag on downgaze (von Graefe’s sign)
  - Exophthalmos
  - Proptosis
  - Double vision
  - Visual loss from optic nerve compression and edema

- **Voice**
  - Hoarse
  - Stridor

- **CVS**
  - Palpitations, hypertension, tachycardia
  - Deep tendon reflexes

- **GI**
  - Dysphagia
  - Diarrhea/constipation
  - Weight change

- **CNS**
  - Tremor
  - Weakness
  - Hypo-/hyperrflexia

- **Skin**
  - Sweating

**SO YOU WANT TO BE AN ENDOCRINOLOGIST!**

Q: The less important eponyms related to thyroid eye disease

A: 
- Infrequent blinking – Stellwag’s sign
- Tremor of gently closed eyelids – Rosenbach’s sign
- Difficulty in everting upper eyelid – Gifford’s sign
- Absence of wrinkling of forehead on sudden upward gaze – Joffroy’s sign
- Impaired convergence of the eyes following close accommodation – Möbius’ sign
- Weakness of at least one of the extraocular muscles – Ballet’s sign
- Paralysis of extraocular muscles – Jendrassik’s sign


**Thyroid nodule and goiter**

8. Take a directed history and perform a focused physical examination to determine if a thyroid nodule is likely to be malignant.

- **History**
  - Age < 30 years old or > 60 years old
  - Single nodules
  - History of head or neck irradiation
  - Compressive symptoms (pain, dysphagia, stridor, hoarseness)

- **Physical exam**
  - Fixed and firm solitary nodule with enlarged regional lymph nodes

- **Type of thyroid disease**
  - Benign nodules
    - Hashimoto’s thyroiditis
    - Hürthle adenoma
    - Follicular adenoma
    - Multinodular goitre
  - Malignant nodules
- Papillary
- Follicular
- Medullary, anaplastic
- 1˚ lymphoma
- Metastatic (breast or kidney tumours)

- Causes of a diffuse goitre (patient often euthyroid)
  - Idiopathic (majority)
  - Puberty or pregnancy
  - Thyroiditis
    - Hashimoto’s
    - Subacute (gland usually tender)
  - Simple goitre (iodine deficiency)
  - Goitregens, e.g. iodine excess, drugs (lithium, phenylbutazone)
  - Inborn errors of thyroid hormone synthesis, e.g. Pendred’s syndrome
    (an autosomal recessive condition associated with nerve deafness)

- Causes of a solitary thyroid nodule
  - Benign
    - Dominant nodule in a multinodular goitre
    - Degeneration or hemorrhage into a colloid cyst or nodule
    - Follicular adenoma
    - Simple cyst (rare)
  - Malignant
    - Carcinoma – primary or secondary (e.g. renal wall carcinoma)
    - Lymphoma (rare)

- Ultrasound
  - Solitary nodule
  - Irregular halo
  - Hypoechoic
  - Punctuate calcification
  - Increased blood flow

Useful background: Likelihood ratios for palpable thyroid gland indicating a goiter

<table>
<thead>
<tr>
<th>Palpable thyroid</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>o Adults</td>
<td>3.8</td>
<td>0.37</td>
</tr>
<tr>
<td>o Children</td>
<td>3.0</td>
<td>0.30</td>
</tr>
<tr>
<td>o Pregnancy</td>
<td>4.7</td>
<td>0.08</td>
</tr>
</tbody>
</table>

Abbreviations: NLR, negative likelihood ratio; PLR, positive likelihood ratio


Useful background:

- A normal thyroid gland weighs <20 gm (in iodine non-deficient regions), and is usually not palpable
- Goiter does not reflect function or neoplasia; goiter is hypertrophy or degeneration of the thyroid gland, with normal or abnormal thyroid function
- Pemberton's sign
  - Hands above the head for 30 min causing
    - Blue/ pink face/ neck from venous stasis and ↑JVP
    - Head congestion, dizziness, stiffness
  - From a retrosternal goiter, or any cause of reversible SVC syndrome (tumor)
- Compression complications of a thyroid goitre
  - Trachea (stridor)
  - Esophagus (dysphagia)
  - Recurrent laryngeal nerve (palsy)

9. Take a directed history for the factors which increase the pretest probability of a goiter being present.

- Children, especially those in endemic iodine deficiency locales
- Pregnant and lactating women
- Elderly patients
- Symptoms of hyperthyroidism or hypothyroidism
- Patients with excessive radiation exposure
- Patients with Down Syndrome

10. Perform a focused physical examination to distinguish between Grave’s disease (GD) and Toxic Nodular Goitre (TNG).

<table>
<thead>
<tr>
<th>Sign</th>
<th>GD</th>
<th>TNG</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Younger</td>
<td>Older</td>
</tr>
<tr>
<td>Thyroid</td>
<td>Diffuse goiter</td>
<td>Nodular, enlarged thyroid gland</td>
</tr>
<tr>
<td>Eye signs</td>
<td>Common</td>
<td>Rare</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>Rare</td>
<td>Common (~40%)</td>
</tr>
<tr>
<td>Associated autoimmune disease</td>
<td>Common</td>
<td>Rare</td>
</tr>
</tbody>
</table>


**Hypothyroidism**

11. Perform a focused physical examination for hypothyroidism.

- **Face**
  - Weight gain
- **Eyes**
  - Periorbital and facial puffiness, lose of outer portion of eyebrows
- **Skin/hair**
  - Coarse, sandpaper like, dry, hair breaks easily
- **Nails**
  - Thick
- **Hands**
  - Doughy skin (glucosaminoglycan deposit)
- **Mood**
  - Lethargic, disinterested
- **CNS**
  - Slow reflex relaxation
  - Cerebellar syndrome
  - Psychosis
  - Coma
  - Unmasking of myasthenia gravis
  - Cerebrovascular disease
  - High cerebrovascular fluid protein
  - Nerve deafness
  - Peripheral neuropathy
- **MSK**
  - Entrapment, carpal tunnel, tarsal tunnel
  - Muscle cramps
  - Proximal myopathy
  - Hypokalemic periodic paralysis

- **CVS**
  - Bradycardia
  - CCF


Useful background: Performance characteristics of physical findings for hypothyroidism

- Periorbital puffiness and slow movements (> 1 minute to fold a bed sheet) are not clinically significant physical findings for hypothyroidism.

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Skin</strong></td>
<td></td>
</tr>
<tr>
<td>Cool and dry skin</td>
<td>4.7</td>
</tr>
<tr>
<td>Coarse skin</td>
<td>3.4</td>
</tr>
<tr>
<td>(Cold palms)</td>
<td></td>
</tr>
<tr>
<td>(Dry palms)</td>
<td></td>
</tr>
<tr>
<td>Puffiness of wrists</td>
<td>2.9</td>
</tr>
<tr>
<td>Hair loss of eyebrows</td>
<td>1.9</td>
</tr>
<tr>
<td>(Pretibial edema)</td>
<td></td>
</tr>
<tr>
<td><strong>Speech</strong> - slow “Hypothyroid” speech</td>
<td>5.4</td>
</tr>
<tr>
<td><strong>Pulse</strong> &lt;60 bpm</td>
<td>4.1</td>
</tr>
<tr>
<td><strong>Thyroid</strong> enlarged</td>
<td>2.8</td>
</tr>
<tr>
<td><strong>Neurologic</strong></td>
<td></td>
</tr>
<tr>
<td>Delayed ankle reflexes</td>
<td>3.4</td>
</tr>
</tbody>
</table>

Abbreviation: likelihood ratio (LR) if finding present = positive PLR

Useful background: Physical examination for hypothyroidism.

- **CNS**
  - Mentally slow
  - Depression
  - Psychosis (‘myxedema madness’)
  - Cerebellar disturbance
  - Deafness
- **Face**
  - Puffy
  - Weight gain
  - Cold intolerance
  - Hair loss
  - Dry skin
- **CVA**
  - Bradycardia
  - Pericardial effusion
  - Premature ischaemic heart disease
- **GI/GU**
  - Constipation
  - Menstrual disturbance
    - Menorrhagia
    - Amenorrhea
- **MSK**
  - Bilateral carpal tunnel syndrome
  - Reflexes slow to relax
- **Myxedema**

Abbreviations: CNS, central nervous system; CVS, cardiovascular system; GI/GU, gastrointestinal/ genitourinary; MSK, musculoskeletal

Useful background: Billewicz diagnostic index for hypothyroidism

<table>
<thead>
<tr>
<th>Finding</th>
<th>Points scored if finding is</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>SYMPTOMS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diminished sweating</td>
<td>+6</td>
<td>-2</td>
<td></td>
</tr>
<tr>
<td>Dry skin</td>
<td>+3</td>
<td>-6</td>
<td></td>
</tr>
<tr>
<td>Cold intolerance</td>
<td>+4</td>
<td>-5</td>
<td></td>
</tr>
<tr>
<td>Weight increase</td>
<td>+1</td>
<td>-1</td>
<td></td>
</tr>
<tr>
<td>Constipation</td>
<td>+2</td>
<td>-1</td>
<td></td>
</tr>
<tr>
<td>Hoarseness</td>
<td>+5</td>
<td>-6</td>
<td></td>
</tr>
<tr>
<td>Paresthesia</td>
<td>+5</td>
<td>-4</td>
<td></td>
</tr>
<tr>
<td>Deafness</td>
<td>+2</td>
<td></td>
<td>0</td>
</tr>
<tr>
<td>PHYSICAL SIGNS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slow movements</td>
<td>+11</td>
<td>-3</td>
<td></td>
</tr>
<tr>
<td>Coarse skin</td>
<td>+7</td>
<td>-7</td>
<td></td>
</tr>
<tr>
<td>Cold skin</td>
<td>+3</td>
<td>-2</td>
<td></td>
</tr>
<tr>
<td>Periorbital puffiness</td>
<td>+4</td>
<td>-6</td>
<td></td>
</tr>
<tr>
<td>Pulse rate &lt;75/ min</td>
<td>+4</td>
<td>-4</td>
<td></td>
</tr>
<tr>
<td>Slow ankle jerk</td>
<td>+15</td>
<td>-6</td>
<td></td>
</tr>
</tbody>
</table>


<table>
<thead>
<tr>
<th>Finding</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Billewicz score</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than −15 points</td>
<td>3-4</td>
<td>28-68</td>
<td>0.1</td>
<td>-</td>
</tr>
<tr>
<td>-15 to +29 points</td>
<td>35-39</td>
<td>…</td>
<td>NS</td>
<td>-</td>
</tr>
<tr>
<td>+30 points or more</td>
<td>57-61</td>
<td>90-99</td>
<td>18.8</td>
<td>-</td>
</tr>
</tbody>
</table>


What’s “the best”? The “best” clinical tests for hypothyroidism are: slow speech, cool, dry and course skin, and brachycardia or a Billewicz diagnostic scale ≥ 30 points.
Useful background: The causes of hypothyroidism

- **Primary**
  - Without a goitre (decreased or absent thyroid tissue)
    - Idiopathic atrophy
    - Treatment of thyrotoxicosis, e.g. iodine, surgery
    - Agenesis or a lingual thyroid
    - Unresponsiveness to TSH
    - Elderly people; inhibitory autoantibody to TSH
  - With a goiter (decreased thyroid hormone synthesis)
    - Chronic autoimmune diseases, e.g. Hashimoto’s thyroiditis
    - Drugs, e.g. lithium, amiodarone
    - Inborn errors (enzyme deficiency)
    - Endemic iodine deficiency or iodine-induced hypothyroidism
    - Riedel’s thyroiditis

- **Secondary**
  - Pituitary lesions

- **Tertiary**
  - Hypothalamic lesions

- **Transient**
  - Thyroid hormone treatment withdrawn
  - Subacute thyroiditis
  - Postpartum thyroiditis


**Hyperthyroidism**

12. Perform a focused physical examination for hyperthyroidism.

- **Face**
  - Anxious, nervous, restless, frightened faces, energetic

- **Eyes**
  - Stare
  - Wide palpebral fissures
  - Lid lag
  - Inability to wrinkle brow on upward gaze
  - Ophthalmopathy
  - Exophthalmos (forward protrusion of eyeball >18 mm from the orbit)
- Conjunctivitis
- Conjunctival edema (chemosis)
- Periorbital edema
- Papilledema optic atrophy
- Extraocular defects
  - Muscle weakness
  - Amblyopia
  - Impaired upward gaze
  - Impaired convergence
  - Strabismus
  - Restricted gaze and visual acuity
  - Visual field competence defects

➢ Mood
  - Apathy, depressed mood (especially in elderly)

➢ Skin/Hair
  - Fine skin and hair
  - Hyperpigmentation at pressure points
  - Pretibial myxedema (non-pitting edema, pigmented, pruritic); note: myxedema of hypothyroidism is more general
  - Warm, moist, velvety skin

➢ Nails
  - Broken
  - Onycholysis (Plummer’s nails, IVth digit, separation of nail from nailbed)

➢ Hands
  - Palmar erythema
  - Fine tremor
  - Thyroid periostitis

➢ CNS
  - Hyperreflexia
  - Fine tremor

➢ GI/ GU
  - Diarrhea
  - Amenorrhea

➢ Muscle
  - Myopathy
  - Proximal muscle weakness

➢ Heart
  - Cardiomyopathy
- CCF (high output)
- Atrial fibrillation
- Flow murmur
- Tachycardia
- Increased pulse pressure

➤ Lung
- Means-Lerman scratch sound (high-pitch pulmonic sound similar to pericardial rub)
- Primary
  - Graves' disease
  - Toxic multinodular goitre
  - Toxic uninnodular goitre: (usually a toxic adenoma)
  - Hashimoto's thyroiditis (thyrotoxicosis early in its course; later H thyroiditis causes hypothyroidism)
  - Subacute thyroiditis (transient)
  - Postpartum thyroiditis (non-tender)
  - Iodine-induced ('Jod-Basedow' phenomenon – iodine given after a previously deficient diet)
- Secondary
  - Pituitary (very rare): TSH hypersecretion
  - Hydatidiform moles or choriocarcinomas: by HCG secretion (rare)
  - Struma ovarii (rare)
  - Drugs, e.g. excess thyroid hormone ingestion, amiodarone


“Our lives begin to end the day we become silent about the things that matter.”
Martin Luther King
Useful background: Complications of hyperthyroidism

**Thyroid eye disease**

- Swollen extra-ocular muscles
- Compression of optic nerve → blindness
- Eye project beyond line (proptosis)
- Corneal ulcers
- Warm, sweaty skin
- Pretibial myoedema infiltration with mucopolysaccharides
- Fine tremor
- Anxiety, irritability
- Palpitations
- Weight loss
- Heat intolerance
- Increased sweating
- Resting tachycardia proportional to severity of thyrotoxicosis

**Proximal myopathy**

- Difficulty on stairs
- Difficulty reaching for top shelf

**Menstrual disturbance**

- Difficulty reaching for top shelf

**Diarrhea**

- Resting tachycardia proportional to severity of thyrotoxicosis

- Difficulty reaching for top shelf

- Warm, sweaty skin

- Thyroid eye disease

- Goiter ± bruit

- JVP

- Eye moves forward

- Eye project beyond line (proptosis)

- Corneal ulcers

- Eyelid retract

- Cornea exposed

- Eyelid retraction

- \[ T_4 \]

- Normal

- Heart failure due to:
  - Atrial fibrillation
  - Cardiomyopathy

Useful background: Performance characteristics of physical findings for hyperthyroidism

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Pulse</td>
<td></td>
</tr>
<tr>
<td>o Pulse &gt; 90 beats/min</td>
<td>4.4</td>
</tr>
<tr>
<td>➢ Skin</td>
<td></td>
</tr>
<tr>
<td>o Moist and warm</td>
<td>6.7</td>
</tr>
<tr>
<td>➢ Thyroid</td>
<td></td>
</tr>
<tr>
<td>o Enlarged thyroid</td>
<td>2.3</td>
</tr>
<tr>
<td>➢ Eyes</td>
<td></td>
</tr>
<tr>
<td>o Eyelid retraction</td>
<td>31.5</td>
</tr>
<tr>
<td>o Eyelid</td>
<td>17.6</td>
</tr>
<tr>
<td>➢ Neurologic</td>
<td></td>
</tr>
<tr>
<td>o Fine finger tremor</td>
<td>11.4</td>
</tr>
</tbody>
</table>


Useful background: Wayne diagnostic index for hyperthyroidism*

<table>
<thead>
<tr>
<th>Symptoms of recent onset or increased severity</th>
<th>Present</th>
<th>Signs</th>
<th>Present</th>
<th>Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td>o Dyspnea on effort</td>
<td>+1</td>
<td>o Palpable thyroid</td>
<td>+3</td>
<td>-3</td>
</tr>
<tr>
<td>o Palpitations</td>
<td>+2</td>
<td>o Bruit over thyroid</td>
<td>+2</td>
<td>-2</td>
</tr>
<tr>
<td>o Tiredness</td>
<td>+2</td>
<td>o Exophthalmos</td>
<td>+2</td>
<td></td>
</tr>
<tr>
<td>o Preference for heat</td>
<td>-5</td>
<td>o Lid retraction</td>
<td>+2</td>
<td></td>
</tr>
<tr>
<td>o Preference for cold</td>
<td>+5</td>
<td>o Lid lag</td>
<td>+1</td>
<td></td>
</tr>
<tr>
<td>o Excessive sweating</td>
<td>+3</td>
<td>o Hyperkinetic</td>
<td>+4</td>
<td>-2</td>
</tr>
<tr>
<td>o Nervousness</td>
<td>+2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Appetite increased</td>
<td>+3</td>
<td>o Fine finger tremor</td>
<td>+1</td>
<td></td>
</tr>
<tr>
<td>o Appetite decreased</td>
<td>-3</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Weight increased</td>
<td>-3</td>
<td>- Hot</td>
<td>+2</td>
<td>-2</td>
</tr>
<tr>
<td>o Weight decreased</td>
<td>+3</td>
<td>- Moist</td>
<td>+1</td>
<td>-1</td>
</tr>
<tr>
<td>o Casual pulse rate:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Atrial fibrillation</td>
<td>+4</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
- <80, regular  -3
- 80-90, regular  0
- >90, regular  +3

Finding | Sensitivity (%) | Specificity (%) | PLR
---|---|---|---
- Wayne index
  - <11 points  1-6  13-32  0.04
  - 11-19 points  12-30  …  NS
  - >20 points  66-88  92-99  18.2


**SO YOU WANT TO BE AN ENDOCRINOLOGIST!**

- Distinguish clubbing and bony enlargement from thyroid acropachy from pulmonary hypertrophic osteoarthropathy (periostitis)
- Thyroid periostitis – hands and feet, asymptomatic
- Pulmonary hypertrophic osteoarthropathy – long bones


Useful background: Causes of hyperthyroidism

- Graves’ Disease
- Goiter
  - Multinodular
  - Uninodular (usually an adenoma)
  - Toxic uninodular goiter (usually a toxic adenoma)
- Thyroiditis
  - Hashimoto’s
  - Subacute
  - Postpartum
- Drugs
  - Iodine (after previous dietary deficiency)
  - Thyroxine excess
Amiodosone

- Tumor
  - Pituitary (TSH production)
  - Ovarian (struma ovarii; extrathyroidal T4)
  - Hydatid moles; choriocarcinoma (HCE secretion)


Useful background: Causes of exophthalmos

- Bilateral
  - Graves’ disease

- Unilateral
  - Cavernous sinus thrombosis
  - Tumours of the orbit, (e.g. dermoid, optic nerve glioma, neurofibroma, granuloma)
  - Pseudotumours of the orbit
  - Graves’ disease


Adrenal disease

13. Take a directed history for the causes of hypoadrenalism (Addison’s disease).

- Acute
  - Septicemia (especially meningococcal)
  - Adrenalectomy
  - Any stress in a patient with chronic hypoadrenalism or abrupt cessation of prolonged high-dose steroid therapy

- Chronic
  - Primary
    - Acute or chronic gland destruction
      - Idiopathic atrophy (Addison’s disease)
      - Infection: TB, fungal
      - Infiltration: metastasis, amyloidosis, etc.
      - Hemorrhage (especially Waterhouse-Friderichsen syndrome)
      - Surgery
    - Metabolic failure
      - Virilizing hyperplasia, e.g. C21-hydroxylase deficiency
- Enzyme inhibitors, e.g. metopirone
- Drugs, e.g. OPDDD (cytotoxic)
- Drugs (warfarin)
  - Secondary
    - Hypopituitarism
    - Suppression of hypothalamic-pituitary axis
      - Exogenous glucocorticoids
      - Endogenous glucocorticoids, e.g. Cushing’s syndrome following tumour removal


“The meaning of life is to fill your three score and ten with love, respect and compassion, for others.”

ABRT
14. Perform a focused physical examination for hypoadrenalism (Addison’s disease).

- **Fatigue**
- **Pigmentation**
  - Buccal
  - Scars
  - Palmar creases
  - Generalized
- **GI**
  - Abdominal pain
  - Diarrhea
  - Anorexia
  - Weight loss
- **Associated disease**
  - Hypothyroid
  - Diabetes type I
  - Pernicious anemia
  - Vitiligo

Abbreviation: GI, gastrointestinal

15. Perform a focused physical examination for pheochromocytoma (catecholamine-secreting tumour).

- Headache
- Sweating
- Anxiety

- Hypotension
  - Myocardial damage and CHF
  - Palpitations
  - Hypotensive attacks (very rarely, if mainly dopamine secreted)

- Paroxysmal symptoms

- Weight loss

Abbreviations: CHF, congestive heart failure


Useful background: Causes of Cushing’s syndrome

- Adrenocorticotropic hormone (ACTA) dependent

- Exogenous administration of excess steroids or ACTH (most common)

- Adrenal hyperplasia
  - Secondary to increased pituitary ACTH production (Cushing’s disease)
    - Microadenoma
    - Macroedema
    - Pituitary-hypothalamic dysfunction
Secondary to ACTH-producing tumours (e.g. small cell lung carcinoma)

- Adrenal neoplasia
  - Adenoma
  - Carcinoma (rare)

- Pituitary adenoma (Cushing’s disease) (70%) F>M
  - Ectopic ACTH (14%)
  - Bronchial carcinoma
  - Carcinoid- lung, gastrointestinal tract, thymus

- ACTH independent
  - Adrenal adenoma (10%)
  - Adrenal carcinoma (5%)
  - Adrenal hyperplasia (1%)

- Differential diagnosis
  - Pseudo-Cushing’s due to alcoholism or depression


Useful background: Performance characteristics for physical examination for Cushing’s syndrome

Although moon facies, generalized obesity, hirsutism (in women), striae, proximal muscle weakness, and peripheral edema have been suggested to be signs of Cushing’s syndrome, these are in fact either non-significant or have positive likelihood ratios (PLR) < 2.

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vital signs</strong></td>
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<tr>
<td>Hypertension</td>
<td>2.3</td>
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<tr>
<td><strong>Body habitus</strong></td>
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<tr>
<td>Central obesity</td>
<td>3.0</td>
</tr>
<tr>
<td><strong>Skin findings</strong></td>
<td></td>
</tr>
<tr>
<td>Thin skinfold</td>
<td>115.6</td>
</tr>
<tr>
<td>Plethora</td>
<td>2.7</td>
</tr>
<tr>
<td>Ecchymoses</td>
<td>4.5</td>
</tr>
<tr>
<td>Acne</td>
<td>2.2</td>
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</tbody>
</table>

16. Perform a focused physical examination for Cushing’s syndrome.

Abbreviations:
CNS, central nervous system; CVS, cardiovascular system; GI/ GU, gastrointestinal/genitourinary; MSK, musculoskeletal

Useful background: Distribution of adipose tissue in Cushing’s syndrome

- Rounding of cheeks and prominent bitemporal fat produces the characteristic ‘moon facies’. Fat also may accumulate bilaterally above the clavicles (‘supraclavicular collar’). In front of the sternum (episternal area, or ‘dewlap’), and over the back of the neck (dorsal cervical fat pad, or ‘buffalo hump’). In these drawings, the dotted line depicts normal contours of patients without Cushing’s Syndrome.

Adapted from: McGee S. R. Saunders/Elsevier 2007, Figure 12-1, page 111.

**Hyperlipidemia**

17. Take a directed history to determine the causes of secondary hyperlipidemia.

- Nutrition
  - Anorexia nervosa
  - Hypertriglyceridemia
  - Obesity
  - Hypothyroidism

- Endocrine
  - Diabetes mellitus
  - Hypothyroidism

- Immune
  - Dysglobulinemias
  - Systemic lupus erythematosus
Liver
  - Cholestasis
  - Acute intermittent porphyria

Kidney
  - Nephrotic syndrome
  - Renal insufficiency

Drugs
  - Progestins
  - Anabolic steroids, glucocorticoid therapy
  - Estrogens
  - Thiazides, steroids
  - β-blocking agents

Life style
  - Sedentary lifestyle
  - Alcohol
  - Cigarette smoking

Miscellaneous

Abbreviations: HDL-C, high density lipoprotein cholesterol; LDL-C, low density lipoprotein cholesterol

Adapted from: Ghosh AK. Mayo Clinic Scientific Press 2008, Table 6-4, page 233.

Metabolic bone disease and calcium disorders

18. Take a directed history for the causes of hypo- and hypercalcemia.

<table>
<thead>
<tr>
<th>Hypocalcemia</th>
<th>Hypercalcemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parathyroid</td>
<td>- Primary hyperparathyroidism</td>
</tr>
<tr>
<td>Thyroid</td>
<td>- Thyrotoxicosis</td>
</tr>
<tr>
<td>Gut</td>
<td>- Hypothyroidism in infants</td>
</tr>
<tr>
<td>Pancreas</td>
<td>- Milk – alkali syndrome</td>
</tr>
</tbody>
</table>
Kidney  
- Chronic renal failure - Associated with renal failure (e.g. severe secondary hyperparathyroidism)

Drugs
- Thiazide diuretics
- Vitamin D excess
- Milk-alkali syndrome
- Steroid withdrawal syndrome

Malignancy
- Hypocalcemia of malignant disease (with or without metastases)
  - Carcinoma (bone metastases or humoral mediators)
  - Excessive intake production of vitamin D metabolites
  - Vitamin D sensitivity
  - Sarcoidosis
  - Multiple myeloma
  - Reticulosis

Inactivity
- Prolonged immobilization or space flight
  - “steroid withdrawal syndrome”

Miscellaneous
- Paget’s disease
- Infantile hypercalcemia

Note:
- Hypercalciuria occurs with all causes of hypercalcemia if renal function is normal.
- Causes of hypercalciuria without hypercalcemia include osteoporosis and renal tubular acidosis (RTA), idiopathic.

Useful background: Causes of hypoparathyroidism

- **Idiopathic**
  - Dysembryogenesis (DiGeorge syndrome)

- **Iatrogenic**
  - Radioactive iodine therapy
  - External neck irradiation
  - Damage during thyroid or neck surgery

- **Infiltration**
  - Metastatic disease (breast, lung, lymphoproliferative disorder)

- **Immune**
  - Polyglandular autoimmune syndrome (PGA type 1) aka autoimmune polyendocrinopathy-candidiasis-ectodermal dystrophy (APECED)

- **Metabolic**
  - Hemochromatosis and Wilson’s deficiency


Useful background: Causes of hyperparathyroidism

- **Primary**
  - Adenoma (80%)
  - Hyperplasia
  - Carcinoma (rare)

- **Secondary**
  - Hyperplasia associated with chronic renal failure

- **Tertiary**
  - Autonomous hyperparathyroidism is a complication of secondary hyperparathyroidism


Useful background: Causes of radiographic punctate translucencies in skull

- **Malignancy**
  - Myelomatosis
  - Metastatic deposits

- **Metabolic**
- Hyperparathyroidism
- Cushing’s

- Hematological
  - Sickle-cell anemia
  - Leukemia
  - Histiocytosis X


<table>
<thead>
<tr>
<th>Condition</th>
<th>Serum Calcium</th>
<th>Serum Phosphate</th>
<th>Parathormone Infusion, response</th>
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<tr>
<td>Hypoparathyroidism</td>
<td>↓</td>
<td>↑</td>
<td>o Marked</td>
</tr>
<tr>
<td>Pseudo-hypoparathyroidism</td>
<td>↓</td>
<td>↑</td>
<td>o None</td>
</tr>
<tr>
<td>Pseudo- pseudo-hypoparathyroidism</td>
<td></td>
<td></td>
<td>o None</td>
</tr>
</tbody>
</table>


19. Take a directed history to determine the cause of osteoporosis.

- Inherited
  - Osteogenesis imperfecta
- Ideopathic (young persons)
- Immobility, senile
- Immune
  - Rheumatoid arthritis
- Infiltration
  - Systemic mastocytosis
  - Multiple myeloma
- Endocrine
  - Deficiency of estrogen, androgen, protein, vitamin C or calcium
  - Hyperthyroidism
  - Diabetes mellitus
  - Acromegaly
  - Cushing’s or glucocorticoid therapy
- Liver
  - Chronic cholestasis
Steroid use

- Kidney
  - Azotemic osteodystrophy
  - Liver
  - Glycogen storage disease
  - Cirrhosis in children

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 100.

Useful background: Likelihood ratios for physical examination manoeuvres suggesting presence of osteoporosis or spinal fracture

<table>
<thead>
<tr>
<th></th>
<th>PLR</th>
<th>NLR</th>
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<tbody>
<tr>
<td>Weight &lt; 51 kg</td>
<td>7.3</td>
<td>0.8</td>
</tr>
<tr>
<td>Wall-occiput distance &gt;0cm</td>
<td>3.8</td>
<td>0.6</td>
</tr>
<tr>
<td>Rib- Pelvis distance &lt;2 fingerbreadths</td>
<td>3.8</td>
<td>0.6</td>
</tr>
<tr>
<td>Tooth count &lt;20</td>
<td>3.4</td>
<td>0.8</td>
</tr>
<tr>
<td>Height loss &gt;3 cm</td>
<td>3.2</td>
<td>0.4</td>
</tr>
<tr>
<td>Self reported humped back</td>
<td>3.0</td>
<td>0.85</td>
</tr>
</tbody>
</table>


Useful background: Causes of metabolic bone disease

- Causes of osteomalacia and rickets

- Skin
  - pigmented skin
  - lack of UV light

- Pregnancy
  - multiple pregnancy
  - prolonged breast feeding

- GI
  - Malnutrition deficiency of vitamin D
  - Post-gastrectomy (probably dietary)
  - Malabsorption

- Renal
- Chronic renal failure
- Idiopathic hypercalcuria
- Fanconi syndrome
- Tubular acidosis
- Hypo-phosphatasia

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 98.

Useful background: Causes of secondary hyperuricemia

<table>
<thead>
<tr>
<th>Hyperuricemia</th>
<th>Hypouricemia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nutrition</strong></td>
<td></td>
</tr>
<tr>
<td>- Obesity</td>
<td></td>
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<tr>
<td>- Increased purine ingestion</td>
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<tr>
<td><strong>Tumor</strong></td>
<td></td>
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<tr>
<td>- Myeloproliferative disorders</td>
<td></td>
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<tr>
<td>- Polycythemia, primary or secondary</td>
<td></td>
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<tr>
<td>- Myeloid metaplasia</td>
<td></td>
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<tr>
<td>- Chronic myelocytic leukemia</td>
<td></td>
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<tr>
<td>- Lymphoproliferative disorders</td>
<td></td>
</tr>
<tr>
<td>- Chronic lymphocytic leukemia</td>
<td></td>
</tr>
<tr>
<td>- Plasma cell proliferative disorders</td>
<td></td>
</tr>
<tr>
<td>- Multiple myeloma</td>
<td></td>
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<tr>
<td>- Disseminated carcinoma and sarcoma</td>
<td></td>
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<tr>
<td><strong>Anemia</strong></td>
<td></td>
</tr>
<tr>
<td>- Sickle cell anemia</td>
<td></td>
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<tr>
<td>- Thalassemia</td>
<td></td>
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<tr>
<td>- Chronic hemolytic anemia</td>
<td></td>
</tr>
<tr>
<td><strong>Skin</strong></td>
<td></td>
</tr>
<tr>
<td>- Psoriasis</td>
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</tr>
<tr>
<td><strong>Drugs / toxins</strong></td>
<td>Drug-induced</td>
</tr>
<tr>
<td>- Cytotoxic drugs</td>
<td>- Thiazide diuretics</td>
</tr>
<tr>
<td></td>
<td>- Furosemide</td>
</tr>
<tr>
<td></td>
<td>- Ethacrynic acid</td>
</tr>
<tr>
<td></td>
<td>- Ethambutol</td>
</tr>
</tbody>
</table>
- Pyrazinamide
- Low-dose aspirin
- Cyclosporine
- Nicotinic acid
- Laxative abuse
- Levodopa

➢ Infections
  o Infectious mononucleosis

➢ Kidney
  o Intrinsic renal disease
    - Chronic renal insufficiency of diverse cause
    - Saturine gout (lead nephropathy)

➢ Endocrine
  o Endocrine conditions
    - Adrenal insufficiency
    - Nephrogenic diabetes insipidus
    - Hyperparathyroidism
    - Hypoparathyroidism
    - Pseudohypoparathyroidism
    - Hypothyroidism
    - Diabetic ketoacidosis
    - Lactic acidosis
    - Starvation
    - Ethanolism
    - Glycogen storage disease type I
    - Bartter syndrome

➢ Genetic
  o Other
    - Sarcoidosis
    - Down syndrome
    - Beryllium disease

Gynecomastia

20. Take a directed history and perform a focused physical examination to determine the causes of gynecomastia.

- Hermaphroditism or pseudo-hermaphroditism
- Endocrine
  - Normal puberty and neonatal
  - Hypothyroidism
  - Thyrotoxicosis
  - Acromegaly
  - Testicular atrophy
  - Testicular and adrenal tumours
- Klinefelter’s syndrome
- Cirrhosis
- Cancer of bronchus
- Malnutrition
- Paraplegia
- Generalized skin disease
- Drugs
  - Spironolactone
  - Amphetamine
  - Reserpine
  - Digitalis
  - Methyldopa


Amenorrhea

21. Take a directed history and perform a focused physical examination to determine the cause of amenorrhea.

- Physiological
  - Pre-pubertal
  - Pregnancy
  - Infarction or pituitary appoplexy
  - Menopausal
  - “Functional”
    - change in environment
    - emotional upset
- rapid change in weight
- aggressive training

➢ Uterus
   o Uterine anomaly
   o Hysterectomy

➢ Ovary
   o Stein-Leventhal syndrome, tumours, e.g. arrhenoblastoma, hilus-cell
   o Oophorectomy
   o Follicular or lutein retention cyst
   o Granulosa cell tumour
   o Irradiation

➢ Endocrine
   o Thyrotoxicosis
   o Cong. Adrenogenital syndrome, acquired virilizing hyperplasia, adenoma or Ca.

➢ Head
   o Mid brain tumor
   o Trauma
   o Surgery radiation infection
     - Pelvic TB
     - Systemic infection
➢ Drugs
   o Estrogen, progesterone, testosterone
   o Glucocorticoids, spironolactone


\textbf{Hirsutism}

Useful background: Causes of hirsutism

➢ Ovarian
   o Tumour
   o Stein-Leventhal syndrome
   o Polycystic ovary syndrome (commonest cause)

➢ Endocrine
   o Adrenal
     - Cushing’s syndrome
     - Congenital adrenal hyperplasia
     - Virilising tumour (more often a carcinoma than an adenoma)
     - Acromegaly
Porphyria cutanea tarda

Drugs
  - Phenytoin, diazoxide, streptomycin, minoxidil, anabolic steroids


**Pituitary disease**

Useful background: Causes of hypopituitarism

- Infiltration
  - Pituitary tumour (non-secretory or secretory)
  - Other tumours
    - Carniopharyngioma
    - Metastatic carcinoma
    - Sarcoma

- Infection
  - Granulomata
    - e.g. Sarcoid, tuberculosis

- Idiopathic


“Let’s change the interactions between alerting, orienting and executive functions, and control in a standard cuing paradigm.”

Grandad
Useful background: Acromegaly

- Eye
  - Bitemporal hemianopia
  - Papilloedema
  - Angiod streaks

- Head and Neck
  - Prognathism
  - Enlarged tongue
  - Transfrontal scar
  - Frontal bossing

- Molluscum fibrosum

Paraneoplastic syndromes

22. Perform a focused physical examination for paraneoplastic syndromes and hormone producing cancers.

- CNS
  - Confusion/Dementia
  - ↓ Na+ (SIADH)
  - ↑ Ca2+ (ectopic PTH)
  - Hyperviscosity syndrome (myeloma, Waldenstroms)
  - Cerebral cortex auto antibodies

- Hematology
  - Anemia
  - Autoimmune haemolytic anaemia-mucin producing cancers
  - Aplasia: Thymic tumor related
  - Polycytemia
  - Renal cancer
  - Cerebellar

- Cachexia

- Nephrotic syndrome
  - Glomerulonephritis (GN) due to tumor Ag-Ab complex deposition
  - Minimal change GN (Hodgkin’s disease)
  - Membranous GN (many cancers)
  - Membranoproliferative GN (non Hodgkin’s lymphoma)

- Muscle weakness
  - Fatigue
  - Cachexia
  - Polymyositis/dermatomyositis
  - ↓ K+ (2.0 to ectopic ACTH production)
  - ↑ Ca2+ (2.0 to ectopic PTH production)
  - Eaton-Lambert syndromes
  - Guillain – Barre syndrome

- Spinal cord syndromes
  - Myelitis, often in thoracic region, → rapid paralysis + death
  - ALS (=motor neuron disease) 5-10% are cancer related

- Hypertrophic pulmonary osteoarthropathy

- Hypercalcaemia
  - Ectopic PTH secretion
  - PTH related hormone syndromes

- Deep vein thrombosis

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<td>Acute and chronic liver disease</td>
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<td>Pruritus</td>
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<td>Gallbladder</td>
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<td>Suggested practice case scenarios for OSCE</td>
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<td>examinations</td>
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OSCE Questions in Gastroenterology Chapter

1. Perform a focused physical examination to determine the causes of stomatitis.
2. Take a directed history to determine the causes of halitosis.
3. Perform a focused physical examination to determine the causes of salivary gland swelling.
4. Perform a focused physical examination to determine the causes of parotid gland enlargement.
5. Take a directed history for dysphagia.
6. Take a directed history to determine the causes of RUQ pain.
7. Take a directed history and perform a focused physical examination for appendicitis, and stratify the risk and need for surgery.
8. Perform a focused physical examination to determine the causes of abdominal masses.
9. Take a directed history and perform a focused physical examination for the cause of abdominal bruit.
10. Take a directed history for bowel obstruction.
11. Take a directed history for diarrhea.
12. Take a directed history to determine the cause of infertility in men with inflammatory bowel disease.
13. Take a directed history of alcohol abuse.
14. Perform a directed physical examination for alcohol withdrawal (SSH-DTs: shake, seizure, hallucinates, DTs).
15. Perform a focused physical examination hepatosplenomegaly.
16. Perform a physical examination for acute liver disease (acute hepatitis and fulminant liver failure).
17. Perform a focused physical examination for signs of chronic liver disease (portal hypertension).
18. Take a directed history and perform a focused physical examination for ascites.
19. Perform a focused physical examination to determine the cause of pruritus.
20. Prepare a patient for informed consent for the use of steroids (GCS, glucocorticosteroids) in a patient with IBD, explaining the adverse effects.
21. Provide a patient with informed consent prior to their having possible bariatric surgery.

22. Prepare a patient for informed consent for the use of nonsteroidal anti-inflammatory drugs, explaining the potential adverse effects.

23. Perform a directed examination of an abdominal x ray (‘flat plate’).
Mouth

1. Perform a focused physical examination to determine the causes of stomatitis.

- General
  - Malnutrition: protein, vitamins, minerals (esp. iron)
  - Smoking
  - Alcoholism
  - Infections
  - Drugs
    - Antibiotics
    - Atotoxic drugs
    - Phenytoin
    - Heavy metals
  - Trauma
    - Poorly fitting dentures
  - Tumour

- Systemic diseases
  - GI
  - Crohn’s
  - Ulcerative colitis
  - Celiac disease
  - MSK
    - Behcet’s
    - Reiter’s
    - Lupus
  - Hematology
    - Leukemia and its treatment
    - Neutropenia
    - Iron deficiency

- Skin diseases (associated, including angular stomatitis)
  - Eczema
  - Erythema multiforme (Stevens-Johnson)
  - Pemphigus vulgaris
  - Pemphigoid
  - Benign pemphigoid of mucous membranes
  - Epidermolysis bullosa
  - Lichen planus
  - Dermatitis herpetiformis

- HIV/AIDS associated infections
  - Viral
    - Herpes simplex
- Herpangina
- Hand, foot and mouth disease
- Exanthemata
  - Bacterial
    - Pyorrhea and alveolar abscess
    - Vincent’s angina
    - TB
    - Syphilis
  - Fungal
    - Candidiasis
    - Actinomycosis

Adapted from: Burton J.L. *Churchill Livingstone* 1971, pages 36 and 37.

2. Take a directed history to determine the causes of halitosis.

- **Infection**
  - Poor oral hygiene
  - Putrid (due to anaerobic chest infections with large amounts of sputum)

- **Metabolic**
  - Fetal hepaticus (a sweet smell)
  - Ketosis (diabetic ketoacidosis results in excretion of ketones in exhaled air, causing a sickly sweet smell)
  - Uremia (fish breath: an ammoniacal odour)

- **Drugs**
  - Alcohol (distinctive)
  - Paraldehyde
  - Cigarettes, tobacco


Useful background: Causes of gum hypertrophy

- **Gingivitis**
  - from smoking
  - calculus
  - plaque
  - Vincent’s angina (fusobacterial membranous tonsillitis)

- **Drugs** (Phenytoin)
- Scurvy (vitamin C deficiency: the gums become spongy, red, bleed easily and are swollen and irregular)
- Leukemia (usually monocytic)


Useful background: Causes of pigmented lesions in the mouth, including tongue

- Drugs/Toxins
  - Heavy metals: lead or bismuth (blue-black line on the gingival margin), iron (hemochromatosis- blue-grey pigmentation of the hard palate)
  - Drugs
    - Antimalarials
    - Oral contraceptive pill (brown or black areas of pigmentation anywhere in the mouth)

- Endocrine
  - Addison’s disease (blotches of dark brown pigment anywhere in the mouth)

- Tumor
  - Malignant melanoma (raised, painless black lesions anywhere in the mouth)

- Genetic
  - Peutz-Jeghers syndrome (lips, buccal mucosa or palate)


Useful background: The tongue

- Brown mottling of enamel (Fluorosis)
- Stippling of gums (Pb, bismuth poisoning)
- Furred tongue-fever (acute abdomen, uremia, cholemia)
- Magenta-colored tongue, angular stomatitis, cheilosis (cracked lips).
- Red side and tip of tongue (nicotinic acid deficiency)
- Dipapillating glossitis (antibiotic therapy).
- Large tongue-myxedema, acromegaly, amyloid, Down syndrome
- Jaundice (frequently appears first and disappears last from frenum of tongue)
- Scrotal tongue (normal or in Down syndrome)
- Geographical tongue-patchy redish depapillation surrounded by “fur” (of no importance)


**Salivary gland**

3. Perform a focused physical examination to determine the causes of salivary gland swelling.

- Unilateral swelling
  - Ductal calculus, with staphylococcus or streptococcus viridians infection

- Bilateral swelling
  - Malnutrition; anorexia nervosa/bulimia, starvation, Kwashiorkor
  - Alcoholism
  - Keratoconjunctivitis sicca (dry eyes and mouth)
    - Autoimmune (Sjögren’s syndrome)
    - Mikulicz’s syndrome (non-autoimmune keratoconjunctivitis sicca): TB, lupus, sarcoid, Waldenstrom’s macroglobulinemia
  - Diabetes
  - Thyrotoxicosis
  - Infection - mumps, HIV
  - Infiltration (leukemia, lymphoma)
  - Drugs (PTU, sulfonamides, lead, mercury, iodide)


4. Perform a focused physical examination to determine the causes of parotid gland enlargement.

- Bilateral
  - Infection
    - Mumps (may be unilateral)
    - Sarcoidosis
  - Infiltration
    - lymphoma
  - Immune
- Mikulicz syndrome (painless enlargement of all three salivary glands, probably an early stage of Sjogren’s syndrome)
  o Drugs / toxin
    - Alcohol-associated parotitis
  o Malnutrition

➤ Severe dehydration

➤ Unilateral
  o Tumor
    - Mixed, parotid, occasionally bilateral
    - Infiltration (check for signs of VII nerve palsy)
  o Duct blockage, e.g. salivary calculus


Dysphagia

➤ Orapharyngeal diseases
  o Symptoms
    - Choke, cough, nasal regurg’ drool, wheeze, CNS signs
    - Food sticks immediately after swallowing
    - Folt in the neck
  o Tests
    - VFSS - ENT consult
    - FEES fibospastic endoscopic evaluation of swallowing
    - Modified borium swallow bolus challenge esophagram (bolus transfer assessment)
  o “buried BE” (submucosal squamous metaplasia) baseline (no Px)
    25%

After RFA
76%
5. Take a directed history for dysphagia.

- **Algorithm for symptomatic assessment**
  - **Solids**
    - Suggests mechanical (e.g. stricture, ring, malignancy)
  - **Liquids**
    - Suggests motility problem (e.g. achalasia, diffuse esophageal spasm [DES], scleroderma)

Progressive
- Stricture

Intermittent
- DES
- Lower esophageal ring

Painful
- Esophagitis

Painless
- NERD (normal endoscopy reflux disease)

Nasal/ ENT symptoms
- Suggests orophangeal or CNS cause

Causes
- CNS
  - E.g. CVA, Parkinsonisms
- Lung
  - Compression
- Stomach
  - Tumor
  - Gastric retention
- Colon
  - Constipation (rare)

Drugs
- Pregnancy
- Diet
- MSK
  - Scleroderma
  - Skeletal muscle disease

Complications
- ENT
- Hemorrhage
- Obstruction
- Perforation
- Malnutrition
- Quality of life
- Drug treatment
- Lifestyle modification

What is “the best”? The “best” four clinical signs for bowel obstruction are: visible peristalsis, distended abdomen, hyperactive and/or abnormal bowel sounds.
Abdominal pain and masses

6. Take a directed history to determine the causes of RUQ pain.

- Heart
  - CHD
  - Pericarditis

- Lung
  - Pleurisy
  - Pneumonia
  - PE

- Aorta
  - Dissection / rupture

- Stomach/ duodenum
  - GU
  - DU

- Liver
  - Hepatitis

- Gallbladder
  - Cholecystitis
  - Choledocholithiasis

- Pancreas
  - Pancreatitis

- Kidney
  - Colic

Abbreviations: CAD, coronary artery disease; DU, duodenal ulcer; GU, gastric ulcer; PE, pulmonary embolism
SO YOU WANT TO BE A GASTROENTEROLOGIST!

Q1: A sexually active woman of reproductive potential presents with acute abdominal pain three weeks after a missed menses. What are the most likely diagnoses?

A1:  
- Ectopic pregnancy
- PID (pelvic inflammatory disease)
- Ovarian cyst or tortion

Q2: What are the names of the two signs of abdominal wall discolouration which suggest the presence of an ectopic pregnancy and ruptured AAA?

A2:  
- Cullen’s Sign: Purple-blue discoloration around umbilicus; peritoneal hemorrhage; caused by acute pancreatitis, ectopic pregnancy
- Grey-Turners Sign: Flank discoloration retroperitoneal hemorrhage; caused by acute pancreatitis, ruptured abdominal aortic aneurysm (AAA), strangulated bowel

Adapted from: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, page 37.

Appendicitis and peritonitis

Useful background: Terminology

- McBurney’s point tenderness
  - Tenderness 1/3 along line from ASIS to umbilicus

- Rovsing’s sign
  - LLQ palpation causes RLQ pain (indirect tenderness)

- Rectal tenderness
  - In patients with appendicitis whose inflammation is confined to the pelvis, rectal examination may reveal tenderness, especially on the right side, and some patients with perforation may have a rectal mass (i.e. pelvic abscess).

- Psoas sign
  - The patient lies down on their left side and the clinician hyperextends the right hip. Painful hip extension is a positive psoas sign, suggesting acute appendicitis.

- Obturator sign
  - Flex the patient’s right hip and knee and then internally rotate the right hip; eliciting pain suggests acute appendicitis.
7. Take a directed history and perform a focused physical examination for appendicitis, and stratify the risk and need for surgery.

- The symptoms with the highest PLR are right lower quadrant (RLQ) pain (PLR, 7.3 to 8.5), migration of pain (3.2), and pain before vomiting (2.8).
- The signs with the highest PLR are severe RLQ tenderness (7.3 to 8.5), rigidity (3.8), tenderness at McBurney's point (3.4), rebound abdominal tenderness (1.1 to 6.3), rectal tenderness (0.8 to 5.3), Rovsing's sign (2.5), and psoas sign (PLR, 2.0 to 2.4).

Abbreviation: likelihood ratio (LR) if finding is present= positive LR (PLR)


Useful background: Alvarado clinical decision rule (mnemonic “Mantrels”) provides guidance to operate for suspected acute appendicitis.

- History
  - MAN – migration of pain (1), anorexia-acetone (1), nausea/vomiting (1)
  - TRE – tenderness in RLG (2), rebound (1), elevated temperature (fever) (1)
  - LS – leucocytosis (2), shift-to-the-left (1)

<table>
<thead>
<tr>
<th></th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alvarado score ≥ 7</td>
<td>3.1</td>
<td>0.26</td>
</tr>
</tbody>
</table>

Abbreviation: NLR, negative likelihood ratio; PLR, positive likelihood ratio.


Note that many historical points, symptoms and signs on physical examination have a PLR < 2 (and are not included here).

What is "the best"? The "best" clinical tests for appendicitis in a person with RLQ pain are: McBurney's point tenderness, Rovsing's sign, and Psoas sign; the Alvarado score ≥ 7 (is also useful).
Useful background: Performance characteristics of physical findings that suggest the presence of peritonitis

- Finding:
  - Guarding
  - Rigidity
  - Rebound tenderness
  - Percussion tenderness
  - Abnormal bowel sounds
  - Positive abdominal wall tenderness test
  - Positive cough test
  - Rectal tenderness

- None of the physical findings which have been taught to be clinically useful to diagnose peritonitis have a positive likelihood ration (PLR) greater than 3.9. the signs with a PLR > 2 include abdominal rigidity (3.9), guarding (2.6), percussion tenderness (2.4), and rebound tenderness (2.1).

Abbreviation: likelihood ratio (LR) if finding is present= positive LR (PLR)


8. Perform a focused physical examination to determine the causes of abdominal masses.

- Upper abdomen
  - Retroperitoneal lymphadenopathy (eg lymphoma, teratoma)
  - Left lobe of the liver
  - Abdominal aortic aneurysm (expansile)
  - Carcinoma of the stomach
  - Pancreatic pseudocyst or tumour
  - Gastric dilatation (e.g. pyloric stenosis, acute dilatation in diabetic ketoacidosis or after surgery)
  - Carcinoma of the transverse colon
  - Omental mass (eg metastatic tumour)
  - Small bowel obstruction

- Right upper quadrant (palpable gallbladder)
  - With jaundice
    - Carcinoma of the head of pancreas
    - Carcinoma of the ampulla of Vater*
    - In-situ gallstone formation in the common bile duct
    - Mucocele of the gallbladder due to a stone in Hartmann’s pouch and a stone in the common bile duct (very rare)
Without jaundice
- Mucocoele or empyema of the gallbladder
- Carcinoma of the gallbladder (stone hard, irregular swelling)
- Acute cholecystitis

- Right lower quadrant
  - Appendiceal abscess or mucocoele of the appendix
  - Carcinoma of the cecum or cecal distension due to distal obstruction
  - Crohn’s disease (usually when complicated by an abscess)
  - Ovarian tumour or cyst
  - Carcinoid tumour
  - Amebiasis
  - Psoas abscess
  - Ileocecal tuberculosis
  - Hernia
  - Transplanted kidney

- Left lower quadrant
  - Feces
  - Carcinoma of sigmoid or descending colon
  - Diverticular abscess
  - Ovarian tumour or cyst
  - Psoas abscess
  - Hernia
  - Transplanted kidney

- Pelvis
  - Bladder
  - Ovarian tumour or cyst
  - Uterus (e.g. pregnancy, tumour, fibroids)
  - Small bowel obstruction

- Groin
  - Above the inguinal ligament
    - Inguinal hernia
    - Undescended testis
    - Cyst of the canal of Nuck
    - Encysted hydrocele or lipoma of the cord
    - Iliac node
    - Large femoral hernia (rare)
  - Below the inguinal ligament
    - Femoral hernia
    - Lymph node
    - Saphena varix (sensation of a ‘jet of water’ on palpation, disappears when supine)
    - Femoral aneurysm (pulsatile)
- Psoas abscess (associated with fever, flank pain and flexion deformity)

Adapted from: Talley N. J., et al. *Maclennan & Petty Pty Limited* 2003, Table 5.14 to Table 5.19, pages 174, 175, 179, 184 and 199.

SO YOU WANT TO BE A GASTROENTEROLOGIST!

Q: What are the uses of examining the “belly button”?
A:  
- Direction of flow of blood in veins of abdominal wall: flow below umbilicus is down into saphenous veins, above umbilicus is upwards into veins of thoracic wall. In portal hypertension, dilated veins show normal direction of flow, but in IVC obstruction, flow in veins below umbilicus is reversed, i.e. Flows upward.
- Umbilicus is common site of infiltration by cancer metastases (Sister Mary Joseph’s nodule)
- Protuberance from ascites (an “out-ie”)

Useful background: Causes of anterior abdominal wall masses

- Umbilicus
  - Malignant deposits – e.g. melanoma, carcinoma

- Rectus sheath
  - Umbilical, paraumbilical hernia, or epigastric hernia
  - Incisional hernia
  - Rectus sheath divarication
  - Rectus sheath hematoma

- Skin
  - Lipoma
  - Sebaceous cyst
  - Dermal fibroma
  - Epigastric hernia

Useful background: Causes of a palpable mass in the rectum

- In lumen
  - Feces
  - Foreign body

- In rectal wall
  - Hypertrophied anal papilla
  - Rectal or polyp carcinoma or rectal or sigmoid colon carcinoma prolapsed into the pouch of Douglas
  - Diverticular phlegmon (recent or old)

- Outside rectal wall
  - Metastatic deposits in the pelvis
  - Uterine or ovarian malignancy
  - Prostatic or cervical malignancy (direct extension)
  - Endometriosis
  - Pelvic abscess or sarcoma
  - Amebic granuloma


What is “the best”? The “best” four clinical tests for the presence of peritonitis are: rigidity, guarding, rebound and percussion tenderness.

**Abdominal aorta**

9. Take a directed history and perform a focused physical examination for the cause of abdominal bruit.

- Kidney
  - Renovascular disease
  - Unilateral renal hypertrophy
  - Bruit is rare in primary systemic hypertension
  - Normal variant (10% of normal persons)

- Liver
  - Hepatitis
  - Cirrhosis
  - Hepatoma (HCC)
  - AV fistula

- Pancreas
Achieving Excellence in the OSCE Part 1

- Neoplasia

- **Spleen**
  - AV fistula
  - Tortuous arteries

- **Vessels**
  - AAA (abdominal aortic aneurysm); aka “triple ‘A’”
  - Celiac artery compression, stenosis
  - Chronic intestinal ischemia
  - *An AB is rare in a person with essential hypertension. In persons with renovascular hypertension, an AB is found in 77% with fibromuscular disease, and 35% of those with atherosclerotic disease.*

Adapted from: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, page 33.

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**SO YOU WANT TO BE A GASTROENTEROLOGIST!**

**Q:** What is the clinical use if auscultating an hepatic bruit or friction rub?

**A:**
- Hepatic arterial bruit
  - Alcoholic hepatitis
  - HCC (hepatocellular cancer)
  - Metastases
- Venous hum
  - Portal hypertension (PHT, usually from cirrhosis, aka Cruveilhier-Bamgarten syndrome)
- Venous hum plus hepatic arterial bruit (PHT + alcoholic hepatitis, or PHT + HCC)
- Rub and bruit + venous hum
  - Cirrhosis and HCC
- Friction rub
  - Infection in and around liver (e.g. gonococcal perihepatitis [Fitz-Hugh-Curtis syndrome])
- Rub + bruit
  - HCC
Useful background: Performance characteristics of auscultation and palpation of abdomen for AAA*

- The value of the positive likelihood ratio (PLR) for AAA depends on the size of the lesion (see below), but overall the physical signs which have the highest PLR values include detecting renovascular hypertension (PLR, 38.9), palpating an AAA (8.0), and auscultating an abdominal bruit (5.6).

*sensitivity depends on size of AAA: > 3 cm, ~29%; > 4 cm, ~50%; >5 cm, 75%

Abbreviations: AAA, abdominal aortic aneurysm; PLR, positive likelihood ratio

Adapted from: McGee S. R. Saunders/Elsevier 2007, Box 49-1, page 589; and Box 47-3, page 561.

**Bowel obstruction**

10. Take a directed history for bowel obstruction.

- Abdominal pain – where, when, what, why, how

- Associated symptoms
  - Appetite
  - Weight change
  - Nausea, vomiting (bilious, feculent, bloods)
  - Jaundice, dark urine
  - Recent weight
  - Pale stools
  - Bloating
  - Borborigmi

- Changes in bowel movements
  - Constipation/ obstipation/ no flatus
  - Diarrhea
  - Tenesmus
  - Calibre of stool
  - Melena
  - Hemochezia
  - Flatus

- Medical history
  - Previous surgeries
  - Abdominal hernia
- Gallstones
- Colorectal cancer
- Drugs (opiates, anticholinergics, antipsychotics)
- Diagnoses – inflammatory bowel disease, colorectal cancer, diverticulitis, gallstones, abdominal hernia, vascular ischemia, endometriosis

Causes/Associations
- Small bowel
  - Adhesions
  - Hernias
  - Strictures from IBD
  - Gallstone ileus
  - Mesenteric artery syndrome
  - Small bowel tumours
  - Metastatic cancer
  - Cystic fibrosis
  - Volvulus
  - Crohn’s disease
- Large bowel
  - Cancer
  - Volvulus
  - Diverticulitis
  - Ileus
  - Narcotics ileus
  - Mesenteric ischemia
  - IIBD with stricture
  - Ogilvie’s syndrome
  - Adhesions
  - Intussusception
  - Endometriosis

Adapted from: Jugovic P.J., Et al. Saunders/ Elsevier 2004, pages 58 and 59.

SO YOU WANT TO BE A GASTROENTEROLOGIST!

Q: Which is the better test to assess the size of the liver in a patient with suspected cirrhosis, abdominal ultrasound with doppler, or an ultrasound.

A: Meta-analysis supports transient elastography to diagnose cirrhosis with a high diagnostic accuracy independent from the underlying liver disease (Friedrich-Rust, et al. GE 2008;134:960-974), and is not used to assess liver size.
Useful background: Performance characteristics of physical examination for bowel obstruction.

- While there may be numerous abnormal physical findings which have been traditionally thought to be useful to diagnose a bowel obstruction, the four best tests in terms of the value of the value of their positive likelihood ration (PLR) are the findings on inspection of visible peristalsis (PLR, 18.8) and distended abdomen (9.6) and on auscultation hearing hyperactive (5.0) or abnormal bowel sounds (3.2)

Abbreviation: Likelihood ratio (LR) if finding present= positive PLR

Diarrhea

11. Take a directed history for diarrhea.

- Stools
  - Timing (duration, onset, and course)
  - Frequency
  - Aggravating/alleviating factors (milk, coffee, drugs)
  - Blood
  - Fat droplets
  - Food particles
  - Floating stools
  - Foul odour
  - Difficult to flush
  - Excess flatus

- Associated symptoms
  - Fever
  - Weight loss
  - Change in appetite and diet
  - Vomiting, nausea
  - Urination changes
  - Tenesmus
  - Extraintestinal symptoms of inflammatory bowel disease
  - Arthritis
    - Ulcerative colitis
    - Crohn disease
    - Whipple disease
    - Yersinia infection
  - Lymphadenopathy
    - Lymphoma
    - Whipple disease
— Neuropathy
  - Diabetic diarrhea
  - Amyloidosis
— Postural hypotension
  - Diabetic diarrhea
  - Addison disease
  - Idiopathic orthostatic hypotension
  - Autonomic dysfunction
— Flushing
  - Malignant carcinoid syndrome
— Hyperpigmentation
  - Whipple disease
  - Celiac disease
  - Addison disease
  - Pancreatic cholera
  - Eosinophilic gastroenteritis
— Dyspepsia
  - Peptic ulcer disease
  - Zollinger-Ellison syndrome

➢ Risk factors
  o Travel history
  o Outbreak (friends/family)
  o Seafood (food poisoning)
  o Extraintestinal symptoms (eye, skin, and joint)
  o Diet changes
  o Laxative use
  o Steatorrhea
  o Celiac disease
  o Immunosuppression, including HIV/AIDS
  o Medications and allergies
  o Anal intercourse
  o Family history (inflammatory bowel disease (IBD), bowel cancer, celiac disease, lactose intolerance)
  o Personal history of IBD, celiac disease, pancreatic or hepatobiliary disease, bowel surgery
  o Medications

**Inflammatory bowel disease**

12. Take a directed history to determine the cause of infertility in men with inflammatory bowel disease.

- Medications
- Active inflammatory bowel disease
- Poor nutritional status
- Tobacco use
- Alcohol use
- Postsurgical states


**Alcohol abuse**

Useful background: The continuum of alcohol abuse

- At risk drinking- drinking more than the recommended levels of alcohol (no more than 2 standard drinks per day to a maximum of 9 standard drinks a week for females and 14 standard drinks for males) but no apparent physical or social problems related to alcohol
- Problem drinking- same as at risk drinking but with one or more alcohol related social or physical problems and no clinical features of dependency
- Alcohol dependency- a maladaptive pattern of alcohol use leading to clinically significant impairment or distress with 3 or more of the following criteria in the past 12 months:
  - Tolerance
  - Use of larger amounts of alcohol for longer periods of time
  - Presence of withdrawal
  - Symptoms, excessive amount of time spent in obtaining alcohol
  - Important activities missed or reduced due to alcohol use
  - Persistent desire but unsuccessful efforts to cut down alcohol

13. Take a directed history of alcohol abuse.

- Alcohol
  - Drinking behavior – where, when, what, who, why
  - CAGE questionnaire
    - Have you ever felt the need to Cut down on your drinking?
    - Have you ever felt Annoyed by criticism of your drinking?
    - Have you ever had Guilty feelings about drinking?
    - Have you ever had an Eye opener (a drink first thing in the morning?)
  - Medical complications and associations
    - Fatigue
    - Seizures
    - Blackouts
    - Sleep disturbances

- Alcohol withdrawal
  - Shakes, seizures, hallucinations, DTs

- Liver – cirrhosis, hepatitis

- Cardiac – hypertension, cardiomyopathy

- GI- esophagitis, gastritis, hepatitis, fatty liver, liver cirrhosis, pancreatitis, peptic ulcers, esophageal or rectal varices, oral and esophageal cancers, malabsorption, UGIB

- Cardiac- alcoholic cardiomyopathy, arrhythmias

- Peptic ulcer disease, variceal or non-variceal upper GI bleeding

- Neurologic- Wernicke’s syndrome, Korsakoff’s syndrome, cerebellar degeneration, peripheral neuropathy, myopathy

- Hematologic- iron or folate anemia, thrombocytopenia, coagulopathies

- Endocrine- impotence, hyperlipidemia

- Immunologic- immune system impairment

- Electrolytes- symptoms of low serum calcium, magnesium, or phosphate; ketosis

- Mental
  - Depression
  - Attempted suicide
  - Family Hx of alcoholism
  - Other substance abuse
  - Stressors
    - Family
    - Spouse
- Friends
- Stress
- Money
  o Social consequences
    - Occupation
    - Family
    - Leisure
    - Financial problems
    - Legal problems
    - Accidents and fights
    - Driving intoxicated


14. Perform a directed physical examination for alcohol withdrawal (SSH-DTs: shake, seize, hallucinates, DTs).

<table>
<thead>
<tr>
<th>Time after stopping alcohol</th>
<th>Name of withdrawal syndrome</th>
<th>Clinical picture</th>
</tr>
</thead>
<tbody>
<tr>
<td>- 6-8 hours</td>
<td>Shakes</td>
<td>- Tremor</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Nausea/vomiting</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Insomnia, agitation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Tachycardia (autonomic hyperactivity), fever</td>
</tr>
<tr>
<td>- 8 hours to 2 days</td>
<td>Seizures</td>
<td>- Multiple tonic-clonic seizures</td>
</tr>
<tr>
<td>- 1-2 days</td>
<td>Hallucinations</td>
<td>- Visual hallucinations</td>
</tr>
<tr>
<td>- 3-5 days</td>
<td>DTs</td>
<td>- Fluctuating LOC, coma, agitation/anxiety, death</td>
</tr>
</tbody>
</table>

- Autonomic hyperactivity (tachycardia, pyrexia, sweating, tremor, irritability)
- Depressed mood
- Nausea, vomiting
- Insomnia
- Anxiety
- Seizures
- Delirium tremens
- Hallucinations (usually visual, but auditory and tactile are possible).
SO YOU WANT TO BE A HEPATOLOGIST!

Q: Patients with NAFLD may have elevated serum levels of ALT and AST. What level of these liver enzymes should signal the need to refer the patient for a liver transplantation (L-Tx)?

A: No level! The decision to refer the NAFLD patient for L-Tx is based on their risk factors for progressing to NASH, and not on the level of their ALT or AST. In fact, ~60% of persons with NASH-cirrhosis have normal transaminases.

<table>
<thead>
<tr>
<th>Benefit</th>
<th>AEs</th>
</tr>
</thead>
<tbody>
<tr>
<td>o ↓ adipokinins</td>
<td>↑ BMI</td>
</tr>
<tr>
<td>o ↑ adiponection (a protector of the liver)</td>
<td>↑ CV event</td>
</tr>
<tr>
<td>o ↑ β oxidation</td>
<td>↑ osteoporosis</td>
</tr>
<tr>
<td>o ↓ TG, ↓ VLDL</td>
<td>↑ bladder cancer</td>
</tr>
<tr>
<td>o Converts “bad” into “good” fat</td>
<td></td>
</tr>
<tr>
<td>o ↓ insulin resistance</td>
<td></td>
</tr>
</tbody>
</table>

➢ Caution: Autoimmune markers may be falsely positive in NAFLD

**Hepatosplenomegaly**

Useful background: Causes of hepatomegaly

➢ Raised venous pressure
  - Congestive cardiac failure
  - Constrictive pericarditis
  - Tricuspid stenosis
  - Hepatic vein thrombosis

➢ Degenerative conditions
  - Fatty infiltration and early cirrhosis

➢ Storage disorders
  - Amyloidosis
  - Gaucher’s
  - Niemann-Pick’s
  - Histiocytosis X
  - Glycogen storage disease
  - Haemochromatosis
  - Hurler’s (Gargoyleism)

➢ Infections
  - Viral
    - Infective and serum hepatitis
- Infectious mononucleosis
  o Bacterial
    - Hepatic abscess
    - TB
    - Syphilis
    - Weil’s disease
  o Protozoal
    - Amoebic abscess
    - Malaria
    - Toxoplasmosis
    - Kala-azar
  o Fungal
    - Histoplasmosis
  o Parasitic
    - Hydatid cyst

Source: Burton J.L.  *Churchill Livingstone* 1971, pages 43 and 44.

15. Perform a focused physical examination hepatosplenomegaly.

- Liver
  o Palpable liver in patients with chronic liver disease
  o Palpable liver in epigastrium in patients with chronic liver disease
  o Liver edge firm to palpation in patients with chronic liver disease
  o Palpable liver in patients with jaundice, detecting hepatocellular disease
  o Palpable liver in patients with lymphadenopathy, detecting serious disease
  o Liver tenderness in patients with jaundice, detecting hepatocellular disease nonobstructive jaundice

- Spleen
  o Palpable spleen in returning travellers with fever, detecting malaria
  o Palpable spleen in patients with non-obstructive jaundice, detecting hepatocellular disease
  o Palpable spleen in patients with chronic liver disease, detecting cirrhosis

Because of the wide values of the reported sensitivity and specificity for detecting hepatosplenomegaly, none of the values of the positive likelihood ratios (PLR) are much greater than 2: palpation of a firm liver edge in a person with chronic liver disease (PLR, 2.7), palpable liver in
the epigastric area in a person with chronic liver disease (2.6), or any palpable liver again in a person with chronic liver disease (2.0).

- A palpable liver is not necessarily enlarged, but increases the likelihood of hepatomegaly (LR if present, 2.5 [95% CI, 2.2-2.8]). A non palpable liver edge does not rule out hepatomegaly, but reduces its likelihood (LR is absent, 0.45 [95% CI, 0.38-0.52]).

- The likelihood of a high PLR for splenomegaly depends on the clinical setting for example, in a returning traveler and fever where there is splenomegaly from malaria (PLR,6.6), with much lower values to detect hepatocellular disease in a person with non-obstructive jaundice (2.9), or detecting cirrhosis in the person with chronic liver disease (2.3).

Adapted from: McGee S. R. *Saunders/Elsevier* 2007, Box 47.2, pages 556 to 559.

Useful background:

- Causes of a hard knobbly liver
  - Carcinoma metastases
  - Post-necrotic cirrhosis
  - Congenital cystic liver
  - Hydatid cysts
  - Hepar lobatum (syphilis)

- Causes of hepatosplenomegaly
  - Infective e.g. infectious mononucleosis
  - Myeloproliferative e.g. myelofibrosis, chronic myeloid leukaemia
  - Some causes of portal hypertension e.g. Budd-Chiari syndrome
  - Reticuloses
  - Storage diseases e.g. Gaucher’s disease, amyloidosis
  - Anemia e.g. PA, sickle cell anemia in children

Source: Burton J.L. *Churchill Livingstone* 1971, page 44.

What is “the best”? In the patient with chronic liver disease, the best tests for determining if there is hepatomegaly are palpating a firm liver edge, palpating the liver in the epigastrium, or just palpating an enlarged liver.
Acute and chronic liver disease

16. Perform a physical examination for acute liver disease (acute hepatitis and fulminant liver failure).

17. Perform a focused physical examination for signs of chronic liver disease (portal hypertension).

- Fever
- Jaundice
- Spider navi (upper body only)
- Hypotension
- Liver may be small or large
- Caput medusa
- Hemorrhoids
- Edema
- Hepatic encephalopathy
- Progressive coma
  - Grade I altered mood
  - Grade II drowsy
  - Grade III stupor
  - Grade IV coma
- Gynecomastia
- Splenomegaly
- Easy bruising (coagulopathy)
- Palmar erythema
- Ascites
- Clubbing
- Metabolic flap (asterixis)
- Brisk reflexes
- Plantar response (late)

**Causes of acute deterioration in known chronic liver disease**
- Drugs (including alcohol)
- Electrolyte disturbance
- Sepsis (especially spontaneous bacterial peritonitis)
- GI bleed
- Hepatoma

*Note that the signs of hepatic encephalopathy (HE), hypotension, fever, acidosis and coagulopathy may occur in acute liver failure. The liver may normal size or small early on. Jaundice may be present. The patient may also experience seizures, which is not a feature of HE.*

Useful background: Findings cirrhosis in patients with chronic liver disease

- There are numerous findings on physical examination of a patient with chronic liver disease which give a positive likelihood ration in the 2’s or 3’s (spider angiomata, 3.7; peripheral edema, 3.0; liver edge firm to palpation, 2.7, or just palpable in the epigastrium, 2.6; palmar erythema, 2.6; splenomegaly (2.3 or hepatomegaly, 2.0), but it is the presence of encephalopathy, ascites, and dilated abdominal wall veins (PLR of 8.8, 6.6 and 5.4, respectively), which have the best performance characteristics.

Abbreviation: likelihood ratio (LR) if finding present = positive PLR


- Ascites

18. Take a directed history and perform a focused physical examination for ascites.

<table>
<thead>
<tr>
<th>Item</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Increased girth</td>
<td>4.2</td>
<td>0.2</td>
</tr>
<tr>
<td>o Recent weight gain</td>
<td>3.2</td>
<td>0.4</td>
</tr>
<tr>
<td>o Hepatitis</td>
<td>3.2</td>
<td>0.8</td>
</tr>
<tr>
<td>o Ankle swelling</td>
<td>2.8</td>
<td>0.1</td>
</tr>
<tr>
<td>o Heart failure</td>
<td>2.0</td>
<td>0.7</td>
</tr>
<tr>
<td>Physical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Bulging flanks</td>
<td>2.0</td>
<td>0.2</td>
</tr>
<tr>
<td>o Flank dullness</td>
<td>2.0</td>
<td>0.3</td>
</tr>
<tr>
<td>o Shifting dullness</td>
<td>2.7</td>
<td>0.3</td>
</tr>
<tr>
<td>o Fluid wave</td>
<td>5.0-6.0</td>
<td>0.4</td>
</tr>
</tbody>
</table>

Note that a history of alcoholism or carcinoma, and the * Puddle sign and auscultatory percussion have a PLR < 2, and are not included here.

Abbreviations: CI, confidence interval; PLR, positive likelihood ratio; NLR, negative likelihood ratio

Useful background: Factors which increase the pretest probability of finding ascites (existing disease)

- Heart
  - CHF
  - Pericarditis

- Liver – cirrhosis

- Kidney – nephrotic syndrome

- GI
  - Protein losing enteropathy
  - Malabsorption
  - Malnutrition

- Cause
  - Systemic infection
  - Blunt abdominal trauma

What is “the best” test? Only the finding of a fluid wave and shifting dullness have a LR>2 for the detection of ascites.
Useful background: Findings predicting hepatocellular jaundice in patients with jaundice

- The peripheral findings on physical examination of dilated abdominal wall veins (17.5), palmar erythema (9.8) and spider angiomata (4.7) have the highest values for positive likelihood ratios (PLR) for predicting a hepatocellular cause of jaundice. The PLRs for the presence of ascites and palpable spleen are much lower, 4.4 and 2.9, respectively. All other findings have PLR less than 2.0.

Abbreviation: LR, likelihood ratio; if finding present = positive LR: PLR


**Fingernails**

Useful background: Conditions altering the normal appearance of the fingernails

- Azure half moons in nail beds: the nails of Wilson’s disease (hepatolenticular degeneration). The lunulae are not white but light blue.

- Beau’s lines: transverse grooves on the fingernails of patients recovering from a serious illness such as myocardial infarction.

- Brittle nails: seen in various dysmetabolic states such as hyperthyroidism, malnutrition, and iron or calcium deficiency. They are characterized by irregular, frayed, and torn nail borders.

- Leuconychia-white nails, beginning at the lunula-may be normal; seen in cirrhosis, leprosy, arsenic poisoning, vasomotor disturbance of fingers

- Mees lines (also called Reynolds or Aldrich lines): transverse white lines distal to the cuticle. They are seen in arsenical or thallium poisoning, cancer chemotherapy, Hodgkin’s lymphoma, and other systemic disorders, such as severe cardiac or renal disease.

- Muehrcke’s lines: two arcuate white lines parallel to the lunula and separated by normal nail. Because they are located in the nail bed (not the nailplate). Muehrcke’s lines do not progress with the growth of the nail. They are seen in patients with hypoalbuminemia (<2 gm/100 ml) and disappear with its resolution.
Nail pitting: an early (but non-specific) sign of psoriasis.

Red half moons in nail beds (variety of Terry’s nails): characterized by a lunula that is not white but red. They also are called the nails of cardiac failure.

Splinter hemorrhages: linear red hemorrhages, extending from the free margin of the nail bed toward the proximal margin; typical finding of subacute bacterial endocarditis, or trichinosis, trauma.

Terry’s nails: characterized by whitening of the proximal 80% of the nail, leaving a small rim of peripheral reddening. They are seen in older people or patients with heart failure, cirrhosis, or non-insulin dependent diabetes.

Yellow nail syndrome: characterized by a yellowish colour of the plates due to abnormal lymphatic circulation.

<table>
<thead>
<tr>
<th>Abnormality</th>
<th>Characteristics</th>
<th>Common associations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onycholysis</td>
<td>Separation of nail from nail bed</td>
<td>Fungal infection, Thyrotoxicosis, Psoriasis, Drugs</td>
</tr>
<tr>
<td>Lindsay’s nails</td>
<td>½ and ½ nails, pink white proximally and brown distally</td>
<td>Chronic liver disease, Azotemia</td>
</tr>
<tr>
<td>Terry’s nails</td>
<td>White nail beds with 1-2 mm of distal border of the nail</td>
<td>Cirrhosis, Hypoalbuminemia</td>
</tr>
</tbody>
</table>
Adapted from; Mangione S. *Hanley & Belfus* 2000, page 412.; and Filate W., et al. *The Medical Society, Faculty of Medicine, University of Toronto*, 2005, Table 5, page 15.

**Pruritus**

19. Perform a focused physical examination to determine the cause of pruritus.

- Various skin diseases
  - Infection
  - Eczema
  - Urticaria
  - Lichen simples
  - Dermatitis herpetiformis

- Systemic
  - Hepatic
    - Obstructive jaundice
    - Recurrent pruritus of pregnancy
  - Blood disorders
    - Reticuloses
    - Leukaemia
    - Polycythaemia
    - Fe-deficiency
    - Mastocytosis
  - Endocrine
    - Diabetes mellitus
    - Diabetes insipidus
    - Myxoedema
    - Hyperthyroidism
- Gout
- Carcinoid
  - Neurological
    - Tabes
    - GPI
    - Thalamic tumour
  - Carcinoma (especially lung, stomach, colon, breast, prostate)
  - Chronic renal failure (probably due to secondary hyperparathyroidism)
  - Psychogenic
  - Drugs
    - Cocaine
    - Morphine
    - Allergic drug reactions


20. Prepare a patient for informed consent for the use of steroids (GCS, glucocorticosteroids) in a patient with IBD, explaining the adverse effects.

- **Psyche**
  - Depression, hallucinations, psychosis, insomnia, aggravation of pre existing mood, instability or psychotic tendencies

- **Neurological**
  - Transient worsening of myasthenia gravis
  - Syncope
  - Seizures
  - Pseudotumor cerebri (with GCS withdrawal)
  - Neuritis
  - Paresthesias

- **Eyes**
  - Cataracts (posterior subcapsular)
  - Glaucoma
  - Activation of ocular herpes simplex
  - May enhance establishment of secondary ocular infections due to fungi or viruses

- **Face**
  - Puffiness

- **Dermatological**
  - Buffalo hump striae
- Necrotizing angiitis
- Impaired wound healing
- Petechiae, ecchymoses
- Facial erythema
- Hirsutism
- Acne
- Allergic dermatitis, urticaria, angioneurotic edema
- Perianal burning
- Hypo/ hyper pigmentation
- Scarring, induration
- Sterile abscesses
- Cutaneous/ subcutaneous atrophy

- **MSK**
  - Muscle wasting (steroid myopathy)

- **Cardiovascular**
  - Hypertension
  - Thromboembolism, fat embolism
  - Hypercholesterolemia
  - Accelerated atherosclerosis
  - K$^+$ deficiency → cardiac arrhythmias
  - Cardiac rupture after MI

- **GI**
  - Stomach/ duodenum possible activation of peptic ulcer disease
  - Cautions concurrent use of steroids with ASA/NSAIDs in persons with increased INR
  - Anorexia/ nausea/ vomiting
  - Increased/ decreased appetite- change in body weight
  - Increased/ decreased BMs
  - Ulcerative esophagitis
  - Perforation of small bowel/ colon (especially in IBD)

- **Adrenal glands**
  - Suppression, risk of adrenal insufficiency if GCS is quickly withdrawn)

- **Kidney**
  - In the presence of a fixed or decreased GFR, edema may develop
  - Use with caution in presence of renal insufficiency, acute glomerulonephritis and chronic nephritis
  - Sodium and water retention- edema
  - Aggravation of CHF
  - Hypokalemic alkilosis
  - Hypocalcemia
- **Bone**
  - Osteoporosis, fractures, avascular (aspectic) necrosis (more likely in rheumatoid arthritis or lupus)

- **Endocrine**
  - Pancreas
  - Type II diabetes, adipose cells
  - Obesity
  - Increased requirement for insulin or oral hypoglycemics in diabetics
  - Secondary adrenocortical and pituitary responsiveness in stress situations
  - Menstrual irregularities
  -Increased sweating
  - Protein catabolism

- **Infections**
  - Activation of amebiasis, TB
  - Suppression of patch tests

- **Enhanced risk of steroid AEs with cirrhosis or hypothyroidism**

- **Children**
  - Suppression of growth

- **Drug interactions look up numerous drug interactions in CPS (2011, page 623)**

- **Hematology**
  - ↑WBC, ↓ lymphs, ↓ platelets
  - Thrombophlebitis

- **Pregnancy**
  - Possible increase of oral cleft in fetus
  - Possible hypercortisolism effects in fetus of pregnant mothers on high dose steroids
21. Provide a patient with informed consent prior to their having possible bariatric surgery.

- Perioperative surgical complications
  - Surgery/anesthesia
    - Anastomatic leaks
    - Hernia
  - Lung
    - Pulmonary embolism
    - Aspiration
  - Heart
    - Dysrhythmia
    - Cardiac arrest
  - GI
    - Hemorrhage
    - Incidental splenectomy
    - Small-bowel obstruction
    - Dumping syndrome
      - Flushing, palpitations, light-headedness, fatigue, diarrhea
    - Strictures
    - Cholelithiasis
  - Nutrition
    - Deficiencies Fe, B12, Ca, folate; vitamins A, D, E, K
    - Electrolyte disturbance
    - Protein deficiency

- Risks of not having obesity corrected


22. Prepare a patient for informed consent for the use of nonsteroidal anti-inflammatory drugs, explaining the potential adverse effects.

- Gastrointestinal
  - Constipation or diarrhea
  - Peptic ulcer disease; anemia, bleeding, perforation
  - Colitis
  - ↑, ↓ bowel habit
  - Hemorrhage from diverticulae
  - Obstruction
    - “diaphragm” disease

- Renal
  - ↓ renal blood flow
Achieving Excellence in the OSCE Part 1

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- ↓ glomerular filtration rate
- ↑ creatinine clearance
- Pyuria
- Interstitial nephritis
- Papillary necrosis
- Nephrotic syndrome
- Hyperkalemia
- Type IV renal tubular acidosis
- Fluid retention

- Hematologic
  - Bone marrow suppression
    - Agranulocytosis
    - Aplastic anemia
  - Iron deficiency anemia
  - Platelet-aggregating defect

- Cardiac
  - Peripheral edema
  - Pulmonary hypertension

- Neurologic
  - Delirium/ confusion
  - Headache
  - Dizziness
  - Blurred vision
  - Mood swings
  - Aseptic meningitis

- Dermatologic
  - Urticaria
  - Erythema multiforme
  - Exfoliative syndromes (toxic epidermal necrolysis)
  - Stevens Johnson syndrome
  - Oral ulcers
  - Dermatitis

- Pulmonary
  - Nasal polyps
  - Pulmonary infiltrates
  - Noncardiac pulmonary edema (aspirin toxicity)
  - Anaphylaxis
  - Bronchospasm

- Drug interactions
  - ↑ effect of warfarin
- ↑ antihypertensive effect of diuretics, beta-blockers, angiotensin-converting enzyme inhibitors
- Influence drug metabolism
  - Methotrexate (high doses only)
  - Lithium
  - Oral hypoglycemic agents

Adapted from: Ghosh A.K. Mayo Clinic Scientific Press 2008, Table 24-21, page 999.

**Gallbladder**

Useful background: Terminology

- **Murphy’s sign**  
  - Breathing in suddenly stops with RUQ palpation, suggesting cholecystitis

- **Courvoisier’s sign**  
  - Painless, palpable distended gallbladder, suggesting pancreatic cancer

- **Cullen’s sign**  
  - Bruising of periumbilical area from retroperitoneal due to acute hemorrhagic pancreatitis or ectopic pregnancy

- **Gray-Turner’s sign**  
  - Bruising of the abdomen and flanks due to acute hemorrhagic pancreatitis, ruptured abdominal aortic aneurysm or strangulated bowel

- **Rebound tenderness**  
  - Pain on quick withdrawal of palpation, suggesting peritonitis
Useful background: Performance characteristics for the palpation of gallbladder

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Palpable gallbladder</td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Detecting obstructed bile ducts in patients with jaundice</td>
<td>26.0</td>
<td>0.7</td>
</tr>
<tr>
<td>o Detecting malignant obstruction in patients with obstructive jaundice</td>
<td>2.6</td>
<td>0.7</td>
</tr>
</tbody>
</table>

Note that Murphy’s sign and back tenderness have a PLR <2, and are not included here as signs for cholecystitis.

Abbreviation: NLR, negative likelihood ratio; PLR, positive likelihood ratio.


‘Sweet Nothings’

- Gall bladder (GB) may be often enlarged without a palpable liver; feel GB better with patient on left side.
- Obstructive jaundice plus palpable GB-unlikely to be due to stones (unless stones in cystic duct or Hartmann’s pouch).
- Finger clubbing with portal cirrhosis. Dupuytren’s contracture in alcoholic cirrhosis
- Patients with hemolytic jaundice do not have pruritus or bradycardia
- Biliary cirrhosis-1°, 2°-GB disease, methyltestosterone, chlorpromazine; very occasionally due to severe infection, hepatitis
- Parotid enlargement is common in liver disease, as is fever, even in absence of infection (look for spontaneous bacterial peritonitis)
- Knobbly liver with umbilication – is pathognomonic of hepatic metastases (2°); jaundice with hepatic 2° is usually due to lesions at hepatic fissure; ascites due to portal vein obstruction by glands, or peritoneal deposits
- Impossible to insert a finger between kidney and erector spinae muscle; there is a band of resonance anteriorly over an enlarged kidney
- Pancreatic cysts may be palpable, but tumors rarely are
- Ovarian tumors may be palpated in the midline, including at the umbilicus
- Distended bladder is symmetrical, unless a diverticulum is present
Abdominal X-ray

23. Perform a directed examination of an abdominal x ray (‘flat plate’).

- General
  - Patient demographics (age, sex)
  - Type of study, study date and time
  - Patient’s clinical history, if any provided
  - Obtain previous films for comparison
  - Critique quality of film

- Supraphrenic structures
  - Pleural effusions
  - Consolidation
  - Rib fractures

- Bones
  - Lower thoracic spine, hips, pelvis
  - Lumbosacral vertebrae
    - Lytic or sclerotic lesions
    - Degenerative changes

- Soft tissue
  - Fat stripe
  - Flank stripe
  - Psoas muscles

- Gas pattern
  - Intestinal dilation (Small bowel, <3 cm; large bowel [transverse colon] <5 cm), cecum < 7 cm)
  - Air fluid levels
  - Mucosal thickening
  - Intramural gas
  - Extraintestinal
    - Pneumoperitoneum
    - Pneumobilia
    - Portovenous gas
    - Abscess

Useful background: Causes of calcification on abdominal X-ray

- Lumen of bowel - Fecoliths
- VEIN - Phleboliths
- Nodes - Calcified lymph nodes
- Stones (Calculi) renal, gall bladder, prostatic
- Glands - Calcified pancreas, adrenal, liver (see below), kidney, aorta, psoas muscle, costal cartilage
- Tumor - Calcified tumor-dermoid, fibroid
- Fetus
- Abdominal wall - Calcification in abdominal wall, e.g. cysticerci
- Foreign body on abdominal wall

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 49.

Useful background: Causes of radiological hepatic calcification

- Infection
  - Hydatid cysts
  - Amoebic abscess
  - TB
  - Histoplasmosis
  - Gumma
  - Brucellosis

- Tumor
  - Hepatoma (HCC)

- Veins
  - Hemangioma

- Intrahepatic bile ducts
  - Calculi

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 49.
Malnutrition

Useful background:

- Likelihood ratios of malnutrition screening tool for adult malnutrition, as compared with subjective global assessment

<table>
<thead>
<tr>
<th>Combination of findings</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum albumin &lt; 3.0 g/dL</td>
<td>3.3</td>
<td>0.88</td>
</tr>
<tr>
<td>LAW criteria</td>
<td>6.1</td>
<td>0.10</td>
</tr>
<tr>
<td>Malnutrition screening tool (score &gt;2)</td>
<td>13</td>
<td>0.27</td>
</tr>
</tbody>
</table>

Item score

- Have you lost weight without trying?
  - No
  - Unsure
  - Yes
    - Use question 2 instead

- If No.1 is ‘yes’, use the question, How much weight (kg) have you lost?
  - None
  - 1-5
  - 6-10
  - 11-15
  - >15
  - Unsure

- Have you been eating poorly because of a decreased appetite?
  - No
  - Yes

- Malnutrition screening score
  - Sum of above

Note that many historical points, symptoms and signs on physical examination have a PLR < 2 (and are not included here)

Abbreviations: LAW criteria: discriminant function using Lymphocyte count, Albumin, percentage Weight loss; NLR, negative likelihood ratio; PLR, positive likelihood ratio

Useful background: Increased pretest probability for presence of malnutrition

- **↓ Intake**
  - ↓ appetite
  - Psychiatric illness
  - Conditions requiring a change to a suboptimal solid diet (eg liquid diets, tube diets)
  - Elderly patients

- **↓ digestion/ absorption**
  - Gastrointestinal tract illness

- **↑ requirements**
  - Malignancy
  - Disorders affecting metabolism

- **↑ looses**
  - Patients with unintentional weight loss of more than 5%, a major category of individuals for whom additional testing is warranted.

### Suggested practice case scenarios for OSCE examinations

<table>
<thead>
<tr>
<th>Primary Stem</th>
<th>Secondary Stem</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ø Abdominal pain</td>
<td>Is an alcoholic</td>
<td>- Pancreatitis</td>
</tr>
<tr>
<td></td>
<td>With jaundice and R upper quadrant pain</td>
<td>- Acute cholelithiasis</td>
</tr>
<tr>
<td></td>
<td>With jaundice and fever in a diabetic</td>
<td>- Ascending cholangitis</td>
</tr>
<tr>
<td></td>
<td>Acute epigastric with guarding</td>
<td>- Perforated DU</td>
</tr>
<tr>
<td></td>
<td>Chronic epigastric</td>
<td>- DU and H-pylori</td>
</tr>
<tr>
<td></td>
<td>Acute epigastric with hypotension in elderly male</td>
<td>- Rupture AAA</td>
</tr>
<tr>
<td></td>
<td>Diffuse pain with atrial fibrillation &amp; diarrhea</td>
<td>- Intestinal ischemia</td>
</tr>
<tr>
<td></td>
<td>Lower abdominal pain &amp; fever in older person</td>
<td>- Diverticulitis</td>
</tr>
<tr>
<td></td>
<td>Lower abdominal pain &amp; fever</td>
<td>- Appendicitis</td>
</tr>
<tr>
<td>Ø Hematochezia</td>
<td>Massive rectal bleeding in elderly person</td>
<td>- Diverticulosis</td>
</tr>
<tr>
<td></td>
<td>Spotting blood &amp; mucous in young person</td>
<td>- IBD</td>
</tr>
<tr>
<td></td>
<td>Blood in stool of older person</td>
<td>- Rectal CA</td>
</tr>
<tr>
<td>Ø Abdominal mass</td>
<td>Renal mass</td>
<td>- PKD/Renal cell carcinoma</td>
</tr>
<tr>
<td></td>
<td>Cecal mass</td>
<td>- Ca, Crohn’s disease</td>
</tr>
<tr>
<td></td>
<td>Pulsatile midline mass</td>
<td>- Abdominal aortic aneurysm</td>
</tr>
<tr>
<td></td>
<td>LUQ mass</td>
<td>- Splenomegaly</td>
</tr>
<tr>
<td>Ø Diarrhea</td>
<td>Acute following travel</td>
<td>- Giardia</td>
</tr>
<tr>
<td></td>
<td>In elderly</td>
<td>- Drugs</td>
</tr>
<tr>
<td></td>
<td>With blood in stool</td>
<td>- Bacterial</td>
</tr>
</tbody>
</table>
- Chronic diarrhea in Asian Lactose intolerance
- Chronic diarrhea, microcytic anemia & rash in Caucasian Celiac
- Chronic diarrhea, bronchospasm, murmur & flushing Carcinoid
- Floating stools, pain, weight loss, alcohol abuse Pancreatic insufficiency

### Abnormal lab tests

<table>
<thead>
<tr>
<th>Primary Stem</th>
<th>Secondary Stem</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jaundice</td>
<td>Painless with pruritus, in older person</td>
<td>Pancreatic cancer</td>
</tr>
<tr>
<td></td>
<td>Painless with pruritus, fatty diarrhea in middle aged female</td>
<td>Primary biliary cirrhosis</td>
</tr>
<tr>
<td></td>
<td>Painless with pallor</td>
<td>Hemolytic anemia</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Abdominal distension</th>
<th>Signs of portal hypertension</th>
<th>Ascites</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heartbeat</td>
<td></td>
<td>Pregnancy</td>
</tr>
<tr>
<td>Android obesity</td>
<td></td>
<td>Cirrhosis 2° chronic hepatitis</td>
</tr>
<tr>
<td>Large kidneys</td>
<td></td>
<td>Syndrome X</td>
</tr>
<tr>
<td>Massive splenomegaly</td>
<td></td>
<td>PKD</td>
</tr>
</tbody>
</table>

### Other

Source: Kindly provided by Dr. P Hamilton (U of Alberta)
OSCE - HEMATOLOGY
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<td>White blood cells</td>
<td>269</td>
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<tr>
<td>Splenomegaly</td>
<td>273</td>
</tr>
</tbody>
</table>
OSCE Questions in Hematology Chapter

1. Take a directed history of thrombocytopenia
2. Take a directed history and perform a focused physical examination of the patient with lymphadenopathy:
3. Perform a directed physical examination for lymph nodes in the neck and axilla.
4. Perform a focused physical examination for pernicious anemia.
5. Take a directed history for causes of hemolytic anemia.
6. Perform a focused physical examination for anemia.
Bleeding Disorders

1. Take a directed history of thrombocytopenia.

- Ideopathic

- Dilutional
  - Massive transfusion/infusion
  - Pregnancy

- ↑ destruction
  - Autoimmune
  - Drugs induced
  - Connective tissue diseases
  - Consumptive (DIC)
  - Sepsis

- ↓ production
  - Anaplastic anemia
  - Metastatic disease
  - Hematologic malignancies (marrow replacement)
  - Nutritional
    - Vitamin B12 & folate deficiency
  - Viral infections (HIV, CMV, hepatitis)

Abbreviations: CMV, Cytomegalovirus; DIC, Disseminated intravascular coagulation; HIV, Human immunodeficiency virus

Adapted from: Ghosh A.K. Mayo Clinic Scientific Press 2008, Table 11-17, page 452.

Useful background: Inherited and acquired thrombophilias

- Inherited thrombophilia
  - Prothrombin G20210A mutation
  - Anticoagulant deficiencies (Antithrombin, protein C, protein S)
  - Selected dysfibrinogenemia

- Acquired thrombophilia
  - Immune
    - Lupus anticoagulant or antiphospholipid antibody syndrome
  - Infiltration
    - Solid organ malignancy
    - Myeloproliferative diseases
  - Drugs
    - Estrogens (oral contraceptives, hormone replacement therapy)
o Pregnancy
o Obesity
o Travel
o Trauma
  - Trauma
  - Postoperative state
o Senescence
o Idiopathic
  - Paroxysmal nocturnal hemoglobinuria (PNH)

➢ Mixed Risk Factors
  o Hyperhomocysteinemia
  o Elevated levels of factors VII, IX, & XI


Useful background: Causes of DIC

➢ Infection
  o Infection or sepsis (bacterial)

➢ Infiltration
  o Malignancies (hematologic & solid organs)
  o Solid tumors

➢ Drugs/ toxins
  o Snake bite

➢ Liver
  o Advanced liver disease

➢ Hemolysis
  o Hemolytic transfusion reaction

➢ Blood vessels
  o Aortic aneurysm
  o Giant hemangiomas

➢ Trauma
  o Massive trauma
  o Burns

➢ Obstetrical disorders
  o Obstetric complications (abruption, amniotic fluid embolism)
  o Obstetric complications (retained dead fetus)
Abbreviations: DIC, disseminated intravascular coagulation.


Useful background: Causes of acquired coagulation factor deficiencies

- **Vitamin K-dependent factors**
  - Warfarin
  - Decreased nutritional intake or malabsorption

- **Factor V**
  - Myeloproliferative disease

- **Von Willebrand factor & factor VIII**
  - Acquired von Willebrand syndrome

- **Factor X**
  - Amyloid

- **Multiple factors**
  - Liver failure
  - Disseminated intravascular coagulation (DIC)


Useful background:

- **Causes of purpura**
  - ↓ platelets
    - marrow aplasia
  - abnormal vessel wall
    - Senile purpura
  - drugs
    - steroids
    - anticoagulants

- **Vascular defects**
  - Senile purpura
  - Steroid-induced purpura
  - Henoch-Schonlein purpura
  - Scurvy
  - Von Willebrand’s disease
  - Uremia
Coagulation defects
  - Hemophilia
  - Anticoagulants
  - Christmas disease


**SO YOU WANT TO BE A HEMATOLOGIST!**

Q: In the setting of a patient with purpura, what is Moschcowitz’s syndrome?
A: Speak English. Moschcowitz’s syndrome is simply TTP (thrombotic thrombocytopenic purpura), an acute disorder characterized by:
  - Thrombocytopenic purpura
  - Microangiopathic haemolytic anemia
  - Transient and fluctuating neurological features
  - Fever
  - Renal impairment


Useful background: The classes of drugs that interact with warfarin (“8 A’s”)

<table>
<thead>
<tr>
<th>Drug or drug class</th>
<th>Risk of hemorrhage</th>
<th>Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antibiotics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Most agents, but especially co-trimoxazole, metronidazole, macrolides and fluoroquinolones</td>
<td>↑</td>
<td>↓ vitamin K synthesis ↓ hepatic warfarin metabolism</td>
</tr>
<tr>
<td>o Rifampin</td>
<td>↓</td>
<td>↑ cytochrome P450 (CYP)</td>
</tr>
<tr>
<td><strong>Antifungals</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Fluconazole, miconazole</td>
<td>↑</td>
<td>↓ CYP 2C9</td>
</tr>
<tr>
<td><strong>Antidepressants</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>o Serotonergic agents (selective serotonin reuptake inhibitors [SSRIs])</td>
<td>↑</td>
<td>↓ primary hemostasis (may also inhibit CYP2C9)</td>
</tr>
</tbody>
</table>

**Antiplatelet agents**
Acetylsalicylic acid, clopidogrel, ticlopidine  

- Amiodarone  
- Anti-inflammatory agents 
  - All, including selective NSAIDs, Coxibs  
- Acetaminophen  
- Alternative remedies 
  - *Ginkgo biloba*, dong quai, fenugreek, chamomile  
  - St. John’s wort

Adapted from: David N. J. *CMAJ* 2007;177(4):369-371, Table 1, page 370.

**Lymphadenopathy and mass in head, neck and axilla**

Useful background: Generalized lymph node enlargement

- Examine the mouth for the following signs: 
  - Tonsillar lymph nodes  
  - Palatal petechiae and pharyngitis (glandular fever)  
  - Neoplastic tumours and ulcers

- Examine other lymph node areas in a systemic manner: submental, submandibular, deep cervical (upper and lower), occipital, posterior triangle, supraclavicular, axillary, epitrochlear and inguinal

- Upper cervical lymph nodes: examine the chest, breast and upper limbs. Also, perform an ear, nose and throat (ENT) examination for nasopharyngeal carcinoma

- Lower cervical and supraclavicular lymph nodes: examine the thyroid, chest, abdomen for gastric carcinoma (Virchow’s nodes) and testis

- Axillary lymph nodes: examine the chest, breast and upper limbs

- Inguinal lymph nodes: examine the lower limbs and external genitalia
Lymph nodes of head, neck and axilla

Useful background: Lymph node drainage

<table>
<thead>
<tr>
<th>Location</th>
<th>Area drained</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervical</td>
<td></td>
</tr>
<tr>
<td>Anterior</td>
<td>- Pharynx, tonsils, face, scalp</td>
</tr>
<tr>
<td>Posterior</td>
<td>- Posterior scalp, ear</td>
</tr>
<tr>
<td>Upper Extremities</td>
<td></td>
</tr>
<tr>
<td>Superior to the clavicle</td>
<td>- Head, neck &amp; axillary nodes</td>
</tr>
<tr>
<td>Posterior to the clavicle</td>
<td>- Head, neck &amp; axillary nodes</td>
</tr>
<tr>
<td>At apex of axilla</td>
<td>- All other axillary nodes</td>
</tr>
<tr>
<td>High in axilla, deep to pectoralis minor</td>
<td>- Pectoral, subscapular and lateral nodes</td>
</tr>
<tr>
<td>Along lower border of pectoralis major, inside anterior axillary fold</td>
<td>- Anterior chest wall, most of breast</td>
</tr>
<tr>
<td>Along lateral border of scapula, deep in posterior</td>
<td>- Anterior chest wall, most of breast</td>
</tr>
</tbody>
</table>

Reproduced with the permission of Dr. B. Fisher, University of Alberta
axillary fold
- Upper humerus - Most of arm
- Epitrochlear (cubita) - Lower arm
- Above medial epicondyle - Ulnar side of hand & forearm

- Lower extremities
  - Upper portion of leg - Superficial tissue of upper portion of leg
  - Below inguinal ligament - Skin of
    - lower abdominal wall
    - external genitalia (not testes)
    - lower 1/3 of vagina
    - gluteal area
  - Medial aspect of femoral vein - Popliteal node and superficial inguinal nodes
  - Popliteal fossa - Heel and outer aspect of foot

- Lymph node area, source of drainage, and causes of lymphadenopathy

<table>
<thead>
<tr>
<th>Lymph node area</th>
<th>Source of drainage</th>
</tr>
</thead>
<tbody>
<tr>
<td>➢ Head and neck</td>
<td></td>
</tr>
<tr>
<td>o Pre-/ postauricular</td>
<td>Eye, scalp</td>
</tr>
<tr>
<td>o Occipital</td>
<td>Posterior scalp</td>
</tr>
<tr>
<td>o Submental</td>
<td>Lower face, floor of mouth</td>
</tr>
<tr>
<td>o Submandibular</td>
<td>Face, oral cavity</td>
</tr>
<tr>
<td>o Cervical</td>
<td></td>
</tr>
<tr>
<td>- Anterior</td>
<td>Pharynx, tonsils, face, scalp</td>
</tr>
<tr>
<td>- Posterior</td>
<td>Posterior scalp, ear</td>
</tr>
<tr>
<td>➢ Clavicular/ axillary</td>
<td></td>
</tr>
<tr>
<td>o Clavicular</td>
<td>Cervical lymph node chains, abdomen, thorax, arm and breast</td>
</tr>
<tr>
<td>- Supra-/ infraclavicular</td>
<td></td>
</tr>
</tbody>
</table>

What is “the best”? The 'best test' for anemia is pallor in conjunctive, palms, face.
Achieving Excellence in the OSCE Part 1

Lymph node area | Source of drainage
---|---

- Axillary
- Central
- Lateral
- Posterior (subscapular)
- Anterior (pectoral)
- Other axillary nodes
- Most of arm
- Posterior chest wall, upper arm
- Anterior chest wall, most of breast

- Common causes
  - Head and neck
    - Infections
    - Malignancy
    - Hodgkin’s, non-Hodgkin’s lymphoma
  - Axilla
    - Infections
    - Malignancy
    - Hodgkin’s, non-Hodgkin’s lymphoma
    - Carcinoma of breast, melanoma
  - Epitrochlear
    - HIV
    - Infectious mononucleosis
    - Sarcoidosis
    - Connective tissue diseases
    - Lymphoma/ CLC

- Cause
  - Cancer
    - Myeloproliferative disorders
    - Myeloma
    - Carcinoma
  - Nutrients
    - B12, folic acid deficiency
    - Pyridoxine deficiency
  - Collagen vascular disorders
  - Poison - Lead poisoning

SO YOU WANT TO BE A HEMATOLOGIST!

Q: In the context of regional lymphadenopathy, what is Virchow’s node, and Delphian node, and what are their common causes and what are its causes?

A:

- **Virchow’s node** (anterior left supraclavicular lymph node).
  (Also known as Troiser’s ganglion)
  - Carcinoma of breast, bronchus, lymphomas and gastrointestinal neoplasms
- **Delphian node** (a midline prelaryngeal lymph node)
  - Laryngeal malignancy
  - Heralds thyroid disease
  - Lymphoma

Adapted from: Baliga R.R. *Saunders/Elsevier* 2007, pages 570 and 571.

2. Take a directed history and perform a focused physical examination of the patient with lymphadenopathy.

- Use the pneumonic **ALL AGES** to approach a patient with lymphadenopathy

- **History**
  - **Age** at presentation (e.g. infectious mononucleosis is commoner in younger age groups; Hodgkin’s disease has a bimodal peak).
  - **Location(s)** of lymph nodes (lymph nodes present outside the inguinal regions, for longer than one month and measuring 1 cm x 1 cm or larger without an obvious diagnosis should be considered for biopsy)
  - **Length of time** the lymph nodes are present
  - **Associated symptoms and signs** including fever (‘B’ symptoms: temp >38°C, drenching night sweats, unexplained weight loss >10% body weight)

- **Examination**
  - **Generalized lymph node enlargement**
  - **Extranodal organ involvement**
  - **Splenomegaly** (rare in metastatic cancer; consider infectious mononucleosis lymphoma, chronic lymphocytic leukemia, and acute leukemia)

3. Perform a directed physical examination for lymph nodes in the neck and axilla.

- **Occipital lymph nodes**  
  - Located at the junction between head and neck, common in childhood infections  
  - In adults, a sign of scalp infection  
  - In the absence of infection, they usually reflect a generalized lymphadenopathy, such as may be encountered in HIV infection

- **Posterior cervical lymphadenopathy**  
  - Dandruff or nasopharyngeal tumor

- **Preauricular nodes**  
  - Lymphoma or on the same side of conjunctivitis (often referred to as Parinarud’s syndrome)

- **Nodes scattered around the two branches of the mandible**  
  - Localized pathology, such as periodontitis or other teeth infection  
  - Or submental and submandibular nodes reflect cancer of the nose, lip, anterior tongue, or anterior floor of the mouth

- **Midjugular nodes**  
  - Cancer of the base of the tongue or larynx

- **Lower jugular nodes**  
  - Primary cancer of the thyroid or cervical esophagus


**Red blood cells**

Useful background: Causes of macrocytic anemia with normoblastic bone marrow

- **Nutrition**  
  - Protein deficiency  
  - Scurvy

- **White blood cell**  
  - Leukemia

- **Red blood cell**  
  - Hemolysis
Hemorrhage

- Marrow
  - Aplastic anemia
  - Marrow infiltration or replacement

- Liver
  - Cirrhosis

- Endocrine
  - Myxedema or hypopituitarism

Adapted from: Burton J.L. *Churchill Livingstone* 1971, pages 25 and 58.

4. Perform a focused physical examination for pernicious anemia.

- General
  - Middle age
  - Blue eyes
  - Hair
    - Blondish
    - Prematurly grey

- Signs of anemia

- CNS/PNS
  - Mental changes
  - ↓ position and vibration sensation (dorso-lateral column changes)
  - Peripheral neuropathy

- Eyes
  - Optic atrophy
  - Nystagmus

- Hepatosplenomegaly

- Causes of vitamin B12 deficiency
  - Acquired
    - Lack of intrinsic factor
      - Pernicious anemia
      - Partial or total gastrectomy
    - Changed intestinal flora
      - Stricture
      - Blind-loop syndrome
      - Diverticulosis of small bowel
      - Fistulae
    - Ileal damage
Crohn's disease
- Resection
  - Parasites: Diphyllobothrium latum
  - Dietary (rare)
  - Pancreatitis (rare)

Abbreviations: UMN, upper motor neuron; LMN, lower motor neuron

Adapted from: Burton J.L. *Churchill Livingstone* 1971, pages 57 and 58.

- Causes of polycythemia
  - Absolute
  - Primary
- Secondary
  - Hypoxic
    - Intake
      - High altitude (Monge's disease)
      - Cerebral (decreased respiratory drive)
      - Obesity
    - Circulation
      - Cardiac or pulmonary disease
    - Blinding
      - Methemoglobinemia and sulphemoglobinemia
  - ↑ Erythropoietin
    - CNS
      - Cerebellar hemangioblastoma
    - Kidney disease
    - Liver
      - Carcinoma of liver
    - Uterus
      - Uterine myomata
    - Adrenal
      - Virilizing tumour
      - Pheochromocytoma
  - Relative
    - Dehydration
    - ‘Stress’ polycythemia

Useful background:

- **Causes of sideroblastic anemia (iron accumulates in RBC precursors)**
  - Hypochromic anemia with large numbers of normoblasts containing many iron granules in the marrow
  - Congenital (Pseudothalassemia)
  - Refractory normoblastic anemia of adults
  - Lead poisoning
  - Nutritional
    - $B_{12}$ folate deficiency, pyridoxine (INH therapy)

- **Miscellaneous blood dyscrasias**
  - Myeloproliferative disease
  - Myelomatosis
  - Collagen disease
  - Carcinoma

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 53.

- ‘Tart cell’: A monocyte or neutrophil which has phagocytosed another cell or nucleus. Mimics the LE cell, but occurs in health and in disorders with raised immuno globulins.

- **Howell-Jolly bodies**: Nuclear remnants seen as small dense purple particles at the periphery of RBCs
  - Causes
    - Splenectomy
    - Dyshemopoietic states: leukemia, megaloblastic anemia, etc

- **Pappenheimer bodies**: Fe-containing granules in siderocytes
  - Causes
    - Lead poisoning
    - Hemolytic anemia, which continues after splenectomy

- **Heinz bodies**: Peripheral rounded dark blue bodies in reticulocyte
  - Causes
    - Hemolytic anemia due to drugs and chemicals
    - Familial RBC defects (e.g. G6PD deficiency)
    - Rare hemoglobinopathies (e.g. Hb Koln) after splenectomy

- **Causes of target cells**
  - Iron
    - Iron deficiency anemia
  - Hemolysis
    - Thalassemia
    - Sickle-cell anemia
o Hemoglobin
  - Hemoglobin –C disease
o Liver/spleen
  - Liver disease and obstructive jaundice
  - Splenectomy
o Dehydration


- **Causes of hypochromic anemia**

- **Sideropenic, non sideropenic, sideroblastic**
  - Non-sideropenic anemia
    - Anemia of chronic disease (may be normochromic) eg, RA, Ca, CRF
    - Thalassemia
    - Sideroblastic anemias
  - Sideroblastic anemia
    - Myeloproliferative disorders

- **Causes of aplasia anemia**
  - Idiopathic
  - Drugs, chemicals (e.g., iodine)

5. Take a directed history for causes of hemolytic anemia.

  - Paroxysmal nocturnal hemoglobinuria
  - Hemolytic disease of the newborn
    - Rhesus
    - ABO

- **Inherited**
  - Hereditary spherocytosis, elliptocytosis
  - Hereditary non-spherocytic anemia
  - Thalassemia and Thal. like disorders
  - Sickle-cell disease and S.C disease - like hemoglobinopathies

- **Immune**
  - Idiopathic (warm or cold antibodies)
  - Viral or mycoplasma infection
  - Paroxysmal cold hemoglobinuria (syphilitic or non-syphilitic)

- **Infiltration**
  - Hematological
    - Malignant disease of lympho- reticular system
Achieving Excellence in the OSCE Part 1

- Solid
  - Myeloproliferative disorders
  - Carcinomatosis
  - Ovarian tumours
  - Atrial myxoma

- Infections
  - Bacterial
    - Coccal septicaemia
    - Clostridium welchii
    - Oroya fever
    - TB.
    - Typhoid
    - ‘H.influenzae’ meningitis
  - Protozoal
    - Malaria (Blackwater fever)
    - Kala-azar

- Renal
  - Chronic renal failure
  - ‘Hemolytic-uraemia’ syndrome (infants and children)
  - Thrombotic thrombocytopenic purpura (TTP)
  - Malignant hypertension
  - Eclampsia, or post-partum

- Pregnancy

- Endocrine disease
  - Myxedema
  - Hypopituitarism
  - Hypoadrenalism

- Hypersplenism

- Liver disease
  - Hepatitis
  - Cirrhosis

- Inflammatory
  - Crohn’s disease, ulcerative colitis

- Immune
  - SLE

- Nutritional
  - Protein deficiency
  - Scurvy
Megaloblastic anemia

- Trauma
  - Cardiac surgery
  - ‘March hemoglobinuria’
  - Burns
  - Radiation

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 54.

6. Perform a focused physical examination for anemia.

- Eyes
  - Palor
- Mouth
  - Glossitis
- Thyroid
  - Goiter
- Hands
  - Palor,
  - Lose of palmar crease
- Heart
  - CHF
  - Atrial myxoma
- Lung
  - Bronchiectasis
  - Absess
  - Cancer
- Liver
  - Cirrhosis
- Spleen
  - Splenomegaly
- Nodes
  - Lymphadenopathy
- MSK
  - Rheumatoid arthritis
  - Lupus
- Malnutrition

Useful background: Performance characteristics of findings of anemia

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conjunctival rim pallor</td>
<td>16.7</td>
</tr>
<tr>
<td>Palmar crease pallor</td>
<td>7.9</td>
</tr>
<tr>
<td>Palmar pallor</td>
<td>5.6</td>
</tr>
<tr>
<td>Conjunctival pallor</td>
<td>4.7</td>
</tr>
<tr>
<td>Pallor at any site</td>
<td>4.1</td>
</tr>
<tr>
<td>Facial pallor (but not nail bed pallor)</td>
<td>3.8</td>
</tr>
</tbody>
</table>

Abbreviations: likelihood ratio (LR) if finding present= positive LR (PLR)


Useful background: Comparisons of most common hypochromic microcytic anemias.

<table>
<thead>
<tr>
<th>Disease state</th>
<th>MCV</th>
<th>RBC</th>
<th>TIBC</th>
<th>Transferri saturation</th>
<th>Serum Ferritin</th>
<th>Bone Marrow Iron</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iron deficiency anemia</td>
<td>↓</td>
<td>↓</td>
<td>↑</td>
<td>Low</td>
<td>Low</td>
<td>0</td>
</tr>
<tr>
<td>Anemia of chronic disease</td>
<td>N/</td>
<td>↓</td>
<td>N</td>
<td>N/↑</td>
<td>N/↑</td>
<td>N/↑</td>
</tr>
<tr>
<td>Thalassemia minor</td>
<td>↓</td>
<td>Usually ↓</td>
<td>N</td>
<td>N</td>
<td>N /↑</td>
<td>N</td>
</tr>
</tbody>
</table>

**White blood cells**

Useful background:

- Causes of eosinophilia (>440/cu.mm.)
  - Allergy
  - Infection: Hookworms, tapeworm, hydatid, ascaris, bilharzias, strongyloides, filarial, trichina, Post-infectious rebound
  - Drugs: penicillin, streptomycin, chlorpromazine
  - Skin diseases
    - Scabies
    - Dermatitis herpetiformis
    - Atopic eczema
    - Erythema neonatorum
  - Pulmonary eosinophilia
    - Asthma (including aspergillosis)
    - Polyarteritis nodosa
    - Tropical eosinophilia
    - Loeffler’s
  - Hematological
    - Blood dyscrasias (including eosinophilic leukemia)
  - Tumor
    - Malignancy
  - Miscellaneous
    - Eosinophilic granuloma
  - Gastrointestinal syndromes
    - Post splenectomy
    - Eosinophilic gastroenteritis


- Causes of pancytopenia, neutropenia
  - Marrow infiltration
  - Hypersplenism
  - Deficiency
    - Megaloblastic anemia
    - Iron deficient anemia
  - PNH (paroxysmal nocturnal hemoglobinuria)
  - Endocrine
    - Hypo-/hyperthyroidism
    - Cirrhosis
  - Immune
    - Lupus

- Causes of neutropenia
Infection
- Viral
- Chronic bacterial,

Malignancy infiltration
- Carcinomatous metastasis to bone
- Myelosclerosis
- Myeloma
- Malignant lymphoma

Non-malignant infiltration
- Gauchers
- Niemann-Picks
- Histiocytosis X

Causes of monocytosis (>800/cu.mm)
- Infectious
  - Viral –infectious mononucleosis
  - Rickettsial- Rocky Mountain spotted fever
  - Bacterial- Listeria monocytogenes
  - TB
  - Brucellosis
  - Typhoid
  - SBE
  - Protozoal- Malaria, kala-azar, trypanosomiasis
- Malignancy
  - Hodgkin’s disease
  - Monocytic leukemia

Causes of leukocytosis (>10,000/cu.mm in adults)
- Physiological
  - Infancy
  - Pregnancy and post-partum
- Infection
- Hemorrhage
- Trauma, burns, surgery
- Myocardial infarction and paroxysmal tachycardia
- Toxins: steroids, digitalis, adrenaline, lead, mercury, carbon monoxide
- Collagen vascular diseases
- Infiltration
  - Tumor
  - Myeloproliferative disorders
- Metabolic disorders: renal failure, gout, diabetic coma, eclampsia
- Miscellaneous
  - Hemolysis
  - Serum. Sickness
  - Acute anoxia
- Spider venom

- Causes of myeloid leukamoid reaction (WCC>50,000/ cu.mm or myelocytes or myeloblasts present in peripheral blood)
  - Infections
  - Malignancy
  - Acute hemolysis
  - Leuco-erythroblastic anemia
  - Marble bone disease (Albers-Schonberg)

- Lymphocytosis (>3500/cu.mm)
  - Infections
    - Viral: infectious mononucleosis; infective hepatitis; infectious lymphocytosis, influenza, exanthemata
    - Bacterial: pyogenic infections in young children, convalescence from acute infections, pertussis, typhoid, brucellosis, TB, Syphilis
    - Protozoal: toxoplasmosis
  - Infiltration
    - Lymphatic leukemia
    - Carcinoma
    - Myeloma
  - Endocrine
    - Myasthenia gravis
    - Thyrotoxicosis
    - Hypopituitarism
  - Physiological (in early childhood)

- Causes of agranulocytosis
  - Drugs
  - Aplastic anemia
  - Leukemia in subleukemic phase
  - Hypersplenism
  - Idiopathic

Adapted from: Burton J.L. *Churchill Livingstone* 1971, pages 59 to 61.
SO YOU WANT TO BE A HEMATOLOGIST!

Q1: What is the difference between “myeloid metaplasia” and “extramedullary hematopoiesis”?

A1: None: they both represent ectopic hematopoietic activity, usually in liver and spleen, and may not be associated with myelofibrosis (bone marrow fibrosis).

Q2: OK. What is “myelofibrosis with myeloid metaplasia”?

A2: By convention, is idiopathic myelofibrosis

Q3: Got any more for me: how ‘bout Question 3? – What are the “chronic myeloid disorders”?

A3: o Chronic myeloid leukemia
   o Myelodysplastic syndrome
   o Atypical chronic myeloid disorder
   o Chronic myeloproliferative disease – Polycythemia vera
   – Myelofibrosis with myeloid metaplasia
   – Essential thrombocythemia
   – Agnogenic myeloid metaplasia
   – Post-polycythemic myeloid metaplasia
   – Post-thrombocythemic myeloid metaplasia


“It is very simple to simplify complex issues.”

Grandad
Splenomegaly

Useful background: Factors which increase the pretest probability of finding splenomegaly

- Suspected or proven viral illness, lymphoproliferative disorder, or malignancy
- Cirrhosis (portal hypertension)
- Suspected or proven malaria
- Connective tissue disorders associated with splenomegaly


Useful background: Causes of splenomegaly

- Ideopathic
  - Viral: infective hepatitis, infectious mononucleosis
  - Bacterial: septicaemia, SBE, TB, syphilis brucellosis, typhoid
  - Rickettsial: typhus
  - Fungal: histoplasmosis
  - Protozoal: malaria*, kala-azar*, trypanosomiasis
  - Parasitic: hydatid cyst disease

- Infections
- Infiltration
  - Lymphoma
  - Leukemia (especially CML*)
  - Amyloid
  - Sarcoidosis
  - Gaucher, Nieman Pick disease
  - Benign tumors/ cysts
  - Myelofibrosis*

- Immune
  - Rheumatoid arthritis (Felty syndrome)
  - SLE

- Hematological
  - Hemolytic anemia
  - Myelofibrosis
  - Polycythemia rubra vera
  - Occasionally in
    - ITP
    - Myelomatosis
    - megaloblastic anemia
    - chronic Fe-deficiency anemia
Liver portal hypertension

Endocrine
  - Hyperthyroidism

*hugh spleen


Useful background: Examination of the spleen

Clinical detection of splenomegaly
- Several percent of the normal presumably healthy population may have palpable spleens. The “normal” spleen lies posterior to the left mid-axillary line, and between the 9th and 11th ribs. Normal dimensions are 3 x 7 x 12 cm or less.
- Because the spleen enlarges anteriorly and posteriorly, spleen size must increase by 40% before becoming palpable.
- Only a small portion of the spleen protrudes beneath the costal margin, even when considerably enlarged.
- Inspection should involve asking the patient to breath in deeply several times as well as looking at the “static” abdomen.

Percussion in Traube’s Space and at Castell Spot
- Traube’s space is defined by the sixth rib superiorly, the left anterior axillary line and the costal margin inferiorly. Castell spot is located at the junction of the intercostal space and the left anterior axillary line. The value of Traube’s space percussion (TSP) increases when combined with palpation.
  - Castell’s sign: Percuss over the lowest intercostal space in the left anterior axillary line, asking the patient to inhale and exhale slowly and deeply. Resonance on expiration, replaced by dullness to percussion on inspiration suggests splenomegaly.
Palpation
- Percussion is more sensitive but less specific than palpation as a diagnostic test for splenomegaly. Percussion (Castell’s sign, Traube’s space or Nixon method) should be done first, followed by palpation. If both percussion and palpation are positive, the diagnosis of splenomegaly can be ruled in, provided there is a pre-test probability of at least 10%.
- Done with the patient supine, with the knees slightly flexed.
- Start from the RLQ and work towards the LUQ, again assessing the effect of deep inspiration.
- Remember that the left hand is not pulling the spleen forward to the right (examining) hand, but rather pulling the overlying skin forward to give enough slack for the right hand to properly feel under the costal margin.
- Palpation is most useful in patients who have percussion dullness.
- Discriminate from other masses such as an enlarged kidney:
  - Feel for medial side splenic notching
  - The spleen moves towards the RLQ with inspiration, the kidney moves inferiorly
  - Below the costal margin the relatively superficial spleen is dull to percussion; because of overlying bowel the enlarged kidney may sound resonant to percussion
- You cannot interpose your fingers between an enlarged spleen and the costal margin, it is however possible to palpate “above” a kidney.
- You may hear a “splenic rub”.


---

**SO YOU WANT TO BE A HEMATOLOGIST!**

Q. Percussion in Traub’s space is not specific for splenomegaly. What other conditions cause dullness here?

A. o Left pleural effusion
   o Large pericardial effusion
   o Massive cardiomegaly
   o Stomach full of food
   o Splenic flexure of colon full of feces
   o Enlarged left kidney

Useful background: Perform characteristics for detection of enlarged spleen

<table>
<thead>
<tr>
<th>Finding</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Palpation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>One handed</td>
<td>8.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Two handed</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Percussion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nixon method</td>
<td>6.5</td>
<td>0.2</td>
</tr>
<tr>
<td>Traube’s space dullness</td>
<td>2.1</td>
<td>0.8</td>
</tr>
</tbody>
</table>

Note that the Castell sign has a PLR < 2, and is not included here. Also, the spleen examination has a low sensitivity, especially if the pre test probability for splenomegaly is < 10%.

Abbreviation: NLR, negative likelihood ratio; PLR, positive likelihood ratio

Adapted from: McGee S. R. *Saunders/Elsevier* 2007, Box 47.1, page 554.
Useful background: Performance characteristics for palpation of spleen in various disorders

- Palpable spleen in returning travellers with fever, detecting malaria
- Palpable spleen in patients with non-obstructive jaundice, detecting hepatocellular disease
- Palpable spleen in patients with chronic liver disease, detecting cirrhosis

- The likelihood of a high PLR for splenomegaly depends on the clinical setting for example, in a returning traveler and fever where there is splenomegaly from malaria (PLR,6.6), with much lower values to detect hepatocellular disease in a person with non-obstructive jaundice (2.9), or detecting cirrhosis in the person with chronic liver disease (2.3).

Abbreviation: likelihood ratio (LR) if finding present = positive LR (PLR)

Adapted from: McGee S. R. Saunders/Elsevier 2007, Box 47.2, pages 556 and 557.

Useful background: Examples of drug interactions with warfarin

- Direct gastrointestinal injury (e.g. nonsteroidal anti-inflammatory drugs)
- Altered gut vitamin K synthesis (e.g. antibiotics)
- Altered warfarin metabolism (e.g. co-trimoxazole, metronidazole, fluconazole, amiodarone)
- Interference with vitamin K cycle (e.g. acetaminophen)
- Altered platelet function (e.g. acetylsalicylic acid, clopidogrel)

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<td>Disorders of acid-base balance</td>
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</tbody>
</table>
OSCE Questions in Nephrology Chapter

1. Perform a directed physical examination systemic hypertension.
2. Take a directed history for the causes of systemic hypertension.
3. Take a focused history for complications of malignant hypertensive emergency.
4. Perform a focused physical examination for an abnormally widened pulse pressure.
5. Perform a directed history for the causes of renal colic.
6. Take a directed history of causes of acute interstitial nephritis.
7. Take a directed history to determine the causes of acute renal failure (ARF).
8. Perform a focused physical examination for the causes of acute renal failure.
9. Take a directed history to determine the causes of chronic renal failure.
10. Perform a focused physical examination for chronic renal failure and its causes.
11. Take a directed history for hyponatremia.
12. Perform a focused physical examination for dehydration (extracellular volume depletion)
13. Take a focused history and perform a focused physical examination for obstructive sleep apnea (aka Pickwickian Syndrome).
Systemic hypertension

Useful background: Definitions

- Asymptomatic: systolic BP >200 mm Hg +/- diastolic BP > 120 mm Hg: Needs therapy to prevent potential complications of a malignant hypertensive crisis.

- Malignant hypertension: Symptomatic accelerated hypertension (hypertension plus end organ damage)

Definition: SBP ≥ 180 mm Hg, DBP > 100 mm Hg

- End-organ damage
  - Eye
    - Retinal hemorrhages
    - Optic nerve edema
    - Blurred vision
  - Brain
    - CVA (thrombosis; intracerebral / or subarachnoid hemorrhage)
    - Confusion
    - Headaches
    - Seizures
  - Heart
    - Ischemia
    - L-CHF
    - Dissecting aortic aneurysm
  - Lung
    - Acute pulmonary edema
  - Kidney
    - Rapid ↑Cr, ↓CCR
    - Acute renal failure

- May be a disconnect between BP and end-organ damage

Abbreviations: DBP, diastolic blood pressure; SBP, systolic blood pressure; Cr, Creatinine; CCR, creatinine clearance rate; CVA, cerebrovascular accident; L-CHF, left-sided congestive heart failure.

Classification of blood pressure for adults 18 years of older

<table>
<thead>
<tr>
<th>Category</th>
<th>Blood Pressure Level, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Systolic</td>
</tr>
<tr>
<td>o Normal</td>
<td>&lt;120</td>
</tr>
<tr>
<td>o Prehypertension</td>
<td>120-139</td>
</tr>
<tr>
<td>o Hypertension</td>
<td>- Stage 1 140-159</td>
</tr>
<tr>
<td></td>
<td>- Stage 2 ≥160</td>
</tr>
</tbody>
</table>


1. Perform a directed physical examination systemic hypertension.

- Eye
  - Vessels
    - Arteriolar narrowing
    - A-V nicking
    - Hemorrhages
    - Exudates
    - Papilledema

- Retina
  - Papilledema (blurring of disc margins, no venous pulsations)
  - Narrowing/irregularity of arterioles
  - AV nicking
  - Hemorrhages
  - Cotton wool spots

- CNS
  - Evidence of CVA (effect on cranial nerves, cerebellum, motor/ sensory systems)

- Neck
  - Thyromegaly
  - Carotid bruits
  - ↑ JVP

- Cardiovascular examination:
  - evidence of
    - Left ventricular hypertrophy
    - Congestive heart failure

- Peripheral vasculature
  - Bruits
    - Abdominal aorta
    - Arteries - Renal arteries, femoral arteries, popliteal arteries, posterior tibial artery, dorsalis pedis artery
  - Absent or diminished peripheral pulses
> Abdomen
>  
>  - Ankle to brachial index (peripheral vascular disease or coarctation of the aorta)
>  
>  - Skin
>    - Striae of Cushing’s syndrome
>  
>  - Kidney
>    - Renal bruits to suggest renovascular hypertension
>    - Renal masses to suggest PCK disease
>  
>  - Arteries
>    - Abnormal aortic pulsations
>    - Femoral bruits to suggest peripheral vascular disease
>    - Radio-femoral delay to suggest coarctation of the aorta

Abbreviations: AV, aortic valve; CHF, congestive heart failure; CNS, central nervous system; CVA, cerebrovascular accident; JVP, jugular venous pressure; LVH, left ventricular hypertrophy; PCK, polycystic kidney disease.


2. Take a directed history for the causes of systemic hypertension.

  ➢ Primary

  ➢ Secondary
  
  - Renal
    - Ischemia
    - Pyelonephritis
    - Glomerulonephritis
    - Polycystic kidney disease
    - Hydronephrosis
    - Diabetes, gout, amyloidosis, nephrocalcinosis
    - Collagen vascular disease
  
  - Endocrine
    - Pheochromocytoma
    - Hypo-/ hyperthyroidism
    - Hypercalcemia
    - Hypoglycemia
    - Cushing’s syndrome
    - Conn’s syndrome
  
  - Toxemia of pregnancy
  
  - Heart
    - Coarctation of aorta
o Neurogenic
  - Brain tumor
  - Spinal cord trauma
  - Sleep apnea
  - Porphyria
  - Psyche
  - Anxiety
  - Pain
  - Bulbar polio
  - Head injury
  - Hypothalamic tumor

o Drug
  - Alcohol
  - Cocaine
  - Lead poisoning
  - Oral contraceptives
  - Hormone replacement therapy
  - NSAIDs
  - Ephedrine
  - Corticosteroids
  - Monamine oxidase inhibitors

Abbreviations: NSAIDs, non-steroidal anti-inflammatory drugs


3. Take a focused history for complications of malignant hypertensive emergency.

➢ CNS
  o Confusion
  o Seizures
  o Headaches
  o Visual changes
  o Cerebral thrombosis (TIA/CVA)
  o Intracerebral or subarachnoid hemorrhage

➢ Heart
  o Unstable angina
  o Myocardial infarction
  o Dissecting aortic aneurysm

➢ Lung
  o Acute pulmonary edema
Kidney
  o Acute renal failure

Genitourinary
  o Severe pre-eclampsia and eclampsia

Endocrine
  o Pheochromocytoma

Abbreviations: CNS, central nervous system.

4. Perform a focused physical examination for an abnormally widened pulse pressure.

Definition: pulse pressure > 50% of systolic blood pressure

Causes
  o Hyperdynamic heart syndrome (↑SV, ↓PVR)
  o Aortic regurgitation
  o Patent ductus arteriosus (PDA)
  o Exercise
  o Anemia
  o Arteriovenous fistulas
  o Beriberi
  o Paget’s disease
  o Cirrhosis
  o Pregnancy
  o Thyrotoxicosis
  o Severe exfoliative dermatitis

Abbreviations: PVR, peripheral vascular resistance; SV, stroke volume.
Renal calculi

5. Perform a directed history for the causes of renal colic.

- **History**
  - **Pain**
    - Location of pain (unilateral vs. bilateral, radiation to groin)
    - Duration
    - Course over time
    - Onset
    - Course (ureteral colic from intermittent ureteral distention, constant flank pain from renal capsular distention)
  - **Irritative urinary symptoms**
    - Increased urinary frequency/nocturia
    - Urgency
    - Dysuria
  - **Obstructive urinary symptoms**
    - Hesitancy
    - Diminished stream
    - Postvoid dribbling
    - Postvoid suprapubic fullness
  - **Conditions predisposing to renal stones**
    - Metabolic syndromes
      - Chronic hypercalcemi
      - Hypercalciuria
- Hyperoxaluria (idiopathic, genetic [type I or II] or secondary to GI disease)
- Hypocitraturia (distal renal tubular acidosis, K+ depletion, renal failure)
- Genetic metabolic diseases (e.g. cystinuria)
  - Repeated infections

  o Concentrated urine
    - Chronically low fluid intake (<2 L/day)/ high insensible
    - Structural abnormality
    - Renal tract obstruction (e.g. sloughed papilla, prostate enlargement, neurogenic bladder)
    - Nephrocalcinosis
    - Renal tubular acidosis (distal-type I)

  o Associated symptoms
    - Hematuria
    - Diaphoresis
    - Constitutional (fever/chills/night sweats/weight loss)
    - Abdominal (nausea/vomiting)

  o Risk factors
    - Diet high in oxalates (spinach, rhubarb, nuts, tea, cocoa)
    - Calcium or excess vitamin C administration
    - Prolonged immobilization
    - Meds (chemotherapy, furosemide, hydrochlorothiazide, indinavir)
    - Family Hx of kidney stones
    - Inflammatory bowel disease (Crohn’s disease)
    - Recurrent urinary tract infections

> Causes
  - Ca^{2+} hypercalcemia
  - Hypervitaminosis C
  - Oxalates – diet (spinach, rhubarb, nuts, tea, cocoa)
  - Dehydration
  - Urinary tract infection
  - Previous urinary tract infections
  - Drugs (furosemide, hydrochlorothiazide, chemotherapy, indinavir)
  - Inflammatory bowel disease
  - Family history of kidney stones

Abbreviation: GI, gastrointestinal tract

Nephrotic syndrome

Useful background: Causes of nephrotic syndrome

- Glomerulonephritis
  - Minimal lesion
  - Membranous
  - Proliferative
  - Mixed

- Metabolic
  - Kimmelstiel-Wilson disease (diabetes)
  - Amyloidosis
  - Multiple x
  - Reticulum cell sarcoma
  - Sickle cell anemia
  - Myxodema

- ‘Collagen vascular’ disease
  - SLE
  - Polyarteritis
  - Henoch-Schoenlein purpura
  - Sjogren’s syndrome

- Infection
  - CMV
  - Syphilis
  - Bacterial endocarditis
  - Staphylococcal septicemia
  - Malaria

- Obstruction
  - Renal artery stenosis
  - Renal vein thrombosis
  - Inf. Vena caval thrombosis
  - CCF, constrictive pericarditis
  - Renal carcinoma
  - Chyluria

- Toxins/Drugs
  - Mercurials
  - Tridione
  - Penicillamine
  - Serum
  - Sickness
  - Bee stings
Poison ivy
Smallpox vaccination

Congenital and familial

Diseases causing nephrocalcinos and renal stones
Primary hyperparathyroidism
Renal tubular acidosis
Sarcoidosis
Medullary sponge kidney
Primary hyperoxaluria
Idiopathic hypercalciuria

Abbreviations: CCF, congestive cardiac failure; CMV, cytomegalovirus SLE, systemic lupus erythematosus

Adapted from: Burton J.L. *Churchill Livingstone* 1971, page 103.

Useful background: Causes of radiologically visible nephrocalcinos

Diffuse, cortical
- Chronic glomerulonephritis
- Old cortical necrosis

Coarse, medullary
- Primary hyperparathyroidism
- Primary renal tubular acidosis
- Chronic pyelonephritis
- Idiopathic hypercalciuria
- Idiopathic

Localised
- Medullary sponge kidney
- Renal neoplasm
- Cysts
- Papillary necrosis
- TB
- Hydatid cyst

Radio-opaque
- Calcium (oxalate, phosphate, mixed)
- Cystine
- Silicate
- Calcified uric acid stone

Non-opaque
- Uric acid
- Xanthine
- Matrix

Abbreviation: TB, tuberculosis

Useful background: Differential diagnosis of non-opaque nephrocalcinosis

- Stone (uric acid, xanthine)
- Tumor, cyst
- Clot
- Papillae
- Varix

Useful background: Differential diagnosis of renal calculi

- Blood clot
- Sloughed papillae
- Tumour
- Varices


**Renal insufficiency (‘failure’)**

Useful background: Causes of renal failure

- Pre-renal
  - Hypovolemia
  - Sepsis
  - Post surgery
  - Cardiogenic shock
  - Hepatic failure
  - Drugs (e.g. NSAIDs)
  - Renal artery/vein obstruction

- Renal
  - Glomerulonephritis
  - Nephrotoxic drugs (e.g. gentamycin, NSAIDs)
  - Rhabdomyolysis
  - Interstitial nephritis
  - Myeloma
  - Hemolytic-uremic syndrome

- Post-renal
  - Ureteric stones (reflex anuria)
  - Retroperitoneal fibrosis
  - Tumours of prostate, bladder, cervix, ureters
  - Benign prostatic hypertrophy (BPH)

- CNS
- Headache
- Lethargy
- Dizziness and ataxia
- Mild confusion
- Psychosis
- Seizures
- Coma

- Dehydration
  - Decreased fluid intake
  - Vomiting
  - Diarrhea
  - Excessive sweating
  - Use of diuretics

- Expanded fluid volume
  - CHF
  - Cirrhosis
  - Nephrotic syndrome

- Drugs causing hyponatremia

- SIADH

Abbreviations: CCF, congestive cardiac failure; CNS, central nervous system; SIADH, syndrome of inappropriate secretion of antidiuretic hormone; NSAIDs, non-steroidal anti-inflammatory drugs

Adapted from Davey P. *Wiley-Blackwell* 2006, pages 250 and 268.

**Acute interstitial nephritis**

6. Take a directed history of causes of acute interstitial nephritis.

- Ideopathic

- Infections
  - Bacteria: *Legionella, Brucella, Streptococcus, Staphylococcus, Pneumococcus*
  - Virus: Epstein-Barr, CMV, *Hantavirus*, HIV, Hepatitis B, *Polyomavirus*
  - Fungus: *Candida, Histoplasma*
  - Parasites: *Plasmodium, Toxoplasma, Schistosoma*
Drugs
  - Antibiotics- penicillin, methicillin (anti-tubular basement membrane antibodies), ampicillin, rifampin, sulfa drugs, ciprofloxacin, pentamidine
  - Diuretics- thiazides, furosemide, bumetanide (sulfa derivatives)
  - Cimetidine
  - Allopurinol, phenytoin, phenindione
  - Cyclosporine
  - Sulfasalazine, mesalamines (5-ASAs)

Immune
  - Systemic lupus erythematosus (SLE)
  - Sjogren syndrome
  - Sarcoidosis
  - Renal transplant rejection

Infiltration
  - Lymphoma
  - Leukemia

Abbreviations: ASAs, acetylsalicylic acids; CMV, cytomegalovirus; HIV, human immunodeficiency virus

Adapted from: Ghosh A.K. Mayo Clinic Scientific Press, 2008, Table 18-6, page 703.

7. Take a directed history to determine the causes of acute renal failure (ARF).

Pre-renal
  - CHF
    - ↓ intake
    - ↑ losses, vomiting, diarrhea, bleeding
    - Fluid redistribution – internal bleeding, sepsis, CHF
  - Hypertension

Post-renal (including reflex anuria due to stones, etc.)

Renal
  - Glomerulus – acute glomerulonephritis
  - Tubule – ATN
  - Cortex – cortical necrosis (dehydration in children, ante-partum hemorrhage)
  - Vascular
    - Hypertension
    - Hypotension (ischemia)
- Renal artery thrombosis
- Stenosis
- IVC thrombosis
- Fat emboli

- Infection – sepsis
  - Systemic infections (OM, lupus)

- Metabolic
  - Diabetes
  - Hypothyroidism
  - Hypoadrenalism
  - Hypercalcemia
  - Hyperuricemia
  - Diabetes mellitus
  - Hepatorenal syndrome
  - Hypercalcemia
  - Toxemia of pregnancy

- Acute collagen-vascular disease

- Acute or chronic (often precipitated by infection or electrolyte disturbance)

- Medications
  - Drugs (aminoglycosides, contrast, NSAIDs, sulfanilamides, thiazides, rifampin, allopurinol, cimetidine, phenytoin, analgesics, chemotherapy)
  - Infections - renal (post streptococcal glomerulonephritis; pyelonephritis)

Abbreviations: ARF, acute renal failure; ATN, acute tubular necrosis; CCF, congestive cardiac failure; IVC, inferior vena cava

8. Perform a focused physical examination for the causes of acute renal failure.

- Acute- on-chronic renal failure (often precipitated by infection or electrolyte disturbance)

- Acute cortical necrosis
  - Ante-partum hemorrhage
  - Excessive dehydration in children

- Acute 'tubular necrosis' (ATN)
  - Toxins (eg. drugs, metals)
  - ‘Shock’
  - ‘Crush syndrome’
  - Transfusion reactions
  - Heat stroke
  - Acute hemolysis

- Acute glomerulonephritis

- Ischemia
  - Renal artery thrombosis
  - Progressive stenosis
  - Inferior vena caval thrombosis
  - Fat emboli

- Immune
  - Acute ‘collagen vascular’ disease

- Infection
  - Acute fulminating

- Metabolic, eg. hypercalcemia

- Pregnancy (Toxemia)

- Hepato-renal syndrome

- Reflex anuria due to obstruction (eg renal stone)

9. Take a directed history to determine the causes of chronic renal failure.

- **Renal**
  - Glomerulus
    - Glomerulonephritis
  - Tubule
    - Renal tubular acidosis
    - Chronic hypokalemia
    - Fanconi syndrome (generalized proximal tubular damage)
  - Obstruction
    - Stones
    - Tumours
    - Fibrosis
    - BPH
  - Vascular
    - Hypertension
    - Hypotension (ischemia)
    - Hypovolemia
  - Infection
    - Chronic pyelonephritis
    - Renal TB
    - Sarcoidosis

- **Cardiac**
  - CHF

- **Metabolic**
  - Diabetes
  - Hypercalcemia
  - Amyloid
  - Hypothyroidism
  - Hypoadrenalism
  - Hyperuricemia

- **Medications**
  - Phenacetin
  - Radiation

- **Collagen-vascular**
  - Polyarteritis nodosa
  - SLE
  - Scleroderma
  - Wegeners
  - Goodpaster’s disease
  - Henoch-Schonlein
  - Hemolytic uremia syndrome
- Thrombotic thrombocytopenia

- Congenital
  - Polycystic renal disease
  - Hypoplasia

Abbreviations: BPH, benign prostatic hyperplasia; CRF, chronic renal failure; SLE, systemic lupus erythematosus; TB, tuberculosis


“Trustworthiness is a gating mechanism for social interactions.”

Grandad
**Hyponatremia**

Useful background: Approach to hyponatremia

Hyponatremia - [Na⁺] < 130 mmol/L

---

**Abbreviation:** SIADH, syndrome of inappropriate secretion of antidiuretic hormone

10. Take a directed history for hyponatremia.

Clinical features of hyponatremia and varying values of serum sodium concentration

<table>
<thead>
<tr>
<th>140</th>
<th>135</th>
<th>130</th>
<th>125</th>
<th>120</th>
<th>115</th>
<th>110</th>
<th>105</th>
<th>100</th>
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<tbody>
<tr>
<td>Mild</td>
<td></td>
<td></td>
<td></td>
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<td>Moderate</td>
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<td></td>
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<td></td>
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<tr>
<td>Severe</td>
<td></td>
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<td></td>
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<td></td>
</tr>
</tbody>
</table>

- Headache
- Lethargy
- Anorexia
- Nausea
- Vomiting

- Confusion
- Muscle cramps
- Muscle weakness
- Ataxia

- Coma
- Convulsion
- Death


Useful background: Causes of the syndrome of inappropriate antidiuretic hormone (SIADH)

- **CNS**
  - Infection
  - Stroke
  - Neoplasia
  - Trauma
  - Alcohol withdrawal
  - Acute psychosis
  - Meningitis
  - Encephalitis

- **Lung**
  - Infection
    - Viral/bacterial pneumonias
    - Bronchogenic tumor
    - TB
  - COPD + lung infection

- **GI**
  - Cancer

- **Renal**
  - Prostate cancer
- Blood
  - Malignancies

- Endocrine
  - Acute intermittent porphyria

- Infection
  - HIV/ AIDS, aka ADH
  - ↑H2O permeability of nephron (e.g. vasopressin)
  - ↑ADH release (e.g. carbamazepine)
  - ↑ADH action (e.g. cyclophosphamide)
  - ↓Prostaglandin synthesis (e.g. aspirin)

- Ideopathic

Abbreviations: ADH, antidiuretic hormone; AIDS, acquired immunodeficiency syndrome; CNS, central nervous system; COPD, chronic obstructive pulmonary disease; GI, gastrointestinal tract; HIV, human immunodeficiency virus; TB, Tuberculosis


**Hypovolemia and dehydration**

Useful background: Detecting the likelihood of hypovolemia caused by blood loss (e.g. risk of serious amounts of blood loss)

- Cause by blood loss
  - Pulse increment 30/min or postural dizziness
  - Sensitivity (%) Specificity (%)
    - Moderate blood loss (450-630 mL) 22 98
    - Larger blood loss (630-1150 mL) 97

- Findings
  - Urine specific gravity > 1.020
    - Young, healthy college wrestlers
    - Dehydration secondary to sweating
  - Dry axilla

<table>
<thead>
<tr>
<th>Findings</th>
<th>PLR</th>
<th>NLR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine specific gravity &gt; 1.020</td>
<td>11</td>
<td>0.09</td>
</tr>
<tr>
<td>Young, healthy college wrestlers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dehydration secondary to sweating</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dry axilla</td>
<td>2.8</td>
<td>0.6</td>
</tr>
</tbody>
</table>
Note that a postural increase in the pulse rate by > 30 bpm when moving from the supine to the standing position has a PLR < 2, as also does the finding of normal electrolytes and creatinine levels, or the fact that the patient who is pregnant. For this reason, these are not included here.

Abbreviations: CI, confidence interval; Abbreviation: NLR, negative likelihood ratio; PLR, positive likelihood ratio.


11. Perform a focused physical examination for dehydration (extracellular volume depletion).

- **CNS**
  - Confused
  - Weakness
  - Speech not clear or expressive

- **Mouth**
  - Dry mucous membranes

- **Eye and face**
  - Decreased eyeball pressure
  - Sunken eyes
  - ‘gaunt’ face

- **JVP**
  - Collapsed veins

- **CVS**
  - Tachycardia
  - Postural hypotension

- **Skin**
  - Reduced skin turgor (elasticity) especially arms, forehead, chest, abdomen
  - Dry axilla
  - Dry mucous membranes of mouth and nose
  - Longitudinal furrows on tongue

- **Oliguria**
  - <400 mL urine/24 hours
Total, body water in a male of 70 kg is about 40 L


➢ The finding of either postural dizziness (preventing measurement of vitals while standing) or a postural rise in the heart rate greater than 30 beats per minute is both sensitive (97%) and specific (98%), for large volume blood loss. In patients with volume depletion due to vomiting, diarrhea, or decreased oral intake, few findings have proven utility.

Source: Filate W., et al. The Medical Society, Faculty of Medicine, University of Toronto, 2005, page 326.
Hypernatremia

Hypernatremia - [Na⁺] >148 mmol/L


Useful background: Causes of hypokalemia (K⁺ <3.5 mmol/L)

- ↓ Intake
  - Lack of K⁺ containing food in diet
  - Malabsorption- resins

- ↑ Gut loss
  - Vomiting
  - NG suction
  - Diarrhea
  - Fistulae
→ Renal loss
  - Diabetes mellitus
  - K+ losing diuretic
  - Excess mineralocorticoid- steroids, Cushing's syndrome, Conn's tumor
  - Renal disease
    - ARF followed by diuretic
    - RTA
    - Fanconi syndrome
    - Renal ischemia

→ Shift
  - Familial periodic paralysis
  - IV insulin, glucose, HCO$_3$-

Abbreviations: ARF, acute renal failure; IV, intravenous; NG, nasogastric tube; RTA, renal tubular acidosis


Useful background: Causes of proteinuria

<table>
<thead>
<tr>
<th>Cause</th>
<th>Pathophysiological features</th>
</tr>
</thead>
<tbody>
<tr>
<td>➤ Primary or secondary glomerulopathy</td>
<td>o ↑ Glomerular capillary permeability to protein</td>
</tr>
<tr>
<td>➤ Tubular or interstitial disease</td>
<td>o ↓ Tubular reabsorption of proteins in glomerular filtrate</td>
</tr>
<tr>
<td>➤ Monoclonal gammopathy, leukemia</td>
<td>o ↑ Production and overflow of low molecular weight proteins</td>
</tr>
</tbody>
</table>

Useful background: Take a directed history for the causes of lactic acidosis*

<table>
<thead>
<tr>
<th>Mechanism</th>
<th>Associations</th>
</tr>
</thead>
<tbody>
<tr>
<td>↑ Production</td>
<td>o Hypoxia</td>
</tr>
<tr>
<td></td>
<td>o ↑ Skeletal muscle activity (e.g. status epilepticus or marathon runners)</td>
</tr>
<tr>
<td></td>
<td>o ↑ Destruction of large tumour masses (e.g. lymphoma or leukaemia)</td>
</tr>
<tr>
<td></td>
<td>o Poisoning (e.g. CO or cyanide)</td>
</tr>
<tr>
<td>↓ Transport</td>
<td>o ↓ Cardiac output</td>
</tr>
<tr>
<td>↓ Metabolism</td>
<td>o Liver failure</td>
</tr>
<tr>
<td></td>
<td>o Liver hypoxia</td>
</tr>
<tr>
<td></td>
<td>o Intoxication (phenformin or alcohol)</td>
</tr>
<tr>
<td></td>
<td>o Diabetes mellitus</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>o Hemofiltration with lactate buffer</td>
</tr>
<tr>
<td></td>
<td>o Pregnancy</td>
</tr>
</tbody>
</table>

* Types A ↓ tissue perfusion/oxygenation  
Types B No hypoperfusion or ↓ oxygenation
B1. Association with underlying disease  
B2. Drugs and toxins  
B3. Inborn errors of metabolism


**Disorders of acid-base balance**

12. Take a focused history and perform a focused physical examination for obstructive sleep apnea (aka Pickwickian syndrome).

<table>
<thead>
<tr>
<th>Sleep</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>o Night</td>
</tr>
<tr>
<td></td>
<td>- Unrefreshing sleep</td>
</tr>
<tr>
<td></td>
<td>- Snoring</td>
</tr>
<tr>
<td></td>
<td>- Poor quality of life</td>
</tr>
<tr>
<td></td>
<td>o Day</td>
</tr>
<tr>
<td></td>
<td>- Daytime somnolence</td>
</tr>
<tr>
<td></td>
<td>- Daytime fatigue</td>
</tr>
<tr>
<td>CNS</td>
<td></td>
</tr>
</tbody>
</table>
- Headache, particularly in the morning
- Poor concentration

> CVS
  - Shortness of breath
  - Systemic hypertension
  - Swelling of feet

> GI
  - Gastroesophageal reflux disease

> Physical examination
  - ↑ BMI
  - Short, stocky neck
  - Lung
    - ↓ ventilation
  - CVS
    - R-CHF
    - Pulmonary hypertension

Adapted from: Burton J.L. *Churchill Livingstone* 1971, pages 290 and 291.
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OSCE Questions in Miscellaneous Chapter

1. Take a directed history for unexplained fever.
2. Perform a focused physical examination for fever of unknown origin.
3. Perform a directed physical examination for fever and infection in a patient in hospital.
4. Take a directed history and perform a focused physical examination for postoperative (post-op) fever.
5. Perform a directed physical examination for flushing.
6. Take a directed history for urinary tract infection.
7. Take a directed history of lifestyle issue.
Fever

Useful background: Common causes of fever of unknown origin

- **Infections**
  - Abscess
  - Mycobacteria
  - Endocarditis

- **Neoplasms**
  - Lymphoma
  - Solid tumours (gastrointestinal tract, liver, renal cell, sarcoma)
  - Leukemia, and other hematological tumours
  - Atrial myxoma
  - Inflammatory - IBD, connective-tissue diseases
  - Vascular – pulmonary emboli
  - Iatrogenic – drug-induced (malignant hypertrophy)
  - Congenital – familial Mediterranean fever

- **Connective tissue disease**
  - Temporal arteritis/polymyalgia rheumatica
  - Polyarteritis nodosa
  - Systemic lupus erythematous (SLE)
  - Still’s disease


“Reward anticipation activates a reward network: that is the success of the not-so-common random acts of kindness.”

Grandad
1. Perform a focused physical examination for fever of unknown origin.

2. Perform a directed physical examination for fever and infection in a patient in hospital.

**Staphylococcus aureus**
- Fever often >40°C
- Unwell
- Often no specific symptoms/signs

**Gram negative septicemia**
- Confusion/delirium often prominent
- Septic shock in 25-40%
- Fever
- Hypotension
- Tachypnea → ARDS

**Bacteremia**
- Positive blood cultures + no/trivial symptoms

**Osteomyelitis**

**Endocarditis**

**Enter bloodstream**

**Occasionally arise from GI tract**

**Usually arise from urinary tract**

**Joint infection**

**Death rate 20-40%**

Useful background: Fever in the returned traveller

- Jaundice
  - Hepatitis
  - Malaria
  - Leptospirosis
  - Yellow fever
- Cognitive impairment/delirium
  - Malaria
  - Fulminant hepatic failure
  - Viral encephalitis
- Vomiting
  - GI pathogens
  - Malaria
- Hepatomegaly
  - Amebic liver abscess
    (often tender)
  - Hepatitis
  - Typhoid
  - Malaria
  - Leptospirosis
- Spleomegaly
  - Malaria
  - Typhoid
  - Brucellosis
  - Visceral leishmaniasis
  - Rickettsial diseases
- Lymphadenopathy (common in many infections)
  - Rickettsial infections
  - HIV
  - Plague (localized, tender)
  - Brucellosis
  - Filarial
  - Visceral leishmaniasis
- Diarrhoea
  - GI pathogens
- Skin lesion
- Rose spots
- Eschar
- Petechiae/hemorrhage
- Maculopapular
- Chancre
- Typhoid
- Tick and scrub typhus
- Anthrax
- Viral hemorrhagic fevers
- Leptospirosis
- Dengue, tick typhus, syphilis, arboviral infections, leptospirosis, HIV
- African trypanosomiasis

Useful background: The presence of a skin rash in the patient with fever helps to narrow the likely conditions causing a fever. Causes of fever and rash include:

- **Specific**
  - **Vesicobullous**
    - Herpes viruses (particularly varicella zoster virus)
    - Coxsackie
    - Enterovirus
    - Mycoplasma
  - **Petechia, purpura or skin hemorrhage**
    - Meningococcal septicemia
    - Staphylococcus aureus
    - Viral hemorrhagic fevers
    - Typhus
    - Leptospirosis
    - Gram-negative septicemia
  - **Nodular rashes**
    - Erythema nodosum
  - **Diffuse generalized erythema**
    - Scalded skin syndrome
    - Toxic shock syndrome
      - Usually due to staphylococcal colonization of tampons
      - Symptoms during or shortly after menstruation
      - Fever, hypotension, shock
      - Scarlet skin eruption
      - Desquamation
      - Multi organ failure
  - **Maculo-papular rashes**

- **Non-specific**
  - Drug reaction
  - Infection
  - Self-limiting viral infections
  - HIV seroconversion
  - Dengue fever

Useful background: The septic patient

3. Perform a directed physical examination for flushing.

- Anxiety

- Skin disease
  - Acne
  - Rosacea
  - Photosensitive dematosis

- Drugs
  - Alcohol
  - Ca$^{2+}$ channel blockers

- Food
  - Scromboid poisoning

- Tumor
  - Carcinoid tumors
- Medullary thyroid cancer
- Systemic mastocytosis

Note that skin conditions or self-limiting infections may cause sweating without flushing


Useful background: Reasons why a senior may have urinary incontinence ("DRIP")

- Delirium/diabetes mellitus (hyperglycemia)
- Restricted mobility/retention
- Infections (UTI)/Impaction of stool
- Psychological/pills (long acting sedatives, diuretics, anticholinergic agents)


“It is highly feasible to simplify complex issues.”

Grandad

“We are inherently critical as scientists, and inherently kind as physicians.”

Grandad
**Viral infections**

Useful background: Three viral infections at a glance

- Epstein Barr virus (EBV)
  - Fever
  - CNS
    - Encephalitis
    - Myelitis
  - Jaundice
  - Sore throat
  - Abnormal LEs (90%)
  - Generalized lymphadenopathy
  - Splenomegaly (in 50%)
    - Thrombocytopenia
    - Hemolytic anemia
    - Cold agglutinin
  - Myalgia
  - Maculopapular rash with ampicillin (common)

EBV infection may be associated with cancer in certain populations:
- Nasopharyngeal cancer (Chinese)
- Burkitt’s lymphoma (African)
- PTLD (post transplant lymphoproliferative disorder)

Cytomegalovirus (CMV)

- Neurological disease in HIV
  - ↓ visual acuity
  - Painless
- Pharyngitis
- Esophageal ulcers (HIV)
- Hepatitis (transplant)
- Pneumonitis (transplant)
- Gut (transplant/HIV)
- Retinitis (HIV)
  - ↓ visual acuity
  - Painless

Glandular fever syndrome

50% of adults have been infected: usually asymptomatic if immunocompetent

Varicella zoster virus (VZV)

- Pneumonia
  - Dyspnea, ↑RR
  - Cough
- Hepatitis
- Characteristic rash
- Rash: more severe + widespread in immune-suppressed with more satellite lesions
- 1st infection (chickenpox)
- Reactivation (Zoster)
- Rash, usually confined to one dermatome

Useful background: Sexually transmitted pathogens and the disease which each causes

- Chlamydia trachomatis (chlamydia)
- Neisseria gonorrhoeae (gonorrhea)
- Herpes simplex virus (HSV; herpes)
- Hepatitis B and C (hepatitis)
- Human Immunodeficiency Virus (HIV/AIDS)
- Treponema pallidum (syphilis)
- Human papilloma virus (genital warts)


Useful background: Complications of sexually transmitted diseases in females

- Acute salpingitis
- Pelvic inflammatory disease
- Infertility
- Ectopic pregnancies
- Arthritis
- Conjunctivitis
- Urethritis
- Fitz-Hugh-Curtis syndrome GC/ (chlamydial infection of the liver capsule)


**Lifestyle issues**

4. Take a directed history of lifestyle issue.

- **HPI – L DOCC SPARC CIP**
  - **Location**
    - Where is the chief complaint experienced?
  - **Duration**
    - How long does the chief complaint last?
  - **Onset**
    - When did the chief complaint start?
- Course
  - What are the changes in the chief complaint over time?
- Character
  - Describe the quantity and quality of the chief complaint
- Severity
  - Grade the chief complaint on a scale from 0 (no pain) to 10 (worst pain the patient can imagine) both for its time of onset and the present
- Palliating/provoking
  - What makes the chief complaint better and worse?
- Associated S&S
  - What are the signs and symptoms presenting as a complex with the chief complaint?
- Risk factors
  - What are the factors known to enhance chances of having the chief complaint?
- Constitutional signs
  - Fever, chills, night sweats, changes in sleep, energy level, weight, and appetite
- Causation
  - What does the patient think the cause is?
- Impact on the patient
  - How has the illness affected the patient?
- Patient's action
  - What has the patient done for the complaint (s)?

PMH: SHIAMS
- Surgeries
  - Type, when, outcome (s)
- Hospitalizations
  - Condition, when hospitalized, outcome (s)
- Illnesses
  - In adults, always ask about HTN, DM, Hx of cancer, as well as duration and treatments
- Allergies
  - Drugs, descriptions of reaction (s), MedAlert?, EpiPen?
- Medications
  - Types and dosing
- Sins
  - Smoking/ alcohol/ drug use

- Family history
- Causes
- Complications
Abbreviations: HPI, history of present illness; PMH, past medical history


Useful background: Reasons for falls in seniors

- **Physiologic**
  - ↓ visual acuity
  - ↓ night vision
  - ↓ sensory awareness, touch
  - ↑ body sway, ↓ righting mechanisms

- **Pathologic**
  - Cardiac
    - Myocardial infarction
    - Orthostatic hypotension
  - Neurological
    - Stroke
    - TIA
    - Dementia
    - Parkinsons disease
  - Metabolic
    - Hypoglycemia
    - Anemia
    - Dehydration
  - MSK
    - Arthritis
    - Muscle weakness
  - Drug induced
    - Diuretics
    - Antihypertensives
    - Sedatives
    - Analgesics

Abbreviations: MSK, musculoskeletal; TIA, transient ischemic attack

Useful background: Describe the classic stages of grief

- Patient attempts to limit awareness of condition (shock, denial, and isolation)
- Patient has awareness and emotional release
- Patient experiences depression
- Patient has acceptance and resolution


Useful background:

- Feelings that many grieving people experience.
  - Anger
  - Hopelessness
  - Guilt
  - Worthlessness

- Three tasks of bereavement.
  - Accept the reality of loss
  - Working through the pain of grief
  - Adjusting to life without the deceased


Abuse

Useful background: What physicians should do for a patient after their disclosure of abuse (“DSM”)

- Document abuse completely and accurately
- Support the abused patient (indicate it ‘s not her/his fault, validate feelings)
- Make a safety plan (help person gain access to shelters, legal options, etc)

Useful background: People who have suffered sexual abuse as children may experience “sexual abuse accommodation syndrome”

- Secrecy and silence
- Helplessness and vulnerability
- Entrapment and accommodation
- Delayed, conflicted, and unconvincing disclosure
- Retraction


Useful background: Suggested practice case scenarios for OSCE examinations

<table>
<thead>
<tr>
<th>Primary Stem</th>
<th>Secondary Stem</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
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<td>Weight loss</td>
<td>o With tremor, tachycardia, heat intolerance</td>
<td>- Thyrotoxicosis</td>
</tr>
<tr>
<td>Pregnancy</td>
<td>o Hypertension (HTN)</td>
<td>- HTN in pregnancy</td>
</tr>
<tr>
<td></td>
<td>o Palpations, poor weight gain</td>
<td>- Thyrotoxicosis</td>
</tr>
<tr>
<td></td>
<td>o Excess weight gain &amp; polyuria</td>
<td>- Diabetes</td>
</tr>
<tr>
<td></td>
<td>o With dyspnea &amp; a murmur</td>
<td>- Mitral stenosis</td>
</tr>
<tr>
<td></td>
<td>o Previous abortion &amp; DVT</td>
<td>- Lupus Anticoagulant</td>
</tr>
<tr>
<td></td>
<td>o RVQ tenderness, jaundice</td>
<td>- HAV, ABV, HVC, AFLP</td>
</tr>
<tr>
<td>Perioperative care</td>
<td>o Aortic ejection murmur</td>
<td>- Aortic stenosis</td>
</tr>
<tr>
<td></td>
<td>o Diabetes mellitus</td>
<td></td>
</tr>
<tr>
<td></td>
<td>o Steroid dependent</td>
<td></td>
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<td></td>
<td>o DVT prophylaxis</td>
<td></td>
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<td></td>
<td>o Risk assessment</td>
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Source: Kindly provided by Dr. P Hamilton (U of Alberta)
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